Reevaluation of Predictive Factors for Complete Recovery in Dogs With Nonambulatory Tetraparesis Secondary to Cervical Disk Herniation

The vast majority of dogs with cervical disk herniation experience cervical pain and only mild motor deficits; therefore, not much is known about the factors that predict recovery in dogs with nonambulatory tetraparesis (NAT) secondary to cervical disk herniation. In this retrospective study, we tested the hypothesis that two previously reported prognostic factors, site of disk herniation and severity of neurological deficits, are useful predictors of complete recovery. Overall, 20 (62%) of 32 dogs with cervical disk herniation-associated NAT had complete recovery. Site of disk herniation was not a significant predictor of complete recovery; dogs with high cervical lesions (C2 to C3, C3 to C4) did not have a higher likelihood of complete recovery than other dogs. Likewise, severity of neurological deficits (i.e., intact voluntary motor function versus absent voluntary motor function) was not a significant predictor of complete recovery. Using stepwise logistic regression, two significant predictors of complete recovery were identified. Small dogs (≤15 kg body weight) were six times more likely to achieve complete recovery than larger dogs. Dogs that regained the ability to walk within 96 hours after surgery were seven times more likely to completely recover than dogs not walking 96 hours after surgery. We conclude that neither the site of disk herniation nor severity of neurological deficits assists the clinician in predicting postoperative outcome in dogs with cervical disk herniation-associated NAT. Reliable preoperative predictors of complete recovery are needed to advance current diagnostic and treatment protocols to improve overall prognosis. J Am Anim Hosp Assoc 2009;45:155-163.

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Introduction

In the field of veterinary neurology, prognostic factors for complete recovery of dogs after surgical treatment of thoracolumbar disk disease are well established. Reliable predictors include severity of neurological deficits1-3 and duration of neurological signs prior to loss of ambulation.4 In dogs with thoracolumbar disk disease, site of herniation (upper motor neuron lesion versus lower motor neuron lesion) does not influence prognosis after decompressive surgery.3,5

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In contrast, little is known regarding prognostic factors for surgically treated dogs with nonambulatory tetraparesis (NAT) secondary to cervical disk herniation. Only one previous case series, published almost 20 years ago, has focused on this understudied subset of dogs in which cervical disk herniation is accompanied by profound motor deficits. In that study, lower cervical (lower motor neuron) lesions and severity of neurological deficits were associated with a poorer prognosis for complete recovery. Neither duration of signs prior to NAT nor duration of NAT prior to surgery was a significant predictor of outcome.

The purpose of this study was to reevaluate factors that predict complete recovery in surgically treated dogs with cervical disk herniation-associated NAT. Herein, we report the results of the first multivariate analysis of prognostic factors in 32 dogs.

Materials and Methods

Case Selection

The Veterinary Medical Data Base was searched for dogs admitted to the Purdue University Veterinary Teaching Hospital between January 1, 1990, and December 31, 2005, that had a diagnosis of cervical disk disease, were unable to walk, and underwent decompressive spinal surgery. In each case, myelogram showed an extradural compressive lesion centered over the disk space. Diagnosis of cervical disk herniation was confirmed at the time of surgery by the identification of disk material within the vertebral canal. For each dog, information was tabulated from medical records and telephone contact with owners and referring veterinarians. Information included the following: signalment; history of trauma; site of disk herniation; duration of signs prior to NAT; duration of NAT prior to surgery; degree of sensory and motor deficits in the thoracic and pelvic limbs; date and type of decompressive surgery; corticosteroid treatment; time to reach ambulatory status; and long-term outcome, including recurrence of clinical signs. To determine the percentage of dogs that achieved complete recovery, cases without complete recovery were excluded from analysis if follow-up of at least 12 months after surgery could not be obtained. Doberman pinchers were also excluded, because cervical disk disease in this breed is a complex syndrome associated with vertebral instability. Thirty-two dogs with cervical disk herniation-associated NAT that satisfied the inclusion criteria are the subject of this report.

Analysis of Potential Prognostic Factors

Degree of neurological recovery was recorded for each dog. The primary endpoint of this study, complete recovery, was defined as resolution of cervical pain and regaining the ability to walk normally. Cases were analyzed to determine if the following potential prognostic factors were predictive of complete recovery: body weight (≤15 kg versus >15 kg); severity of preoperative neurological deficits (e.g., absent voluntary motor function in thoracic limbs, absent voluntary motor function in pelvic limbs, intact voluntary motor function); duration of signs prior to NAT (tertiles); duration of NAT prior to surgery (tertiles); site of disk herniation (“high” lesions [intervertebral spaces C2 to C3 and C3 to C4] versus “low” lesions [intervertebral spaces C4 to C5, C5 to C6, or C6 to C7]); history of trauma (yes versus no); time to reach ambulatory status after surgery (≤96 hours versus >96 hours); and chronological age (≤8.8 years versus >8.8 years). To obtain a closer look at the relationship between age and outcome, the likelihood of complete recovery was compared in the youngest and oldest dogs after adjusting the chronological age of each dog to physiological age, a process which takes into account breed-specific differences in life expectancy. The association between date of surgery (three categories) and complete recovery was also evaluated to rule out a significant cohort effect.

Data were analyzed using SPSS (version 15.0) for Windows and SAS System (version 9.1) for Windows. Odds ratios (ORs) were calculated using unconditional logistic regression analysis to identify which factors predicted the likelihood of complete recovery. Odds ratios were considered significant if 95% confidence intervals (CIs) did not include 1.0. Stepwise multivariate logistic regression analysis was used to evaluate the influence of additional variables on those predictors found to be significant at P<0.10 in univariate analysis. Two-by-two tables were constructed, and chi-square analysis was used to evaluate the association between site of herniation and severity of neurological deficits. Fisher’s exact test was used to compare the likelihood of complete recovery in dachshunds versus other breeds. For chi-square and Fisher’s exact tests, P values <0.05 were considered significant.

Results

Clinical Features of Dogs With Cervical Disk Herniation-associated NAT

The clinical features of 32 dogs with NAT secondary to cervical disk herniation are summarized in Table 1. Median age at surgery was 8.8 years (range 3.7 to 15.0 years). Median body weight was 15 kg (range 4 to 50 kg). The dachshund was the most commonly affected breed (n=6). The most common site of disk herniation was the C3 to C6 intervertebral space (n=9 dogs), accounting for 28% of dogs in this series. Twelve (38%) of 32 dogs had high cervical lesions (upper motor neuron, C2 to C3 and C3 to C4 intervertebral spaces), whereas 20 (62%) dogs had low cervical lesions. One dog (case no. 2) had disk herniation at two sites.

All dogs had preoperative thoracic limb and pelvic limb neurological deficits severe enough to render them unable to walk. In three dogs, medical records did not specify the presence or absence of voluntary motor function. Voluntary motor function was intact in 21 (72%) of 29 dogs. Six dogs were tetraplegic, whereas two dogs had absent forelimb voluntary motor function. Deep pain perception in the thoracic limbs was absent in only one dog (case no. 11); all dogs had intact deep pain perception in the pelvic limbs. Fifteen dogs had some degree of neck pain noted in the medical record.

Six (19%) dogs had a history of trauma immediately preceding their neurological signs. Median duration of neuro-
### Table 1

**Clinical Features of 32 Dogs With Nonambulatory Tetraparesis (NAT) Secondary to Cervical Disk Herniation**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Breed</th>
<th>Age (y)</th>
<th>Body Weight (kg)</th>
<th>Site of Cervical Disk Herniation</th>
<th>Duration of Signs Prior to NAT</th>
<th>Thoracic Limb</th>
<th>Pelvic Limb</th>
<th>Time to Regain Ambulatory Status</th>
<th>Follow-up (mos)</th>
<th>Case Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mixed-breed dog</td>
<td>7.1</td>
<td>17</td>
<td>C2-C3</td>
<td>1 wk</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>48 h</td>
<td>35</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>2</td>
<td>Dalmatian</td>
<td>6.5</td>
<td>36</td>
<td>C2-C3, C3-C4</td>
<td>24 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>71</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Dachshund</td>
<td>6.6</td>
<td>6</td>
<td>C3-C4</td>
<td>2 wks</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>85</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Dalmatian</td>
<td>12.4</td>
<td>26</td>
<td>C3-C4</td>
<td>1 wk</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>89</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Dachshund</td>
<td>10.6</td>
<td>8</td>
<td>C3-C4</td>
<td>0</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>20</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Pug</td>
<td>9.3</td>
<td>9</td>
<td>C3-C4</td>
<td>7 d</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>&lt;72 h</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Pekingese</td>
<td>3.7</td>
<td>5</td>
<td>C4-C5</td>
<td>2 wks</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>24</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Dachshund</td>
<td>9</td>
<td>6</td>
<td>C4-C5</td>
<td>0</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>84</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Mixed-breed dog</td>
<td>12</td>
<td>29</td>
<td>C4-C5</td>
<td>1 wk</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>4-14 d</td>
<td>6</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>10</td>
<td>Mixed-breed dog</td>
<td>10.3</td>
<td>19</td>
<td>C4-C5</td>
<td>0</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>48-96 h</td>
<td>65</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>11</td>
<td>Dachshund</td>
<td>7.4</td>
<td>8</td>
<td>C4-C5</td>
<td>6 mos</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
<td>57</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Dachshund</td>
<td>9.8</td>
<td>10</td>
<td>C4-C5</td>
<td>0</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>53</td>
<td>Complete recovery</td>
<td></td>
</tr>
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(Continued on next page)
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Breed</th>
<th>Age (y)</th>
<th>Body Weight (kg)</th>
<th>Site of Cervical Disk Herniation</th>
<th>Duration of Signs Prior to NAT</th>
<th>Duration of NAT Prior to Surgery</th>
<th>Thoracic Limb Deep Pain</th>
<th>Voluntary Motor Function</th>
<th>Pelvic Limb Deep Pain</th>
<th>Voluntary Motor Function</th>
<th>Time to Regain Ambulatory Status</th>
<th>Follow-up (mos)</th>
<th>Case Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>Yorkshire terrier</td>
<td>7</td>
<td>4</td>
<td>C5-C6</td>
<td>NA†</td>
<td>NA</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>2 wks</td>
<td>19</td>
<td>Complete</td>
<td>Trauma (fell down stairs); reoperated for recurrence of NAT; DDL</td>
</tr>
<tr>
<td>14</td>
<td>Mixed-breed dog</td>
<td>12</td>
<td>8</td>
<td>C5-C6</td>
<td>12 h</td>
<td>36-48 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>3-14 d</td>
<td>72</td>
<td>Complete</td>
<td>Trauma (hit with golf club); neck pain</td>
</tr>
<tr>
<td>15</td>
<td>Dachshund</td>
<td>8.4</td>
<td>6</td>
<td>C5-C6</td>
<td>24 h</td>
<td>48 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>6 d</td>
<td>56</td>
<td>Complete</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Mixed-breed dog</td>
<td>10</td>
<td>9</td>
<td>C5-C6</td>
<td>2 wks</td>
<td>3 d</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>48 h</td>
<td>30</td>
<td>Complete</td>
<td>Recurrence of NAT 2 y postoperatively</td>
</tr>
<tr>
<td>17</td>
<td>Beagle</td>
<td>9</td>
<td>15</td>
<td>C5-C6</td>
<td>6 mos</td>
<td>&lt;24 h</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>72 h</td>
<td>5</td>
<td>Complete</td>
<td>Neck pain</td>
</tr>
<tr>
<td>18</td>
<td>Labrador retriever</td>
<td>8.9</td>
<td>31</td>
<td>C5-C6</td>
<td>3 d</td>
<td>48 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>7 d</td>
<td>40</td>
<td>Complete</td>
<td>Neck pain; DDL</td>
</tr>
<tr>
<td>19</td>
<td>Beagle</td>
<td>8</td>
<td>18</td>
<td>C6-C7</td>
<td>1 wk</td>
<td>4 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>72 h</td>
<td>24</td>
<td>Complete</td>
<td>Neck pain</td>
</tr>
<tr>
<td>20</td>
<td>Yorkshire terrier</td>
<td>9.2</td>
<td>6</td>
<td>C6-C7</td>
<td>0</td>
<td>4 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>24 h</td>
<td>67</td>
<td>Complete</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>Australian shepherd</td>
<td>4.7</td>
<td>32</td>
<td>C2-C3</td>
<td>0</td>
<td>&lt;24 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>&gt;3 mos</td>
<td>127</td>
<td>Residual</td>
<td>Residual deficits; Trauma (collision with tree); neck pain; residual right hemiparesis</td>
</tr>
<tr>
<td>22</td>
<td>Rottweiler</td>
<td>7.3</td>
<td>42</td>
<td>C2-C3</td>
<td>24 h</td>
<td>48 h</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>6 d</td>
<td>25</td>
<td>Residual</td>
<td>Residual deficits</td>
</tr>
<tr>
<td>23</td>
<td>Rottweiler</td>
<td>7</td>
<td>36</td>
<td>C3-C4</td>
<td>NA</td>
<td>NA</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>96 h</td>
<td>24</td>
<td>Residual</td>
<td>Euthanized due to recurrent NAT; no necropsy</td>
</tr>
<tr>
<td>24</td>
<td>Beagle</td>
<td>8.1</td>
<td>19</td>
<td>C3-C4</td>
<td>24 h</td>
<td>&lt;24 h</td>
<td>Present</td>
<td>NA</td>
<td>Present</td>
<td>NA</td>
<td>1 mo</td>
<td>53</td>
<td>Residual</td>
<td>Residual deficit; mild pelvic-limb ataxia</td>
</tr>
</tbody>
</table>

(Continued on next page)
### Table 1 (cont’d)

Clinical Features of 32 Dogs With Nonambulatory Tetraparesis (NAT) Secondary to Cervical Disk Herniation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Breed</th>
<th>Age (y)</th>
<th>Body Weight (kg)</th>
<th>Site of Cervical Disk Herniation</th>
<th>Duration of Signs Prior to NAT</th>
<th>Duration of NAT Prior to Surgery</th>
<th>Thoracic Limb</th>
<th>Pelvic Limb</th>
<th>Time to Regain Ambulatory Status</th>
<th>Follow-up (mos)</th>
<th>Case Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>Dalmatian</td>
<td>7.5</td>
<td>32</td>
<td>C₃-C₄</td>
<td>0</td>
<td>8 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>24-48 h</td>
<td>69</td>
<td>Residual deficits Neck pain</td>
</tr>
<tr>
<td>26</td>
<td>Mixed-breed dog</td>
<td>7.8</td>
<td>31</td>
<td>C₅-C₆</td>
<td>4 d</td>
<td>48 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>3-14 d</td>
<td>24</td>
<td>Residual deficits Neck pain; euthanized 18 mos postoperatively due to recurrent NAT</td>
</tr>
<tr>
<td>27</td>
<td>Welsh corgi</td>
<td>12.2</td>
<td>16</td>
<td>C₅-C₆</td>
<td>0</td>
<td>24 h</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>&lt;3 wks</td>
<td>18</td>
<td>Residual deficits Trauma (collision with bed)</td>
</tr>
<tr>
<td>28</td>
<td>Rottweiler</td>
<td>7.3</td>
<td>50</td>
<td>C₆-C₇</td>
<td>0</td>
<td>4 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>&gt;4 wks</td>
<td>35</td>
<td>Residual deficits</td>
</tr>
<tr>
<td>29</td>
<td>Tibetan spaniel</td>
<td>12.1</td>
<td>7</td>
<td>C₆-C₇</td>
<td>6 y</td>
<td>&gt;5 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>1 wk</td>
<td>47</td>
<td>Residual deficits</td>
</tr>
<tr>
<td>30</td>
<td>Mixed-breed dog</td>
<td>8.8</td>
<td>16</td>
<td>C₂-C₃</td>
<td>0</td>
<td>3 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Never</td>
<td>1</td>
<td>Never walked Trauma (hit by car); neck pain</td>
</tr>
<tr>
<td>31</td>
<td>Mixed-breed dog</td>
<td>15</td>
<td>9</td>
<td>C₄-C₅</td>
<td>7 wks</td>
<td>4 d</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Never</td>
<td>2</td>
<td>Never walked Trauma (fell down stairs); euthanized due to renal failure</td>
</tr>
<tr>
<td>32</td>
<td>Maltese</td>
<td>9.9</td>
<td>5</td>
<td>C₅-C₆</td>
<td>2 wks</td>
<td>48 h</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Never</td>
<td>5 d</td>
<td>Never walked Euthanized due to postoperative dyspnea, respiratory failure; no necropsy</td>
</tr>
</tbody>
</table>

*DDL=dorsal decompressive laminectomy
†NA=not available in medical record
logical signs prior to NAT was 24 hours (range 0 hours to 6 years). Median duration of NAT prior to surgery was 3 days (range <24 hours to 1 month). Dogs were treated with ventral slot decompression (n=29) or dorsal laminectomy (n=3). Twenty-seven dogs received intraoperative methylprednisolone succinate at a dose of 30 mg/kg body weight. Twenty-five dogs received corticosteroids for a period of days to weeks prior to surgery.

Functional Outcome in Dogs With Cervical Disk Herniation-associated NAT

Overall, 20 (62%) of 32 dogs with NAT secondary to cervical disk herniation had complete recoveries. Median duration of follow-up for this group was 51 months (range 5 to 111 months). Twelve dogs did not achieve complete recovery. Nine (28%) dogs in this series became ambulatory but had residual paresis and/or ataxia. Median duration of follow-up for this group was 47 months (range 18 to 127 months). Median time to reach ambulatory status after surgery was 6 days.

Three (9%) dogs never walked after surgery. One dog (case no. 32) was euthanized on the 5th postoperative day because of progressive dyspnea. One dog (case no. 30) was euthanized 1 month after surgery for failure to ambulate. One dog (case no. 31) was euthanized 2 months after surgery because of renal failure. None of these dogs underwent necropsy.

Overall, recurrence of signs consistent with intervertebral disk herniation occurred in three of 20 dogs that had complete recoveries. Recurrence of intervertebral disk herniation was documented in one dog and suspected in two other dogs. One dog (case no. 3) had two episodes of severe neck pain that responded to corticosteroids and cage rest. One dog (case no. 13) developed NAT 19 months after dorsal laminectomy; ventral slot decompression was performed at the same site as the initial surgery, and a large amount of fibrous material grossly consistent with disk material was removed from the vertebral canal. One dog (case no. 16) had recurrent NAT 30 months after surgery and was euthanized. Two (22%) of nine dogs with residual neurological deficits were euthanized because of recurrent NAT approximately 2 years after surgery (case nos. 23, 26). None of these dogs underwent necropsy.

