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RETROSPECTIVE STUDY

Retrospective evaluation of fresh platelet concentrate administration in dogs: Patient characteristics, outcomes, and transfusion practices in 189 transfusion episodes (2008–2019)

Laurence M. Saint-Pierre DVM[1] | Kate S. Farrell DVM, DACVECC[2] | Kate Hopper BVSc, PhD, DACVECC[2] | Krystle L. Reagan DVM, PhD, DACVIM[3]

[1]William R. Pritchard Veterinary Medical Teaching Hospital, University of California, Davis, California, USA

[2]Department of Veterinary Surgical and Radiological Sciences, University of California, Davis, California, USA

[3]Department of Medicine & Epidemiology, School of Veterinary Medicine, University of California, Davis, California, USA

Correspondence
Laurence M. Saint-Pierre, William R. Pritchard Veterinary Medical Teaching Hospital, University of California, Davis, One Garrod Ave, Davis, CA 95616, USA.
Email: lmsaintpierre@ucdavis.edu

Abstract

Objective: To describe patient characteristics, underlying disease processes, clinical outcomes, transfusion dose and type (therapeutic or prophylactic), platelet count changes, and adverse events associated with platelet concentrate (PC) administration in dogs.

Design: Retrospective study.

Setting: University teaching hospital.

Animals: A total of 149 dogs, representing 189 PC transfusion episodes.

Interventions: None.

Measurements and Main Results: In this population, 39 of 149 dogs (26.2%) were diagnosed with primary immune-mediated thrombocytopenia, 22 of 149 (14.8%) had decreased bone marrow production, 12 of 149 (8.0%) received PC during a massive transfusion, 3 of 149 (2.0%) had congenital thrombocytopathia, 59 of 149 (39.6%) had severe thrombocytopenia of other causes, and 14 of 149 (9.4%) underwent transfusion for miscellaneous causes without a documented severe thrombocytopenia. In 117 of 149 dogs (78.5%), >1 site of hemorrhage was noted. The most common sites of hemorrhage were the gastrointestinal (GI) tract in 89 of 149 (59.7%) and the skin in 78 of 149 (52.3%). Overall survival to discharge was 59.1% (88/149). The median PC dose was 0.8 units per 10 kg of body weight per transfusion episode (range: 0.2–6.7). Of 189 episodes, 29 of 189 (15.7%) were prophylactic, and 158 of 189 (83.6%) were therapeutic. For 99 of 189 transfusion episodes, paired pre- and postplatelet counts were available within 24 hours. The median platelet count change was 5.0 × 10^9/L (5000/μL; range: −115 × 10^9/L to 158 × 10^9/L [−115,000 to 158,000/μL]); the post-transfusion platelet count was significantly higher than pretransfusion ($P < 0.0001$). The increase in platelet count after transfusion was greater in the prophylactic group

Abbreviations: BSA, body surface area; DEA, dog erythrocyte antigen; FWB, fresh whole blood; GI, gastrointestinal; IMTP, immune-mediated thrombocytopenia; PC, platelet concentrate; pRBCs, packed RBCs.

than the therapeutic group ($P = 0.0167$). Transfusion reactions were suspected during 2 of 168 episodes (1.2%).

Conclusions: Immune-mediated thrombocytopenia was the most common disease process that resulted in PC transfusion. PC was more frequently administered to animals with active hemorrhage rather than prophylactically, and most dogs had evidence of hemorrhage in multiple organ systems, particularly the GI tract and skin. PC transfusions typically appeared safe, and the median platelet count increased after transfusion.

KEYWORDS
active bleeding, canine, platelet, transfusion

1 | INTRODUCTION

Platelet transfusions can be administered in an effort to prevent hemorrhage or treat active bleeding in patients with thrombocytopenia or thrombocytopathia. In veterinary medicine, most platelet transfusions are therapeutic, being administered to patients with uncontrolled hemorrhage, particularly in cases of CNS, pulmonary, or cardiac hemorrhage.[1] In contrast, people more commonly receive platelet transfusions as a prophylactic measure to prevent hemorrhage in those at increased risk of spontaneous bleeding due to their disease process or invasive procedures.[2–4] In human medicine, published guidelines for prophylactic and therapeutic platelet transfusion are available, with criteria for administration and target platelet counts associated with a concomitant decrease in hemorrhage risk.[3,4] There is a paucity of studies describing platelet transfusions in dogs with thrombocytopenia or thrombocytopathia; therefore, platelet transfusion thresholds, efficacy, and adverse events have not been well described.

Platelet-containing products available for dogs include fresh whole blood (FWB), fresh platelet products such as platelet-rich plasma and platelet concentrate (PC), cryopreserved platelets, and lyophilized platelets. Large volumes of FWB are required to increase the platelet count significantly, and patients who do not require red blood cells and plasma may be at risk for polycythemia, circulatory overload, and additional transfusion reactions.[7] PC offers the benefit of containing a higher concentration of platelets as compared to FWB; however, this product is not widely accessible and has a short shelf life. Cryopreserved and lyophilized platelet products offer longer storage times and immediate availability, but the duration of survival and function of transfused platelets may not equal those observed with PC, which remains the product of choice for use in people.[1] Despite the recognized utility of PC, large-scale studies evaluating its clinical use in dogs are lacking.[5,6]

The objective of this study was to describe the clinical characteristics, underlying disease processes, and clinical outcomes of dogs receiving PC transfusions. A secondary objective was to describe the transfusion dose, classification of prophylactic or therapeutic administration, platelet count changes posttransfusion, and adverse events associated with PC administration.

