Postoperative thrombocytosis and thromboelastographic evidence of hypercoagulability in dogs undergoing splenectomy for splenic masses

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OBJECTIVE

To determine the frequency and severity of thrombocytosis and thromboelastographic evidence of hypercoagulability during the first 2 weeks after splenectomy in dogs with splenic masses and to investigate relationships between platelet counts and thromboelastography values.

ANIMALS

34 dogs undergoing splenectomy for splenic masses.

PROCEDURES

Blood samples for platelet counts and thromboelastography were obtained at induction of anesthesia (day 0) prior to splenectomy and on days 2, 7, and 14.

RESULTS

Mean platelet counts were 167.9 X 10^3/μL, 260.4 X 10^3 μ/L, 715.9 X 10^3/μL, and 582.2 X 10^3/μL on days 0, 2, 7, and 14, respectively, and were significantly higher at all postoperative assessment points than on day 0. Thrombocytosis was observed in 3% (1/34), 6% (2/33), 81% (21/26), and 69% (18/26) of dogs on days 0, 2, 7, and 14. Platelet counts > 1,000 X 10^3/μL were observed in 1 dog on day 2 and in 5 dogs on day 7. One or more thromboelastography values suggestive of hypercoagulability were observed in 45% (15/33), 84% (26/31), 89% (24/27), and 84% (21/25) of dogs on days 0, 2, 7, and 14. At each assessment point, higher platelet counts were correlated with thromboelastography values suggestive of hypercoagulability.

CONCLUSIONS AND CLINICAL RELEVANCE

Marked thrombocytosis and thromboelastography values suggestive of hypercoagulability were common during the first 2 weeks after splenectomy for the dogs of this study. If present, hypercoagulability could increase the risk for development of postsplenectomy thrombotic conditions such as portal system thrombosis and pulmonary thromboembolism. (*J Am Vet Med Assoc* 2020;256:85–92)

Splenic masses are a potentially life-threatening condition in aging dogs and are the most frequent indication for splenectomy. The most common types of splenic masses are hemangiosarcoma, which is a highly metastatic tumor, and benign lesions, such as hematoma and nodular hyperplasia.[1,2] Many dogs with splenic masses, particularly hemangiosarcoma, are evaluated because of acute and severe systemic clinical signs caused by sudden intra-abdominal hemorrhage.[1,2]

Reported perioperative mortality rates associated with splenectomy for splenic masses in dogs range from 7.6% to 33%.[1,3–8] Studies[5–7] have provided evidence that thromboses of major venous systems, including PST and PTE, are important causes of perioperative death. In a retrospective study[7] of 33 dogs with PST, 4 dogs had previously undergone splenectomy; in 3 of these dogs, the procedure was performed within the previous 3 months. Development of postoperative respiratory disease caused by suspected PTE or acute respiratory distress syndrome was associated with failure to survive to hospital discharge in a study[5] of 83 dogs with hemoperitoneum, 67 of which underwent splenectomy. In a study[6] of 539 dogs with splenic masses undergoing splenectomy at the authors' institution, the perioperative mortality rate was 7.6%, and the most common causes of death were known or suspected PST (22% of deaths) and PTE (10% of deaths).

In people, splenectomy is most commonly performed for blunt or penetrating abdominal trauma and hematologic disorders such as hemolytic anemia

ABBREVIATIONS

α	Clot formation angle
G	Clot strength
K	Clot formation time
MA	Maximum amplitude
PST	Portal system thrombosis
PTE	Pulmonary thromboembolism
R	Reaction time

and immune thrombocytopenic purpura.[8] People who have undergone splenectomy are predisposed to PST[9–12] and chronic PTE.[13,14] Portal system thrombosis develops in approximately 5% to 10% of patients, typically within 1 to 4 weeks after splenectomy,[9–12] whereas PTE typically develops at later points.[13,14] Although the causes of postsplenectomy venous thromboses are complex and incompletely understood, many people and dogs undergoing splenectomy have the Virchow triad of broad risk factors for thrombosis—inflammatory or other endothelial injury, hemodynamic stasis, and hypercoagulability.[15,16] For example, the presence of marked splenic enlargement may cause regional stasis of blood flow and has been identified as a risk factor for PST in human splenectomy patients.[9,10,17,18] Manipulation and ligation of the splenic vein, particularly if it is markedly enlarged, may cause endothelial damage and stasis of blood flow within the splenic vein remnant, leading to formation of a thrombus that may expand directly into the portal vein.[8,17] Loss of the filtering function of the spleen may allow particulate matter and abnormal cells to persist in the circulation, leading to hypercoagulability resulting from endothelial damage.[8]