Predictive Factors for Complete Recovery in Dogs With Cervical Disk Herniation-associated NAT

The association between possible prognostic factors and complete recovery was analyzed in 32 dogs [Table 2]. Neither the site of disk herniation nor severity of neurological deficits were significant predictors of complete recovery in dogs with cervical disk herniation-associated NAT. Six (50%) of 12 dogs with high cervical lesions had complete recovery, whereas 14 (70%) of 20 dogs with low cervical lesions had complete recovery (P=0.26). Five (83%) of six tetraplegic dogs and both dogs with absent voluntary motor function in the thoracic limbs had complete recovery. The one dog with absent deep pain perception in the thoracic limbs made a complete recovery.

In dogs with cervical disk herniation-associated NAT, two significant predictors of complete recovery were identified: small body size and regaining ambulatory status within 96 hours after surgery. Univariate analysis showed small dogs (≤15 kg body weight) were more than five times more likely to have complete recovery than larger dogs (OR, 95% CI = 5.6, 1.3 to 27.5). Six of six dachshunds had complete recovery, whereas 14 of 26 other breeds had complete recovery (P=0.06). Incidence of complete recovery was not different between dachshunds and other small breeds (P=0.25, Fisher’s exact test).

In univariate analysis, dogs that regained the ability to walk within 96 hours after surgery were more than six times more likely to have complete recovery, compared with dogs that remained nonambulatory at 96 hours (OR, 95% CI = 6.1, 1.1 to 35.3). Eleven (85%) of 13 dogs that regained ambulatory status within 96 hours after surgery went on to make a complete recovery, whereas only nine (47%) of 19 dogs that were still unable to walk at 96 hours completely recovered.

In stepwise logistic regression analysis, the relationship strengthened between small body size and complete recovery (OR, 95% CI = 6.8, 1.1 to 39.9) and between regaining ambulatory status within 96 hours after surgery and complete recovery (OR, 95% CI = 7.5, 1.1 to 51.6). No other factors, including chronological age, physiologic age, history of trauma, duration of signs prior to NAT, duration of NAT prior to surgery, or date of surgery were significant predictors of complete recovery.

Discussion

Whereas the prognostic factors for surgically treated dogs with thoracolumbar disk herniation are well established,1-4 not much is known about which factors predict complete recovery in dogs with NAT secondary to cervical disk herniation.6,10-17 Two prognostic factors, site of disk herniation and severity of neurological deficits, are stated in veterinary textbooks.18,19 but these factors are based upon a single study of 12 cases.6 This prompted us to reevaluate prognostic factors in this subset of dogs having cervical disk herniation and severe motor deficits. From this first multivariate analysis of prognostic factors in 32 dogs with NAT, we reject the notion that site of disk herniation and severity of neurological deficits are useful predictors of outcome. Instead, our results suggest that small body size and regaining ambulatory status within 96 hours postoperatively are the strongest predictors of complete recovery in dogs with cervical disk herniation-associated NAT.

We tested the hypothesis that high cervical lesions (C2 to C3, C3 to C4) are associated with a more favorable prognosis than lower cervical lesions (C4 to C5, C5 to C6, C6 to C7). This association was suggested by a previous report in which three of three dogs with high cervical lesions had complete recoveries, whereas only five (56%) of nine dogs with lower cervical lesions had complete recoveries.6 In the
Table 2
Univariate and Multivariate Analyses of Predictive Factors for Complete Recovery in 32 Surgically-treated Dogs With Nonambulatory Tetraparesis (NAT) Secondary to Cervical Disk Herniation

<table>
<thead>
<tr>
<th>Prognostic Factors</th>
<th>Univariate OR† (95% CI‡)</th>
<th>Multivariate OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Site of disk herniation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C2-C3, C3-C4 &quot;High&quot; (n=12)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>C4-C5, C5-C6, C6-C7 &quot;Low&quot; (n=20)</td>
<td>2.3 (0.53-10.27)</td>
<td></td>
</tr>
<tr>
<td><strong>Severity of neurological deficits</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic limb voluntary motor function absent (n=8)</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Thoracic limb voluntary motor function present (n=21)</td>
<td>0.2 (0.02-1.83)</td>
<td>NS</td>
</tr>
<tr>
<td>Pelvic limb voluntary motor function absent (n=6)</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Pelvic limb voluntary motor function present (n=23)</td>
<td>0.3 (0.03-3.12)</td>
<td></td>
</tr>
<tr>
<td><strong>Body weight</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;15 kg (n=16)</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>≤15 kg (n=16)</td>
<td>5.6 (1.1-27.5)</td>
<td>6.8 (1.1-39.9)</td>
</tr>
<tr>
<td><strong>Ambulatory status at 96 h postsurgery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonambulatory (n=19)</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Ambulatory (n=13)</td>
<td>6.1 (1.1-35.3)</td>
<td>7.5 (1.1-51.6)</td>
</tr>
<tr>
<td><strong>Chronological age at surgery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤8.8 y (n=16)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>&gt;8.8 y (n=16)</td>
<td>3.0 (0.7-13.4)</td>
<td></td>
</tr>
<tr>
<td><strong>Physiological age at surgery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Youngest (n=16)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Oldest (n=16)</td>
<td>0.6 (0.1-2.5)</td>
<td></td>
</tr>
<tr>
<td><strong>Duration of neurological signs prior to NAT</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1 wk (n=18)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>≥1 wk (n=12)</td>
<td>2.4 (0.5-11.9)</td>
<td></td>
</tr>
<tr>
<td><strong>Duration of NAT prior to surgery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤48 h (n=12)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>72-96 h (n=8)</td>
<td>1.7 (0.3-10.3)</td>
<td></td>
</tr>
<tr>
<td>&gt;96 h (n=10)</td>
<td>4.0 (0.6-27.2)</td>
<td></td>
</tr>
<tr>
<td><strong>Trauma history</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No trauma (n=26)</td>
<td>1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Trauma (n=6)</td>
<td>0.2 (0.03-1.47)</td>
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</tbody>
</table>

(Continued on next page)
present study, however, site of herniation was not a predictor of complete recovery. Dogs with lower cervical lesions were not less likely to have complete recoveries. In this report, lower cervical lesions were associated with an increased likelihood of recovery, but this difference was not statistically significant. The finding that site of herniation fails to impact prognosis in dogs with cervical disk herniation-associated NAT mirrors the senior author’s observations of surgically treated dogs with thoracolumbar disk herniation. Although counterintuitive, it seems that dogs with lower motor neuron lesions secondary to disk herniation do not have a poorer prognosis than dogs with upper motor neuron lesions.

Severity of neurological deficits is considered a robust predictor of outcome in surgically treated dogs with thoracolumbar disk herniation. In contrast, in dogs with NAT secondary to cervical disk herniation, we found no significant association between severity of neurological deficits and complete recovery. Absence of voluntary motor function in the thoracic or pelvic limbs was not a reliable predictor of poor outcome; 83% of tetraplegic dogs recovered. In a previous report, two of 12 dogs with NAT had absent deep pain perception in the thoracic limbs; both failed to achieve complete recovery. In the present series, however, the only dog that had sensory deficits had complete recovery. Our results indicate that fewer than one-third of dogs that are nonambulatory secondary to cervical disk herniation experience loss of voluntary motor function; sensory deficits are encountered even less frequently. While the hypothesis that complete sensorimotor loss portends a poor prognosis in these dogs remains untested, we advise clinicians to refrain from using severity of neurological deficits as a proxy for poor postoperative outcome in dogs with cervical disk herniation-associated NAT.

In contrast to the null findings for site of disk herniation and severity of neurological deficits, small body size was found to be a significant predictor of complete recovery. Since the 1989 report, additional data probing the association between signalment and complete recovery in dogs with cervical disk herniation-associated NAT are limited to eight new cases. All weighed ≤15 kg, and seven (86%) of eight dogs had complete recovery. Twenty-three of 23 small, tetraparetic dogs that were reported as nonambulatory by Cherrone et al became ambulatory after surgical treatment of cervical disk herniation. The likelihood of these dogs to completely recover (i.e., walk normally) was not reported. The reason small body size confers a more than five-fold advantage for complete recovery in our series is not clear. Small dogs with NAT were more likely to have more severe preoperative neurological deficits. Seven (47%) of 15 dogs weighing ≤15 kg had absent voluntary motor function, whereas only one (7%) of 14 larger dogs had absent voluntary motor function (P=0.04).

Dachshunds accounted for almost 40% of small dogs, and each of the six dachshunds in our series had complete recovery. This 100% complete recovery rate for dachshunds

<table>
<thead>
<tr>
<th>Prognostic Factors</th>
<th>Univariate OR† (95% CI‡)</th>
<th>Multivariate OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery date category</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1990-1993 (n=10)</td>
<td>1.0 NS</td>
<td></td>
</tr>
<tr>
<td>1994-2000 (n=9)</td>
<td>0.5 (0.08-3.5)</td>
<td></td>
</tr>
<tr>
<td>2001-2005 (n=13)</td>
<td>0.7 (0.1-4.0)</td>
<td></td>
</tr>
</tbody>
</table>

* The relationship between prognostic factors and complete recovery was first evaluated in univariate analysis. For each prognostic factor, odds ratio and 95% confidence interval (OR, 95% CI) for complete recovery were expressed relative to a reference category assigned an odds ratio of 1.0. The wide confidence intervals observed for odds ratios are likely a consequence of small sample size. Only prognostic variables whose 95% confidence interval did not include 1.0 were considered statistically significant. Multivariate analysis was carried out using a stepwise logistic regression model. The model selected only two prognostic factors: body size and ambulatory status at 96 hours after surgery. The multivariate OR and 95% CI for these variables are shown. No other variables were selected by the model.
† OR=odds ratio
‡ CI=confidence interval
§ NS=not selected in multivariate model
far exceeds the 50% complete recovery rate for non-dachshunds in this series and the 58% complete recovery rate reported previously for all breeds with NAT. Data from dachshunds reported in the literature are limited to only six dogs; four achieved complete recovery. It is possible that anatomical factors make this breed more likely to recover completely. Deserving further study are the vertebral canal:spinal cord ratio and the nature of disk herniations in dachshunds that experience severe motor deficits.

In the dogs reported here, the most powerful predictor of complete recovery was regaining ambulatory status within 96 hours of surgery. Dogs that walked within 96 hours were more than six times more likely to have complete recovery than dogs that were still nonambulatory at 96 hours after surgery. Eighty-five percent of dogs that regained ambulatory status at 96 hours went on to complete recovery, whereas the majority (53%) of dogs that were still nonambulatory at 96 hours failed to completely recover. Many dogs with NAT secondary to cervical disk herniation regain ambulatory status rapidly; this is consistent with previous observations. Our findings emphasize, however, that complete recovery is also possible (albeit less likely) in dogs that do not rapidly regain the ability to walk.

Conclusion

Nonambulatory tetraparesis is an infrequent manifestation of cervical disk herniation in dogs. The prognosis for affected dogs after surgical treatment is variable; only about 60% of dogs experience complete recovery. Neither site of disk herniation nor severity of preoperative neurological deficits assists the clinician in predicting outcome. Although dachshunds and other small breeds may have a higher likelihood of favorable outcome, regaining the ability to walk within 96 hours after surgery is the strongest predictor of complete recovery. Future studies should focus on identifying reliable preoperative factors associated with poor outcome in this subset of dogs with cervical disk herniation. Success in this effort may guide the development of new diagnostic and treatment protocols that will improve overall prognosis.

References

Combination Auriculopalpebral Nerve Block and Local Anesthesia for Placement of a Nictitating Membrane-to-Superotemporal Bulbar Conjunctiva Flap in Dogs

The purpose of this study was to evaluate the efficacy of combined local anesthesia in dogs undergoing nictitating membrane (NM)-to-superotemporal bulbar conjunctiva flap construction. Medical records of 47 dogs that had received local anesthesia for NM-to-superotemporal bulbar conjunctiva flap were reviewed. Combined local anesthetic technique included auriculopalpebral nerve block, topical anesthesia of the eye, and infiltration anesthesia of the superotemporal bulbar conjunctiva and palpebral surface of the NM. Forty-two (89.3%) dogs complied with the anesthetic procedures and underwent NM flap without general anesthesia or sedation. No complications were related to the combined local anesthesia. Combined local anesthesia for NM-to-superotemporal bulbar conjunctiva flap may be a time- and cost-effective method that produces both analgesia of the surgical site and akinesia of the eyelid. J Am Anim Hosp Assoc 2009;45:164-167.

Introduction

Nictitating membrane (NM) flap is a simple and rapid procedure that is frequently performed in dogs with corneal ulcers. The NM-to-superotemporal bulbar conjunctiva flap is one of several NM flaps available, and it can be pursued in the interest of avoiding the complications associated with other NM flaps. One complication associated with NM-to-superior-lid technique is necrosis of the upper lid; complications of NM-to-episclera technique include inadvertent penetration of the globe and premature flap failure.1 This procedure is usually performed under general anesthesia, even though some dogs with corneal ulcers may have clinical conditions that increase their general anesthetic risk.

Many reports on topical, sub-Tenon’s capsule, intracameral, retrobulbar, and regional anesthesia for ocular surgery in humans and animals have been published.2-6 Nictitating membrane flaps can likely be performed in conscious dogs under local anesthesia with adequate analgesia, but to our knowledge, the clinical evaluation of NM flaps without general anesthesia has not been reported. We constructed the NM-to-superotemporal bulbar conjunctiva flap in conjunction with auriculopalpebral nerve block, topical anesthesia of the eye, and infiltration anesthesia of the superotemporal bulbar conjunctiva and palpebral surface of the NM. Forty-two (89.3%) dogs complied with the anesthetic procedures and underwent NM flap without general anesthesia or sedation. No complications were related to the combined local anesthesia. Combined local anesthesia for NM-to-superotemporal bulbar conjunctiva flap may be a time- and cost-effective method that produces both analgesia of the surgical site and akinesia of the eyelid. J Am Anim Hosp Assoc 2009;45:164-167.

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Address all correspondence to Dr. Seo.

This study was supported through the BK21 Program for Veterinary Science and through the Research Institute for Veterinary Science (RIVS), College of Veterinary Medicine, Seoul National University, Seoul, Korea.
nerve block, topical anesthesia of the eye, and infiltration anesthesia of the superotemporal bulbar conjunctiva and palpebral surface of the NM. The purpose of this study was to evaluate the efficacy of combined local anesthesia in dogs undergoing NM-to-superotemporal bulbar conjunctiva flap construction.

Materials and Methods

We reviewed medical records of 47 consecutive dogs that had received local anesthesia for NM-to-superotemporal bulbar conjunctiva flap between January 2006 and December 2007. Signalment, reason for combined local anesthesia administration for NM flap, temperament of the dog, and compliance with the anesthetic procedure and surgery were investigated.

Auriculopalpebral nerve block and topical and infiltration anesthesia were performed to achieve combined local anesthesia of the eye. Auriculopalpebral nerve block was induced by inserting a 1.2-cm, 26-gauge needle through the skin dorsal to the zygomatic process at its caudal one-third and injecting 0.4 mL of 2% lidocaine subcutaneously [Figure 1A]. The palpebral reflex was tested at the initial ophthalmic examination and 5 minutes after the auriculopalpebral nerve block was carried out.

Immediately following the auriculopalpebral nerve block, one drop of topical anesthetic (0.5% proparacaine) was applied to the eye. After 1 minute, 0.1 mL of 2% lidocaine was injected subconjunctivally at the projected suture sites in the superotemporal bulbar conjunctiva and palpebral surface of the NM, approximately 3 to 5 mm ventral to the margin of the nictitans [Figure 1B]. Five minutes after the injection, the NM injection site was lightly pinched with mosquito hemostat forceps in order to evaluate analgesia. When the dog no longer reacted to the stimulus, we proceeded with NM-to-superotemporal bulbar conjunctiva flap construction using 4-0 polygalactin 910. The dog was placed in a sitting position, under minimal head restraint, with the eyelids manually held open by an experienced handler.

Corneal ulcers were treated by debridement, punctate keratotomy, and grid keratotomy when needed. The entire surgical procedure lasted <5 minutes from beginning to the completion of suture placement. Treatment success was defined as compliance of the dog during the anesthetic procedures and surgery, without the need for sedation or general anesthesia. Postoperative palpebral reflex was assessed 1 hour after the operation to evaluate for any residual akinesia.

Results

Combined local anesthesia for NM-to-superotemporal bulbar conjunctiva flap was performed in 47 dogs with corneal ulcer. Mean age at the time of the procedure was 8.3±3.9 years (range 2 months to 17 years). The most common dog breed was the shih tzu (n=23) [see Table]. The main reasons for choosing combined local anesthesia included owner reluctance for general anesthesia to be used (n=15), senility (n=10), hepatic problems (n=6), cardiovascular problems (n=4), and renal problems (n=4).

All dogs that received combined local anesthesia had a negative or significantly reduced palpebral reflex 5 minutes after the auriculopalpebral nerve block was achieved. Complete recovery of the reflex was observed 1 hour after the operation in all dogs. The overall success rate of combined local anesthesia for NM flap was 89.3% (n=42). During the procedure, no anesthesia- or surgery-related complications were noted. The causes of treatment failure included resistance to injection of lidocaine for the auriculopalpebral nerve block (n=1), resistance to infiltration anesthesia of the superotemporal bulbar conjunctiva (n=2), and lack of compliance with the surgery after the local anesthesia was achieved (n=2). These dogs required general anesthesia (n=2) or sedation (n=3) in order to complete the surgery. The NM-to-superotemporal bulbar conjunctiva flap construction was performed in 42 dogs (89.3%).

Figures 1A, 1B—(A) Auriculopalpebral nerve block was achieved by inserting a 1.2-cm, 26-gauge needle through the skin dorsal to the zygomatic process at its caudal one-third and injecting 0.4 mL of 2% lidocaine. (B) For infiltration anesthesia, 0.1 mL of 2% lidocaine was injected subconjunctivally at the projected suture sites in the superotemporal bulbar conjunctiva and palpebral surface of the nictitating membrane, approximately 3 to 5 mm ventral to the margin of the nictitans. Illustrations by Hae Kyeong Min and Dr. Se Eun Kim.
anesthesia in humans and large animals. Auriculopalpebral nerve block is necessary for general anesthesia. Local anesthesia can also serve as an alternative in veterinary practices that have limited access to the resources extra time, money, and effort necessitated by general anesthesia. Second, local anesthesia saves the time and resources needed for general anesthesia of the upper eyelid. Ocular surgery in dogs and cats to achieve immobilization and relaxation. However, NM flap construction does not require complete immobilization, because it is a less delicate procedure than most other ocular surgeries, and it is short in duration—typically <5 minutes. Therefore, we believed NM flap construction could be performed successfully under combined local anesthesia.

We did not note any complications related to NM-to-superotemporal bulbar conjunctiva flap construction in conscious dogs in this study. Inadvertent globe penetration is the major complication that can occur when this surgery is performed in conscious dogs. However, with the NM-to-superotemporal bulbar conjunctiva flap technique, the bulbar conjunctiva is picked up with forceps, and the needle is passed through the conjunctiva so that the needle point is unlikely to penetrate the globe.