2 | METHODS

2.1 | Data collection

All canine PC transfusions performed at the William R. Pritchard Veterinary Medical Teaching Hospital at the University of California, Davis, between January 2008 and December 2019 were retrospectively evaluated. The electronic medical records of patients that were billed and received ≥1 PC transfusions were reviewed, and the following patient information was retrieved: breed, sex, age, weight, and clinical diagnosis assigned by the attending clinician.

Pretransfusion platelet count was documented in the 24 hours preceding transfusion, and posttransfusion platelet count was documented in the 24 hours after PC transfusion. The platelet count closest in time to the transfusion was recorded if >1 platelet count was performed in the 24 hours before or after a transfusion. The platelet change was defined as the difference between the pre- and posttransfusion platelet counts; the value was positive if the patient's platelet count increased posttransfusion and negative if the platelet count decreased posttransfusion. All platelet counts were measured with a hematology analyzer[a] or estimated via blood smear evaluation by the primary clinician or the reference laboratory.[7] For automated platelet counts, a blood smear was reviewed to assess for platelet clumping and the presence of macroplatelets. If clumps were present, the value recorded represents the lowest potential platelet count. Thrombocytopenia was defined as a platelet count $<150 \times 10^9$/L (150,000/μL),[8] and severe thrombocytopenia was defined as a platelet count of $\leq 50 \times 10^9$/L (50,000/μL).[7]

Additional data retrieved from the medical record included sites of hemorrhage, based on patient examination, noted on diagnostic tests, or observed on necropsy. Patients with documented hemorrhage at any point during their visit were included in the following categories: CNS, pulmonary, gastrointestinal (GI), ocular, epistaxis, cutaneous,

urinary tract, intracavitary (abdominal, pleural, or pericardial), or other. Patients were included in multiple categories if >1 site of hemorrhage was documented. Further information recorded included the pretransfusion HCT or PCV (the closest value to the time of transfusion), dog erythrocyte antigen (DEA) 1 blood type, transfusion rate, adverse events during transfusion (as described in the following section), number and types of other blood products administered, discharge status for the visit (alive, euthanized, or died in hospital), and concurrent administration of vincristine.[9]

2.2 | Disease mechanisms resulting in platelet transfusion

For each dog, the disease process that necessitated a PC transfusion was assigned to 1 of the following categories: primary immune-mediated thrombocytopenia (IMTP), decreased bone marrow production, congenital thrombocytopathia, massive transfusion, severe thrombocytopenia of other cause, and miscellaneous causes without a documented severe thrombocytopenia. Each patient was only assigned to 1 category.

Primary IMTP was diagnosed in patients with a platelet count $<40 \times 10^9$/L (40,000/μL) at the time of presentation and no concurrent illnesses known to be associated with thrombocytopenia, including malignant neoplasia, bone marrow disease, severe systemic infections, or other immune-mediated diseases.[10–12] Exclusion of these illnesses was determined based on physical examination, CBC, serum chemistry analysis, serology for infectious disease as indicated, and thoracic and abdominal imaging. Patients vaccinated or treated with medications[14] associated with secondary IMTP in the preceding 42 days were excluded from this category. Patients were only included in the IMTP category for this study if they had completed the diagnostics as mentioned previously. Patients that did not meet the full diagnostic workup were included in the category of severe thrombocytopenia of other causes.

Patients were considered to have decreased bone marrow production if their platelet count was below the laboratory reference range of 150×10^9/L (150,000/μL) and if an underlying cause of bone marrow suppression was documented. These causes included previous exposure to cytotoxic drugs, including chemotherapeutics[14–17] or estrogens.[18] Dogs were also classified in this category if infiltrative bone marrow disease, including neoplasia, myelodysplastic syndromes, megakaryocytic aplasia, or hypoplasia, was diagnosed on cytological or histological bone marrow examination.

The diagnosis of congenital thrombocytopathia included patients identified with intrinsic platelet defects, such as Glanzmann thrombasthenia[19] or Scott syndrome,[20] based on genetic testing, ELISA, or flow cytometry.

Massive transfusion was defined as transfusion of a volume of whole blood or blood components greater than the patient's estimated blood volume within a 24-hour period, or replacement of half of the patient's estimated blood volume within a 3-hour period.[21] Total blood volume for dogs was estimated to be 90 mL/kg. Animals receiving blood products as part of therapeutic plasma exchange were not included in this category.

Severe thrombocytopenia of other causes included patients with an initial platelet count $\leq 50 \times 10^9$/L (50,000/μL) that did not fulfill criteria for other categories. This cutoff was chosen due to the increased concern for hemorrhage during invasive procedures in these patients.[3,22]

Patients that did not fulfill the criteria for the categories listed previously or that received platelets without a documented platelet count were included in a category known as miscellaneous causes without a documented severe thrombocytopenia.