Hypercoagulability may also be promoted by a transient thrombocytosis that frequently develops as a sequela to splenectomy in people.[8,19] Thrombocytosis is thought to develop because the spleen functions as a reservoir for platelets and usually removes senescent and damaged platelets from the circulation.[19] Platelet counts typically become increased relative to presplenectomy values 2 to 10 days after splenectomy, peak at 7 to 20 days, and then return to presplenectomy values over a period of weeks to months.[19] Postsplenectomy thrombocytosis has been identified as a risk factor for PST[9,10,12] and has been documented in up to 75% of human splenectomy patients.[9,10,12,19]

Little information is available regarding the possibility of a predilection of dogs to postsplenectomy thrombocytosis or hypercoagulability. In a previous clinical study[20] in which CBCs were performed 3 and 10 days after surgery in 12 dogs undergoing splenectomy for various reasons, platelet counts > 500 × 10^3/µL were found in 1 dog at the 3-day assessment point and in 5 additional dogs at the 10-day assessment point. In an experimental study[21] of platelet life span following splenectomy in 4 clinically normal dogs, mean platelet life spans were determined at various times between approximately 8 and 46 weeks after surgery. Mean platelet life span was 47% longer in splenectomized dogs (193 hours) than nonsplenectomized control dogs (131 hours). High mean platelet counts were observed in the splenectomized dogs 1 to 3 weeks and 3 months after surgery (993 × 10^3 platelets/µL and 701 × 10^3 platelets/µL, respectively).[21] Because dogs undergoing splenectomy often have hemoperitoneum and consumptive thrombocytopenia resulting from rupture of a splenic mass, any tendency for postsplenectomy thrombocytosis might be worsened by reactive increases in platelet production and release.[22] In addition, many dogs undergoing splenectomy have known risk factors for systemic inflammation and resultant hypercoagulability, including malignancy involving the endothelium, blood loss, blood transfusions, and major surgery.[15,16]

Although routine tests of coagulation such as prothrombin time and activated partial thromboplastin time are valuable for identifying hypocoagulable states, they have limited value in predicting hypercoagulability.[23] Thromboelastography, a dynamic in vitro test of the speed and strength of clot formation, is a superior indicator of thrombotic tendencies.[23] Overviews of thromboelastography methods and interpretation are available.[24,25] In brief, key thromboelastography parameters used in the identification of hypercoagulability are as follows: R, the time until initial formation of fibrin; K, the time to achieve a defined level of clot strength; α, a measure of the speed and strength of clot formation; MA, a measure of the ultimate strength of the fibrin clot; and G, a representation of clot strength and platelet function derived from the MA.[24,25] Associations between various thromboelastography parameters and platelet counts have been documented.[25–27] An important potential limitation of thromboelastography is that in vitro clot formation occurs under conditions that may not perfectly replicate clotting conditions in vivo.[28–31] For example, studies[28–31] involving healthy dogs have shown that because thromboelastography is performed by use of a constant volume of blood, low PCVs may result in a relative increase in the total mass of coagulation factors within the evaluated samples, causing an artifactual tendency for the resulting values to suggest levels of hypercoagulability that may not be present in the patient.

The primary purpose of the study reported here was to determine the frequency and severity of thrombocytosis and thromboelastographic evidence of hypercoagulability before and during the first 2 weeks after splenectomy in dogs with splenic masses. Secondary objectives were to evaluate relationships between platelet counts and thromboelastography parameters at each assessment point and to evaluate associations between selected preoperative clinical variables and postoperative platelet counts and thromboelastography parameters.