The three anesthetic steps described in the present study were auriculopalpebral nerve block, topical anesthesia of the conjunctiva and cornea, and infiltration anesthesia of the superotemporal bulbar conjunctiva and the NM. Auriculopalpebral nerve block is a widely used method for paralyzing the orbicularis muscle in horses and cattle to facilitate ophthalmic examination and ocular surgery. In this study, the palpebral reflex was suppressed significantly or completely in all dogs, and adequate akinesia was achieved concurrently. Persistent paralysis of the orbicularis muscle may cause corneal problems in other procedures, but the cornea was protected by the NM in the procedure discussed in this study. However, orbicularis paralysis must be considered carefully during other ophthalmic procedures in order to avoid corneal damage; generous application of eye lubricant is recommended. Topical instillation of 0.5% proparacaine facilitated the subconjunctival injections of 2% lidocaine and the operations on the cornea. A combination of topical and infiltration anesthesia is considered to provide adequate analgesia for the flap.

When auriculopalpebral nerve block and infiltration anesthesia of the conjunctiva are performed, the administrator should remember that toxic doses of lidocaine can cause systemic and local side effects in the central nervous system, cardiovascular system, and skeletal muscles. The maximum dose of lidocaine allowed in healthy dogs is 12 mg/kg. The total dose of lidocaine used in auriculopalpebral nerve block and infiltration anesthesia for NM flap construction in this study was 12 mg. Therefore, when this anesthetic procedure is performed in dogs weighing ≤1 kg, the lidocaine dose should be reduced according to body weight of the dog. In order to have an adequate volume for local infiltration, the calculated dose of lidocaine can be diluted in an equal or greater volume of sterile saline.

Injecting anesthetic around the eyes of two aggressive dogs and one hyperactive young dog was impossible. Two other dogs became agitated after completion of combined

### Table

<table>
<thead>
<tr>
<th>Breed</th>
<th>No. of Dogs</th>
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</thead>
<tbody>
<tr>
<td>Shih tzu</td>
<td>23</td>
</tr>
<tr>
<td>Yorkshire terrier</td>
<td>6</td>
</tr>
<tr>
<td>Maltese</td>
<td>4</td>
</tr>
<tr>
<td>Pekingese</td>
<td>4</td>
</tr>
<tr>
<td>Mixed-breed dog</td>
<td>3</td>
</tr>
<tr>
<td>Miniature poodle</td>
<td>2</td>
</tr>
<tr>
<td>Chihuahua</td>
<td>2</td>
</tr>
<tr>
<td>American cocker spaniel</td>
<td>2</td>
</tr>
<tr>
<td>Miniature schnauzer</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>47</strong></td>
</tr>
</tbody>
</table>

flaps remained in place for the intended period (10 to 19 days) in 43 dogs. Premature flap failure occurred in four dogs; the NM flap held for 5 to 7 days in these cases. Causes of premature flap loosening were buphthalmia in two cases and inexperience of the operator in two cases.

**Discussion**

The combined local anesthetic technique reported here provides adequate analgesia for the surgical site and induces akinesia of the eyelid to facilitate NM-to-superotemporal bulbar conjunctiva flap placement under minimal physical restraint in dogs. Ocular surgery in a conscious patient under local anesthesia offers many advantages. First, the surgery can be performed in cases that would be at high risk under general anesthesia. Second, local anesthesia saves the extra time, money, and effort necessitated by general anesthesia. Local anesthesia can also serve as an alternative in veterinary practices that have limited access to the resources necessary for general anesthesia.

Ocular surgery is commonly performed without general anesthesia in humans and large animals. Auriculopalpebral nerve block is used for minor corneal procedures; supraorbital, lacrimal, and infratrochlear nerve blocks are used for upper eyelid repair; and infratrochlear nerve block is used for enucleation and radiation therapy in cattle with squamous cell carcinoma. In humans, various local anesthesia techniques (e.g., sub-Tenon’s capsule, intracameral, peribulbar, retrobulbar, and topical anesthesia) have been reported, especially in the setting of cataract surgery. Regional nerve block of the upper eyelid has also been reported in the setting of oculoplastic surgery in humans.

Conversely, general anesthesia is usually required for ocular surgery in dogs and cats to achieve immobilization and relaxation. However, NM flap construction does not require complete immobilization, because it is a less delicate procedure than most other ocular surgeries, and it is short in duration—typically <5 minutes. Therefore, we believed NM flap construction could be performed successfully under combined local anesthesia.

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Injecting anesthetic around the eyes of two aggressive dogs and one hyperactive young dog was impossible. Two other dogs became agitated after completion of combined
local anesthesia, and they failed to tolerate surgery. These dogs were either sedated or generally anesthetized in order to have surgery completed. Temperaments of animals must be carefully considered before the procedure. When combined with sedation, the NM flap procedure is better tolerated than when the combined local anesthetic technique is used alone, especially in dogs with poor temperament.

Conclusion
This study showed that combined local anesthesia is a time- and cost-effective procedure in NM-to-superotemporal bulbar conjunctiva flap construction, and it provides both analgesia of the surgical site and akinesia of the eyelid. This anesthetic technique would be particularly useful in dogs that are compliant and may be at high risk for general anesthesia. Furthermore, the technique may be beneficial in providing prolonged postoperative analgesia of the surgical site in dogs undergoing general anesthesia. For this supplementary postoperative analgesia, the local anesthetic can be chosen based on the specific needs of duration and onset of anesthetic effect.

Footnotes
a Daehan Lidocaine HCl 2%; Dai Han Pharm. Co., Ltd., Seoul, 150-100, Korea
b Alcaine 0.5%; Alcon, Puur, B-2870, Belgium
c Vicryl; Ethicon, Livingstone, EH54 OAB, United Kingdom

References
Primary Omental Abscessation or Omental Infarction in Four Dogs

Primary omental abscessation was diagnosed in three dogs after laparotomy. Laparotomy was performed to explore an abdominal mass of unknown origin and chronic fat necrosis diagnosed in one dog as an incidental finding during ovariohysterectomy. Primarily hypoechoic masses not connected to any abdominal structures were visualized with abdominal ultrasound in three dogs. Suppurative inflammation was diagnosed from fine-needle aspirate evaluation in two dogs. Bacteria were cultured from two abscesses despite the absence of organisms on Gram stain, cytology, and histopathology. Foreign material was found in one abscess. All four dogs experienced weight loss and/or an episode of gastrointestinal signs. Primary omental abscessation may be preceded by omental vascular compromise and/or interaction of the omentum with foreign material. J Am Anim Hosp Assoc 2009;45:168-175.

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Introduction

Omental abscessation in the absence of recent surgery or intraabdominal infection is uncommon in veterinary and human cases. This may be due to the extensive immune system within the omentum itself. Indeed, omentation (i.e., the deliberate placement of the omentum into a desired location by the surgeon) has been used to successfully treat non-resectable abscesses of the canine prostate, uterine stump, and pancreas. The same properties of the omentum that allow it to effectively wall off areas of infection can also make an omental abscess difficult to detect until it leaks and causes peritonitis, erodes health due to chronic inflammation, and/or creates a space-occupying effect. Suggested etiologies for a primary omental abscess in humans include compromise of omental vasculature by infarction or torsion, foreign body reaction, and hematogenous spread. The purpose of this paper is to describe the clinical presentation, diagnostic assessment, and treatment of three dogs with primary omental abscesses and one dog with omental infarction.

Case Reports

Case No. 1

A 33-kg, overweight, spayed female golden retriever known to be at least 13 years old was presented after an episode of vomiting. The referring veterinarian identified a large abdominal mass, suspected to be splenic in origin, on abdominal palpation and radiographs. The dog was referred to the Washington State University Veterinary Teaching Hospital the following day.

Case no. 1 had an extensive medical history, with previous diagnoses of cardiomegaly, pulmonary edema, hypertension, arthritis, lumbosacral spondylosis, hypothyroidism, uveitis, glaucoma (with enucleation of the right eye), oral ossifying epulis, and vulvar trichoblastoma. Abdominal ultrasonography was performed as part of the workup for hypertension 16 months earlier, and no significant findings were seen. Fourteen weeks earlier, case no. 1 was presented to the referring veterinarian because of
diarrhea, hair loss, difficulty going upstairs, unsteady hindquarters, and a 5-kg weight loss. Current medications were enalapril, furosemide, digoxin, carprofen, levothyroxine sodium, and prednisolone acetate eye drops. Mucous membranes were pale, heart rate was regular with good pulses, lung sounds were clear, and the abdomen was pendulous. A fluid wave and possible intraabdominal mass were palpated. A complete blood count (CBC) was normal. Abnormalities on a serum biochemical panel were elevated blood urea nitrogen (BUN) concentration (34.8 mg/dL, reference range 9 to 27 mg/dL) and amylase activity (1801 U/L, reference range 500 to 1500 U/L). The digoxin dose was reduced and the dog improved.

Upon the dog’s arrival at the Washington State University Veterinary Teaching Hospital, the owner reported the dog had eaten well the night before and was no longer vomiting. Physical examination revealed marked abdominal distension and a nonpainful, approximately 8-cm diameter, round movable mass in the cranioventral abdomen. Other abnormalities were a grade I/VI systolic heart murmur, mild to moderate dental disease, and ossifying epulis. A CBC revealed a mature neutrophilic leukocytosis (17,600 white blood cells [WBCs]/µL, reference range 5800 to 11,700 WBCs/µL; 15.664 neutrophils/µL, reference range 3000 to 7100 neutrophils/µL) and mild microcytic, hypochromic anemia (packed cell volume [PCV] 37%, reference range 38% to 59%; mean corpuscular volume [MCV] 52 µ3, reference range 64 to 73 µ3; mean corpuscular hemoglobin 18 picograms, reference range 21 to 26 picograms). Large platelets and a few keratocytes, blister cells, target cells, and schistocytes were seen on a blood smear. Serum biochemical abnormalities were high alkaline phosphatase (ALP) activity (120 U/L, reference range 14 to 72 U/L); high concentrations of BUN (68 mg/dL, reference range 9 to 27 mg/dL), creatinine (2.6 mg/dL, reference range 0.7 to 1.2 mg/dL), and phosphorus (6.9 mg/dL, reference range 3.3 to 6.0 mg/dL); and low concentrations of albumin (2.4 g/dL, reference range 2.5 to 3.8 g/dL), sodium (149 mEq/L, reference range 163 to 168 mEq/L), and chloride (113 mEq/L, reference range 117 to 120 mEq/L). Activated partial thromboplastin time (aPTT) and partial thrombin time (PTT) were within reference ranges.

Echocardiography revealed moderately increased left ventricular wall thickness with normal systolic function and no obvious cardiac masses. Systolic blood pressure was elevated at 180 mm Hg. Chest radiographs revealed a prominent aortic arch and left ventricular hypertrophy, both consistent with systemic hypertension. An approximately 11-cm diameter spherical mass was seen in the ventral aspect of the cranial third of the abdomen with ultrasound. The mass was composite in nature with hyperechoic swirls of tissue embedded in an essentially hypoechoic tissue matrix. Repeated efforts to show attachment of the mass to the spleen or to the liver were unsuccessful. A slight amount of free abdominal fluid was present.

Based on the dog’s signalment, red blood cell (RBC) changes, and ultrasound findings, hemangiosarcoma was the primary differential diagnosis for the abdominal mass. Digoxin and furosemide were discontinued because of normal cardiac function and to allow more accurate assessment of renal function. Enalapril was continued for systemic hypertension, but the dose was decreased. Because iatrogenic hyperthyroidism was a potential cause of the hypertension, the thyroxine dose was halved.

Six days later, the dog returned for a scheduled exploratory laparotomy. Packed cell volume was 41%, and total protein was 7.4 g/dL. Serum BUN (31 mg/dL) and creatinine (1.5 mg/dL) concentrations were improved but still elevated. Urine specific gravity was 1.017, confirming renal compromise. Blood pressure was 172 mm Hg systolic, 109 mm Hg diastolic, and 129 mm Hg mean.

A ventral midline abdominal exploratory was performed. A 15-cm diameter mass, estimated to weigh approximately 2 kg, was present in the greater omentum [Figure 1A]. No connections were between the mass and any other organs. Purulent fluid oozed out of the mass during manipulation. A partial omentectomy was performed, and the large vessels were ligated with 2-0 silk suture. A few thin, flexible, linear, blue fragments (approximately 1 × 0.1 cm) of unknown composition were found in association with the mass. A copious amount of purulent material leaked out of the excised mass after it was incised [Figure 1B]. Fluid cytology revealed a pyogranulomatous inflammation with previous hemorrhage and secondary fibroplasia. Rare pieces of extracellular, angular, blue-green material consistent with plant material were also seen.

Cefazolin (22 mg/kg intravenously [IV]) was given at anesthesia induction, every 1.5 hours intraoperatively (two doses), and every 6 hours postoperatively (two doses). Amoxicillin-clavulanic acid (20.5 mg/kg per os [PO] q 12 hours) was then administered for 14 days postoperatively. Postoperative management also included IV fluids and analgesics. Recovery was uneventful with the exception of persistent hypertension.

Histological examination of the omental mass revealed thick bands of connective tissue partially surrounding a central area of inflammation that contained neutrophils, macrophages, and a few eosinophils. Occasional foreign matter consisting of bright pink, thin, angular material was contained in dense granulomas of neutrophils and macrophages. Small numbers of multinucleated giant cells and aggregates of streaming cellular debris were adjacent to some of the foreign material. The occasional honeycombed appearance of the material was suggestive of plant matter. The histological diagnosis was chronic, active, locally extensive omental abscess associated with foreign material. The individual fragments of blue material recovered intraoperatively did not survive the histological preparation process. Other histological findings from biopsies taken intraoperatively included moderate, diffuse, hydropic degeneration with iron granulomas in the liver; moderate, chronic medullary fibrosis in the kidney; and normal pancreas. No organisms were identified on cytology, Gram stain, histological section, aerobic culture, or anaerobic culture of the omental mass or its contents.
Two weeks postoperatively, the dog was doing well. No abnormalities were detected on abdominal palpation. A serum biochemical panel found elevated BUN (45 mg/dL), creatinine (2.6 mg/dL), and potassium (6.0 mEq/L, reference range 4.4 to 5.3 mEq/L) concentrations. Systolic blood pressure ranged from 190 to 220 mm Hg. The dog was started on atenolol for hypertension. Eight days later, the dog became very weak and collapsed. Physical examination by the referring veterinarian revealed a left head tilt, nonpositional nystagmus, disorientation, and severe weakness. The heart rate was 120 beats per minute and steady, pulses were strong, and mucous membranes were pink. After treatment with IV diazepam, intramuscular dexamethasone, and subcutaneous fluids, the dog appeared more comfortable; however, neurological signs and weakness persisted. The dog died later at home. No necropsy was performed.

**Case No. 2**

A 10-year-old, spayed female, miniature schnauzer was presented for assessment of urinary incontinence. Abnormal findings on physical examination were a 15% weight loss compared to the previous year (from 6.7 kg to 5.7 kg); bilateral lenticular sclerosis; a grade 2/6 heart murmur with the point of maximal intensity over the right apex; and a firm, round abdominal mass that did not appear to be associated with any organ. The dog was tense on abdominal palpation but did not appear painful.

A CBC was unremarkable. Alkaline transferase (ALT) activity (142 U/L, reference range 12 to 67 U/L) and glucose (125 mg/dL, reference range 70 to 117 mg/dL) concentrations were elevated. Fecal matter prevented clear visualization of the mass on abdominal radiographs. Results of a fine-needle aspiration (FNA) of the mass, guided by palpation, were consistent with a pyogranulomatous inflammatory response. The owner declined further workup at that time. Diethylstilbestrol was dispensed for presumptive urinary sphincter mechanism incompetence.

Two months later, the dog was presented again for further assessment of the abdominal mass. The dog had been doing well, and urinary incontinence had improved. Body weight had increased to 6.5 kg, and the body condition score was 3 out of 5. Grade 3/5 dental disease was present. On abdominal palpation, the dog tensed such that the mass could not be palpated; but once again, the dog did not seem overtly painful.

A CBC indicated mild hypochromic anemia (PCV 36%; RBCs 5.55 × 10⁶/µL, reference range 5.6 to 8.5 × 10⁶/µL; hemoglobin [Hb] 12.2 g/dL, reference range 13 to 19 g/dL); normal total WBC (10,200 cells/µL) with normal neutrophil (6426 cells/µL) and lymphocyte (2556 cells/µL) concentrations; monocytosis (1020 cells/µL); eosinophilia (204 cells/µL, reference range <100 cells/µL); and thrombocytosis (488,000 platelets/µL, reference range 157,000 to 394,000 platelets/µL). Slight anisocytosis and large platelets were seen on a blood smear. Abnormalities on a serum biochemical panel were elevated ALT (162 U/L) and ALP (165 U/L) activities, increased globulin (4.3 g/dL, reference range 2.7 to 3.8 g/dL) concentration, and decreased BUN (7 mg/dL) and calcium (8.4 mg/dL, reference range 9.1 to 12 mg/dL) concentrations.

Abdominal ultrasound revealed a round mass with a maximum diameter of 2.8 cm cranial to the bladder and in isolation from other abdominal structures. The rim of the mass had alternating hypoechoic and hyperechoic layers suggestive of an intestinal wall, but no intestinal loop could be traced to this structure. The center of the mass was hypoechoic. The spleen contained multiple masses of mixed echogenicity; the largest mass was 2.1 cm in diameter. Enlarged lymph nodes in the mesenteric root retained central hyperechogenicity, suggestive of reactive lymphadenopathy.

**Figures 1A,1B**—(A) A 15-cm diameter mass was found in the greater omentum of an approximately 13-year-old, spayed female golden retriever (case no. 1). No connections to any other organs were present. (B) After an incision was made into the excised mass with a scalpel blade, the abscess deflated and released a copious amount of purulent material.
Ultrasound-guided FNAs were indicative of a suppurative, possibly septic inflammatory response in the mass and mild extramedullary hematopoiesis in the spleen. Chest radiographs were within normal limits.

At surgery, a 5-cm diameter, spherical mass was found in the omentum cranial to the bladder; it was removed via partial omentectomy performed with monopolar electrocautery. A partial splenectomy and liver biopsy were performed. The dog received one dose of cefazolin (22 mg/kg IV) intraoperatively. No additional antibiotics were administered.

Postoperative management included IV fluids and analgesics. One day postoperatively, a CBC and blood smear revealed a neutrophilic leukocytosis (24,500 WBCs/µL; 17,640 neutrophils/µL) with a left shift (3185 band cells/µL, reference range <300 band cells/µL); thrombocytosis (594,000 platelets/µL, reference range 157,000 to 394,000 platelets/µL); hyposegmented polymorphonuclear cells; Döhle bodies; and platelet clumps. The dog was discharged on the second postoperative day, and recovery was uneventful.