2.3 | Platelet concentration preparation

Preparation of PC was performed according to University of California, Davis Blood Bank standard protocols. Blood was collected from healthy canine donors that met the UC Davis Canine Community Blood Donor Program enrollment criteria. A physical examination, CBC, serum chemistry analysis, and relevant infectious disease testing were performed yearly for each donor. After standard aseptic preparation of the skin, a jugular venipuncture was performed, and approximately 450 mL of whole blood was collected into a polyvinyl chloride bag containing a citrate phosphate dextrose solution.[b] Whole blood was centrifuged[c] ($1000 \times g$, 24°C) for 6 minutes and 45 seconds (1 min, 45-s acceleration, 5-min run time). This platelet-rich plasma was isolated in a satellite bag[d] and centrifuged ($2000 \times g$, 24°C) for 12 minutes (2-min acceleration, 10-min run time). Most of the supernatant was expressed into a transfer bag, leaving approximately 60 mL of plasma in the satellite bag along with the platelet pellet. After both centrifugation protocols, the PC was kept at room temperature for 1 hour and then gently agitated[e] to resuspend the platelets in the remaining plasma before subsequently being stored at 22°C[f] with constant gentle agitation. Units were generally stored for a maximum of 5 days prior to disposal if they were unused. When available, characteristics of PC units, including platelet count, storage time, platelet culture, and DEA 1 blood type of the donor, were retrieved from the electronic medical record system.

2.4 | PC transfusion

For this study, a transfusion episode was defined as all PC units transfused to a patient within a 24-hour interval beginning with the first PC unit transfused. If additional PC units were transfused beyond the first 24-hour interval, a new transfusion episode would begin at the start of the next PC transfusion. All transfusion episodes that occurred during the study period were included. The PC units were administered using an Hemo-Nate filter[g] while under constant agitation with a commercial rocker, and this was followed with a 0.9% saline flush. PC units were transfused at a rate of 3 mL/kg/h for 10 minutes, which was increased to 10 mL/kg/h until completion in the absence of any transfusion reaction, unless the primary clinician specified a different rate

for administration. Patients were monitored for the presence of transfusion reactions such as an increase in rectal temperature of >1°C, tachycardia, tachypnea or respiratory distress, nausea, hypersalivation, acute vomiting or diarrhea, urticaria, facial swelling, pruritus, or other concerns noted during the transfusion episode.[2]

In this study, a therapeutic transfusion was defined as a PC transfusion episode administered to a patient to reduce current hemorrhage. Prophylactic transfusion referred to the administration of PC to patients without symptomatic blood loss at the time of PC transfusion; these patients were considered by the primary clinician (and stated in the medical record) to be at increased risk of spontaneous bleeding due to their disease process, thus necessitating a PC transfusion before an impending invasive procedure or discharge home. If a patient received both a therapeutic and a prophylactic transfusion in the same transfusion episode (ie, in the same 24-h period), that transfusion episode was considered therapeutic for the study purposes. If retrospective evaluation of the medical record could not determine whether a transfusion episode should be characterized as therapeutic or prophylactic, the type of transfusion was listed as unknown.

2.5 | Statistics

Population demographics are presented using nonparametric descriptive statistics. Conditions for normality were tested using the D'Agostino and Pearson test and the Shapiro–Wilk test. The data did not meet conditions for normality, so nonparametric testing was performed. A chi-squared test was used to compare categorical variables. Pre- versus posttransfusion platelet counts were compared between groups, and the degree of platelet count changes was compared between the therapeutic and prophylactic groups using a nonparametric, paired Wilcoxon rank-sum test.[h] Significance was set at $P < 0.05$.

3 | RESULTS

3.1 | Patient characteristics and underlying disease mechanisms

Over the 11-year study period, 189 platelet transfusion episodes were performed in 149 dogs. Of these 149 dogs, there were 77 neutered females (51.7%), 50 neutered males (33.6%), 9 entire females (6.0%), and 13 entire males (8.7%). The median age at presentation for all dogs was 8.1 years (range: 0.5–15.0). The median weight on presentation was 20.8 kg (range: 1.7–85.5). The most commonly represented breeds included the German Shepherd Dog (10/149 [6.7%]), Labrador Retriever (10/149 [6.7%]), Golden Retriever (7/149 [4.7%]), and mixed breed dogs (28/149 [18.8%]).

Of the 149 dogs in this population, 39 (26.2%) were diagnosed with primary IMTP, 22 (14.8%) were diagnosed with decreased bone marrow production, 3 (2.0%) had congenital thrombocytopathia, 12 (8.0%) received PC as part of a massive transfusion, 59 (39.6%) were diagnosed with severe thrombocytopenia of other causes, and 14 (9.4%) underwent PC transfusion for miscellaneous causes without a documented severe thrombocytopenia. Within the category of severe thrombocytopenia of other causes, the most commonly reported diseases were secondary IMTP (28/59 [47.4%]) and sepsis (12/59 [20.3%]). The miscellaneous category included patients with neoplastic disease (6/14 [42.8%]), most of which received a PC transfusion perioperatively (5/6 [83.3%]), and patients with known ingestion of aspirin or anticoagulants (4/14 [28.5%]). All patients in the massive transfusion category underwent surgical intervention for neoplastic disease (6/12 [50.0%]), cardiac surgery (3/12 [25.0%]), or liver surgery (3/12 [25.0%]).