Materials and Methods

Animals

Dogs undergoing splenectomy for a splenic mass at the Foster Hospital for Small Animals, Cummings School of Veterinary Medicine, Tufts University between December 2015 and May 2017 were enrolled in the study with owner consent. No a priori sample size calculation was performed because the authors were unable to identify any large clinical studies in which the frequency of postsplenectomy thrombocytosis or thromboelastography results suggestive of hypercoagulability were reported. To ensure that the included dogs were representative of the general population of dogs undergoing splenectomy for splenic masses, no limitations were placed on treat-

ments administered, including IV fluid therapy, blood products or anticoagulants, anesthetic protocols, and additional surgical procedures performed, such as liver biopsy or prophylactic gastropexy. Dogs with 1 or more bleeding extrasplenic masses identified at surgery were excluded because of the possibility that such dogs would not survive the full duration of the study. Dogs with hyperadrenocorticism and dogs receiving corticosteroid drugs were also excluded because both are prothrombotic conditions that cause evidence of hypercoagulability on thromboelastography.[32,33] In addition, dogs receiving NSAIDs were excluded because of the potential of such drugs to alter platelet function.[34] The study protocol and related informed consent document for owners were approved by the institution's Clinical Studies Review Committee.

Data collection

Blood samples for measurement of PCV, platelet count, serum total protein concentration, and thromboelastography parameters were obtained at the time of anesthetic induction (day 0) and 2, 7, and 14 days following splenectomy. All samples were obtained by cephalic, saphenous, or jugular venipuncture with 21-gauge needles or from freshly placed IV catheters. Samples were placed in EDTA collection tubes (PCV and platelet count), collection tubes without anticoagulants (serum total protein concentration), or 3.2% sodium citrate collection tubes (thromboelastography). Samples for platelet counts were stored at 2° to 8°C, and samples for thromboelastography were stored at room temperature (approx 21°C [69.8°F) until analysis. The timing of sample collection was chosen on the basis of the previously described progression of postsplenectomy thrombocytosis in people.[19]

Results of histologic evaluation were obtained for the splenic mass and any other tissues biopsied or resected at the time of surgery. The largest diameter of the splenic mass and the volume of any abdominal fluid were recorded during surgery.

Platelet counts and thromboelastography

Platelet counts were performed with an automated analyzer[a] between 1 and 18 hours after blood sample collection. Samples collected on day 0 had variable storage times because these samples were occasionally collected at night or on weekends when clinical pathology services were unavailable. At later assessment points, all samples were collected during the day and were analyzed within 1 to 3 hours after collection. Dogs were considered to have thrombocytosis if the platelet count was > 486 × 10^3/µL.

Thromboelastography was performed by trained clinicians or technicians between 30 minutes and 2 hours after sample collection. A hemostasis analyzer[b] and kaolin activation were used, and testing was performed at 37°C. Blood samples obtained from an IV catheter or from dogs receiving anticoagulant treatment were placed in cups containing heparinase to negate the effect of any residual heparin prior to thromboelastography. Recorded parameters included R, K, α, MA, and G. Values for G were calculated by the analyzer software with the following formula: G = (5,000 × MA/[100 – MA])/1,000. The reference intervals for all thromboelastography parameters were those that had been previously established at our institution by use of blood samples from 76 clinically normal dogs. Any K value below the lower reference limit or any α, MA, or G value above the upper reference limit was considered to be evidence of hypercoagulability.[35] The R value was not considered in statistical analyses because kaolin-activated thromboelastography had been performed.

Statistical analysis

The paired Student *t* test was used to determine whether mean platelet counts and thromboelastography values at each assessment point were significantly different from those on day 0. Proportions of dogs with thrombocytosis and thromboelastography values suggestive of hypercoagulability were compared among assessment points with the χ^2 test. Relationships between platelet counts and thromboelastography values at each assessment point were examined by means of linear regression analysis, in which Pearson correlations (*r*) were determined.

