Primary splenic torsion in dogs: 102 cases (1992–2014)

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OBJECTIVE
To determine the percentage of dogs surviving to hospital discharge and identify factors associated with death prior to hospital discharge among dogs undergoing surgery because of primary splenic torsion (PST).

DESIGN
Retrospective case series.

ANIMALS
102 client-owned dogs.

PROCEDURES
Medical records of dogs with a confirmed diagnosis of PST that underwent surgery between August 1992 and May 2014 were reviewed. History, signalment, results of physical examination and preoperative bloodwork, method of splenectomy, concurrent surgical procedures, perioperative complications, duration of hospital stay, splenic histopathologic findings, and details of follow-up were recorded. Best-fit multivariate logistic regression was performed to identify perioperative factors associated with survival to hospital discharge.

RESULTS
93 of the 102 (91.2%) dogs survived to hospital discharge. German Shepherd Dogs (24/102 [23.5%]), Great Danes (15/102 [14.7%]), and English Bulldogs (12/102 [11.8%]) accounted for 50% of cases. Risk factors significantly associated with death prior to hospital discharge included septic peritonitis at initial examination (OR, 32.4; 95% confidence interval [CI], 2.1 to 502.0), intraoperative hemorrhage (OR, 22.6; 95% CI, 1.8 to 289.8), and postoperative development of respiratory distress (OR, 35.7; 95% CI, 2.7 to 466.0). Histopathologic evidence of splenic neoplasia was not found in any case.

CONCLUSIONS AND CLINICAL RELEVANCE
Results suggested that the prognosis for dogs undergoing splenectomy because of PST was favorable. Several risk factors for death prior to discharge were identified, including preexisting septic peritonitis, intraoperative hemorrhage, and postoperative development of respiratory distress. (J Am Vet Med Assoc 2016;248:661–668)

Isolated torsion of the splenic pedicle, or PST, is a rare condition in dogs and occurs when the spleen rotates around the gastrosplenic and phrenicosplenic ligaments.1,2 It is most commonly reported in large- or giant-breed, deep-chested dogs, particularly Great Danes and German Shepherd Dogs.3 Acute and chronic forms of PST have been described.4–5 Acute PST can cause signs of severe abdominal pain, weakness, and cardiovascular collapse, which can occur rapidly, manifesting in some cases in a matter of hours.3,5,6 Chronic PST can be a diagnostic challenge in some cases, with clinical signs including vomiting, lethargy, weakness, abdominal pain, hematuria, and diarrhea tending to be vague or intermittent.6,8 The etiology of PST in dogs is poorly understood. Proposed causes include congenital absence or weakness of the supporting ligaments of the spleen, or acquired laxity of these ligaments as a result of surgery, trauma, or previous episodes of GDV.5–7,9 Although successful splenic derotation and reposi- tioning have been reported,10 splenectomy is the most commonly performed treatment for PST in affected dogs.4,7,11,12 It has been suggested that transection of the gastrosplenic ligament during splenectomy can result in increased mobility of the stomach within the peritoneal cavity, which may increase the risk for subsequent GDV.10,13,14 and that dogs with a history of PST will be predisposed to developing GDV later in life.13,15 However, this association was not found to be significant in a recent report.11 Nonetheless, because
of this potential association, prophylactic gastropexy at the time of splenectomy for treatment of PST has been recommended by some clinicians.13,15

There are few published reports of the management of PST in dogs; thus, information on predisposing factors and perioperative outcome is limited. The largest retrospective study of 19 dogs by Neath et al3 found an increased risk in Great Danes and German Shepherd Dogs and suggested that male dogs (castrated and sexually intact) were more frequently affected than females. In that study, splenectomy led to a successful outcome (ie, the patient was discharged from the hospital) in all 18 dogs in which it was performed, but no patient variables significantly associated with an increased odds of morbidity or death were identified.3

The objectives of the study reported here were to determine, for a large cohort of dogs undergoing splenectomy because of PST, the percentage of dogs surviving to hospital discharge and to identify factors associated with death prior to hospital discharge. An additional objective was to determine the proportion of dogs undergoing splenectomy for PST that develop GDV in later life.