In 117 of 149 dogs (78.5%), >1 anatomic site of hemorrhage was noted. The most common sites were the GI tract in 89 (59.7%) and the skin in 78 (52.3%). Of the 39 dogs with primary IMTP, the most commonly reported sites of hemorrhage were the skin in 32 (82.0%) and the GI tract in 31 (79.4%). The skin and GI tract were also the most common sites for hemorrhage in patients with decreased bone marrow production (12/22 [54.5%] for each site) and severe thrombocytopenia of other causes (31/59 [52.5%] and 34/59 [57.6%], respectively). Cavitary bleeding was the most common site of hemorrhage in dogs that received a massive transfusion (10/12 [83.3%]) and those with miscellaneous causes without a documented thrombocytopenia (9/14 [64.3%]). Only 9 of 149 patients (6.0%) experienced no known hemorrhage before platelet transfusion. Table 1 summarizes the sites of hemorrhage for each underlying disease mechanism.

The overall survival to discharge was 59.1% (88/149). The survival for patients with primary IMTP was 66.7% (26/39). Dogs with severe thrombocytopenia of other causes had the lowest survival, at 52.5% (31/59) (Table 1). Among nonsurvivors, 72.1% (44/61) were euthanized, and the remaining 27.9% (17/61) died.

3.2 | PC transfusion episodes

In this study, 271 PC units were transfused, of which 130 were DEA 1 positive and 141 were DEA 1 negative. For 250 units, a platelet count was measured. The median platelet concentration in the transfused units was 870×10^9/L (870,000/μL; range: 442×10^9/L to 2042×10^9/L [442,000–2,042,000/μL]), which represents a median of approximately 52×10^9 platelets transfused per unit. The median storage time of the PC units was 3 days (range: 0–7). In this study, 5 units (4 DEA 1 negative and 1 DEA 1 positive) were transfused after being stored for >5 days. Information regarding platelet culture was available for 209 of 271 PC units. The culture was positive in 7 of 209 PC units (3%), with all of these units suspected of being contaminated during plating of the PC unit, as noted in the medical record by the laboratory and based on the organism identified and its growth pattern. Organisms isolated were *Bacillus* sp. (3/7), *Aspergillus* sp. (1/7), *Micrococcus* sp. (1/7), *Streptococcus viridans* (1/7), and coagulase-negative *Staphylococcus* sp. (1/7).

Patients received a median of 1.0 PC unit per transfusion episode (range: 0.5–6.0). The median PC dose was 0.8 units per 10 kg of body weight per transfusion episode (range: 0.2–6.7). Twenty dogs

TABLE 1 Sites of hemorrhage and survival rate for dogs undergoing platelet concentrate transfusions based on the underlying disease mechanism.

		All patients transfused (*N* = 149)	Primary IMTP (*N* = 39)	Massive transfusion (*N* = 12)	Decreased bone marrow production (*N* = 22)	Congenital thrombocytopathia (*N* = 3)	Severe thrombocytopenia of other causes (*N* = 59)	Miscellaneous causes *N* = 14
Site of hemorrhage	CNS	27	14	0	1	0	10	2
	GI	84	31	4	12	0	34	3
	Pulmonary	19	6	2	3	0	6	2
	Ocular	25	17	0	1	0	6	1
	Cutaneous	78	32	0	12	1	31	2
	Epistaxis	29	9	2	7	0	8	3
	Urinary	19	4	2	1	0	12	0
	Cavitary[a]	29	1	10	4	0	5	9
	Other[b]	27	5	3	4	1	13	2
	None	9	1	0	1	1	5	1
Survival (%)		59.1	66.7	58.3	54.5	100	52.5	57.1

Note: *N* is the number of dogs.
Abbreviations: GI, gastrointestinal; IMTP, immune-mediated thrombocytopenia.
[a]Cavitary bleeding includes abdominal, thoracic, and pericardial hemorrhage.
[b]Other includes pancreatic, heart, prostate, oral cavity, lymph node, and adrenal gland hemorrhage.

underwent >1 transfusion episode. One dog received 10 PC units over 39 days. The PC dose for each underlying disease mechanism is listed in Table 2.

Of the 189 PC transfusion episodes, 29 (15.7%) were considered prophylactic, whereas 158 (83.6%) were therapeutic transfusions. For 2 of these transfusion episodes, the reason for transfusion was not clearly expressed in the medical record. Therapeutic transfusions represented 94.0% (47/50) of the transfusion episodes for primary IMTP. In contrast, prophylactic transfusions were more commonly administered to patients with thrombocytopenia of other causes, such as suspected secondary IMTP (8/15) and sepsis (5/15) (Table 2).

A platelet count in the 24 hours before a PC transfusion was available in 172 of 189 transfusion episodes (91.0%). Of these, 10 of 172 platelet counts (5.8%) were obtained by blood smears by the primary clinician; the remainder were automated platelet counts and reviewed by a reference laboratory. For these 172 transfusion episodes, the median platelet count before transfusion was 8.0×10^9/L (8000/μL; range: 0.0–519 $\times 10^9$/L [0–519,000/μL]). The median platelet count was 14×10^9/L (14,000/μL; range: 2.0×10^9/L to 211×10^9/L [2000–211,000/μL]) prior to prophylactic transfusions and 8.0×10^9/L (8000/μL; range: 0.0–519 $\times 10^9$/L [0–519,000/μL]) prior to therapeutic transfusions. For 149 of 172 samples (86.6%), information regarding platelet clumping was available, and 14 of 149 samples (9.4%) were found to have clumps. An HCT or PCV was available in the 24 hours before transfusion for 185 of 189 episodes (97.9%). The median HCT (or PCV) was 21.0% (range: 7.0%–55.0%). A platelet count measured within 24 hours after a PC transfusion was available in 105 of 189 episodes (55.5%). For these 105 episodes, the median platelet count after transfusion was 16×10^9/L (16,000/μL; range: 2.0×10^9/L to 468×10^9/L [2000–468,000/μL]). For 61 of 105 samples, information regarding platelet clumping was available, and 12 of 61 samples (19.6%) had clumps. The laboratory data pre- and post-PC transfusion for each underlying disease mechanism are summarized in Table 2.