Relationships between selected preoperative and perioperative clinical variables and postoperative platelet counts and thromboelastography values were evaluated by use of general linear models. Clinical variables considered in these models included PCV, serum total protein concentration and platelet counts on day 0, whether hemoperitoneum was present (yes or no), volume of any abdominal fluid, whether the splenic mass was benign or malignant, diameter of the splenic mass, and whether a perioperative transfusion was given. For dogs that received transfusions prior to blood sample collection on day 0 and again before sample collection on day 2, general linear model comparisons to dogs that received no transfusions were made at each assessment point. For dogs that received transfusions after blood sample collection on day 0 but before sample collection on day 2, comparisons to dogs that received no transfusions were made for days 2, 7, and 14. Whether fluid resuscitation was administered was not considered as a candidate clinical variable because all dogs received IV fluid therapy on admission to the hospital.

For all analyses, values of $P < 0.05$ were considered significant. All analyses were conducted with the aid of statistical software.[c]

Results

Animals

Thirty-four dogs with a median age of 11.0 years (range, 6.7 to 16.5 years) were included in the study. Dogs were classified as mixed-breed dogs (n = 10)

and Labrador Retrievers (6), German Shepherd Dogs (3), Beagles (2), and various other breeds (1 each). Nineteen dogs were castrated males, 14 were spayed females, and 1 was a sexually intact male.

Hemoperitoneum and splenic masses

Seventeen (50%) dogs were confirmed to have hemoperitoneum at the time of splenectomy, and the remaining 17 (50%) dogs had no hemoperitoneum. Findings for the 33 dogs in which histologic evaluation was performed indicated that the splenic mass in 18 (55%) dogs was malignant and in 15 (45%) dogs was benign. Malignant masses included hemangiosarcoma (n = 15), histiocytic sarcoma (1), splenic stromal sarcoma (1), and T-cell lymphoma (1). Benign masses included splenic hematoma (n = 6), primary infarction (2), myelolipoma (2), and fibrohistiocytic nodule, lymphoid hyperplasia, hematoma with extramedullary hematopoiesis and secondary infarction, lymphoid hyperplasia with extramedullary hematopoiesis, and splenic hyperplasia with necrosis (1 each).

Surgeries and related findings

Twenty (59%) dogs underwent additional procedures at the time of splenectomy, and 5 of these dogs underwent multiple additional procedures.