Materials and Methods

Case selection criteria

Medical records of all dogs with a diagnosis of PST that underwent exploratory laparotomy at any of 7 referral hospitals (6 academic institutions and 1 private practice) from August 1992 through May 2014 were retrospectively reviewed. Dogs were included in the study if PST was confirmed at the time of exploratory laparotomy and the medical record was available for review. Dogs were excluded if splenic torsion was not primary (eg, secondary to a diagnosis of GDV).

Medical records review

Preoperative data collected from the medical records included history, initial complaints, signalment, body weight, duration of clinical signs, clinical laboratory findings, physical examination findings on initial examination, and results of diagnostic imaging findings. Intraoperative data collected from the medical records included duration of general anesthesia (minutes), duration of surgery (minutes), surgical complications (if applicable), blood product administration (product administered, if applicable), method of splenectomy (suture ligation only, vessel-sealing device only, a combination of suture ligation and a vessel-sealing device, or a combination of suture ligation and a ligate-divide-staple device), and concurrent surgical procedures performed. Postoperative data collected from the medical records included postoperative complications (if applicable), blood product administration (product administered, if applicable), duration of hospital stay (days), outcome (survival to discharge or died prior to discharge), results of splenic histopathologic evaluation, date of most recent follow-up, occurrence of GDV in the follow-up period (and interval to GDV [months], if applicable), survival time, and cause of death or euthanasia (if applicable).

Statistical analysis

Patient demographics, signalment, and characteristics of diagnosis and treatment were evaluated. Summary statistics were reported for all measured variables, including signalment, clinical laboratory values, and variables associated with treatment and hospitalization. The proportions of patients with various common clinical signs and diagnostic imaging findings were calculated. Distributions of variables were evaluated with tests of skewness and kurtosis, quantile probability plots, and the Shapiro-Wilk test. Categorical variables were expressed as numbers and percentages, and continuous variables were expressed as means, SDs, medians, and ranges. Logistic regression modeling was used to identify factors associated with death prior to hospital discharge. Death prior to hospital discharge was the dependent variable, and country, age, sex, weight, duration of clinical signs, presence of preexisting septic peritonitis (as indicated in the medical record), preoperative PCV, preoperative blood lactate concentration, presence of gas in the spleen on radiographic or ultrasonographic images, receipt of a blood product transfusion at any time during hospitalization, ventricular arrhythmias during hospitalization, excessive intraoperative hemorrhage (as described in the surgical report), and postoperative morbidities including anemia, coagulopathy, hematuria, respiratory distress, and neurologic disorders (including grand mal seizures, signs of cervical pain, ataxia, and diskospondylitis) were independent variables. Stratification by hospital was attempted but not included in the final model because it resulted in dropout of a large portion of observations. Univariate analyses were performed, and covariates with a Wald P value < 0.2 were tested for inclusion in the multivariate model. A forward selection model was used for multivariate modeling, with covariates retained if the likelihood ratio test P value, Wald P value, or both was < 0.05 or if covariates were confounding the association of interest (defined as > 15% change in the OR). In instances when likelihood ratio methods were not applicable because of missing data, Akaicke and Bayesian information criteria were used to compare models. The Kaplan-Meier product-limit method was used to estimate survival time following hospital discharge. Model fit was assessed with the Hosmer-Lemeshow goodness-of-fit statistic. All statistical analyses were performed with computer software.

Results

One hundred two dogs met the study selection criteria. Patient characteristics and clinicopathologic data were summarized (Tables 1 and 2). Information on initial physical examination findings was available for 98 dogs; initial physical examination identified a palpably enlarged spleen in 68 of 98 (69.4%) dogs, hemoperitoneum in 30 (30.6%) dogs, hypovolemic shock in 22 (22.5%) dogs, and septic peritonitis in 8 (8.2%) dogs. Mean arterial blood pressure was recorded at initial examination, prior to initiation of treatment, in
49 dogs; mean ± SD arterial blood pressure was 112.7 ± 31.5 mm Hg. Ventricular premature contractions or tachycardia was recorded at initial examination in 10 of 102 (9.8%) dogs. Preoperative activated clotting time was available for 22 dogs, with a mean ± SD time of 111.7 ± 29.4 seconds. Prothrombin time and partial thromboplastin time were available for 52 dogs; however, institution-specific reference ranges resulted in the inability to compare these data.