In 99 PC transfusion episodes, platelet counts measured both within the 24 hours before and after transfusion were available for comparison. The median platelet count change per transfusion episode was 5.0×10^9/L (5000/μL; range: -115×10^9/L to 158×10^9/L [−115,000 to 158,000/μL]), which corresponds to a median platelet count change of 4.0×10^9/L platelets per unit of PC transfused (4000/μL; range: -57.0×10^9/L to 158×10^9/L [−57,000 to 158,000/μL]). Compared to the pretransfusion platelet count, the posttransfusion platelet count was significantly higher in transfusion episodes with paired values ($P < 0.0001$). The difference was also significant in specific disease categories, including patients with primary IMTP ($P < 0.0001$), thrombocytopenia due to decreased bone marrow production ($P = 0.0008$), and thrombocytopenia of other causes ($P < 0.0001$). There were insufficient data to compare in the other disease categories. Table 3 summarizes the platelet count change for the underlying diseases with sufficient sample sizes for comparison.

Among these 99 transfusion episodes for which a platelet count was measured within the 24 hours before and after PC transfusion, 16 (16.2%) were prophylactic PC transfusions, 81 (81.8%) were therapeutic, and 2 were unknown. The median platelet count change per transfusion episode was $+17.0 \times 10^9$/L (17,000/μL; range: -4.0×10^9/L to 108×10^9/L [−4000 to 108,000/μL]) in patients receiving a prophylactic transfusion and 4.0×10^9/L (4000/μL; range: -115×10^9/L to 158×10^9/L [−115,000 to 158,000/μL]) in patients receiving a therapeutic PC transfusion. There was a significant increase in platelet count

TABLE 2 Patient characteristics and type of transfusion for 189 platelet concentrate transfusion episodes based on the underlying disease mechanism.

	All transfusion episodes ($N = 189$)	Primary IMTP ($N = 50$)	Massive transfusion ($N = 12$)	Decreased bone marrow production ($N = 38$)	Congenital thrombocytopathia ($N = 10$)	Severe thrombocytopenia of other causes ($N = 65$)	Miscellaneous causes ($N = 14$)
Number of PC units per transfusion episode	1.0 (0.5–6.0)	1.0 (0.5–3.0)	1.0 (1.0–2.0)	1.0 (1.0–3.0)	2.0 (1.0–4.0)	1.0 (0.5–6.0)	1.0 (1.0–2.0)
Number of PC units per 10 kg	0.8 (0.2–6.7)	1.2 (0.2–5.7)	1.1 (0.3–6.7)	0.8 (0.2–2.6)	0.7 (0.3–1.4)	0.8 (0.2–4.4)	0.3 (0.2–0.5)
HCT or PCV pretransfusion (%)	21.0 (7.0–55.0)	21.0 (8.0–50.7)	21.0 (13.2–45.0)	18.0 (11.0–29.0)	33.2 (11.8–44.0)	23.1 (12.0–55.0)	26.0 (16.0–47.0)
PLT count pre-PC transfusion ($\times 10^3/\mu$L or $\times 10^9$/L)	8.0 (0.0–519.0) ($n = 172$)	7.0 (0.0–59.0[a]) ($n = 48$)	66.5 (19.0–519.0) ($n = 10$)	6.0 (0.0–56.0) ($n = 37$)	142.0 (62.0–385.0) ($n = 7$)	10.0 (0.0–50.0) ($n = 58$)	93.5 (51.0–335.0) ($n = 10$)
PLT count post-PC transfusion ($\times 10^3/\mu$L or $\times 10^9$/L)	16.2 (2.0–468.0) ($n = 105$)	9.0 (2.0–92.0) ($n = 34$)	81.0 (31.0–123.0) ($n = 6$)	13.0 (3.0–164.0) ($n = 26$)	268.0[c] ($n = 1$)	27.0 (2.0–468.0) ($n = 36$)	70.0 (9.0–126.0) ($n = 3$)
Prophylactic transfusions	29[b]	3	0	8[b]	2	15	1
Therapeutic transfusion	158[b]	47	12	28[b]	8	50	13

Note: *N* is the number of transfusion episodes; *n* is the number of transfusion episodes for which a platelet count was available before or after the PC transfusion. Data represent median (range).

Abbreviations: IMTP, immune-mediated thrombocytopenia; PC, platelet concentrate; PLT, platelet.

[a]This patient with a pre-PC transfusion platelet count of 59,000/μL was included in the IMTP category because the platelet count on admission was <40,000/μL.

[b]For 2 of these transfusion episodes, the reason for transfusion (prophylactic vs therapeutic) was not clearly expressed in the medical record.

[c]For this single patient, platelet counts pre- and posttransfusion were available; the pretransfusion platelet count was 383×10^9/L (383,000/μL).

TABLE 3 Total platelet count change and platelet count change per unit for transfusion episodes with paired values pre- and posttransfusion (within 24 h), based on the underlying disease mechanism and the type of transfusion (prophylactic vs therapeutic).