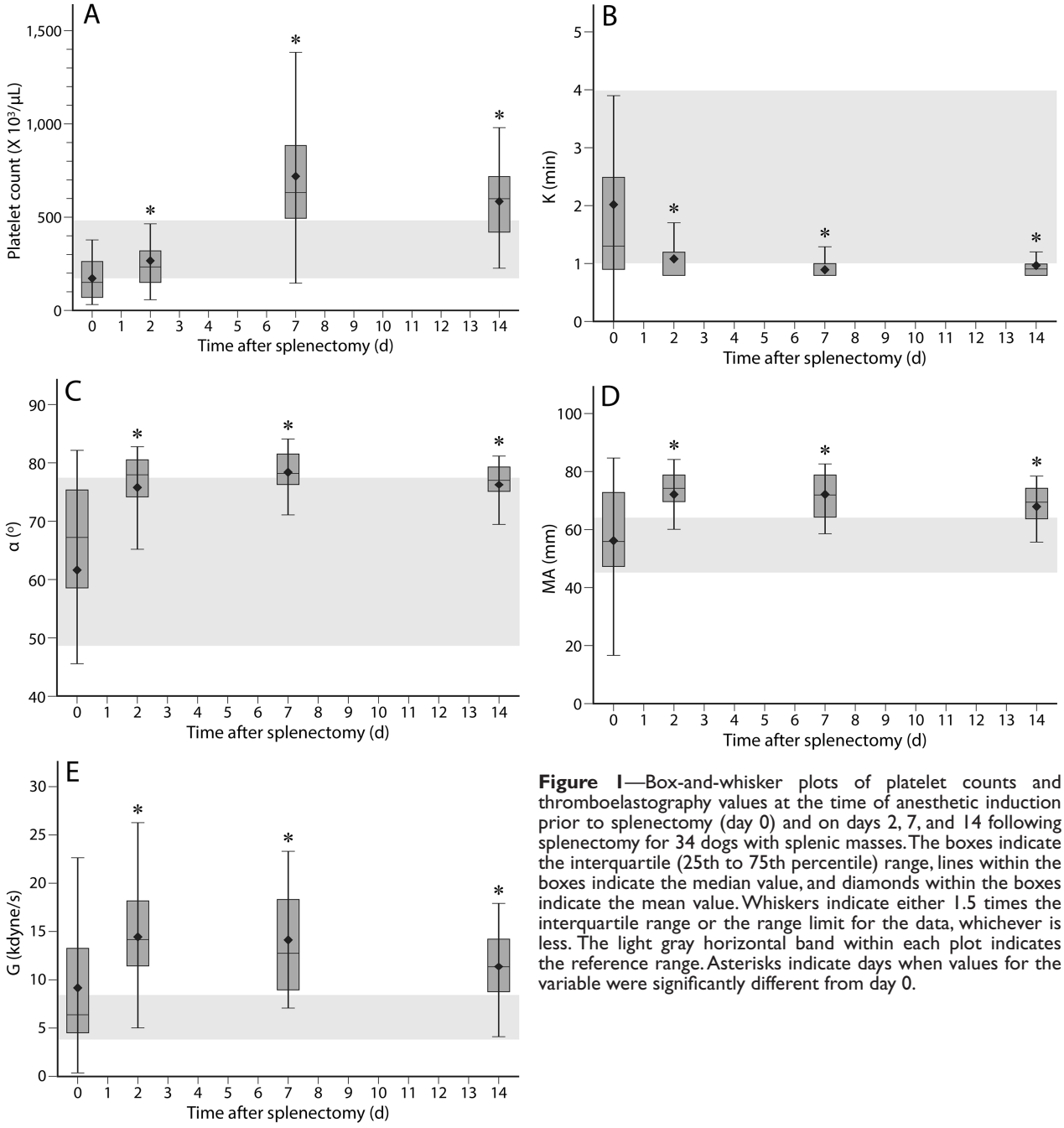

Figure 1—Box-and-whisker plots of platelet counts and thromboelastography values at the time of anesthetic induction prior to splenectomy (day 0) and on days 2, 7, and 14 following splenectomy for 34 dogs with splenic masses. The boxes indicate the interquartile (25th to 75th percentile) range, lines within the boxes indicate the median value, and diamonds within the boxes indicate the mean value. Whiskers indicate either 1.5 times the interquartile range or the range limit for the data, whichever is less. The light gray horizontal band within each plot indicates the reference range. Asterisks indicate days when values for the variable were significantly different from day 0.

These procedures included prophylactic gastropexy (n = 5), partial liver lobectomy (2), gastrotomy (1), and biopsies of the liver (14), intra-abdominal lymph node (1), omentum (1), falciform fat (1), peritoneum (1), or diaphragm (1). Six of the 15 dogs with hemangiosarcoma had histologically confirmed metastases to other organs, including the liver (n = 6) and the omentum, peritoneum, and diaphragm (1 each). The dog with splenic T-cell lymphoma also had histologically confirmed lymphoma in both the liver and a hepatic lymph node.

Other treatments

Both dogs with primary splenic infarction were treated with enoxaparin sodium[d] and clopidogrel bisulfate[e] at the discretion of their attending veterinarians. The medications were first administered after blood sample collection on day 7 and prior to sample collection on day 14 and continued through the end of the 14-day follow-up period.

Eleven dogs received packed RBC transfusions. Five dogs received transfusions before sample collection on day 0 and again before collection on day 2. Six dogs received transfusions after blood sample collection on day 0 but before collection on day 2.

Platelet counts and thromboelastography

Complete data sets on platelet counts and thromboelastography variables were obtained for 26 of 34 (76%) dogs. Owners of 4 of the 8 remaining dogs declined further participation before all blood samples were obtained. Of the other 4 dogs, 3 were euthanized because of documented or suspected progression of their malignant disease, and 1 was euthanized because of suspected intracranial thromboembolism (none of the owners consented to necropsy). Therefore, of 136 total possible platelet counts (34 dogs at 4 assessment points), 119 (87.5%) were obtained; missing values were noted on days 2 (n = 1), 7 (8), and 14 (8). Of 136 total possible thromboelastograms, 116 (85.3%) were performed; missing thromboelastograms were noted on days 0 (n = 1), 2 (3), 7 (7), and 14 (9). Two dogs had missing values for K on day 0; no other individual thromboelastographic values were missing. Platelet counts and thromboelastography values at each assessment point were graphically displayed **(Figure 1)**. Mean platelet counts were significantly ($P < 0.001$) higher at all postoperative assessment points than on day 0 **(Table 1)**. Twenty-two of 34 (65%) dogs were thrombocytopenic (ie, platelet count < 173 X 10^3/µL) on day 0. Mean values for K were significantly ($P < 0.001$) lower at all postoperative assessment points than on day 0, and mean values for the remaining thromboelastography parameters were significantly ($P < 0.001$) higher at all postoperative assessment points than on day 0.