Abdominal radiography was performed in 70 of the 102 dogs, but radiography reports for only 68 dogs were available for review. The most common abnormalities noted were enlarged spleen (n = 46 [67.6%]) and loss of serosal detail (32 [47.1%]). Gastric dilatation (8 [11.8%]), abnormal spleen position (5 [7.4%]), and free gas in the spleen (3 [4.4%]) were reported less commonly.

Abdominal ultrasonography was performed in 83 dogs, including 26 that also underwent abdominal radiography. Ultrasound examination results were available for 82 dogs. The most common abnormalities noted were enlarged spleen (n = 71 [86.6%]), reduced to absent splenic blood flow (60 [73.2%]), hypoechoic splenic parenchyma (38 [46.3%]), and free peritoneal fluid (38 [46.3%]). Less commonly reported were abnormal spleen position (26 [31.7%]), hyperechoic mesentery (23 [28.0%]), and free gas in the spleen (2 [2.4%]).
A diagnosis of PST was confirmed via direct visualization at the time of laparotomy in all 102 dogs. In 1 dog, the splenic pedicle was derotated and the spleen was repositioned and left in situ. In the remaining 101 dogs, splenectomy was performed. The surgical technique employed to ligate the splenic vasculature and adjacent mesentery was recorded in 94 dogs and consisted of suture ligation (n = 36 [38.3%]), suture ligation in combination with a ligate-divide-staple device (28 [29.8%]), suture ligation in combination with a vessel-sealing device (15 [16.0%]), or use of a vessel-sealing device alone (14 [14.9%]).

A prophylactic gastropexy was also performed in 68 (66.7%) dogs; additionally, 4 dogs had an existing gastropexy from prior surgery. The type of gastropexy was not recorded in the medical record. At least 1 additional nongastropexy surgical procedure was performed in 39 (38.2%) dogs (including 24 dogs that also had a gastropexy), including liver biopsy in 11 (11.1%) dogs, resection of necrotic omentum in 5 (4.9%) dogs, partial pancreatectomy in 3 (2.9%) dogs, gastrotomy to remove a foreign body in 3 (2.9%) dogs and gastric resection or invagination in 2 (2.0%) dogs. Other concurrent surgical procedures included subcutaneous mass excision or biopsy in 3 (2.9%), castration in 1 (1.0%), colostomy in 1 (1.0%), ovariohysterectomy in 1 (1.0%), paraprostatic cyst omentalization in 1 (1.0%), mesenteric lymph node biopsy in 1 (1.0%), jejunal resection and anastomosis in 1 (1.0%), gastric biopsy in 1 (1.0%), unilateral arytenoid lateralization in 1 (1.0%), and brachycephalic airway surgery in 1 (1.0%).

Median surgical procedure time was 90 minutes (range, 31 to 265 minutes), and median anesthesia time was 163 minutes (range, 50 to 340 minutes). Intraoperative complications were reported in 29 (28.4%) dogs. These included severe hemorrhage (as stated in the medical record) in 10 (9.8%) dogs, ventricular arrhythmias in 9 (8.8%) dogs, atroventricular block in 4 (3.9%) dogs, thrombosis of the splenic vasculature in 2 (2.0%) dogs, gross spillage of gastric contents in 2 (2.0%) dogs, and iatrogenic injury (inadvertent liver laceration or inadvertent full-thickness gastric laceration) in 2 (2.0%) dogs. Two (2.0%) dogs had cardiac arrest and died under general anesthesia: 1 with ongoing abdominal hemorrhage and 1 with severe pulmonary hemorrhage.

Blood products were administered to 27 (26.5%) dogs during the perioperative period. Packed RBCs were given to 14 (13.7%) dogs, fresh frozen plasma to 6 (5.9%) dogs, whole blood to 2 (2.0%) dogs, both packed RBCs and fresh frozen plasma to 3 (2.9%) dogs, and packed RBCs and whole blood to 2 (2.0%) dogs.