The omental mass was filled with a milky, suppurative fluid. Histological diagnosis was a focal, chronic omental abscess. The abscess was cystic with a discrete, dense collagenous wall infiltrated with numerous small blood vessels and aggregates of lymphocytes admixed with large numbers of fibrocytes. The center of the cyst was filled with necrotic tissue and sheets of neutrophils and macrophages with fragments of mineralized necrotic debris. The omentum surrounding the mass had multifocal, extensive areas of necrosis with saponification of adipose tissue and numerous hemosiderophages. Nodular hyperplasia of the spleen, hydropic degeneration of the liver, and mild suppurative hepatitis were also present. No organisms were seen on a Gram stain, cytology, or histopathology from the omental mass, and aerobic cultures were negative. A moderate number of *Bacteroides ureolyticus* grew on anaerobic culture.

Nine months later, case no. 2 was diagnosed with severe pancreatitis and diabetes mellitus. Abdominal ultrasound showed right lobe pancreatitis with secondary peritonitis and diffuse alterations in hepatic echogenicity. Hepatic FNAs were consistent with mild hepatic vacuolar degeneration, cholestasis, and possible mild hepatic lipidosis. The dog responded to medical treatment for this episode of pancreatitis as well as for another episode experienced 2 months later. One year following the omental abscess, the dog continued under treatment for diabetes mellitus.

**Case No. 3**

A 1.5-year-old, 37.3-kg, neutered male yellow Labrador retriever was referred to the Washington State University Veterinary Teaching Hospital with a 3-day history of anorexia, depression, fever, and abdominal distention; one episode of diarrhea; and a report of loss of detail on abdominal radiographs. Two months earlier, the referring veterinarian had given the dog dipyrone and sulfadiazine/trimethoprim for diarrhea, which resolved. Weight loss (from 39.5 kg to 36.5 kg over 3 months) was documented at that time. The following month, the veterinarian surgically removed an approximately 1.8-cm diameter mass that had been on the left caudal lumbar area for 2 weeks. The histological diagnosis was focally extensive dermal fibrosis with reactive connective tissue and a necrotic tract.

Upon presentation at the Washington State University Veterinary Teaching Hospital, the dog was depressed, tachycardic, tachypneic, normothermic, and had abdominal distension with a palpable fluid wave. The abdomen was not painful. A CBC revealed a leukocytosis (28,100 WBCs/µL) with neutrophilia (17,141 cells/µL); a left shift (2810 band cells/µL); lymphocytosis (5620 cells/µL, reference range 1100 to 5100 cells/µL); monocytes (2529 cells/mL); and a mild hypochromic, microcytic anemia (PCV 34%; Hb 12.4 g/dL; MCV 63 µ3; RBC distribution width 17%, reference range 14% to 16%). Anisocytosis was seen on a blood smear. Elevated ALP activity (106 U/L) and decreased albumin (2.2 g/dL), globulin (2.3 g/dL), calcium (7.8 mg/dL), phosphorus (2.9 mg/dL), sodium (117 mEq/L), potassium (4.1 mEq/L), chloride (94 mEq/L), and carbon dioxide (16.2 mmol/L, reference range 18 to 24 mmol/L) concentrations were present on a serum biochemical panel.

Abdominocentesis recovered 2100 mL of a serosanguineous exudate containing >170,000 cells/µL (97% moderately degenerate neutrophils, 3% macrophages) and 3 g/dL protein. On abdominal ultrasound, a 2.5 × 5 × 2.5-cm hypoechoic cluster of tissue was found immediately dorsal and medial to the spleen; it could not be connected to any abdominal structures. An FNA of the mass recovered 84% markedly degenerate neutrophils and 16% macrophages, indicating a suppurative inflammatory response; no organisms were seen. Thoracic radiographs showed a small caudal vena cava indicative of dehydration. Sternal lymphadenopathy, consistent with cranial abdominal disease, was also seen. An approximately 1.5-cm focus of periosteal reaction was present on the ventral third of the left eighth rib.

At surgery, a firm, red, necrotic-appearing, 5-cm diameter mass containing multiple pockets of purulent material was excised from the greater omentum. The abdomen was lavaged, and two Jackson-Pratt drains were placed. An esophagostomy feeding tube was also placed. Postoperative management included IV fluids, hetastarch, analgesics, ranitidine, sucralfate, enrofloxacin (2.4 mg/kg IV q 12 hours), ampicillin (22 mg/kg IV q 8 hours; the first dose was given intraoperatively), and feedings through the esophagostomy tube. Antibiotics were administered for a total of 2 weeks, with a switch to oral medications (enrofloxacin 3.6 mg/kg PO q 12 hours; ampicillin 20 mg/kg PO q 8 hours) 3 days postoperatively.

The morning following surgery, the dog was alert and able to go on a short walk outside. Fifteen minutes later, the dog was recumbent and severely pale and was diagnosed with hemoabdomen. A CBC showed pronounced neutrophilic leukocytosis (51,200 WBCs/µL; 34,816 neutrophils/µL) with a left shift (10,752 band cells/µL) and monocytes (2048 cells/µL). Neutrophils had moderate...
toxic changes and Dohle bodies. Anemia (PCV 15% and 2.4 × 10^6 RBCs/µL) and hypoproteinemia (1.0 g albumin/dL and 1.7 g globulin/dL) were marked. The PTT was normal, while aPTT was prolonged (18.4 seconds, reference range 8.9 to 12.8 seconds). Treatments included blood transfusions, hetastarch, and lactated Ringer’s solution. The dog was returned to surgery, and bleeding omental vessels were ligated. On the third postoperative day, the dog began to eat voluntarily, and a full recovery eventually occurred.

No organisms were seen on Gram stain of the abdominal fluid sampled during the first surgery. Culture of the abdominal fluid grew many Prevotella species and a few Escherichia coli. Histological examination of the omentum identified severe, chronic, multifocal, pyogranulomatous to abscessing omentitis consistent with a foreign body reaction, although no foreign material was seen. No bacteria or fungal organisms were found with special histological stains.

Four months after surgery for the omental abscess, an approximately 2.5-cm diameter, discoid subcutaneous mass and its accompanying tract (approximately 6.6-cm long) of inflamed tissue was removed from over the left rib cage. A Penrose drain was placed. The quantity and duration (>10 days) of drainage were both greater than expected, and the site took several months to heal. The histological diagnosis was ischemic, fibrotic, moderately inflamed adipose tissue likely secondary to trauma or ischemia. The dog was last seen 3.5 years after the omental abscess and was doing well.

**Case No. 4**

A 1-year-old, intact female Labrador retriever was seen on emergency for a peracute onset of repetitive, profuse vomiting; abdominal pain; and shock (pale mucous membranes, prolonged capillary refill time, tachycardia, weak pulses, tachypnea, and hypotension). The dog had been fine when placed alone in a fenced-in yard 1 hour earlier. Blood smear and CBC were normal. Serum biochemical abnormalities (after fluid treatment for shock) were elevated ALT (1120 U/L), ALP (81 U/L), and amylase (1415 U/L) activities and decreased concentrations of protein (1.1 g albumin/dL; 2.0 g globulin/dL), calcium (8.6 mg/dL), and potassium (3.6 mEq/L). Canine parvoviral antigen test and fecal smear and flotation were negative. On abdominal radiographs, the stomach was displaced dorsally and to the left, with the long axis nearly perpendicular to the ribs, suggestive of an extraluminal mass or local peritonitis. Treatment included IV isotonic fluids, hetastarch, ranitidine, sucralfate, and ampicillin.

The following day, the dog was marked improved. A CBC showed leukocytosis (16,000 cells/µL) and neutrophilia (12,800 cells/µL) and eosinophilia (640 cells/µL). A blood smear revealed slight anisocytosis and polychromasia, a few hyposegmented neutrophils, toxic neutrophils, reactive lymphocytes, rare Howell-Jolly bodies, and giant platelets. Elevated ALT (1879 U/L), ALP (131 U/L), and amylase (1415 U/L) activities and hypoproteinemia (3.0 g albumin/dL; 2.1 g globulin/dL) were still present. On a barium contrast study, the stomach was back in its normal position, gastrointestinal transit time was normal, and no evidence of luminal compromise was seen. The dog was discharged on sucralfate and ranitidine. A bland diet was administered, and recovery was complete.

During routine ovariohysterectomy 1 year later, a 1.5 × 2 × 3-cm omental mass was an incidental finding in the superficial leaf of the greater omentum; the mass was excised after surrounding omental vessels were ligated with polydioxanone suture. The histological diagnosis was chronic fat necrosis with focal eosinophilic and granulomatous peritonitis. These changes were consistent with disruption of the vascular supply, and such findings led to a presumptive diagnosis of omental infarction as the cause of the acute abdomen 1 year earlier.

**Discussion**

Primary omental abscessation and omental infarction are uncommon in dogs. The four dogs of this report were the only cases found during a computerized search of canine and feline medical records from January 1994 to March 2007 at the Washington State University Veterinary Teaching Hospital.

A search of the veterinary literature recovered two other cases of omental abscessation, both in dogs. A 17 × 18-cm, 2.1-kg omental abscess was removed from an 11-year-old, neutered male, German shepherd mixed-breed dog with concurrent discospondylitis. Clinical signs included 6 weeks of weight loss and abdominal distention. Cultures from the omental mass, blood, and peritoneal fluid were all positive for Staphylococcus intermedius. Because the original site of infection was not determined in this dog, it is unknown if the infection in the omentum was primary or secondary.

Staphylococcus intermedius was also cultured from a 2.2-kg omental mass removed from a 6-year-old, intact male German shepherd dog with acute ataxia, weight loss, and a palpable abdominal mass. Botryomycosis (i.e., bacterial pseudogranuloma) was the histological diagnosis in this dog. Neither of these reports focused on primary omental abscessation in their discussions.

The initiating cause of omental abscessation in case nos. 1, 2, and 3 could not be definitively determined. Rodent models demonstrate that a very large inoculum of bacteria is required to create an intraabdominal abscess in a healthy animal; this dose is such that it can lead to fulminant sepsis and death. Based on this observation, together with the rarity of infection in the omentum relative to other sites of the body and the extensive omental antibacterial defense system, the likelihood is strong that omental vascular compromise and/or the presence of foreign material precede most cases of primary omental abscessation. During the several months prior to the diagnosis of omental abscess, case no. 1 had a period of weakness, weight loss, and diarrhea; case no. 2 had weight loss; and case no. 3 had an episode of diarrhea and weight loss similar to signs described for one dog in a previous report. Though unable to be proven, these episodes possibly represented some initial omental compromise or introduction of foreign material into the abdominal cavity, as discussed below.
Foreign material can reach the omentum via penetration of the peritoneum or luminal organs. No history of penetrating injury to the abdomen or recent abdominal surgery (case nos. 1 and 2 were spayed >4 years prior to presentation) was reported for any of the dogs. However, a small gastrointestinal perforation that seals on its own may go unnoticed by the owner or be dismissed as a transient gastrointestinal upset (similar to the gastrointestinal events reported for several of the dogs).9-11 Because marked foreign body reactions can be secondary to miniscule fragments of material, the inability of the surgeon or pathologist to find foreign material does not exclude the possibility that foreign material contributed to abscessation.12

Migrating plant awns, which can penetrate the gastrointestinal tract after ingestion, were suspected in case no. 3 because of subcutaneous masses with associated connective tissue tracts and prolonged drainage after excision.13 One mass was in the lumbar area (a location where inhaled migrating plant awns often lodge), and the other was near a site of costal periosteal proliferation, which can be a sign of foreign body migration.12,14 Ongoing inflammation associated with foreign material would explain why case no. 3 presented with a microcytic, hypochromic anemia despite an apparent acute onset of illness, as well as a historic weight loss.

The presentation and clinical course of illness in case no. 4 resembled that of segmental omental infarction or torsion in humans, who present with an acute abdomen that is often misdiagnosed as appendicitis due to its severity.15 These conditions are usually self-limiting, although they occasionally lead to omental necrosis and abscessation.16 In retrospect, the malposition of the stomach seen on radiographs of this dog was consistent with entrapment and strangulation of the omentum in the epiploic foramen. Resolution of the entrapment would explain the normal stomach position and resolving clinical signs the following day. In a dog operated on for acute abdomen, examination of the omentum for torsion or infarction is advised, especially when no other cause can be found.17

Obesity and cardiovascular disease (both present in case no. 1) are predisposing factors for omental infarction in humans.15 The extensive fat necrosis around the abscess in case no. 2 suggested an earlier bout of pancreatitis may have contributed to omental compromise. This dog later developed diabetes mellitus, which in humans has been associated with impairment of omental vascularization.15

The presentation of dogs with primary omental abscessation varied from acute illness to a persistent abdominal mass with minimal clinical signs. Abdominal distension, but not pain, was reported. When a mass was palpable, no definitive connection with a specific abdominal organ could be made; this suggested an omental origin. The most common abnormalities seen on blood analyses were mild anemia, mild to moderate inflammatory leukogram, and hepatobiliary inflammation, most likely from bacterial or toxic showering from the abscess. Alterations in protein levels were consistent with long-standing inflammation (hypalbuminemia) or bacterial antigen stimulation (hyperglobulinemia). Postoperative exacerbation of neutrophilia, as identified in case nos. 2 and 3, is explained by granulopoiesis that had been upregulated to compensate for WBC sequestration and consumption in the abscess.18

Radiographic signs associated with an abdominal abscess in veterinary cases may include a soft-tissue mass containing gas or a gas-fluid interface, an air-fluid interface seen with a horizontal beam, and/or loss of serosal detail because of associated effusion.6 A soft-tissue mass was visible on abdominal radiographs in case no. 1, and a loss of detail was noted on radiographs from case no. 3 (similar to previous reports6,7), but the omental origin was not apparent and no gas was observed. Because neoplasia is a potential cause of any abdominal mass, performing chest radiography to look for metastatic disease is appropriate.

Similar to previous reports, abnormal tissue was detected on abdominal ultrasounds of all three dogs with omental masses, although none of the masses could be shown to originate from a specific tissue, including omentum.6,7 In these dogs, the dimensions of the omental masses measured intraoperatively were larger than those measured ultrasonographically. These discrepancies may be because measurements were taken from different reference points, precise margins were difficult to determine, and/or because ultrasound can measure directly through the center of a mass while intraoperative measurement of diameter of an intact mass requires a degree of estimation.

Canine abdominal abscesses generally appear as a hypoechoic mass with an irregular border on ultrasound. Ultrasound penetration of the mass is poor.19 Variations in echogenicity can occur because of differences in fluid viscosity and cellularity, density of granulomatous tissues, gas accumulation, presence of foreign material, and/or calcification.19 This ultrasonic appearance is not unique to abscesses; differential diagnoses include hematoma, primary or metastatic neoplasia with necrosis or hemorrhage, and complex cyst with cellular debris or hemorrhage.19 Magnetic resonance imaging, with its excellent delineation of the omentum and other soft tissues, would likely have been a more effective way to localize the mass to the omentum and assess for local metastases. This definitive procedure would increase the preoperative confidence of the surgeon and pet owner that surgery would successfully eliminate disease.20

Percutaneous FNA of abdominal masses is generally associated with high diagnostic accuracy and minimal complications, especially when under ultrasound guidance.21,22 Fine-needle aspiration was not performed in case no. 1 because of the high suspicion that the mass was a hemangiosarcoma.23 Fine-needle aspirates provided evidence of the suppurative nature of the intraabdominal masses in case nos. 2 and 3. This finding lessened the concern for malignancy but did not rule it out, as areas of necrosis and inflammation can mask underlying neoplasia.24

The presence of an abdominal mass of unknown origin was the main indication for exploratory surgery in all dogs.
with omental abscesses. Similar to previous reports, the abscess in case no. 1 weighed about 2 kg, demonstrating the remarkable ability of the omentum to wall off an area of inflammation from the rest of the abdominal cavity, even when the source of the inflammation is within the omentum itself.\textsuperscript{6,7} Partial omentectomy was performed with either electrocautery or sequential suture ligation of omental vessels. A margin of grossly normal omentum should be removed where possible, in case the mass is neoplastic. Perioperative challenges included leakage of purulent material from the mass (which was managed by packing moist laparotomy pads on and around the mass and performing lavage), proximity of the omental mass to the main splenic vessels (which required careful dissection), and postoperative management of abdominal drainage. Care must be taken to ensure that fat in the omentum does not prevent ligatures from completely collapsing encircled vessels. Since some omental tissue remains after removal of the abscess, the potential for future omental disease exists in all cases.

Bacteria cultured from the canine omental abscesses were all normal inhabitants of the oral cavity, intestine, and/or skin. These are all potential entry sites for foreign material, which often carries bacteria indigenous to its point of penetration into deeper tissue.\textsuperscript{12} Hematogenous spread of bacteria from the oral cavity of a dog with dental disease is also possible.\textsuperscript{25} Important to note is that positive cultures were obtained in case nos. 2 and 3 even though no microorganisms were identified with Gram stain, cytology, and histopathology. Thus, omental abscesses should be cultured for aerobic and anaerobic bacteria even in the absence of bacterial detection by other means. All dogs received antibiotics, although the duration of administration varied from a single intraoperative dose to 2 weeks. The indication for ongoing antibiotic administration is clear in dogs with purulent material free in the abdominal cavity. When the omental abscess is well-encapsulated, limiting antibiotics to the perioperative period may be sufficient.

Adverse consequences of omentectomy in otherwise healthy patients are not reported. Omentectomy is a recommended part of the treatment of human neoplasms with a high incidence of peritoneal dissemination (e.g., ovarian and advanced colorectal cancers), because the omentum serves as a site of tumor implantation.\textsuperscript{26} Omentectomy is also performed to prevent blockage of peritoneal catheters in dialysis patients.\textsuperscript{27} In veterinary medicine, omentectomy has been associated with decreased adhesion formation after abdominal surgery in the horse.\textsuperscript{28} However, omentectomy may adversely affect intraabdominal bacterial defenses in rats, and it is associated with increased mortality in dogs with experimentally induced drainage of pancreatic juice into the abdomen.\textsuperscript{29,30} Beneficial properties of the omentum include immune surveillance, sealing of gastrointestinal leaks, promotion of hemostasis, drainage of nonresectable abscesses, and stimulation of wound healing.\textsuperscript{3,4,31,32} Indiscriminate omentectomy is not recommended, because the omentum, via its inherent activity or through surgical omentalization, can be a key factor in the successful management of potentially life-threatening conditions. Also, the incidence of omental disease is low.

**Conclusion**

Dogs with primary omental abscessation or omental infarction may be presented with an acute illness or a persistent abdominal mass with few clinical signs, although a history of weight loss and/or gastrointestinal signs may exist. The leukogram may not accurately reflect the actual degree of inflammation or infection, especially in dogs with a chronic abscess. Ultrasound may be more informative than radiographs regarding the nature of the abdominal mass. Fine-needle aspiration is a useful tool to identify suppurrative contents of the abscess. Despite absence of bacteria on Gram stain, cytology, and histopathology, aerobic and anaerobic cultures should always be performed. Omental abscesses can be cured with surgical resection and appropriate supportive care. Omental infarction may cause an acute abdomen that responds to medical therapy. The omentum should be examined for sites of infection or abscessation during exploratory laparotomy for acute abdomen, especially when no other cause can be found.