Proportions of dogs with thrombocytosis and thromboelastography values suggestive of hypercoagulability as well as comparisons of these proportions over time were summarized **(Table 2)**. Correlations between platelet counts and thromboelastography parameters at each assessment point were also summarized **(Table 3)**. At each assessment point, platelet

Table 1—Mean (SD) PCV, platelet count, and thromboelastography values at the time of anesthetic induction prior to splenectomy (day 0) and days 2, 7, and 14 following splenectomy for 34 dogs with splenic masses.

Variable	Reference interval	Day 0	Day 2	Day 7	Day 14
PCV (%)	39–55	30.7 (9.5)	31.8 (7.4)	37.1 (5.1)	39.1 (4.4)
Platelet count (X 10^3/µL)	173–486	167.9 (141.2)	260.4 (187.8)*	715.9 (332.0)*	582.2 (197.0)*
K (min)	1–4	2.02 (2.0)	1.09 (0.5)*	0.90 (0.2)*	1.00 (0.28)*
α (°)	48–77	61.8 (19.9)	75.9 (6.6)*	78.5 (3.7)*	76.4 (3.9)*
MA (mm)	45–65	56.1 (20.8)	72.0 (8.9)*	71.9 (7.4)*	67.7 (8.2)*
G (kdyne/s)	3.9–8.4	9.1 (6.7)	14.4 (5.4)*	14.0 (5.1)*	11.4 (3.8)*

*The mean value at this assessment point differs significantly ($P < 0.001$) from the mean for the same variable on day 0.

Table 2—Proportion (%) of the dogs of Table 1 with thrombocytosis* or thromboelastography values suggestive of hypercoagulability at various assessment points.

Variable	Day 0	Day 2	Day 7	Day 14	*P* value†
Thrombocytosis	1/34 (3)	2/33 (6)	21/26 (81)	18/26 (69)	< 0.001
K	9/31 (29)	20/31 (65)	20/27 (74)	15/25 (60)	0.008
α	7/33 (21)	19/31 (61)	19/27 (70)	13/25 (52)	< 0.001
MA	13/33 (39)	26/31 (84)	21/27 (78)	18/25 (72)	0.001
G	15/33 (45)	26/31 (84)	22/27 (81)	20/25 (80)	0.001
≥ 1 thromboelastography parameter suggestive of hypercoagulability	15/33 (45)	26/31 (84)	24/27 (89)	21/25 (84)	< 0.001

*Dogs were considered to have thrombocytosis if the platelet count was > 486 X 10^3/µL. †*P* values pertain to comparison of proportions over time.

See Table 1 for remainder of key.

Table 3—Pearson correlations (*r*) between platelet counts and thromboelastography parameters at various assessment points for the dogs of Table 1.

	Day 0		Day 2		Day 7		Day 14	
Parameter	***r***	***P* value**	***r***	***P* value**	***r***	***P* value**	***r***	***P* value**
K	–0.41	0.02	–0.41	0.02	–0.49	0.01	–0.40	0.02
α	0.61	< 0.001	0.52	0.003	0.59	0.002	0.57	0.003
MA	0.66	< 0.001	0.60	< 0.001	0.37	0.06	0.43	0.03
G	0.72	< 0.001	0.70	< 0.001	0.37	0.06	0.44	0.03

Decreases in K or increases in α, MA, or G were considered supportive of hypercoagulability.

counts were negatively correlated with K values and positively correlated with α, MA, and G values. These correlations were significant, except for the correlations between platelet counts and MA and G on day 7.

