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Carbon hemoperfusion markedly reduces serum meloxicam concentrations in dogs with meloxicam toxicosis

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Objective
To investigate the efficacy of synthetically derived activated carbon hemoperfusion (cHP) by measuring serum meloxicam concentration in dogs with meloxicam toxicosis.

Methods
5 dogs with meloxicam overdose that underwent cHP at 3 teaching hospitals between January 2023 and March 2026 were identified. Dogs that had serum meloxicam concentrations measured were included. For case 1, in-series cHP and hemodialysis were performed. Based on case 1 data, standalone cHP was used for cases 2 to 5. Serum meloxicam concentration was measured via ultra-performance liquid chromatography–tandem mass spectrometry.

Results
Median ingested meloxicam dose was 16.7 mg/kg (range, 1.4 to 168.8). Median blood volumes processed were 2 L/kg (range, 1.36 to 2.56) over 2.2 hours (range, 1.5 to 2.6). During cHP, meloxicam half-life was 0.4 to 1 hour versus 5.1 to 16.6 hours post-cHP, and median clearance was 6.52 mL/kg/min (range, 3.81 to 10.42) versus 0.49 mL/kg/min post-cHP (range, 0.22 to 0.72). Median extracorporeal elimination constant from 5 dogs was 1.24 (range, 0.68 to 1.95), 13.8 times faster than the intrinsic elimination constant of 0.09 from 3 dogs (range, 0.04 to 0.14). After cHP, a rebound phenomenon (47.2% increase, approximately 5 hours later) was noted in 3 dogs. All dogs were discharged in 72 hours, with 1 dog developing International Renal Interest Society Grade II acute kidney injury during hospitalization that recovered with supportive care.

Conclusions
Carbon hemoperfusion markedly reduced serum meloxicam concentrations in dogs with meloxicam toxicosis and substantially shortened meloxicam half-life.

Clinical Relevance
Dogs that underwent timely cHP had excellent short-term outcomes, and cHP should be considered in dogs with high-dose meloxicam ingestion.

Keywords: carbon hemoperfusion, dogs, meloxicam, extracorporeal therapy, intoxication

Meloxicam is an NSAID that preferentially inhibits cyclooxygenase-2 activity and prostaglandin synthesis. As with other NSAIDs, meloxicam has analgesic, anti-inflammatory, and antipyretic effects.[1] In dogs, bioavailability of meloxicam is 90.24% after oral administration.[2] Meloxicam has an elimination half-life ($t_{1/2}$) of approximately 24 hours.[3] It is metabolized in the liver and then excreted in urine and feces.[4]

Nonsteroidal anti-inflammatory drug toxicosis is common in veterinary medicine.[5] Clinical signs of NSAID toxicosis include gastrointestinal disturbances, acute kidney injury, and CNS disturbances.[6]

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Elevated liver enzymes and idiosyncratic liver injury have also been reported.[6,7] Treatment of NSAID toxicosis includes gastric decontamination, activated charcoal, gastrointestinal support, intralipid therapy, and fluid therapy.[8] Extracorporeal toxin removal (ECTR), such as hemoperfusion (HP) and therapeutic plasma exchange (TPE), has gained interest and shown good outcomes for NSAID toxicosis in dogs.[8–11] Meloxicam is an ideal drug for HP or TPE because it has a small volume of distribution (0.32 L/kg) with a small molecular weight (351.4 g/mol) and high protein-binding property (97%).[3,12,13]

Data on the efficacy of HP for meloxicam elimination from dogs are limited to a few case reports[9,10] that reported serum concentrations but lacked detailed pharmacokinetic data in meloxicam toxicosis. In addition, HP devices used in the previous case reports[9,10] (AimaLojic Atlas Cartridge V100 and V300; AimaLojic Animal Health) were the prior version of HP devices (AimaLojic Dublin Cartridge V100 and V300; AimaLojic Animal Health) used in the current study. There were also no serial serum meloxicam measurements after HP to assess the rebound phenomenon.[9,10] Hemoperfusion can be delivered in standalone or hemodialysis (HD) platform or combined with TPE.[10,14,15] There is no recommended optimal ECTR protocol for meloxicam toxicosis at the time of this writing. This case series aimed to describe the efficacy of the synthetically derived activated carbon HP (cHP) by measuring serum meloxicam concentration in 5 dogs with meloxicam toxicosis. We also documented extraction ratio (ER; percent), elimination rate constant (k), $t_{1/2}$ (hour), and clearance (milliliters per kilogram per minute) during ECTR and after ECTR. To the authors' knowledge, our case series is the first to compare the efficacy of cHP to HD and provide the most comprehensive description of pharmacokinetics in dogs with meloxicam toxicosis treated with the newer version of cHP devices.