Among the 100 dogs that survived surgery, 35 (35.0%) had postoperative complications, of which 13 (13.0%) experienced > 1 postoperative complication. Anemia (n = 13 [13.0%]) and ventricular arrhythmias (12 [12.0%]) were the most common complications encountered. Respiratory distress (as noted in the medical record) occurred in 8 (8.0%) dogs, including aspiration pneumonia (4 [4.0%]), pleural effusion (2 [2%]), pulmonary hemorrhage (1 [1.0%]), and acute respiratory distress syndrome attributed to pulmonary thromboembolism (1 [1.0%]). Hematuria or pigmenturia occurred in 8 (8.0%) dogs, none of which had received a prior blood transfusion. Suspected coagulopathy (protracted bleeding) occurred in 8 (8.0%) dogs. Four (4.0%) dogs had postoperative neurologic complications, including 1 dog that reportedly had grand mal seizures, signs of cervical pain, ataxia, and diskospondylitis. Other postoperative complications listed in the medical record included regurgitation (n = 3 [3.0%]), icterus (2 [2.0%]), vomiting (2 [2.0%]), hypoglycemia (1 [1.0%]), septicemia (1 [1.0%]), and evisceration of intestines through the abdominal incision (1 [1.0%]).

Splenic histopathologic results were available for 79 (77.5%) dogs. The histopathologic findings in all spleens were consistent with vascular occlusion secondary to torsion; congestion, hemorrhage, necrosis, thrombosis, fibrin, siderotic plaques, and neutrophilic inflammation were commonly identified. Neoplasia or other concomitant splenic abnormalities were not identified in any spleen.

Median duration of hospitalization was 3 days (range, 1 to 6 days). In total, 9 of 102 (8.8%) dogs did not survive to hospital discharge. In addition to the 2 dogs that died while under anesthesia, 7 dogs were euthanized. The cause of euthanasia was attributed to disseminated intravascular coagulation in 3 dogs, pleural effusion in 2 dogs, aspiration pneumonia in 1 dog, and ventricular tachycardia in 1 dog. Three of 8 (37.5%) dogs that had signs of septic peritonitis at initial examination prior to surgery died before hospital discharge.

Univariate logistic regression analysis revealed that body weight, ventricular arrhythmia at any time during hospitalization, preexisting septic peritonitis, intraoperative hemorrhage, postoperative anemia, and postoperative respiratory distress were significantly associated with death prior to hospital discharge. Univariable logistic regression analysis revealed that body weight, ventricular arrhythmia at any time during hospitalization, preexisting septic peritonitis, intraoperative hemorrhage, postoperative anemia, and postoperative respiratory distress were significantly associated with death prior to hospital discharge. In the best-fit multivariate model (Table 3), dogs with signs of preexisting septic peritonitis, dogs that had intraoperative hemorrhage, and dogs that developed postoperative respiratory distress were significantly more likely to die prior to hospital discharge, after adjusting for other confounding variables.

**Table 3**—Results of multivariate logistic regression analysis of risk factors associated with death prior to hospital discharge in dogs (n = 102) undergoing surgery because of PST.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>OR (95% CI)</th>
<th>Wald P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preexisting septic peritonitis</td>
<td>32.4 (2.1 – 502.0)</td>
<td>0.013</td>
</tr>
<tr>
<td>Intraoperative hemorrhage</td>
<td>22.6 (1.8 – 289.8)</td>
<td>0.016</td>
</tr>
<tr>
<td>Postoperative respiratory distress</td>
<td>35.7 (2.7 – 466.0)</td>
<td>0.006</td>
</tr>
<tr>
<td>Weight (&gt;10 kg)</td>
<td>1.6 (0.7 – 3.2)</td>
<td>0.236</td>
</tr>
</tbody>
</table>

Weight was a confounding variable. Hosmer-Lemeshow goodness-of-fit P = 0.630 (8 degrees of freedom).
Follow-up information was available for 64 of the 93 (68.8%) dogs that survived to hospital discharge. Among these dogs, the median duration of follow-up was 472 days (range, 7 to 3,655 days). Dates and causes of death were known for 11 (11.8%) dogs. Six dogs died of neoplasia and 5 died of other causes, including 1 each of cardiac arrest (secondary to dilative cardiomyopathy), GDV, aspiration pneumonia, and euthanasia for nonspecific clinical signs and for aggression. The median survival time for these 11 dogs was 719 days (range, 290 to 2,752 days). Survival time for the entire population of dogs could not be accurately estimated because of heavy censoring and losses to follow-up. Among the 65 dogs for which follow-up information was available, 47 underwent prophylactic gastropexy at the time of PST surgery, 2 had previously been treated with a gastropexy, and 15 had never undergone a gastropexy (the gastropexy status of the remaining dog was unknown). Within the study follow-up period, 1 dog with a gastropexy and 1 dog without a gastropexy developed GDV; the proportions of dogs with and without gastropexy that developed GDV were not significantly different (P = 0.278). There were no reports of death or euthanasia attributed to splenic torsion or surgery following hospital discharge.