Analyses of the effects of the selected pre- and perioperative clinical variables on postoperative platelet counts and thromboelastography parameters revealed that platelet counts on day 2 (but not on days 7 or 14) were positively correlated with platelet counts on day 0 ($P = 0.001$). No significant associations were identified between postoperative platelet counts and other preoperative clinical variables. Among thromboelastography parameters, values for MA and G on day 7 were significantly ($P = 0.008$ and $P = 0.003$, respectively) higher for dogs with (vs without) hemoperitoneum and were significantly ($P = 0.03$ for both parameters) higher for dogs with a malignant (vs benign) splenic mass. On day 14, there was a significant ($P = 0.01$) positive correlation between values for K and PCV on day 0. No other significant associations between clinical variables and postoperative thromboelastography parameters were identified.

Discussion

Results of the present study indicated that thrombocytosis and thromboelastographic evidence of hypercoagulability were common during the first 2 weeks after splenectomy in dogs with splenic masses. If present, hypercoagulability could increase the risk for development of postsplenectomy thrombotic conditions such as PST and PTE.

Wide variability in platelet counts was observed during the study period. Sixty-five percent of dogs had thrombocytopenia on day 0 (at the time of anesthetic induction prior to splenectomy), and although mean platelet count was beginning to increase on day 2, most (94%) dogs had low or normal rather than high platelet counts at that assessment point. By day 7, thrombocytosis was evident in > 80% of dogs, and 5 dogs had platelet counts > 1,000 × 10^3/μL. Mean platelet count and the proportion of dogs with thrombocytosis appeared to be decreasing by day 14; however, the amount of time required for complete resolution of postsplenectomy thrombocytosis remains unknown. The lack of significant associations between platelet counts on days 7 and 14 and platelet counts on day 0 or the presence, absence, or volume of hemoperitoneum suggested that the thrombocytoses observed at these later assessment points may have resulted from the splenectomy rather than from an ongoing reaction to initial thrombocytopenia.

Thromboelastographic values also varied widely in the present study. At each assessment point, platelet counts were negatively correlated with K values and positively correlated with α, MA, and G values, suggesting that increasing platelet counts were a cause of thromboelastographic evidence of hypercoagulability. Although most (≥ 84%) dogs had at least 1 thromboelastography parameter suggestive of hypercoagulability at all postoperative assessment points, smaller proportions of dogs also had thromboelastographic evidence of hypercoagulability on day 0. Thrombocytosis was comparatively uncommon on days 0 and 2, suggesting that early in the follow-up period, thromboelastographic evidence of hypercoagulability was driven largely by factors other than platelet counts. These factors may have included the presence of a large benign or malignant splenic mass, metastases, endothelial damage, blood loss, administration of blood products, or major abdominal surgery, each of which could have contributed to systemic inflammation and an associated risk of hypercoagulability.[15,16,31] By days 7 and 14, the effects of these factors were likely decreasing, and thromboelastographic findings suggestive of hypercoagulability at these assessment points were likely attributable largely to thrombocytosis. Measurement of markers of inflammation that may correlate with thromboelastographic hypercoagulability in dogs,[31] such as plasma fibrinogen and C-reactive protein concentrations, was beyond the scope of the study. The occasional thromboelastographic evidence of hypercoagulability on day 0 suggested the possibility that some dogs with splenic masses could have early PST on admission to the hospital and that assessment of the portal system with color flow Doppler mode during the initial abdominal ultrasonographic examination should be considered when feasible.

Few significant associations were identified between the selected preoperative clinical variables and postoperative thromboelastography parameters, although the power of these analyses was likely limited by small sample sizes. On day 7, values of MA and G were higher for dogs with (vs without) hemoabdo-

men and for dogs with malignant (vs benign) masses. Blood loss and endothelial malignancy are possible risk factors for hypercoagulability in people[15] and dogs[16]; however, the lack of an association between the presence or absence of hemoabdomen or splenic malignancy and MA or G on day 2 suggested that the significant associations on day 7 may have been attributable to type I error. The finding that K was positively correlated with preoperative PCV on day 14 but not at earlier assessment points suggested that this result may also have been attributable to type I error.