**References**

Successful Treatment of a Metacarpal Trophic Ulcer Utilizing a Neurovascular Island Flap

A 4-year-old, neutered male, soft coated wheaten terrier mixed-breed dog was presented for evaluation of a nonhealing ulcer of the metacarpal pad of 10 to 12 weeks’ duration. A diagnosis of an ulnar nerve deficit and secondary trophic ulceration was made. The ulcer was repaired using a neurovascular island flap. Postoperatively, a fiberglass clamshell splint was used to protect the healing flap. The flap healed successfully and developed a highly keratinized epithelium much like a normal footpad.


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Introduction

Trophic ulceration of the metacarpal, metatarsal, and digital pads is an uncommon sequela of peripheral nerve damage. Medical management of cutaneous trophic ulceration is intensive, long term, and frequently unrewarding in both human and veterinary patients. In the veterinary literature, two case series describe the use of neurovascular island flaps to provide sensitized skin coverage to trophic ulcers of the metacarpal pad. Outcomes of these five cases varied from excellent to poor. This case report represents the modification to a previously described technique with a successful outcome for a dog.

Case Report

A 4-year-old, neutered male, soft coated wheaten terrier mixed-breed dog was presented for evaluation and treatment of a nonhealing ulcer on the metacarpal pad of the left thoracic limb. The ulceration had been present for 10 to 12 weeks. Approximately 3 years prior, the dog was hit by a car and suffered neurological dysfunction of the left thoracic limb, consistent with brachial plexus injury. The dog gradually recovered neurological function of the limb but had a persistent, left thoracic limb lameness. The referring veterinarian (RDVM) initially instructed the owner to manage the wound by soaking the foot in a chlorhexidine solution on a daily basis. Four weeks prior to presentation, the RDVM reevaluated the lesion on the metacarpal pad and performed a punch biopsy at the margin of the lesion. Initial histopathological evaluation of the lesion was consistent with a diagnosis of squamous cell carcinoma.

On presentation, a 4-mm, circular ulcer was seen on the proximolateral aspect of the metacarpal pad of the left thoracic limb [Figure 1]. Abnormalities in the neurological examination were limited to the left thoracic limb. The dog exhibited a normal gait; however, mild hyperextension of the left carpus resulted in a slight palmarigrade stance during weightbearing. The withdrawal and panniculus reflexes and muscle tone were normal in the left thoracic limb. Atrophy of the muscles of the brachium and antebrachium was noted when compared to the corresponding musculature of the right thoracic limb. Cutaneous sensation was absent in the area of the ulcer and the lateral aspect of the metacarpal pad.
Cutaneous sensation was normal proximal to the metacarpal pad and normal in the autonomous zone for the radial nerve [Figure 2].

Neuroanatomical diagnosis was consistent with a peripheral neuropathy affecting the cutaneous innervation in the distribution of the ulnar, median, and musculocutaneous nerves. The palmigrade stance suggested an abnormality in the palmar carpal ligament. The underlying cause of the palmar carpal ligament abnormality may be related to a neurological deficit or traumatic injury. It was not possible to determine if this carpal laxity was a direct result of the dog being hit by a car or was a later development, because stressed view radiographs had not been made at the time of injury; however, the injury was not believed to be the cause of the ulcer. Complete blood count and serum biochemical profile were within reference ranges, except for a mild thrombocytopenia (144,000 cells/µL; reference range 235,000 to 694,000 cells/µL) that was considered to be clinically insignificant. Based on the initial histopathological evaluation of the ulceration, fine-needle aspiration was performed on the left prescapular lymph node, and cytological evaluation was normal.

The dog was anesthetized for biopsies of the ulcer and for electrophysiological testing of the nerves supplying the ulcerated skin. Motor nerve conduction studies were performed on the ulnar and radial nerves as previously described. The radial nerve had normal motor nerve conduction velocity (mNCV). The ulnar nerve had a reduced compound muscle action potential. The mNCV of the ulnar nerve proximal to the elbow was normal but was prolonged distal to the elbow. These results were consistent with a distal neuropathy affecting the ulnar nerve. Because of the neurological deficits, a trophic ulcer was suspected. To confirm the diagnosis of squamous cell carcinoma, a wedge incisional biopsy was taken from the junction of normal and abnormal tissue in the area of ulceration on the metacarpal pad. Histopathological evaluation of the specimen was consistent with chronic, focally extensive, ulcerative, hyperplastic, suppurative pododermatitis. The original biopsy was reviewed by two separate pathologists (neither of which was the original pathologist), who both read the sample out as consistent with chronic, ulcerative pododermatitis rather than squamous cell carcinoma.

Based on the history, neurological examination findings, electrophysiological testing, and histological evaluation of both biopsy specimens, the cutaneous ulcer was believed to be the result of chronic denervation, and a diagnosis of a trophic ulcer was made. In order to treat the ulcer, a neurovascular island skin flap was planned, which would preserve the neurovascular supply to the overlying skin. A nonadhesive, soft padded bandage was placed on the paw to protect the sutures at the biopsy site. The bandage was changed every 72 hours and maintained for 15 days until surgery was performed. The biopsy site had healed, but the ulcer remained and appeared unchanged.

Carpal arthrodesis was not considered as an initial therapeutic option, because the palmigrade stance was considered to be mild and not the cause of the ulcer. Additionally, the complication rate reported with carpal arthrodesis is high. Instead, a neurovascular island flap was performed, and carpal arthrodesis was reserved as a salvage procedure if the flap failed to resolve the ulcer.

Neurological testing of the donor site was performed immediately prior to surgery. Stimulation of the skin on the dorsal aspect of the base of the fourth digit with forceps resulted in a withdrawal reflex and a conscious response; this indicated normal sensory innervation supplied by the lateral branch of the superficial branch of the radial nerve.
The left thoracic limb was clipped and prepared for aseptic surgery. An Esmarch tourniquet was applied by tightly wrapping the forelimb with sterile elastic tape, beginning at the toes and wrapping in a proximal direction to the mid antebrachium. The elastic tape was then cut, beginning at the toes and extending proximally. A 3-cm band of tape was left at the proximal-most aspect of the bandage. This acted as a tourniquet to prevent venous congestion and reduce the arterial blood supply, thereby providing a relatively bloodless field that allowed accurate visualization of neurovascular structures. An incision was made through the skin over the dorsal aspect of the fourth metacarpal bone.

The trifurcation of the neurovascular bundle was identified and isolated. This bundle contained the lateral branch of the superficial radial nerve, the cranial superficial antebrachial artery, and the accessory cephalic vein into the dorsal common digital nerves, arteries, and veins (II, III, and IV). The neurovascular bundle containing the dorsal common digital nerve artery and vein IV was dissected from the underlying connective tissue, at the point of the trifurcation to just proximal to the base of the fourth digit. A premade paper template, patterned after the defect in the metatarsal pad, was used to outline the island of skin at the base of the fourth digit that would be used as the island flap. The outlined area of skin was incised and dissected free of the underlying fascia from distal to proximal, with care taken to elevate the neurovascular bundle with the flap. The island and its pedicle were wrapped in a saline-soaked gauze sponge while the recipient site was prepared.

The ulcer was debrided to grossly normal tissue on all margins. The path of the flap was chosen based on the shortest distance to the recipient site. An incision was made through the skin one-third of the way from the recipient site to the base of the pedicle. A subcutaneous tunnel was made under the remaining skin to the pedicle and the recipient site. The flap and pedicle were passed through this tunnel [Figure 3], the island was laid into the recipient site, and the pedicle was laid into the channel created by the skin incision.

Excess tension on the flap was alleviated by additional undermining of the trifurcation of the lateral branch of the superficial radial nerve, the cranial superficial antebrachial artery, the accessory cephalic vein, and the dorsal common digital nerves, arteries, and veins (II, III). The flap was secured in place only with simple interrupted skin sutures (4-0 nylon). The channel for the pedicle was closed using only simple interrupted skin sutures (4-0 nylon). The defect from the donor site was closed with simple interrupted subcutaneous sutures (3-0 polydioxanone), and the skin was apposed with simple interrupted skin sutures (4-0 nylon). A soft padded bandage and fiberglass clamshell splint were applied in such a manner that the dog could walk on the end of the splint, reducing weightbearing on the metacarpal pad.

Hydromorphone (0.05 mg/kg intravenously 4 hours) was administered for analgesia for 18 hours, followed by tramadol (2.5 mg/kg per os 12 hours) for 3 days. The dog was managed in the hospital for 23 days at the owner’s request to enforce strict exercise restrictions. The dog was bearing weight on the splinted left thoracic limb during short leash walks. The bandage was changed every 24 to 48 hours. At each evaluation, the flap appeared healthy and viable; no swelling, discoloration, or discharge was noted. Five days after surgery, a 3- to 4-mm area on the proximal-medial surface of the flap appeared to be separating from the recipient site [Figure 4], but it healed without intervention. Ten days postoperatively, the flap appeared to be healing well with no evidence of necrosis or additional incisional dehiscence; all skin sutures were removed. Twelve days postoperatively, the bandage was changed from a clamshell splint to a soft padded bandage. The bandage was changed every 24 to 36 hours until it was removed permanently 23 days after surgery.

Mild pododermatitis was noted during bandage changes. This was managed successfully with cleansing, using a 2% chlorhexidine solution diluted 1:10 with tap water and talcum powder-coated cotton balls placed between the toes. On day 12 after surgery, a 1 × 1-cm portion of the superficial layer of the heavily keratinized epithelium of the metacarpal pad had sloughed. Also, a 0.5 × 0.5-cm portion of the superficial layer of the heavily keratinized epithelium of the first digital pad had sloughed. These areas redeveloped their tough, heavily keratinized layer sometime between bandage removal 23 days after surgery and recheck.

Figure 3—Flap and its neurovascular pedicle (being held by the surgeon), elevated from the donor site at the base of the fourth digit and passed through the subcutaneous tunnel created under the skin of the dorsolateral manus (highlighted by the thumb forceps passing through the tunnel). Esmarch bandage is in place, minimizing hemorrhage.
examined 55 days after surgery. The superficial sloughing was attributed to disuse of the pads. The dog was discharged 24 days postoperatively, with owner instructions to leave the dog’s paw unbandaged and to use a protective foot cover when walking the dog on rough surfaces.

The dog was reevaluated 55 days after surgery, and the owner reported the dog was using the leg better than prior to surgery. The dog did not lick the donor site excessively, and the flap site appeared normal. On examination, the donor site had healed completely. The surface of the flap appeared to have developed a heavily keratinized layer grossly similar to the normal, heavily keratinized metacarpal pad in the region [Figure 5]. For practical reasons, we were unable to confirm this change histopathologically. Sensation of the flap was not assessed. Mild hyperextension of the left carpus persisted. The dog was discharged with owner instructions to allow the dog to walk with the paw exposed and to monitor the metacarpal pad for signs of ulceration. Telephone contact was made 95 days postoperatively, and the owner reported the dog was doing well with no signs of ulceration of the metacarpal pad.

Discussion

A modification of a previously described technique creating a neurovascular island flap was used to treat a suspected trophic ulcer of the metacarpal pad in this dog. The modifications included passing the island flap and its pedicle through a subcutaneous tunnel and using a clamshell splint to protect the healing flap.

The pathogenesis of trophic ulceration secondary to denervation of the overlying skin is not completely understood. It appears, at least in part, that loss of cutaneous sensation inhibits normal somatosensory reflexes and voluntary changes in posture, allowing for prolonged ischemia and subsequent ulceration of the desensitized tissues. In the veterinary literature, reports of trophic ulcers have been limited to the metacarpal, metatarsal, and digital pads. Medical management of trophic ulcers, both in human and veterinary patients, requires intensive, long-term nursing care and is frequently unrewarding. Reported surgical therapies attempt to provide healthy donor tissue with normal sensory innervation and blood supply. These neurovascular island flaps are based on known neurovascular bundles, but success has been variable in humans and dogs.

Digital pad transposition has been used successfully to treat defects of the metacarpal and metatarsal pads, but it requires the effective amputation of at least one digit. The use of free segmental grafts from the digital paw pads has been shown to be successful in experimentally induced metatarsal pad defects. However, the application of such grafts to trophic ulceration of the metacarpal/tarsal pad may be difficult due to the apparent difficulties in trying to achieve a healthy bed of granulation tissue at the recipient site. Treatment of trophic ulceration by using the fourth dorsal common digital neurovascular bundle as a pedicle for an island flap has been described. Reported complications include subsequent self-mutilation of the fifth digit, necrosis of the flap, and superficial abrasions of the flap from heavy use. The outcomes in five cases varied from excellent to poor. In the two successful cases of island flaps reported by Gourley et al., the dogs were reported to develop abrasions with excessive activity, indicating that the flap had not become heavily keratinized.

The dog of this report had a successful outcome in the short term, with few complications experienced. Sloughing
occurred in the superficial layers of the heavily keratinized epithelium of the metacarpal pad and the first digital pad. These areas redeveloped their heavily keratinized layer without complication. This was considered to be a minor complication secondary to bandaging and not bearing weight on the pads. The mild pododermatitis observed during bandage changes also resolved with conservative management and was considered to be a minor complication.

Wounds of the paw pads can be protected using different types of external coaptation. Soft padded bandages or splints have been used extensively. Recent research indicates a clamshell-type splint significantly reduces the pressure applied to the metacarpal pad during ambulation, and, for this reason, a clamshell splint was used for this dog to protect the flap.\textsuperscript{11} Clamshell splints were not used in the previous case series reported, and only one island flap failed;\textsuperscript{4} however, the authors feel the clamshell splint protected the flap during healing. The development of what grossly appeared to be a heavily keratinized surface on the island flap, similar to the surface of the normal pad, is noteworthy.

Conclusion
The surgical procedure described requires careful dissection of delicate neurovascular structures, but it is relatively straightforward. This technique should also be adaptable for treating cutaneous defects involving the hind paw. Although an uncommon sequela of peripheral neuropathy, neurovascular island flaps appear to be a useful option for the treatment of chronic trophic ulcers of the metacarpal pad when they are unresponsive to conservative therapy. Owners should be made aware of the risks of flap failure, the potential for future ulcers to result from excessive exercise, and the prolonged and involved care needed during healing.

Footnotes
\textsuperscript{a} Vetrap; 3M Animal Care Products, St. Paul, MN 55144-1000
\textsuperscript{b} Chlorhexidine gluconate solution; Phoenix Pharmaceutical, St. Joseph, MO 64507

References
Successful Surgical Treatment of a Suspected Iatrogenic Arytenoid Cartilage Fracture in a Dog

A 3-year-old, intact female golden retriever was presented with a sudden onset of inspiratory obstructive dyspnea following general anesthesia to perform a mastectomy. The cuneiform process of the left arytenoid cartilage was found to be extremely mobile on laryngeal examination. Fracture of the cuneiform process of the left arytenoid cartilage was diagnosed. A combined cricoarytenoid and thyroarytenoid caudolateralization procedure was performed on the left side, and no further dyspnea was observed during a follow-up period of 7 months. Fracture of the cuneiform process of the arytenoid cartilage has not been previously reported in dogs. The condition may respond favorably to cricoarytenoid and thyroarytenoid caudolateralization surgery. J Am Anim Hosp Assoc 2009;45:181-184.

Case Report

A 3-year-old, intact female golden retriever was presented for evaluation of dyspnea. The animal had undergone a left caudal mastectomy 4 days previously for treatment of a necrotic mammary gland that had developed after whelping. The dog was reported to have had a difficult and aggressive recovery following endotracheal extubation after the mastectomy; later in the same day of the procedure, the dog began exhibiting episodes of inspiratory stridor and dyspnea. These episodes worsened in severity over the following day. The dog displayed cyanosis, stridor, and hyperpnea during these periods, but a normal respiratory rate and pattern were noted between these episodes.

The surgeon who had performed the mastectomy performed survey radiography of the neck and chest (right lateral views). The radiographic findings were judged as normal. On the basis of clinical examination findings and radiographic findings, this surgeon made a presumptive diagnosis of laryngeal paralysis. Theophylline, diuretic, and diazepam were administered; however, the dyspneic episodes continued with the same severity.

The dog was referred to another veterinarian 48 hours postoperatively. Survey orthogonal cervical and thoracic radiographs were obtained, and laryngoscopic and bronchoscopic examinations were performed. A tentative diagnosis of left-sided laryngeal paralysis was made, based on the clinical signs of inspiratory stridor and dyspnea and a laryngoscopic finding of a slight asymmetry between the two cuneiform processes. It was noted, however, that a unilateral laryngeal paralysis would be unlikely to account for the dyspneic episodes. Enrofloxacin and meloxicam were added to the dog’s treatments. The dog was discharged with the hope that spontaneous improvement might occur.

The dyspneic episodes continued, and the dog was referred to the University of Bristol Small Animal Hospital. On clinical examination, no stridor or dyspnea was noted. The dog was eupneic with a respiratory rate of 30 breaths per minute. Thoracic auscultation was unremarkable. The
dog was normothermic (rectal temperature 38.2°C). The authors witnessed the dog experiencing several dyspneic episodes following admission. These episodes occurred spontaneously, lasted for a few minutes, and stopped abruptly. They were not preceded by activity or excitement and were not associated with hyperthermia.

Laryngeal examination, under a light plane of general anesthesia, revealed both arytenoid cartilages to abduct briskly during inspiration. No mucosal lesions or vocal cord edema were evident. A subtle asymmetry was noted between left and right arytenoid cartilages, and a slight overlap of the cuneiform processes was seen to occur on adduction. The arytenoid cartilages were probed with a plastic endotracheal tube obturator. The right arytenoid cartilage was considered normal. The cuneiform process of the left arytenoid cartilage was markedly mobile and, with gentle pressure, could readily be deviated medially approximately 130˚ from its resting position to point caudally. Both corniculate processes were probed and found to be unremarkable. Thoracic radiographs were suggestive of a mild bilateral aspiration pneumonia. Cervical radiographs were within normal limits. A fracture of the cuneiform process of the left arytenoid cartilage was suspected.

A combined cricoarytenoid and thyroarytenoid caudolateralization procedure was performed on the left side in a manner similar to that described previously. The dog was positioned in right lateral recumbency, and a left lateral approach to the extrinsic muscles of the larynx was used. The left thyropharyngeus muscle was incised along the left dorsal border of the thyroid cartilage. Care was taken to preserve the left cranial laryngeal nerve. The thyroid cartilage was disarticulated from the cricoid cartilage at its caudal cornu attachment.