Discussion

Results of the present 22-year (1992–2014) multiinstitutional study of 102 dogs undergoing surgical treatment for PST suggested that the prognosis was favorable. However, we identified several variables associated with death prior to hospital discharge in this patient population, including preexisting septic peritonitis, intraoperative hemorrhage, and postoperative development of respiratory distress. In this study, 8.8% (9/102) of dogs did not survive to discharge. Dogs that survived to hospital discharge and for which follow-up information was available had no complications related to surgery, and none died or were euthanized as a result of surgical treatment of PST.

The breed and sex characteristics of dogs in the present study were similar to those described in previous reports.3,13,15 Previously, Great Danes and German Shepherd Dogs have been identified as the most frequently affected, and male dogs (castrated and sexually intact) were more frequently affected than female dogs.3,16 In this study, in addition to Great Danes and German Shepherd Dogs, English Bulldogs represented a notable percentage of the study population (12/102 [11.8%]). These 3 breeds comprised 50% of dogs in the present study. To our knowledge, the English Bulldog breed has not previously been reported to be predisposed to PST; and it has been suggested that deep-chested dogs, with an increased thoracic depth-to-width ratio, are the most susceptible to PST because additional space in the abdominal cavity may allow organs to rotate abnormally.13 A similar mechanism may potentially be present in English Bulldogs because of their barrel-chested conformation; however, further investigation into this potential association is required. Nonetheless, we suggest that PST should be considered an important differential diagnosis for English Bulldogs being examined because of signs of acute abdomen.

In our study population, dogs with septic peritonitis at the time of initial examination were 32.4 times as likely to die prior to discharge as were dogs that lacked this diagnosis. On the basis of this association, we suggest that dogs suspected to have PST should be evaluated for the presence of septic peritonitis, as this diagnosis may discourage dog owners from consenting to surgery because of the potential for a poorer prognosis in this patient population. The pathophysiology of septic peritonitis secondary to PST is poorly understood. With PST, the spleen twists around the splenic pedicle, such that thin-walled veins become occluded, whereas arteries remain patent. This results in vascular stasis and eventual thrombus formation in the splenic vasculature, resulting in splenic necrosis that may allow for bacterial translocation into the peritoneal cavity. There are also several reports17,18 of splenic clostridial infections, which could serve as a source for bacterial translocation. In the present study, results of microbial culture of samples from patients with PST and preexisting septic peritonitis were not investigated. Future studies are necessary to provide further insight into the pathophysiologic mechanisms and origin of septic peritonitis in dogs with PST.

Dogs that had intraoperative hemorrhage in this study were 22.6 times as likely to die prior to discharge as were dogs that did not. A possible explanation for the development of hemorrhage may be that dogs with PST are predisposed to develop disseminated intravascular coagulation,5,12,19 Disseminated intravascular coagulation may occur as a result of activation of the coagulation cascade and fibrinolysis with the release of microemboli and thromboplastic substances from the compromised spleen.12,19 Disseminated intravascular coagulation may also occur as a result of decreased function of the reticuloendothelial system in patients with PST.12,19 Anemia and thrombocytopenia may also occur with sequestration of platelets within the congested spleen, further increasing the risk of hemorrhage in patients with PST.12,20 In the present study, 26.5% (27/102) of dogs required blood product transfusions during hospitalization, suggesting that hemorrhage or anemia may be common in dogs with PST. It is also possible that excessive hemorrhage was related to failure of the sutures or staples or other means of surgical ligation. Because intraoperative hemorrhage was found to be a significant risk factor for death in the present study, procedures to evaluate and improve hemostasis should be considered in dogs undergoing surgery for PST.

An additional consideration in evaluating the association between an increased odds of death and intraoperative hemorrhage in the present study may be the technique of splenectomy in dogs with PST. A variety of techniques for performing splenectomy were reported for the patients in this study; however, we did
not find an association between specific surgical technique and intraoperative hemorrhage. Vessel-sealing devices have been reported to be an efficient and safe method of hemostasis when performing splenectomy in dogs. Splenectomy in patients with PST can be challenging. It has been recommended that the spleen be removed without derotating or untwisting the splenic pedicle to avoid massive release of toxins that have accumulated within the compromised spleen, as these may have direct cardiodepressant effects for anesthetized patients if suddenly released into the systemic circulation. A recent study in dogs demonstrated that the use of a bipolar vessel-sealing device significantly decreased surgery times and reduced blood loss, when compared with a stapling device. Additionally, vessel-sealing devices may allow for minimal manipulation of the torsed splenic pedicle and may result in less hemorrhage and reduced complication rates.