	Pretransfusion platelet count ($\times 10^3/\mu L$ or $\times 10^9/L$)	Posttransfusion platelet count ($\times 10^3/\mu L$ or $\times 10^9/L$)	*P*-value	Platelet count change post PC transfusion ($\times 10^3/\mu L$ or $\times 10^9/L$)	Platelet count change per PC unit transfused ($\times 10^3/\mu L$ or $\times 10^9/L$)
All transfusions episodes (*N* = 99)	7.0 (0.0-383)	16.0 (4.0-268)	<0.0001	+5.0 (-115.0 to +158.0)	+4.0 (−57.5 to +158.0)
Primary IMTP (*N* = 34)	7.0 (0.0–59.0)	9.0 (2.0–92.0)	<0.0001	+2.0 (−5.0 to +75.0)	+1.5 (−5.9 to +71.0)
Decreased bone marrow production (*N* = 25)	6.0 (0.0–56.0)	14.0 (3.0–164)	0.0008	+4.0 (−7.0 to +158.0)	+3.0 (−7.0 to +158.0)
Severe thrombocytopenia of other causes (*N* = 32)	9.0 (0.0–36.0)	29.5 (2.0–121)	<0.0001	+11.5 (−11.0 to +108.0)	+6.0 (−3.3 to +128.0)
Prophylactic PC transfusions (*N* = 16)	8.5 (5.0–59.0)	44.5 (6.0–121)	0.0004	+17.5 (−4.0 to +108.0)	+15.2 (−2.0 to +106.0)
Therapeutic PC transfusions (*N* = 81)	7.0 (0.0–383.0)	12 (2.0–268)	<0.0001	+4.0 (−115.0 to +158.0)	+3.0 (−57.5 to +158.0)

Note: *N* is the number of PC transfusion episodes in which a platelet count measured within the 24 hours before and after PC transfusion was available for comparison. A negative value represents a decrease in the platelet count post-PC transfusion, whereas a positive value represents an increase in platelet count post-PC transfusion. A *P*-value <0.05 represents a statistically significant difference between pre- and posttransfusion platelet counts. Data represent median (range).
Abbreviations: IMTP, immune-mediated thrombocytopenia; PC, platelet concentrate.

after prophylactic transfusions ($P = 0.0004$) and therapeutic transfusions ($P < 0.0001$). The increase in platelet count post-PC transfusion was greater in the prophylactic transfusion group than in the therapeutic group ($P = 0.0167$). Table 3 summarizes the median platelet count change for each type of PC transfusion. For therapeutic transfusions, 93 of 158 patients (58.9%) had $\leq 10.0 \times 10^9$/L (10,000/μL), 111 (70.2%) had $\leq 20.0 \times 10^9$/L (20,000/μL), and 125 (79.1%) had $\leq 50.0 \times 10^9$/L (50,000/μL]) prior to PC transfusion. For prophylactic transfusions, 11 of 29 patients (37.9%) had $\leq 10.0 \times 10^9$/L (10,000/μL), 14 (48.3%) had $\leq 20.0 \times 10^9$/L (20,000/μL), and 20 (69.0%) had $\leq 50.0 \times 10^9$/L (50,000/μL) prior to PC transfusion.

Throughout their hospitalization, 104 of 149 dogs (69.8%) received at least 1 additional type of blood product. This included 99 dogs (66.4%) that were administered packed RBCs (pRBCs), 44 (29.5%) that received plasma products, and 8 (5.3%) that received FWB. For patients receiving a massive transfusion, the median ratio of plasma product:pRBC:PC was 5.5:5.5:1.

3.3 | Transfusion reactions

Information regarding transfusion reactions was available for 168 of 189 PC transfusion episodes (88.9%). A transfusion reaction was suspected during 2 of 168 episodes (1.2%). One patient became apparently nauseated (lip smacking, swallowing) and febrile, although this patient was concurrently receiving 1 unit of pRBCs. The other patient was suspected to have developed mild facial swelling, which resolved without any medication prescribed. This patient had received a PC unit stored for 7 days. No transfusion reactions were noted upon transfusion of the other 4 units stored for >5 days.

In the study period, 8 DEA 1-positive PC units were administered to DEA 1-negative patients. Among these, 1 dog underwent 3 mismatched PC transfusion episodes. No transfusion reactions were noted during these transfusion episodes.

4 | DISCUSSION

This is the largest report of the clinical use of PC transfusion in dogs to date, and the product was found to be typically safe, with a low rate of transfusion reactions. In this population, PC was most commonly administered to animals with active hemorrhage rather than as a prophylactic measure. The median platelet count increased after PC administration to this population, although the efficacy of platelet transfusion in reducing or preventing hemorrhage could not be determined given the retrospective nature of the study.

In veterinary medicine, most platelet transfusions are administered to patients with primary or secondary IMTP, and it was previously reported that 64%–87% of patients undergoing platelet transfusions in the form of fresh, cryopreserved, or lyophilized platelets were primary IMTP patients.[5,23,25] Other underlying diseases that have been reported include sepsis, neoplasia, tick-borne diseases, trauma, GI bleeding associated with nonsteroidal anti-inflammatory administration, and cardiopulmonary bypass.[5,24–26] In this study, primary IMTP only accounted for 26.2% of the PC transfusions, which is lower than previous reports. This finding may be due to the inclusion of patients without thrombocytopenia in the current study or the stringent criteria used to categorize patients in the primary IMTP group. While this report is in agreement with previous studies regarding the general disease processes that result in platelet transfusions, it is the first report

to describe the use of PC as part of a massive transfusion protocol in dogs.