Following the lateral retraction of the left thyroid lamina, examination of the intrinsic muscles of the left larynx revealed a subjectively normal-sized left cricoarytenoideus dorsalis (CAD) muscle with no evidence of muscle fiber atrophy. The insertion of the muscle onto the muscular process of the left arytenoid cartilage was transected. The left arytenoid cartilage was completely disarticulated from its articular facet on the rostral border of the cricoid lamina, and the interarytenoid band was transected. Manipulation of the left arytenoid cartilage confirmed its complete release from the cricoid and contralateral arytenoid cartilages, allowing the structure to be freely moveable in a caudal direction. Lateralization and caudal fixation of the arytenoid cartilage were accomplished by the use of two sutures. The first was a 2-0 polypropylene mattress suture placed through the left caudal cornu of the thyroid cartilage, then through the articular facet and muscular process of the left arytenoid cartilage. The second was a simple interrupted suture of 2-0 polypropylene placed through the caudolateral border of the cricoid cartilage, then through the articular facet and muscular process of the arytenoid cartilage. The second suture is sometimes referred to as a prosthetic CAD muscle suture, because when the suture is tied, its line of tension is in the same direction as the muscle fibers of the left CAD muscle.

The procedure was performed without complication, and the dog did not have any more episodes of dyspnea after recovery from anesthesia. Amoxicillin clavulanate was administered intravenously at 20 mg/kg three times daily to treat the suspected aspiration pneumonia. Nebulization and coupage were also performed three times daily during this period. The dog’s moist cough resolved after 5 days of this protocol, and treatment was discontinued. The owners were contacted by telephone 3 and 7 months after surgery, and they reported the dog had no episodes of dyspnea and normal exercise tolerance.

Discussion

This is the first report, to the authors’ knowledge, of a suspected arytenoid fracture in a dog. In dogs, respiratory obstruction resulting from laryngeal dysfunction can occur secondary to conditions including laryngeal paralysis, laryngeal collapse, and laryngeal neoplasia. Iatrogenic glottic obstruction is usually caused by laryngeal edema following endotracheal intubation. The authors hypothesize that the left arytenoid cartilage fractured as a result of traumatic endotracheal intubation for general anesthesia for the mastectomy. The dog had no history of dyspnea prior to this procedure. The most likely site of fracture is the narrow isthmus of cartilage that connects the cuneiform process to the rest of the arytenoid cartilage [Figure 1]. This is a site of weakness of the arytenoid cartilage, and medial deviation of the cuneiform process is a component of laryngeal collapse syndrome.

Interestingly, the cuneiform process is derived from elastic cartilage while the remainder of the arytenoid cartilage is composed of hyaline cartilage; the two cartilages are embryologically distinct. Instability at this site would permit medial folding of the cuneiform process during inspiration, when air pressure within the upper respiratory tract lumen is at its lowest [Figure 2]. The resultant partial glottic occlusion and consequent increased respiratory effort would act to increase airflow speed at the rima glottidis, further reducing air pressure at this location and exacerbating any medial displacement of the cuneiform process. Not known was whether significant medial excursion of the cuneiform process would still occur in its more caudal location, following arytenoid caudolateralization and stabilization.

Careful attention was given to ensure substantial caudal translocation of the left arytenoid cartilage following tying of the arytenoid caudolateralization sutures. This was achieved by completely disarticulating the left arytenoid cartilage from the cricoid cartilage and by transecting the interarytenoid sesamoid band to gain maximum mobilization of the arytenoid cartilage. The thyroarytenoid suture was placed using the most caudal portion of the cornu of the thyroid wing. Although an arytenoid caudolateralization procedure may be performed without disarticulating the cricoid and thyroid cartilages, achieving better surgical exposure and allowing a more thorough inspection of the region were considered necessary in this case. A goal of the surgery was also to produce enough caudal translocation of the arytenoid cartilage to buttress it between the thyroid wing and the cricoid cartilage.
Two factors likely contributed to the success of the procedure. First, the lateralization of the left arytenoid cartilage widened the rima glottidis; second, the arytenoid was buttressed between the cricoid and thyroid cartilages as the arytenoid cartilage was drawn caudally. The excessively mobile cuneiform process was, thus, wedged between the cricoid and thyroid cartilages, preventing the medial folding that had occurred during inspiration. Two sutures were used to provide two-point fixation; this minimized the risk of cranial displacement of the arytenoid cartilage if one suture were to break or be pulled out.

Laryngeal fractures occur in humans most commonly during car accidents because of poorly positioned seatbelts or from blunt trauma from airbags. Fractures also occur during strangulation or attempted hanging. Laryngeal fractures have not been reported in humans following endotracheal intubation for anesthesia. This may be because the pyramidal shape of the human arytenoid cartilage does not have the same isthmus as the dog; the isthmus in the dog can act as a potential “point of weakness.”

A presumptive diagnosis was made in this dog based on the history and laryngeal examination. While laryngeal paralysis was tentatively diagnosed by another veterinarian, the arytenoids abducted appropriately during inspiration when examined under light anesthesia; this finding ruled out a diagnosis of laryngeal paralysis. The cricoarytenoid articulation was not subluxated or luxated as assessed during the surgical procedure, and no atrophy of the CAD muscle was apparent. Fracture of the left arytenoid cartilage between the cuneiform process and the remainder of the arytenoid appeared to be the most plausible explanation for the dog’s clinical signs of intermittent obstructive inspiratory dyspnea and the profound mobility of the left cuneiform process. The cartilaginous isthmus of the left arytenoid cartilage was not visualized during the surgical procedure, because additional dissection to prove the presence of a fracture was considered unnecessary and potentially detrimental to the outcome. The caudal positioning and fixation of the damaged arytenoid cartilage depended on the presence of intact pericartilaginous structures to ensure that moving the body of the cartilage would result in similar movement of the cuneiform process.

A definitive diagnosis of arytenoid fracture may have been possible using advanced imaging, such as magnetic
resonance imaging. High-resolution spiral computed tomography is the method of choice for imaging traumatized larynges in humans. Standard computed tomography incorporating the window technique can be used, although some laryngeal fractures can be overlooked with this approach.

The authors are optimistic regarding the long-term prognosis for the dog reported, particularly because the arytenoid cartilage contralateral to the one operated on has normal function. This is in contrast to most dogs undergoing laryngoplasty for idiopathic laryngeal paralysis.

**Conclusion**

This report suggests that combined cricoarytenoid and thyroarytenoid caudolateralization may be a useful procedure for treatment of cuneiform process instability resulting from arytenoid cartilage fracture in dogs.

**Footnotes**

a Corvental-D; Novartis Animal Health UK Ltd, Camberley, Surrey, GU16 7SR United Kingdom
b Dimazon; Intervet UK Ltd, Milton Keynes, Buckinghamshire, MK7 7AJ United Kingdom
c Diazepam; Alpharma Animal Health, Antwerp, Antwerp, 2610 United Kingdom
d Baytril; Bayer, Newbury, Berkshire, RG14 1JA United Kingdom
e Metacam; Boehringer Ingelheim Ltd, Bracknell, Berkshire, RG12 8YS United Kingdom
f Prolene; Ethicon Ltd, Edinburgh, Midlothian, EH11 4HE United Kingdom
g Augmentin; GlaxoSmithKline PLC, Brentford, Middlesex, TW8 9GS United Kingdom

**References**

Nonsurgical Treatment of Gastroesophageal Intussusception in a Puppy

A 7-week-old, female Siberian husky was presented to Murdoch University Veterinary Hospital with an acute onset of respiratory distress and regurgitation. Thoracic imaging identified an intraluminal esophageal mass with concurrent aspiration pneumonia. Esophagoscopy identified the mass as stomach, and a diagnosis of gastroesophageal intussusception was made. The intussusception was reduced endoscopically, and fixation of the stomach to the abdominal wall was performed using a tube gastropexy. Gastroesophageal intussusception is an uncommon disease in small animals and traditionally has been managed surgically. This case report describes an alternative method of treatment associated with a good outcome in this puppy. J Am Anim Hosp Assoc 2009;45:185-190.

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Introduction

Gastroesophageal intussusception (GEI) is a rare disorder in which part or all of the stomach invaginates into the thoracic esophagus. In severe cases, additional organs such as the spleen, pancreas, duodenum, and omentum may also involute. Some authors have categorized GEI as a type of hiatal hernia; however, GEI can be differentiated from a sliding hiatal hernia by the position of the gastroesophageal junction and a paraesophageal hiatal hernia by the position of the stomach within, rather than adjacent to, the esophagus. Two types of GEI are reported in the literature: a chronic, recurrent form that causes intermittent gastrointestinal signs and an acute, persistent type that results in signs of acute esophageal obstruction and respiratory distress.

The pathogenesis of GEI is not well understood and is likely multifactorial. Anatomical abnormalities such as esophageal hiatal enlargement, lower esophageal sphincter incompetency, and esophageal motility disorders (such as megaesophagus) are likely to be involved. In humans, risk factors for the development of GEI include conditions that cause an increase in abdominal pressure, such as pregnancy, obesity, overeating followed by rigorous exercise, and chronic dyspepsia. In dogs, increased intraabdominal pressure from disorders such as chronic vomiting or blunt trauma, and negative intrathoracic pressure from inspiratory dyspnea due to chronic upper airway obstruction or severe respiratory disease may also be factors. The latter hypothesis is supported by a study of six of 11 Dalmatian puppies that died from acute respiratory distress syndrome (ARDS) and had developed GEI in the terminal stages of disease. The development of GEI in a dog with laryngeal paralysis and the development of a sliding hiatal hernia in a cat with a laryngeal mass also support this association.

The clinical signs of GEI are usually related to esophageal and gastric outflow obstruction and include vomiting, regurgitation, hypersalivation, dysphagia, abdominal discomfort, and occasionally hematemesis. Concurrent respiratory distress due to the resultant space-occupying intrathoracic mass is often present and may be complicated by secondary
aspiration pneumonia. In acute cases, cardiovascular compromise can result from compression of the great thoracic vessels causing reduced venous return, or from endotoxic shock due to gastric ischemia, necrosis, and release of inflammatory mediators. Such cases deteriorate rapidly and can be considered gastrointestinal emergencies.

Diagnosis of GEI is based on a combination of survey and contrast radiography, fluoroscopy, and esophagoscopy. Survey radiographs may suggest GEI if a large soft-tissue mass is visible in the caudodorsal mediastinum, with displacement of the trachea and cardiac silhouette and loss of the gastric shadow. Contrast radiography often will show an intraluminal filling defect and may outline gastric rugal folds. Fluoroscopy identifies esophageal dysmotility, while esophagoscopy may confirm the diagnosis by showing a mass in the distal esophagus—the appearance of which is consistent with gastric mucosa.

Historically, the treatment of choice for a GEI has involved surgical replacement and fixation of the stomach to the abdominal wall via laparotomy. In acute cases that are presented as emergencies, immediate stabilization of the animal is required, including oxygen support for dyspnea, correction of electrolyte abnormalities, and appropriate management of dehydration and/or shock. Management of secondary complications, such as aspiration pneumonia and esophageal disease, is also required in the perioperative period. Prognosis in early reports was poor, with a mortality rate of 95% in one study. Survival rates have improved in recent years; however, the long-term prognosis often remains guarded because of concomitant esophageal disease.

This case report describes the successful management of an acute onset of esophageal obstruction and respiratory distress secondary to GEI in a puppy, using endoscopic reduction and fixation of the stomach to the abdominal wall with a tube gastropexy.

**Case Report**

A 7-week-old, 2.5-kg, female Siberian husky was presented to Murdoch University Veterinary Hospital with a 12-hour history of vomiting, regurgitation, and respiratory distress. The owner reported that the puppy had intermittent regurgitation from birth that had lessened in frequency after she began eating solid food the week prior to presentation. Physical examination revealed a body condition score of 3/9, heart rate of 220 beats per minute, and a respiratory rate of 60 breaths per minute with increased inspiratory effort. Increased bronchoesuvacular lung sounds were auscultated bilaterally, while pyrexia (body temperature 39.3°C), reduced oxygen saturation (SpO2 79%), and a mean arterial blood pressure of 74 mm Hg were also identified.

Survey radiography performed by the referring veterinarian showed a homogeneous soft-tissue mass in the dorsal thorax extending between the diaphragm and the third rib pair. The cranial margin of the mass was smoothly rounded [Figure 1A]. The mass caused marked ventral displacement of the trachea. Contrast radiography demonstrated distension of the cervical and cranial thoracic esophagus and confirmed obstruction of the caudal esophagus by the mass [Figure 1B]. Based on these findings, a diagnosis of esophageal obstruction was made. Possible causes of the obstruction included GEI and esophageal foreign body.

The puppy was initially treated with oxygen support and intravenous fluid resuscitation (5 mL/kg Dextran 70® and 20 mL/kg lactated Ringer’s solution) for signs of compensated cardiovascular shock. Once the animal was hemodynamically stable, crystalloid solution was continued intravenously (IV) at a maintenance rate (3.6 mL/kg per hour of lactated Ringer’s solution, supplemented with 2.5% glucose and 20 mmol/L potassium chloride).

The puppy was maintained in an oxygen-rich environment (fraction of inspired oxygen between 40% and 60%). Additional medications included metoclopramide (0.4 mg/kg IV q 8 hours), ranitidine (2 mg/kg IV q 12 hours), and...
terbutaline (12 µg/kg subcutaneously q 8 hours), and amoxicillin (24 mg/kg IV q 8 hours).

Thoracic radiography was repeated while the puppy was under sedation with butorphanol (0.03 mg/kg) and acetyl-promazine (0.004 mg/kg). Ventral displacement of the trachea persisted; however, the entire thoracic esophagus was now filled with a heterogeneous soft-tissue opacity. In the ventral lung fields, patchy consolidation and interstitial infiltrates suggested aspiration pneumonia [Figures 2A, 2B]. Fluoroscopy and contrast radiography were performed after the effects of sedation had resolved and confirmed esophageal dilatation, a caudal esophageal mass, and failure of the contrast medium to enter the gastric lumen.

The following day, the puppy underwent general anesthesia (butorphanol 0.01 mg/kg IV, propofol 5 mg/kg IV, inhalational isoflurane). Esophagoscopy revealed a dilated, fluid-filled proximal esophagus. A large, intraluminal mass consistent with the mucosal surface of the stomach was observed in the midthoracic esophagus [Figure 3]. Gentle advancement of the endoscope resulted in replacement of this structure into the abdominal cavity. The region of the esophagus where the invaginated stomach had been located was dilated and inflamed, but no evidence of ulceration or adhesions was seen. A percutaneous endoscopic gastrostomy (PEG) tube was placed through the left abdominal wall to attach the stomach to the abdominal wall and to enable enteral nutritional management. After recovery, the puppy’s respiratory rate had improved (44 breaths per minute with reduced inspiratory effort and an SpO2 of 84%), and it continued to improve over the following 24-hour period. Sucralfate (0.1 g/kg per os [PO]) was administered 12 hours after PEG tube placement, while food and water were withheld for an additional 12 hours. At 24 hours following the procedure, PEG tube feeding was not required, as the puppy ate willingly. Oral feeding from an elevated position was continued during hospitalization, and no further regurgitation occurred. Thoracic radiography was repeated the following day with butorphanol (0.4 mg/kg IV) sedation; radiographs showed a small amount of gas in the cranial thoracic esophagus, resolution of the GEI, and resolving aspiration pneumonia [Figures 4A, 4B].

The puppy was discharged 6 days after admission, with medications including ranitidine (2 mg/kg PO q 12 hours), sucralfate (0.1 g/kg PO q 8 hours), and amoxicillin-clavulanic acid (10 mg/kg PO q 12 hours). The owners were instructed to feed the puppy from an elevated position, and care for the PEG tube site was outlined. Follow-up examination was performed 4 days after discharge, at which time necrosis of the area surrounding the PEG tube was noted. The owner reported intermittent regurgitation of fluid. An esophagram was repeated the following day. Gas and fluid were present within the esophagus, consistent with reduced esophageal motility, but the stomach was located in a normal position in the cranial abdomen. Reassessment 9 days after discharge showed the PEG site to be healing well, but regurgitation remained intermittent, and weight gain was minimal despite a good appetite. The puppy’s daily caloric requirement was recalculated, and a feeding regimen using a highly digestible diet was introduced over a 3-day period. The puppy was returned for PEG tube removal 24 days after discharge, at which time the owner reported that the regurgitation had resolved completely. Abdominal ultrasound confirmed that the fundus of the stomach was positioned adjacent to the left abdominal wall. The PEG tube was removed without complication while the puppy was under brief inhalation anesthesia with sevoflurane.

Follow-up evaluation was performed 48 days after discharge. The animal’s body weight had appropriately
increased to 5 kg. The gastrostomy site had healed well, and the owner reported no further vomiting or regurgitation. Follow-up via telephone 4 months after initial presentation revealed that the dog continued to remain asymptomatic.

**Discussion**

Canine GEI is a rare condition with only 30 cases reported in the veterinary literature. These reports predominantly involve large-breed dogs <3 months of age, with a higher incidence in males and German shepherd dogs. The case reported here has a similar signalment but differs in that it is a female. Interestingly, a previous case has been reported involving the husky breed, also of the female gender. The key to successful treatment of GEI involves the initial identification and treatment of concurrent diseases such as aspiration pneumonia.

**Figures 2A, 2B**—Left lateral (A) and ventrodorsal (B) projections of the thorax. The thoracic esophagus is markedly distended by a heterogeneous, soft-tissue opacity (M) causing ventral displacement of the trachea (arrow). The well-defined margin of the mass seen in the previous study is no longer visible, perhaps due to the accumulation of fluid in the cranial esophagus. In the periphery of the right cranial and caudal lung lobes, an alveolar lung pattern is present. In the left lung, a dense interstitial pattern is present. These changes are consistent with aspiration pneumonia.

**Figure 3**—Endoscopic view of the thoracic esophagus. A soft-tissue mass (M) is seen within the lumen of the esophagus (arrows).
as pneumonia, followed by reduction of the intussusception itself. Historically, surgical exploration has been advocated as the method of choice to achieve this. To date, all reported canine cases of GEI in which treatment has been attempted have been managed in this way.\textsuperscript{3,8,13-16} Following surgical reduction, attachment of the stomach to the abdominal wall with either unilateral or bilateral, incisional, circumcostal, or belt loop gastropexies has been performed. Correction of concurrent anatomical abnormalities, such as an enlarged esophageal hiatus, has also been performed during the surgical procedure. Endoscopic reduction of GEI has been previously reported once in a dog.\textsuperscript{2} In that case, reduction was achieved by insufflating the esophagus while closing off the proximal esophagus. No report was provided as to whether this dog went on to have either surgical or tube gastropexy performed. Endoscopic reduction of GEI was also achieved in a cat\textsuperscript{17} that subsequently had a surgical gastropexy.