Dogs that developed signs of postoperative respiratory distress or disease were 35.7 times as likely to die prior to discharge as were dogs that did not. For the patients in the present study, these signs included included aspiration pneumonia (n = 4 cases), pleural effusion (2), pulmonary hemorrhage (1), and suspected pulmonary thromboembolism (1). Both dogs with pleural effusion and the dog with pulmonary hemorrhage either died or were euthanized prior to hospital discharge. Pulmonary hemorrhage or pulmonary thromboembolism may have developed secondary to disseminated intravascular coagulation as a result of PST, and pleural effusion may occur secondary to pulmonary embolism. On the basis of the association between development of respiratory distress and increased odds of death, in addition to the finding that intraoperative hemorrhage was associated with an increased odds of death, we suggest that prompt diagnosis and surgical treatment may increase chances of survival in dogs with PST by reducing the propensity for thromboembolic disease and platelet sequestration, as both can result in disseminated intravascular coagulation.

Splenic histopathologic evaluation results were available for 79 of the 102 (77.5%) dogs in the present study. The most common histologic findings were combinations of splenic congestion, hemorrhage, necrosis, and the presence of thrombi, fibrin, and neutrophilic inflammation. There was no evidence of neoplasia in any of the cases where the spleen was submitted for histopathologic evaluation. This lends further evidence to the suggestion that PST is an idiopathic disease or at least is not associated with neoplasia. Furthermore, this finding should be considered when discussing etiology and prognosis with owners of dogs with PST, as malignant splenic neoplasia carries a guarded prognosis, which may prompt owners to decide against surgical intervention.

The association between PST and GDV is unclear, with anecdotal evidence suggesting that dogs with a history of PST have an increased risk of developing GDV. The authors of a 1995 case series suggested that all dogs undergoing splenectomy for PST should also be treated with a concurrent prophylactic gastro- pexy if they were hemodynamically stable (ie, could tolerate the additional anesthesia time). This recommendation, however, was made on the basis of only 2 dogs that reportedly developed GDV following splenectomy for PST. In a subsequent retrospective study of 19 dogs, of which 8 underwent concurrent prophylactic gastro- pexy, long-term follow-up information (ranging from 1 to 84 months after surgery) was available for 12 of 19 dogs. Of those 12 dogs, only 1 developed GDV 7 months after surgery. In the present study, follow-up information was available for 68.8% (64/93) of the dogs that survived to hospital discharge (median duration of follow-up, 553 days; range, 7 to 6,562 days). Of those 64 dogs, 49 had a concurrent gastro- pexy performed. In this group of dogs, a single dog (English Bulldog) developed GDV 3 years after surgery. Development of GDV following incisional gastro- pexy has been previously described. Of the 15 dogs in which a concurrent gastro- pexy was not performed, a single dog (Newfoundland) developed GDV 4 months after surgery. The percentages of dogs with and without gastro- pexy that developed GDV were not significantly different, but given the low rate of GDV in both groups and given that long-term follow-up information was not available for all patients in the study, we cannot draw meaningful conclusions regarding the importance of gastro- pexy or the risk of GDV in dogs following splenectomy for PST. On the other hand, because gastro- pexy is an effective, relatively simple procedure that is associated with a low complication rate, we support the previous recommendation to perform a concurrent gastro- pexy following splenec- tomy in dogs with PST.