Interpretation of the thromboelastography data obtained in the present study is complicated by the variability in PCVs observed during the study follow-up period. Previous studies[28–31] involving healthy dogs have demonstrated that thromboelastography parameters suggestive of hypercoagulability can be artifactual when a low PCV is present. In the present study, mean PCV was below the lower reference limit on days 0 and 2, likely owing to intra-abdominal blood loss; closer to the lower reference limit on day 7; and within reference limits on day 14. However, mean thromboelastography values were within or close to reference limits on day 0, when mean PCV was lowest, and were commonly suggestive of hypercoagulability at the postoperative assessment points, whereas PCV was approaching or within reference limits. This finding, combined with the presence of postoperative thrombocytosis and several other possible risk factors for hypercoagulability in the study dogs, supported the conclusion that the observed thromboelastographic evidence of hypercoagulability was not solely an artifact resulting from anemia. Additional research will be required to determine how thromboelastographic evidence of hypercoagulability should be interpreted in dogs with low PCVs.

The clinical implications of our findings for the postoperative monitoring of dogs undergoing splenectomy are currently unclear. First, no combination of thromboelastography findings has been identified that reliably indicates dogs are hypercoagulable.[35] For this reason, we elected to report results for each thromboelastography parameter, as has been recommended.[35] Second, no relationship has been established between thromboelastographic evidence of hypercoagulability and development of thrombotic conditions in dogs. Although significant relationships between thromboelastography parameters and postoperative thrombotic complications have been confirmed in human surgical patients,[36,37] a recent study[38] of critically ill dogs failed to establish an association between evidence of thrombosis at necropsy and thromboelastography parameters measured during the previous 7 days. The large proportions of dogs with postoperative thrombocytosis and thromboelastography parameters suggestive of hypercoagulability in the present study, combined with the comparatively low incidence of clinically apparent thromboses in dogs undergoing splenectomy,[5–7] imply that other important factors are involved in the development of thromboses. Such factors may include the size of the splenic mass or whether it is benign or malignant, severity of endothelial damage and any intra-abdominal hemorrhage, and patient age and comorbidities. Additional research is needed to determine whether platelet counts and thromboelastography parameters can be used to assess the risk of postsplenectomy thromboses or guide decisions regarding institution of thrombotic prophylaxis in dogs. Until such information becomes available, veterinarians should be aware that CBCs performed in dogs for any reason during the first few weeks following splenectomy may reveal elevated platelet counts and that the clinical implications of such increases are currently unknown.

The present study had several limitations in addition to those already described. Because our intent was to investigate postoperative platelet counts and thromboelastographic parameters in a sample representative of the general population of dogs undergoing splenectomy, we did not attempt to control for all clinical variables that may have influenced our results. Platelet counts and thromboelastography values were likely influenced to some degree by variability in splenic mass types, presence and severity of hemoperitoneum, comorbidities, concurrent medications, fluid resuscitation protocols, administration of thrombotic prophylaxis and blood transfusions, sedation and anesthesia protocols, and surgeries performed in addition to splenectomy. Platelet clumping may have caused variable degrees of reduction in platelet counts. Missing data, which was most common on days 7 and 14, likely had a modest effect on our results. Variability in blood collection sites,[39,40] the interval between blood sample collection and thromboelastography,[40,41] and thromboelastography operators[40] may have influenced the thromboelastography results. Postoperative blood samples were collected at intervals of several days, potentially limiting our ability to identify brief episodes of thrombocytosis or thromboelastographic evidence of hypercoagulability, and the limited duration of the follow-up period prevented an assessment of the time required for thrombocytosis and thromboelastography values suggestive of hypercoagulability to fully resolve. In-depth investigations of the causes of postsplenectomy thrombocytosis and thromboelastographic evidence of hypercoagulability were beyond the scope of the study and warrant additional research.

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Footnotes

a. Advia 120, Siemens Corp, Munich, Germany.
b. Haemoscope 5000, Haemonetics Corp, Niles, Ill.
c. SAS, version 14.1, SAS Institute Inc, Cary, NC.
d. Lovenox, Sanofi-Aventis, Bridgewater, NJ.
e. Plavix, Sanofi-Aventis, Bridgewater, NJ.

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