Methods

Case selection

Client-owned dogs with meloxicam overdose that presented to 3 teaching hospitals in the United States between January 2023 and March 2026 were eligible: The Ohio State University, University of Florida, and North Carolina State University. Meloxicam overdose was defined as an estimated dose higher than the known veterinary therapeutic dose. Inclusion criteria included dogs with confirmed meloxicam overdose that received supportive care and ECTR and had serum meloxicam concentrations measured during hospitalization. The potential cases were identified by the authors via direct contact with the referring veterinarians or emergency services. Dogs were excluded if extracorporeal therapy records were incomplete. All dogs were client owned, and hospitalization and extracorporeal therapy were performed with written informed consent from the owners. Prospective sample collection was approved by the IACUC of The Ohio State University (2022A00000040) and North Carolina State University (24-406). At the University of Florida, only residual serum remaining after clinically indicated biochemistry was analyzed; therefore, IACUC approval was not required. No further follow-up was obtained for these dogs after discharge.

Sample collection, handling, and meloxicam analysis

For case 1, in-series cHP and HD were performed. Blood samples were obtained from precarbon, postcarbon, and postdialyzer at intervals (0, 15, 30, 90, and 180 minutes) during ECTR to investigate the meloxicam removal by cHP and HD, respectively. Based on the data from case 1, standalone HP was used for cases 2 to 5. In all 5 cases, blood samples were collected for serum meloxicam concentrations at the beginning and upon completion of HP. For cases 1 and 4, blood samples for meloxicam analysis were collected upon intake. For cases 1, 4, and 5, serum was also collected after ECTR during hospitalization (1 or 4 hours and 8 or 12 hours after ECTR) to assess rebound phenomenon.

For case 1, samples were centrifuged, and serum was frozen and submitted to the Texas A&M Veterinary Medical Diagnostic Laboratory for analysis. For cases 2 to 5, samples were processed as case 1 but submitted to North Carolina State University Baynes Laboratory. Analysis of meloxicam was performed by ultra-performance liquid chromatography–tandem mass spectrometry validated in canine plasma. **Supplementary Material S1** provides the detailed protocol.

Extraction ratio of meloxicam from cHP was calculated using Equation 1 in **Table 1**. Assuming elimination is by first-order kinetics, meloxicam k during ECTR and intrinsic k were calculated using Equation 2. The total elimination rate constant (k_{tot}) is the sum of the extracorporeal elimination rate constant (k_{ect}) and the intrinsic elimination rate constant (k_{int}). The intrinsic meloxicam elimination rate constant was calculated based on meloxicam concentrations obtained after ECTR during hospitalization. The elimination $t_{1/2}$ and clearance of meloxicam during and after ECTR were calculated using Equations 3 and 4, separately.

Table 1—Equations used to calculate the pharmacokinetic parameters of meloxicam during carbon hemoperfusion.

No.	Parameter	Equation
1	Extraction ratio of meloxicam from cHP (%)	$(C_{precarbon} - C_{postcarbon})/C_{precarbon} \times 100$
2	Elimination rate constant (k)	$k = (\ln C_1 - \ln C_2)/(t_2 - t_1)$
3	Half-life ($t_{½}$)	$t_{½} = 0.693/k$
4	Clearance (Cl)	$Cl = V_d \times k = 0.32 \times (\ln C_1 - \ln C_2)/(t_2 - t_1)$

A volume of distribution (V_d) of 0.32 L/kg was used.[12] Time (t) was expressed in hours for calculation of half-life ($t_{½}$) and in minutes for calculation of clearance.

C_1 and C_2 = Serum meloxicam concentrations at times t_1 and t_2, respectively. cHP = Carbon hemoperfusion. $C_{precarbon}$ = Precarbon device serum meloxicam concentration. $C_{postcarbon}$ = Postcarbon device serum meloxicam concentration. ln = Natural logarithm.

Statistical analysis

Due to the descriptive nature and small number of cases, no statistical analysis was performed. All clinical data were extracted from the medical records into a standardized spreadsheet (Excel; Microsoft Corp) using predefined variables and were cross-checked by a second investigator for accuracy and completeness.