To the authors’ knowledge, this is the first case of GEI that has been corrected solely by endoscopic maneuvers. Benefits of this method include avoidance of prolonged general anesthesia in an already compromised animal and providing a method of administering enteral nutrition. A possible disadvantage of this technique is the limitation of having to do a left-sided, unilateral gastropexy; bilateral gastropexies have been advocated by some authors.\textsuperscript{3,13} Others, however, have had success with left unilateral gastropexies.\textsuperscript{8} One report\textsuperscript{16} suggests they may have an anatomical advantage over right-sided gastropexies because of the position of the esophageal hiatus to the left of midline. Contraindications to performing endoscopic reduction would include situations where the gastric wall is compromised or adhesions are present, creating a risk of gastric perforation. Assessment and correction of esophageal hiatal abnormalities are also not possible with this method of correction.

Although the pathogenesis of GEI in small animals is unclear, the veterinary literature has shown a strong association with preexisting esophageal disease. In one report,\textsuperscript{4} >50% of the 23 dogs with GEI had concurrent megaesophagus or other esophageal abnormalities, and this has been a finding in all feline cases reported.\textsuperscript{17-19} This has been suggested as the reason German shepherd dogs are over-represented,\textsuperscript{3} as they also have a predisposition for the congenital form of this disease.\textsuperscript{20,21} In the case presented here, regurgitation had been documented since birth, suggesting the presence of congenital esophageal disease that may have contributed to the development of GEI.

An alternative explanation for the persistent regurgitation is that this puppy initially had the chronic intermittent form of GEI, which then developed into the acute, persistent form. In contrast to feline cases of GEI, which are more often the chronic intermittent type,\textsuperscript{17-19} the acute form is much more common in the dog.\textsuperscript{3,6-8,14,16} However, two canine cases of the chronic intermittent form have been described. One case affected a 2-year-old pug that had previously been diagnosed and treated for a sliding hiatal hernia.\textsuperscript{15} The second case involved an 8-week-old female Siberian husky,\textsuperscript{13} similar to the case reported here.

Unfortunately, it is impossible to confirm which theory might be correct in the case of this report, because no imaging of the puppy had been performed prior to the presentation with acute disease. Clinically, the animal had complete resolution of the regurgitation, suggesting either spontaneous resolution of congenital esophageal disease or chronic intermittent GEI.
Conclusion

Gastroesophageal intussusception is an unusual condition predominantly affecting young, large-breed dogs. It should be considered as a differential diagnosis when an animal exhibits signs of acute or chronic esophageal obstruction, especially with concurrent respiratory distress. Endoscopic intervention may provide an alternative to surgical correction of GEI.

Footnotes
a Surgivet 9200 series; Smiths Medical PM, Inc., Waukesha, WI 53186
b Dextran 70; Baxter Healthcare, Old Toongabbie, New South Wales 2146 Australia
c Compound sodium lactate Hartmann’s solution; Baxter Healthcare, Old Toongabbie, New South Wales 2146 Australia
d Metomide; Delvet, Seven Hills, New South Wales 2147 Australia
e Zantac; Glaxo Smith Kline, Boronia, Victoria 3113 Australia
f Bricanyl; Astra Zeneca, North Ryde, New South Wales 2113 Australia
g Amoxicillin; CSL Ltd., Parkeville, Victoria 3052 Australia
h Torbugesic; Fort Dodge, Baulkham Hills, New South Wales 2153 Australia
i A.C.P. 2; Delvet, Seven Hills, New South Wales 2147 Australia
j Fresofol; Pharmatel Fresenius Kabi Hornsby, New South Wales 2077 Australia
k L.S.O.; VCA, Kings Park, New South Wales 2148 Australia
l Carafate; Aspen Pharmcare, St. Leonards, New South Wales 2065 Australia
m Clavulox; Pfizer, West Ryde, New South Wales 2114 Australia
n Science Diet puppy; Hills, North Ryde, New South Wales 2113 Australia
o SevoFlo; Advanced Anesthesia Specialists, Gladesville, New South Wales 2111 Australia

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References
Dacryops (Lacrimal Cyst) in Three Young Labrador Retrievers

This case series constitutes a report of dacryops in multiple Labrador retrievers and the use of smooth-muscle actin immunostaining to confirm the lacrimal duct origins of the cyst wall. Three Labrador retrievers were presented with a history of a slowly enlarging mass adjacent to the left medial canthus. Ultrasonography of the masses revealed they were each spherical, thin-walled cystic structures. Aspiration cytology was performed in two cases revealing mixed inflammation and absence of detectable microorganisms. Dacryocystorhinography of the left nasolacrimal system performed in two cases revealed a normal nasolacrimal system that was closely associated, but not communicating with, the cystic mass in both cases. Surgical excision of all cysts was curative. Histopathology and positive immunohistochemical staining for smooth-muscle actin confirmed a diagnosis of dacryops in all cases. J Am Anim Hosp Assoc 2009;45:191-196.

Introduction

Dacryops is a cyst or ectasia originating from lacrimal glandular ductal tissue (both intraglandular and extraglandular). Dacryops are rare in domestic animals and are infrequently reported in the veterinary literature.1-6 The exact pathogenesis for the development of dacryops is uncertain, and no specific etiology has been elucidated in reported canine cases.1,2,5,6 In the dog, the main lacrimal gland lies under the periorbita in the dorsolateral aspect of the orbit, and the accessory lacrimal gland is located in the third eyelid. Ectopic or choristomatous lacrimal gland tissue has been reported in dogs.6 Cyst formation may occur in any location where lacrimal gland tissue is present.1,3,4,6 Management of dacryops is primarily surgical, with complete excision being curative.7-9 This case series outlines three cases of dacryops in young Labrador retrievers; the dacryops originated from ectopic periorcular lacrimal gland ductal tissue, and cases were treated successfully by surgical excision. The young age of the dogs, cyst location, and lack of historical trauma suggest a congenital etiology.

Case Reports

Case No. 1

A 1-year-old, castrated male Labrador retriever was referred for evaluation of chronic swelling adjacent to the medial canthus of the left eye (OS) and epiphora OS. The swelling had been enlarging slowly for 6 months prior to presentation. Surgical excision of the mass was attempted by the referring veterinarian 5 days prior to referral, but it was aborted due to the close proximity of the mass to the nasolacrimal apparatus. The dog was not receiving any medications at the time of referral.

Initial ophthalmic examination revealed a well-demarcated, ovoid, raised, turgid, nonulcerated, subcutaneous mass measuring 15 × 10 mm adjacent to the medial canthus and medial inferior eyelid margin OS [Figure 1A]. Neuroophthalmic examination including menace response, direct and consensual papillary light response, palpebral reflex, and vestibular ocular reflex was normal in both eyes (OU). Applanation
Tonometry revealed normal intraocular pressures of 16 and 15 mm Hg in the right eye (OD) and OS, respectively. Values of the Schirmer tear test were 25 and 23 mm per minute OD and OS, respectively. Slit-lamp biomicroscopy and indirect ophthalmoscopy revealed no anterior or posterior segment abnormalities. Jones I test (i.e., fluorescent dye passage) performed by application of fluorescein dye topically to each eye was positive OD and negative OS. Jones II test was performed OS with 5 mL of sterile eye-irrigating solution, and a 24-gauge intravenous (IV) catheter sheath was inserted into the superior canaliculus, revealing positive fluid and fluorescein stain passage through the corresponding ventral punctum and nare. The size of the mass remained unchanged after nasolacrimal flush. No other abnormalities were detected on physical examination.

Results of a complete blood count, serum biochemical analysis, and urinalysis were within reference ranges. Ultrasonography of the medial canthal mass using a 10-MHz probe revealed a spherical, thin-walled cystic structure. A transcutaneous, fine-needle aspirate of the mass was obtained, yielding 0.5 mL of turbid brown fluid. Cytological examination of the aspirate revealed mixed inflammation (primarily nondegenerate neutrophils) with no organisms. Positive contrast dacryocystorhinography of the left nasolacrimal system delineated normal nasolacrimal canaliculus, sac, and duct; no communication with the cystic mass was detected. Contrast media, injected via a 25-gauge needle into the mass, outlined a solitary cystic structure with no communication to adjacent tissues or evidence of foreign body. The cyst was superimposed over the nasolacrimal system on all radiographic views. Further evaluation with computed tomography (CT) scan confirmed a normal nasolacrimal system and an independent, but closely juxtaposed, cystic mass at the medial canthus OS. No bony invasion was apparent [Figure 2].

Surgical removal of the mass was recommended as the most likely means of effecting a cure, to prevent further occlusion of the left nasolacrimal duct, and to provide a histopathological diagnosis. The inferior canaliculus was cannulated with a 3.5 French red-rubber catheter to demarcate proximal nasolacrimal structures during dissection of the cyst. A 1.5-cm curvilinear incision was made parallel to the eyelid margin through the skin, orbicularis oculi, and levator nasolabialis muscles overlying the blue-colored cystic mass. The cyst was carefully dissected free of its soft-tissue attachments using a combination of sharp and blunt dissection, and it was removed in its entirety [Figure 3].

A 1-mm rent in the canaliculus occurred during surgical excision because of the intimate association between the inferior canaliculus and cyst wall. Meticulous apposition of the tissue around the canalicular incision was completed with a simple interrupted suture of 7-0 polygalactin. The subcutaneous tissues were closed in two layers using 4-0 polygalactin in a simple continuous pattern, and the skin was closed with 4-0 braided nylon in a Ford interlocking pattern.
A 10-month-old, spayed female Labrador retriever was referred for evaluation of a swelling of 5 months’ duration, located at the left medial canthus and epiphora OS. Treatment by the referring veterinarian with oral antibiotics and corticosteroids had not been effective. Ophthalmic examination abnormalities were limited to a turgid, round, 15 mm × 20-mm, subcutaneous mass adjacent to the medial canthus and medial inferior eyelid margin OS [Figure 1B]. Intraocular pressure measurements were 16 and 15 mm Hg OD and OS, respectively. Schirmer I tear test values were within normal reference ranges (18.89±2.62 mm per minute),10 and no fluorescein stain uptake was detected in either eye. Jones I test was positive OD and negative OS. Jones II test OS revealed positive fluid and fluorescein stain passage through the corresponding ventral punctum and nare, and the size of the mass remained unchanged after nasolacrimal flush.

Ultrasoundography of the mass revealed a spherical, thin-walled cystic structure. Cytological examination of the aspirated cyst fluid revealed mild suppurative to mixed inflammation with no microorganisms detected. Bacteria were not isolated from the aspirated fluid. Dacryocystorhinography of the left nasolacrimal system delineated normal nasolacrimal structures and demonstrated no communication with the cystic mass, which was located rostral to the canaliculus. A magnetic resonance imaging (MRI) study with existing contrast media still present in the nasolacrimal apparatus was performed with additional injection of contrast into the cystic mass and confirmed isolation of the cyst.

Excision of the mass was performed under general anesthesia, and a 1-mm laceration occurred in the inferior lacrimal canaliculus during mass removal. The laceration was closed, and the indwelling nasolacrimal catheter was left in situ as described for case no. 1. Postoperative care was similar to that described for case no. 1. The nasolacrimal catheter was removed 3 weeks following surgery, and patency of the nasolacrimal system was confirmed by Jones I test. Follow-up information per the owner 1 year after surgery indicated the mass and epiphora had not recurred.

Case No. 3

An 8-month-old, spayed female Labrador retriever was referred for evaluation of a swelling of 6 months’ duration at the left medial canthus. The swelling had been aspirated and drained by the referring veterinarian 1 month prior to presentation, but the mass had refilled completely by the time of examination. Physical examination abnormalities were limited to OS and periorcular structures. Applanation tonometry revealed normal intraocular pressures of 16 and 15 mm Hg OD and OS, respectively. Values of the Schirmer I tear test were 22 and 21 mm per minute OD and OS, respectively. A subcutaneous swelling measuring approximately 7 × 7 mm was found at the medial inferior eyelid, and bilateral retinal dysplasia was present. Positive Jones I and II tests were observed OU. Imaging diagnostics and fluid analysis were recommended to further characterize the lesion, but these were declined by the owner due to financial constraint.

Excision of the mass was completed under general anesthesia after cannulating the left nasolacrimal system with a 3.5 French red-rubber catheter. The deepest portion of the cyst was intimately associated with the inferior nasolacrimal canaliculus, which was incised to ensure complete cyst removal. This rent was repaired with two simple interrupted sutures of 8-0 polygalactin. The subcutaneous tissue and skin were closed routinely. The nasolacrimal catheter was left indwelling and sutured to the skin using a finger trap pattern. Postoperative care was similar to that described for case no. 1. The dog recovered uneventfully, and the indwelling catheter was removed 3 weeks following surgery without complication. No evidence of cyst recurrence has been reported within 1 year of follow-up.
Histopathology of the masses from all three dogs revealed cysts with single to double cell-layered, nonciliated, cuboidal epithelial lining with mild submucosal fibrosis and mixed inflammation composed of lymphocytes, plasma cells, and macrophages [Figures 4A-4C]. In some areas, the epithelium was flattened to a squamous type, and this was attributed to pressure atrophy. Immunohistochemical staining for smooth-muscle actin performed in all cases revealed several layers of attenuated slender cells directly beneath the epithelium, positively expressing this antigen. This finding indicates the presence of myoepithelial cells at the cyst lining, consistent with a glandular duct [Figure 5]. Control samples of a normal canaliculus and lacrimal gland with ductal tissue were subjected to immunohistochemical staining for smooth-muscle actin. The lumen of normal canaliculus was lined by stratified squamous epithelium and showed negative immunoreactivity for smooth-muscle actin [Figure 6A]. The normal lacrimal glandular and ductal tissue was strongly positive for smooth-muscle actin [Figure 6B].

The presence of myoepithelial tissue is a normal finding in lacrimal ductal tissue, but the presence of myoepithelial tissue in the canaliculus has never been evaluated in dogs. Based on this, positive smooth-muscle actin staining in the three cases presented here supported the diagnosis of dacryops (a cyst of lacrimal glandular ductal tissue). The tissue of origin was most likely ductal tissue of ectopic lacrimal tissue since canine lacrimal tissue does not normally exist in the medial canthus.

Discussion

Cysts of the periorbital region are rare in domestic animals. Reported periorbital cysts involving or closely related to the nasolacrimal system include dacryops, cystic dilatation of the canaliculi (canaliculops), and the nasolacrimal duct, periorbital epidermoid cysts, maxillary bone epithelial cysts, and cysts of the frontal and nasal sinuses. In the three Labrador retrievers in this report, the size, anatomical location, and patency of the nasolacrimal system were normal based on positive Jones II tests in all cases and dacryocystorhinography and advanced imaging in two of the cases. No connection between the nasolacrimal system and the cyst was identified in any of the dogs. Histopathology of the cyst was identical to that of normal lacrimal ductal tissue and compatible with previously reported dacryops. Positive immunohistochemical staining for smooth-muscle actin confirmed the presence of

![Figures 4A-4C](Image)

**Figures 4A-4C**—Histological photomicrograph of the dacryops. (A) Case no. 1; (B) case no. 2; (C) case no. 3. Hematoxylin and eosin stain.
myoepithelial cells under the cyst walls, consistent with glandular ductal origin.\textsuperscript{19} To the authors’ knowledge, this finding has not been previously evaluated or reported in the literature. In the cases presented here, cystic lacrimal glandular ductal tissues in the periocular tissue were located ectopically (as choristomas), consistent with other accounts of canine dacryops.\textsuperscript{1,5}

Eight cases of dacryops have been reported in the veterinary literature. Four of the reported canine dacryops cases occurred in young basset hounds, indicating a potential breed predisposition.\textsuperscript{1,2,4-6} This case series represents the first report of Labrador retrievers with dacryops. A previous report of a maxillary bone epithelial cyst in a Labrador retriever has been published. No familial relationship existed between the three dogs. The unilateral presentation and young age of these dogs were consistent with previous reports of canine dacryops.\textsuperscript{2,6}

In this case series, the left periocular area was affected in all dogs; the significance of this finding is unknown. Potential causes for the cystic lesions include congenital malformations, chronic inflammation, traumatic disruption of glandular or ductal tissue, and neoplastic processes.\textsuperscript{1,5,12,20} The exact pathogenesis is unclear. Studies in humans suggest that periductal inflammation or trauma stimulates hypersecretion, destroys neuromuscular contractility of the lacrimal gland duct, weakens the duct walls, and finally results in passive ductal dilatation with cyst formation.\textsuperscript{7} In the dogs reported here, no histological or historical evidence was found that correlated with known inciting causes of dacryops. However, ectopic lacrimal glandular and ductal tissue has only been reported in dogs with dacryops, suggesting that the presence of ectopic lacrimal tissue predisposes the dog to or causes dacryops.\textsuperscript{1,2} The signalment, clinical appearance, and lack of traumatic or inflammatory history were supportive of a developmental defect in this case series.

Various diagnostic imaging modalities were utilized in these dogs to determine the extent of association between the dacryops to the canaliculi and lacrimal sac. Dacryocystorhinography and subsequent intracyst injection of contrast with radiographs, CT scan, or MRI afforded better surgical planning and helped confirm patency of the normal nasolacrimal system and lack of communication between the dacryops and duct prior to surgery.

Dacryops do not typically spontaneously regress, and the natural course of a simple untreated dacryops tends to be chronic with no significant cyst enlargement.\textsuperscript{7} However, some dacryops can become complicated by inflammation, fistulas, or malignant transformation.\textsuperscript{18,21} Enlargement may also lead to occlusion of the nasolacrimal duct and development of dacryocystitis.\textsuperscript{15} In this case series, surgical removal was curative. Laceration of inferior canaliculus occurred in all cases during surgery because of the close association between canaliculi and the cyst wall. Meticulous apposition of the tissue around the canalicular laceration and placement of an indwelling nasolacrimal catheter allowed adequate healing while preserving the patency of the nasolacrimal system. In the year following surgical excision, long-term success of the procedure was confirmed in all cases. Success was evidenced by a patent nasolacrimal system, resolution of epiphora, and excellent eyelid function and cosmesis.

**Conclusion**

In this case series, dacryops were documented and successfully treated in three young Labrador retrievers. This is the first report of dacryops in this breed. Dacryops were located within the inferior, medial eyelid and periocular tissue in all cases. Two of the cases had mild epiphora associated with functional nasolacrimal obstruction. Surgical excision of the cysts necessitated surgical incision with subsequent reconstruction of the inferior lacrimal canaliculus; the procedure was curative in all cases. Laceration of the nasolacrimal system (specifically the inferior canaliculus) is a
potential complication associated with surgical excision of dacryops in this location. Diagnosis of dacryops was confirmed with histopathology and immunohistochemistry for smooth-muscle actin in all cases.