Limitations of the present study are mainly due to its retrospective nature. Data were collected from records intended for patient care; missing data and selective records reporting could introduce bias and result in conclusions that do not accurately reflect the general population of dogs with PST. Data for diagnostic test results, in particular, contained many missing values, which affected variable selection in the regression model and could have biased the results. Variables considered for our regression model had < 5% missing data, except for serum lactate concentration, which was not included in the final model. When identifying preoperative condition and perioperative complications, medical records often identified the condition (eg, septic peritonitis, excessive hemorrhage, and respiratory distress) but confirmatory testing results were not always available. We acknowledge that hemorrhage is a subjective term and use of this term represented a limitation of our data. Cases were accumulated from a number of institutions, resulting in variability in surgeon experience, surgical technique, and patient management before and after surgery. Because of the small number of cases, we were not able to
stratify analyses according to hospital, which could have accounted for regional or institutional differences in patient care and outcome. Furthermore, because the institutions included in the study were secondary or tertiary referral institutions, there may have been delays in identifying a definitive diagnosis or administering treatment, depending on care initiated with the primary care veterinarian prior to referral. However, variability in individual care and referral is typical of real-world veterinary practice. This study specifically examined patients undergoing surgery for PST at referral facilities, and it is certain that some dogs with PST either died or were euthanized prior to surgery at their primary veterinary clinic. Additionally, we have discussed disseminated intravascular coagulation as a proposed mechanism for causing increased intraoperative hemorrhage and respiratory distress. However, in the dogs of this study, data on coagulation parameters (prothrombin time, partial thromboplastin time, activated clotting time, and platelet count) were available for only a small subset of cases, preventing broader determination of hemostatic function and risk of disseminated intravascular coagulation. With respect to breed predispositions, the relative hospital populations of each breed were not examined; thus, particular breeds may have been overrepresented within the hospital population, affecting study results. Uniform follow-up information was not available for all dogs, and it is possible that dogs lost to follow-up had outcomes that would alter our results and conclusions if known. In particular, we could have failed to capture episodes of GDV, given that this condition primarily occurs in older dogs and the median follow-up time in the present study was < 2 years. Finally, despite the inclusion of a large number of cases relative to prior studies, our study could nevertheless have been underpowered to identify factors with small to moderate associations with death prior to hospital discharge.

In this study, results indicated that the prognosis for dogs undergoing splenectomy for PST was favorable. German Shepherd Dogs, Great Danes, and English Bulldogs were commonly represented in the study population. Three perioperative risk factors were identified as increasing the risk of death prior to hospital discharge (presence of septic peritonitis at initial examination, intraoperative hemorrhage, and postoperative development of respiratory distress). Proactive monitoring of coagulation parameters to allow for early recognition of signs of coagulopathy and prompt administration of appropriate treatment may help decrease the mortality rate associated with PST. The role of prophylactic gastropetry in preventing GDV after surgical treatment of PST in dogs is not clear.

Footnotes

1. Stata statistical software, release 12, StataCorp LP, College Station, Tex.

References

25. Hammond TN, Pesilto-Crosby SA. Prevalence of hemangiosarcoma in anemic dogs with a splenic mass and hemoperito-


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**From this month’s *AJVR***

**Effects of a medetomidine-ketamine combination on Schirmer tear test I results of clinically normal cats**

Simona Di Pietro et al

**OBJECTIVE**

To evaluate the effects of a medetomidine-ketamine combination on tear production of clinically normal cats by use of the Schirmer tear test (STT) I before and during anesthesia and after reversal of medetomidine with atipamezole.

**ANIMALS**

40 client-owned crossbred domestic shorthair cats (23 males and 17 females; age range, 6 to 24 months).

**PROCEDURES**

A complete physical examination, CBC, and ophthalmic examination were performed on each cat. Cats with no abnormalities on physical and ophthalmic examinations were included in the study. Cats were allocated into 2 groups: a control group (n = 10 cats) anesthetized by administration of a combination of medetomidine hydrochloride (80 µg/kg) and ketamine hydrochloride (5 mg/kg), and an experimental group (30) anesthetized with the medetomidine-ketamine combination and reversal by administration of atipamezole. Tear production of both eyes of each cat was measured by use of the STT I before anesthesia, 15 minutes after the beginning of anesthesia, and 15 minutes after administration of atipamezole.

**RESULTS**

Anesthesia with a medetomidine-ketamine combination of cats with no ophthalmic disease caused a significant decrease in tear production. The STT I values returned nearly to preanesthetic values within 15 minutes after reversal with atipamezole, whereas the STT I values for the control group were still low at that point.

**CONCLUSIONS AND CLINICAL RELEVANCE**

Results indicated that a tear substitute should be administered to eyes of cats anesthetized with a medetomidine-ketamine combination from the time of anesthetic administration until at least 15 minutes after administration of atipamezole. (*Am J Vet Res* 2016;77:310–314)

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