In the present population with a high overall mortality rate, patients with congenital thrombocytopathia and primary IMTP had the highest survival rate. Dogs with primary IMTP have reported survival rates that vary between 74% and 97%,[5,27–32] which is higher than the 66.7% survival rate reported in this study. This difference likely represents increased severity of disease for dogs in this study that required platelet transfusions. The survival rate for dogs receiving massive transfusions (58.3%) was higher than previously reported (26.6%)[33] but similar to human patients undergoing massive transfusion for nontrauma-related hemorrhagic shock.[34–36] Comparison of outcomes among other disease mechanisms is more challenging given the heterogeneity of those populations.

Common sites of hemorrhage previously reported in dogs receiving platelet transfusions include cutaneous, GI tract, ocular, oral, nasal, respiratory tract, urinary tract, and the CNS.[5,26,37] In the present study, we found a similar distribution of hemorrhage, as has been previously reported.[5] In patients with IMTP, the most commonly reported sites of hemorrhage are the skin followed by the GI tract, which is similar to our findings.[30,31] In our study, 36% of IMTP patients were suspected to have CNS hemorrhage. The incidence of CNS hemorrhage in dogs with IMTP is unknown. In pediatric human patients with IMTP, there is a reported incidence of 0.19%–0.78%.[38] As this study only included animals with IMTP that required a platelet transfusion, the true incidence of CNS bleeding in the general IMTP population is likely overestimated.

Studies comparing prophylactic and therapeutic platelet transfusions in dogs are scarce. In a study of fresh and lyophilized platelet transfusions, 30% of transfusions were administered prophylactically.[5] Another study reported 60% of cryopreserved platelet transfusions were given prophylactically.[26] In our study, only 15% of the transfusions were prophylactic. Given the lack of clear platelet transfusion triggers in veterinary medicine and reliance on clinical acumen and product availability, this difference is not surprising and most likely reflects differences in clinical practice. In human guidelines, prophylactic transfusion of platelets is recommended in patients with $<10 \times 10^9$/L (10,000/μL) to prevent spontaneous hemorrhage and in patients with $<50.0 \times 10^9$/L (50,000/μL) prior to invasive procedures.[3] Regarding therapeutic transfusions, there is little evidence concerning platelet transfusion triggers, and current human guidelines recommend maintaining a platelet count $>30 \times 10^9$/L to 100×10^9/L (30,000–100,00/μL) depending on the localization and severity of the underlying hemorrhage.[4] In our study, the median platelet counts for patients receiving prophylactic and therapeutic transfusions were 14.0×10^9/L (14,000/μL) and 8.0×10^9/L (8000/μL), respectively; nonetheless, clinical decisions to transfuse platelets are multifactorial and not based solely on platelet number. Appropriate platelet transfusion triggers are unknown in veterinary medicine and require further evaluation.

One goal in platelet transfusions is to increase the platelet count, although the ability of various veterinary products to do so is still controversial. While fresh platelet products have been reported to increase platelet counts in experimental animals, there are still limited data in clinical animals that demonstrate this or compares fresh platelets to other products. An experimental study in irradiated dogs demonstrated that fresh PC significantly increased the platelet count in transfused dogs compared to nontransfused patients.[6] Another experimental study in dogs demonstrated a significantly higher recovery of platelets after transfusion of fresh platelets compared to cryopreserved platelets.[34] However, clinical studies have shown mixed results for all platelet products. In a study evaluating patients receiving fresh PC compared to lyophilized platelets, platelet counts immediately after transfusion were variable in both groups; while posttransfusion platelet counts appeared higher with administration of fresh platelets, neither product successfully consistently increased the platelet count.[5] Despite having hemostatic function, lyophilized or cryopreserved platelets have not been demonstrated to significantly change the platelet count in veterinary patients.[24,39,40]

In this study, the increase in platelet count post-PC transfusion was greater in the prophylactic transfusion group than in the therapeutic group. This finding is not surprising as it is expected that platelets would be consumed during active hemorrhage. In addition, most of the IMTP patients in this study were included in the therapeutic transfusion group. These dogs may have had ongoing immune-mediated destruction of transfused platelets, further reducing the posttransfusion platelet count. In human medicine, the lack of a clinically significant increase in platelet count after PC transfusion is often considered the result of immune-based platelet refractoriness in patients undergoing repeated PC transfusions due to the presence of antibodies against various platelet antigens.[41] This phenomenon, which has been previously reported in dogs,[37] is considered unlikely in this study given the use of single-donor PC units and the small number of patients undergoing repeated PC transfusion.

Differences in platelet count based on the dose of platelets received have been evaluated more systematically in human medicine. A large human trial assessed the platelet increment after prophylactic PC transfusion using 3 different PC doses.[42] The increments noted for the low (1.1×10^{11} platelet/m^2 of body surface area [BSA]), median (2.2×10^{11} platelet/m^2 of BSA), and high PC dose (4.4×10^{11} platelet/m^2 of BSA) were 10.0×10^9/L (10,000/μL), 19.0×10^9/L (19,000/μL), and 38.0×10^9/L (38,000/μL), respectively. It must be noted that the low dose used in this large human trial represents significantly more platelets than the units used in our study, which is not surprising because human PC products are derived from apheresis or pooled platelets collected from whole blood of multiple donors. However, despite the higher concentration of platelets in human PC units, the platelet increment posttransfusion for the low dose in the human study is similar to the one reported in this study.