Footnotes

a Tonopen-XL; Mentor O&O Inc., Norwell, MA 02061
b Schirmer Tear Test Strips; Schering-Plough Animal Health Corporation, Union, NJ 07083-1982
c Flor-I-Strip; Wyeth-Ayerst Laboratories, Philadelphia, PA 19101
d Eye Wash solution; Major Pharmaceuticals, Livonia, MI 48150
e Abbocath-T 24-gauge IV Cannula; Abbott Laboratories, Abbott Park, IL 60064-6408
f Hypaque; Nycomed, Princeton, NJ 08540-6231
g Red rubber urethral catheter, 3.5 French; Baxter-Allegiance Healthcare, McGaw Park, IL 60085
h Vicryl Polygalactin 910 suture; Ethicon, Somerville, NJ 08876
i Neomycin and polymyxin B sulfates and gramicidin ophthalmic solution USP; Bausch and Lomb Pharmaceuticals, Rochester, NY 14604-2701

Acknowledgments

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References

Mucinous Gastric Carcinoma With Abdominal Carcinomatosis and Hypergastrinemia in a Dog

A 12-year-old, spayed female Australian cattle dog was evaluated for a 5-month history of progressive vomiting. Abdominal radiographs and ultrasound revealed significant gastric wall thickening and a peripancreatic mass, and serum gastrin concentration was increased (127 pg/mL, reference range 10 to 40 pg/mL). Surgical exploration of the abdomen revealed a thickened, firm, and irregular gastric fundus, pylorus, and antrum; nodules were present throughout the spleen and mesentery adjacent to the left limb of the pancreas. Mucinous gastric carcinoma with carcinomatosis was diagnosed by histopathological examination of surgically excised tissues. Unfortunately, severe postoperative complications resulted in euthanasia 10 days after surgery, and a necropsy was not performed. This case is significant, because it is the first report of a mucinous gastric carcinoma associated with hypergastrinemia in a dog. J Am Anim Hosp Assoc 2009;45:197-202.

Introduction

Gastrinomas in dogs are rare functional tumors of gastrin-secreting cells, usually located in the pancreas; in a single case, a gastrinoma was reported in the duodenum.1-3 Hypergastrinemia results in excess hydrochloric acid production by gastric parietal cells, leading to gastric and duodenal ulceration. Gastrin also has trophic effects on the gastric mucosa, which may result in diffuse gastric wall thickening. Historically the concurrent findings of gastric hyperacidity, gastrointestinal ulceration, diarrhea, and pancreatic islet cell tumor were referred to as Zollinger-Ellison syndrome, and this term is still occasionally used in the literature.2-6

Unlike gastrinomas, gastric carcinomas are primary tumors arising from the epithelium of the stomach, and they are the most common primary gastric neoplasms in dogs.7 Mucinous gastric carcinomas are an uncommon subtype of gastric carcinoma characterized by marked mucin production and the presence of mucinous lakes. These neoplasms arise from the gastric mucosa, particularly the lamina propria, and they frequently invade the underlying submucosa and metastasize to the regional lymph nodes, mesentery, liver, and spleen.8

The veterinary literature includes few case series and case reports of gastrinomas in dogs and cats, and small case series of gastric carcinomas in dogs. The clinical signs from these two tumor types are very similar,
with most dogs evaluated for prolonged inappetence or anorexia and vomiting. Differentiation relies on identification of a gastric mass versus a pancreatic mass, histopathological examination, and measurement of fasting serum gastrin concentrations.\textsuperscript{2,3,7,9,10} The purpose of this paper is to present a case of mucinous gastric carcinoma that mimicked gastrinoma because of increased fasting serum gastrin, diffuse severe gastric wall thickening, and a peripancreatic mass due to carcinomatosis.

**Case Report**

A 12-year-old, spayed female Australian cattle dog was presented to the Purdue University Veterinary Teaching Hospital (PUVTH) for further evaluation of a 5-month history of vomiting. Frequency of vomiting had increased over time, ranging from one to six episodes per day. Vomitus consisted of fluid and/or partially digested food, but vomiting episodes were not associated with feeding or time of day. Despite a normal appetite, the dog had lost 2.3 kg since initial presentation to the referring veterinarian 5 months earlier. Diagnostics performed by the referring veterinarian included complete blood count, serum biochemical panel, abdominal radiographs, and gastroscopy (performed approximately 1 week prior to referral). No significant abnormalities were noted, and no biopsies were obtained during the endoscopic examination. Conservative treatment was attempted, which consisted of an oral antiemetic (i.e., metoclopramide), zinc supplementation, pancreatic enzyme supplementation, a bland diet, and H\textsubscript{2} blocker (i.e., famotidine); however, because owner compliance was lacking, effectiveness was difficult to evaluate. According to the owners, famotidine had not been administered for at least 3 to 4 weeks prior to presentation at the PUVTH. At presentation, the dog was slightly thin and approximately 5% dehydrated, but the remainder of the physical examination was unremarkable.

Complete blood count, serum biochemical panel, and urinalysis revealed lymphopenia and eosinopenia (lymphocytes 730 cells/µL, reference range 1000 to 5000 cells/µL; eosinophils 0 cells/µL, reference range 100 to 1250 cells/µL); increases in alanine transaminase (81 IU/L, reference range 3 to 69 IU/L) and alkaline phosphatase activities (554 IU/L, reference range 20 to 157 IU/L); and decreased blood urea nitrogen concentration (6 mg/dL, reference range 7 to 32 mg/dL). Thoracic radiographs were unremarkable. Abdominal radiographs suggested a thickened stomach wall, and a mixture of soft tissue and fat opacity was seen in the cranial abdomen. This resulted in a moderate loss of serosal margins in the abdomen and a mottled appearance to the peritoneum, particularly on the left side [Figure 1]. Abdominal ultrasonography revealed a small amount of free fluid adjacent to the liver. The stomach wall was thickened with loss of normal layering, measuring approximately 1.4 cm in the fundic region [Figure 2]. In the region of the pancreas, a hypoechoic, mass-like lesion that measured approximately 1 cm in thickness was seen, with adjacent hyperechoic fat. A mesenteric lymph node visualized in the same region was slightly enlarged but of normal shape (0.7 cm wide;
short:long axis ratio 0.37). Both adrenal glands were at the large end of normal size.

Serum pre- and postprandial bile acids, canine pancreatic lipase immunoreactivity, trypsin-like immunoreactivity, and cobalamin concentrations were within reference ranges, but serum fasting folate concentration was mildly decreased (5.3 µg/L, reference range 7.7 to 24.4 µg/L). Fasting serum gastrin concentration was increased (127 pg/mL, reference range 10 to 40 pg/mL). The pH of the vomitus while the dog was fasted was 4 to 5, as measured with both urine dipstick and standard laboratory pH paper. Basal endogenous adrenocorticotropic hormone (ACTH) concentration was mildly decreased (14.2 pg/mL, reference range 20 to 100 pg/mL), and an ACTH stimulation test revealed increased serum postcortisol concentration (precortisol 4.2 µg/dL, reference range 1.0 to 6.0 µg/dL; postcortisol 27.1 µg/dL, reference range 7.0 to 17.0 µg/dL).

The previously noted abdominal fluid was sampled using ultrasound guidance. Fluid analysis revealed a modified transudate (3200 nucleated cells/µL, protein concentration 2.5 g/dL) with carcinomatosis. A differential cell count revealed a predominance of neutrophils (73%) and fewer macrophages (23%), which were often highly vacuolated and displayed cytophagia. The remaining cells noted were neoplastic, occurring both individually and in clusters. These cells had a dark blue cytoplasm with occasional blebbing around the cell margins. Many cells had punctate, clear vacuoles with an eccentrically located oval nucleus. An equal number of cells displayed signet-ring morphology with a rim of dark cytoplasm around the periphery of the cell and a lighter blue central area that appeared mottled with indistinct vacuolization; these neoplastic cells were most consistent with either carcinoma or adenocarcinoma. No bacterial growth was identified in the peritoneal fluid or a previously submitted urine culture.

Gastro-duodenoscopy was performed 2 days after presentation, using an Olympus 1.0 m × 9.8 mm videoscope. The esophageal mucosa had a cobblestone appearance and was diffusely erythematous, particularly oral to the lower esophageal sphincter. Upon entering the stomach, a larger-than-expected volume of bile-tinged fluid was seen, despite 48 hours of fasting. The gastric mucosa was diffusely erythematous and appeared thickened, and the rugal folds were ill-defined even with minimal gastric insufflation. Subjective impression of the endoscopist was that after insufflation with air, the stomach did not distend as uniformly or as much as expected. Ulcers or erosions were not identified in the stomach, and no abnormalities were seen in the duodenum. Smears of gastric pinch-biopsy samples were cytologically consistent with parietal cell hyperplasia with the presence of many fibroblasts. Results of a point-of-care test for detection of Helicobacter sp. were not consistent with infection. Biopsy samples were not submitted for histopathology, because the decision was made to proceed with exploratory laparotomy given the uncertainty of primary gastric disease versus gastrinoma and the owner’s desire for palliative intervention.

Exploratory laparotomy the following day revealed a thickened, firm, and irregular-appearing gastric fundus, pylorus, and antrum [Figure 3]. A nodular, yellow, mesenteric mass extended parallel to the left limb of the pancreas and was closely associated with the vascular root of the spleen. The pancreas itself appeared normal, and no mass lesions were palpable within the parenchyma. Omental adhesions were present between the spleen, stomach, and diaphragm; the jejunum was mildly dilated; and an approximately 7.0 cm-diameter, tan nodule encompassed a large portion of the caudate lobe of the liver. A gastrojejunostomy, splenectomy, and a wedge biopsy of the liver nodule were performed. Because the owners had stressed that they preferred aggressive intervention if it offered hope for control of clinical signs, a subtotal gastrectomy was performed. This left only a relatively small portion of the gastric antrum, which was anastomosed end-to-side to the jejunum.
A majority of the peripancreatic mass was removed, and a jejunostomy tube was placed.

Histopathological examination of the surgically obtained samples revealed mucinous gastric carcinoma with metastasis to the mesentery and spleen. Neoplastic cells occurring in clusters with glandular to solid arrangements originated from the lamina propria of the stomach and extended into the submucosa and muscularis [Figure 4]. The gastric fundus was the most severely affected region, and neoplastic emboli within lymphatic vessels were seen in many cut sections. Variably defined nodules of neoplastic cells with extensive mucin production were seen in the spleen. The masses within the mesentery also contained numerous neoplastic cells admixed with free mucin as well as intravascular neoplastic emboli. The liver biopsy revealed hepatic glycogenic and lipidic degeneration. Immunohistochemical staining failed to reveal gastrin within the neoplastic gastric cells. Because neoplastic cells in all other examined tissues were identical in appearance to the primary mucinous gastric carcinoma, immunohistochemical staining of other tissue sections was not performed.

Multiple complications developed in the immediate postoperative period, including severe pancreatitis with secondary disseminated intravascular coagulation (DIC) and hemothorax. Symptomatic therapy included the following: oxygen therapy via nasal cannulae; withholding of oral food and water; chest tube placement and intermittent thoracentesis in response to dyspnea; and transfusions with packed red blood cells, plasma, whole blood, and serum albumin in response to anemia, anemia with coagulopathy, and severe hypoalbuminemia with secondary subcutaneous edema. Enteral nutrition was provided via the jejunostomy tube. The dog’s hemothorax and clinical signs of pancreatitis slowly decreased in severity over 7 days but never completely resolved. The owners nevertheless insisted on attempting to manage the dog at home, and the dog was discharged 7 days postoperatively with an extremely guarded prognosis. Two days after discharge, the dog became dyspneic because of severe pleural effusion and was euthanized; a necropsy was not performed.

Discussion

This case report describes a dog with mucinous gastric carcinoma that closely mimicked the clinical presentation and diagnostic imaging appearance expected in dogs with gastrinomas. The dog of this report also had a mild increase in fasting serum gastrin concentration that has not been previously reported in a dog with primary gastric neoplasia. As with our case, dogs with gastrinomas classically are presented with vomiting and anorexia presumptively due to hypergastrinemia, thickened gastric mucosa, and gastrointestinal ulceration. Hypersecreting pancreatic masses are frequently identified via laparotomy or necropsy, and fasting serum gastrin concentrations are above the reference range.

The increased serum gastrin concentration in this dog is consistent with previous reports of gastrinoma, even though gastrin concentrations were not markedly increased. Fasting serum gastrin concentrations from previously reported dogs with histologically confirmed gastrinomas range from 2.6 to 31.7 times the upper end of the reference range (n=5; 90 to 2780 pg/mL [n=16]);2,3,5,11-14 with concentrations <200 pg/mL in three cases.5,11 Unfortunately, although absolute values of serum fasting gastrin are often reported, direct comparisons of previous reports are difficult, as various assays have been used; therefore, fold-comparisons above reference range are now conventionally considered more appropriate.
Although some authors have suggested that a fasting serum gastrin concentration >1000 pg/mL is necessary for definitive diagnosis of gastrinoma without further provocative testing, results well below this are often encountered. Serum gastrin concentrations in dogs with primary gastric neoplasms have not been reported. We suspect that gastric mucosal invasion by neoplastic cells may have decreased the stomach’s ability to produce hydrochloric acid, resulting in reduced negative feedback for gastrin production. Gastric pH in our dog was slightly higher than normal, although fasting gastric content pH is slightly more alkaline than after feeding.15

Other reported causes of increased serum gastrin in dogs include gastric ulcers, gastric outflow obstruction, renal failure, hyperparathyroidism, short bowel syndrome, and atrophic gastritis—none of which were present in this dog.16 *Helicobacter pylori* infection in the antral mucosa has also been associated with hypergastrinemia in humans.10 This has not been reported in dogs, and the point-of-care test for detection of *Helicobacter* sp. used here as well as histopathology were not consistent with infection. The point-of-care test for humans used at our institution is 99% sensitive and 95% specific after a 2-hour incubation, although the accuracy of identifying *Helicobacter* sp. depends on the bacterial load present, and to our knowledge this test has not been studied in dogs.17 Ultimately it remains unclear why this particular dog had increased fasting serum gastrin concentration.

Although the clinical presentation, many of the diagnostic imaging findings, and hypergastrinemia were supportive of gastrinoma, in retrospect other findings were more suggestive of a primary gastric neoplasm.18 The reference range for normal canine gastric rugal fold thickness is 0.1 to 0.8 cm — well below the 1.4 cm measured by ultrasonography in this case.18 Abdominal ultrasonographic gastric wall measurements over 0.6 to 0.7 cm in dogs have been proposed as being highly consistent with pathological lesions, although thickening is not considered specific for gastric neoplasia, and nonmalignant lesions should likely still be considered.9,19,20 The severity of gastric wall thickening in dogs with gastrinomas has not been reported.

In addition to the generalized increase in gastric wall thickness, the loss of normal gastric wall layering seen with abdominal ultrasound is also more consistent with a primary gastric neoplasm. In 13 dogs with gastric neoplasia, only one had preserved gastric wall layering.9 We presume that the gastric wall hypertrophy that occurs secondary to gastrinomas should maintain normal gastric layering on sonographic evaluation, but this must be systematically evaluated. Interestingly, in human medicine, endoscopic ultrasonography has been used to differentiate mucinous from nonmucinous gastric carcinomas. Mucinous lakes surrounded by reactive fibrous tissue may correlate with reticular, highly echoic speckles; however, these were not seen in this case.21

Gastroduodenoscopy was attempted in this dog in the hope of differentiating a primary gastric neoplasm from secondary thickening due to gastrinoma, even though an accurate diagnosis of gastric neoplasia may be difficult to obtain unless neoplastic cells invade the superficial mucosa. Interestingly, we did not note any gastric ulcers or erosions. Gastrointestinal ulceration has been reported in five of seven animals with gastrinoma, and in 18 of 23 dogs with gastric carcinoma.2,7 The remaining five dogs with gastric carcinoma in this latter report had blanching of the mucosa with loss of the normal submucosal vascular pattern, which is more consistent with our findings. Diffuse infiltration of the gastric wall by carcinomas is referred to as limitis plastica; it is often evident on endoscopic examination as marked thickening of gastric folds, a difficult-to-distend stomach due to wall rigidity, and a “leopard skin” appearance to the mucosa.22-25 This scirrhous subtype of tumor is highly associated with carcinomatosis in humans, as was seen in this dog.25 Interestingly, in at least one experimental model, hypergastrinemia was associated with development of gastric carcinoma with limitis plastica features in a beagle.22

Mucinous gastric carcinomas in humans comprise 2% to 5% of all gastric neoplasms.20 This subtype of gastric carcinoma is characterized by a substantial number of mucus lakes (i.e., >50% extracellular mucin) resulting from mucin pooling in the tumor stroma; signet ring cells or carcinomatous glands are frequently seen floating within lakes.26,27 The potential prognostic relevance of mucin production by gastric carcinomas is controversial. One report suggests that mucinous gastric carcinomas are more deeply invasive tumors, more commonly have lymph node involvement, and reflect a more advanced stage of disease at the time of diagnosis compared to nonmucinous gastric carcinomas. A significant difference in survival time may also be seen between mucinous and nonmucinous gastric carcinomas, although this may be due to the more advanced stage of disease at which mucinous carcinomas are typically diagnosed.26

Some authors further divide mucinous gastric carcinomas into undifferentiated and differentiated types, which may also be a prognostic indicator.26 Early-stage mucinous gastric carcinomas may have a similar biological behavior as the nonmucinous form. However, in later stages, lymphatic spread is more common than hematogenous spread, likely because of increased lymphatic permeation in mucinous tumors.21,28 Early mucinous gastric carcinomas in humans also demonstrate an increased frequency of submucosal invasion (83%) compared with that (43%) in early nonmucinous carcinomas. Finally, mucinous gastric carcinomas have been proposed to originate as adenocarcinomas and later become mucinous with tumor progression.28

The optimal surgical intervention for such an extensive tumor is unclear. In a case series of 21 dogs with malignant gastric neoplasia, two underwent gastrectomy and were euthanized 4 and 5 weeks postoperatively because of recurrence of vomiting and anorexia.8 Data regarding survival times for dogs with advanced-stage gastric carcinoma are not available. The development of pancreatitis, DIC with hemorrhagic pleural effusion, anemia, and severe hypoalbuminemia were significant complications in our case. Unfortunately,
even though these complications were managed with partial success, the persistent pleural effusion resulted in humane euthanasia being the only viable option because of the dog’s quality of life and owner financial constraints.

**Conclusion**

Although rare, mucinous gastric carcinoma should be a differential diagnosis in dogs with diffuse gastric wall thickening. As demonstrated by this case, dogs with mucinous gastric wall carcinoma may have mild increases in fasting serum gastrin concentration of unexplained etiology. Therefore, an increased fasting serum gastrin concentration, when less than the published recommendation of >1000 pg/mL, should not be automatically considered diagnostic for gastrinoma. Future studies should more thoroughly investigate fasting serum gastrin concentrations in dogs with neoplastic and nonneoplastic primary gastric diseases, in order to better establish the sensitivity and specificity of various values as predictors for gastrinomas.

**Footnotes**

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**References**