There are limited data on the occurrence of transfusion reactions secondary to PC administration in veterinary medicine. In a study of 15 dogs receiving fresh PC transfusion, 13% developed an acute mild transfusion reaction, with clinical signs such as urticaria, periorbital swelling, and emesis.[5] Similar prevalence (17%) and clinical signs were noted in a study of 5 dogs receiving multiple PC transfusions.[6] However, these animals were also receiving whole blood transfusions, so adverse reactions to PC specifically cannot be determined.[6] The

frequency of transfusion reactions reported in these 2 studies is similar to human studies, where the incidence of adverse events after PC transfusion varies between 2.2% and 13.7%.[42–45] In the present study, transfusions reactions were rare, and their evaluation was complicated by the administration of other blood products before or after PC transfusion. However, the prevalence here was lower than previously reported in veterinary medicine. This finding may be due to the retrospective nature of the study, the lack of standardization for recording such events, and the different circumstances under which patients received platelets. For example, some patients underwent PC transfusion while under general anesthesia during surgical intervention, which could have affected evaluation of the temperature and heart rate and therefore underestimated the true incidence of adverse reactions.

One of the commonly described disadvantages of PC is its short storage time of 5 days. This time limit is related to the development of platelet storage lesions, a decrease in platelet function and viability, and potential bacterial proliferation.[1,46,47] In human medicine, the current evidence suggests that prolonged storage times (5–7 days) appear to be associated with dampened platelet count elevations posttransfusion compared to platelets stored for <3 days.[48,49] Some evidence suggests that platelet function in canine PC progressively decreases over a 7-day time frame,[50] but another recent experimental veterinary study showed that platelet function assessed by aggregometry remained acceptable in PC stored up to 7 days despite the presence of storage lesions.[46] The clinical impact of these findings is much harder to evaluate given the difficulty in assessing platelet function and viability in vivo, as well as the efficacy of PC transfusion to resolve active hemorrhage. Gram-positive skin commensals are most frequently implicated in infections associated with platelet transfusions and likely contaminate the unit during blood collection. Gram-negative contamination is less common and might be secondary to transient bacteremia in asymptomatic human donors.[51] Regardless of the cause, bacterial contamination of PC units is rare, with an incidence of <1%, but is still of significant concern in human medicine because of the implications in recipients.[52–54] Information regarding contamination of PC units in veterinary medicine is scarce, and the current evidence supports the fact that bacterial contamination is a rare phenomenon.[6,46,47,54] In our study, the incidence of a positive culture was 3%. These positive cultures were suspected by the laboratory personnel to be bacterial contaminants during plating, as opposed to true contamination of the PC units during collection; however, the clinical implication of such culture results remains difficult to evaluate given the retrospective nature of this study.

This study has several limitations. The retrospective aspect limits the accuracy of categorizing underlying disease mechanisms and their influence on platelet change post-PC transfusion. The retrospective nature also limits standardization of data collection, such as timing and evaluation of platelet count pre- and post-PC transfusion, which was only performed at the primary clinician's discretion. The presence of platelet clumping is another limitation, as it leads to underestimation of the patient's actual platelet count and might interfere with the calculations of platelet change posttransfusion. It is important to note that the descriptive design of this study and the lack of a control group do not allow for the evaluation of the efficacy of PC transfusion to stop hemorrhage or increase platelet count posttransfusion. Prospective studies are required for further evaluation. In this study, most of the nonsurvivors were euthanized, which further limits the evaluation of mortality for this population, as it may not reflect severity of illness but, rather, an owner's decision to halt therapy.

In this retrospective study, dogs undergoing PC transfusion were commonly diagnosed with immune-mediated disease. They frequently had evidence of hemorrhage in multiple organ systems, particularly the GI tract and skin. PC transfusions were most commonly administered therapeutically and led to increased platelet count posttransfusion. However, this increase in platelet count was more substantial in prophylactic transfusions. Administration of PC transfusions appeared relatively safe, with a low reported incidence of transfusion reactions.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ORCID

Laurence M. Saint-Pierre DVM https://orcid.org/0000-0002-9140-9156

Kate S. Farrell DVM, DACVECC https://orcid.org/0000-0002-8536-2443

Krystle L. Reagan DVM, PhD, DACVIM https://orcid.org/0000-0003-3426-6352

ENDNOTES

[a] ADVIA 120 Hematology System, Siemens Healthineers, Erlangen, Germany.
[b] Imuflex WB-SP LGQ506A6 Blood Bag System, Terumo Corporation, Tokyo, Japan.
[c] Sorvall RC 12 BP, Kendro Laboratory Products, Stortford, UK.
[d] Storage Bag XT612, Terumo Corporation, Tokyo, Japan.
[e] PF 15i Platelet Agitator, Noblesville, IN.
[f] PC 100i Platelet Incubator, Helmer, Noblesville, IN.
[g] Hemo-Nate Filter, Utah Medical Products, Midvale, UT.
[h] GraphPad Prism, Version 9, San Diego, CA.

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