Results

Five client-owned dogs with meloxicam toxicosis were identified during the study period. All dogs received supportive care and cHP and had serum meloxicam concentrations measured during hospitalization; therefore, they were all included in the current study.

Case 1

A 1.5-year-old male neutered Bernese Mountain Dog weighing 32.5 kg was presented to The Ohio State University Veterinary Medical Center approximately 2 hours after ingestion of 1.4 mg/kg of meloxicam. Physical examination was unremarkable, and emesis was induced, but no meloxicam tablets were recovered. Antiemetic and activated charcoal were then given. Due to the magnitude of the ingested dose, ECTR with in-series cHP and HD was chosen to expedite clearance of meloxicam. Bloodwork before initiation of ECTR revealed a mild increase in BUN (35 mg/dL; reference interval, 7 to 27 mg/dL), and creatinine was within normal limits (1.5 mg/dL; reference interval, 0.5 to 1.8 mg/dL). The dog was sedated for dialysis catheter placement. The dog was placed in left lateral recumbency, and the lateral cervical region was clipped and prepped aseptically. A temporary dialysis catheter was aseptically placed in the right external jugular vein percutaneously using a modified Seldinger technique. A combination of cHP and HD was initiated using a continuous renal replacement therapy platform (Prismaflex; Baxter Healthcare Corp) with a high-flux dialyzer (Prismaflex M100 set; Baxter Healthcare Corp). A cHP device (AimaLojic Dublin Cartridge V300; AimaLojic Animal Health) was primed according to the manufacturer's instructions before connecting to the dialyzer. Seventeen times the blood volume was processed over 2.6 hours. Blood samples were collected for serum meloxicam concentration on presentation, before ECTR, at intervals during ECTR with predetermined time points, upon completion of ECTR, and during hospitalization (1, 4, and 12 hours after ECTR). The dog was hospitalized with supportive care, and his kidney values (BUN, 13 mg/dL; creatinine, 1.04 mg/dL) were within normal limits before discharge.

Case 2

A 10-month-old female spayed Havanese weighing 6.3 kg was presented to a satellite clinic of the emergency service at the University of Florida for vomiting after ingestion of 16.7 mg/kg of meloxicam. Emesis was induced, and activated charcoal was administered before referral for ECTR. Physical examination was unremarkable, and bloodwork showed normal kidney values (BUN, 20 mg/dL, reference interval, 7 to 29 mg/dL; creatinine, 0.8 mg/dL, reference interval, 0.3 to 1.2 mg/dL), mildly elevated ALT (114 U/L; reference interval, 8 to 75 U/L), and moderate hyperbilirubinemia (3.1 mg/dL; reference interval, 0 to 0.8 mg/dL). Due to the magnitude of the ingested meloxicam dose, HP was recommended. The dog was sedated for dialysis catheter placement using the same methodology as case 1. The HP treatment was performed using a cHP device (AimaLojic Dublin Cartridge V100; AimaLojic Animal Health) on a standalone HP machine (AimaLojic ReVive HP Complete Hemoperfusion System; AimaLojic Animal Health). Fresh frozen plasma (FFP) was primed in the circuit since the extracorporeal circuit volume approximated 15% of the dog's blood volume based on a blood volume of 80 mL/kg in dogs. The HP treatment was uneventful, and 25 times the blood volume was processed over 2.5 hours. Blood samples were collected for serum meloxicam concentration before and upon completion of HP. During hospitalization, recheck bloodwork showed International Renal Interest Society Grade II acute kidney injury (BUN, 31 mg/dL, reference interval, 7 to 27 mg/dL; creatinine, 1.66 mg/dL, reference interval, 0.6 to 1.5 mg/dL) that resolved with supportive care before discharge.

Case 3

A 6-year-old female spayed Biewer Terrier weighing 2.3 kg presented to the primary veterinarian for lethargy, anorexia, and vomiting after ingestion of 6.5 mg/kg of meloxicam approximately 24 hours prior. Bloodwork showed mildly elevated BUN (44 mg/dL; reference interval, 7 to 27 mg/dL), and the dog was given intralipid before being referred for ECTR. On presentation to North Carolina State University, physical examination was unremarkable, and the dog weighed 2.3 kg. Considering the ingested dose of meloxicam, HP was recommended. The dog was sedated for dialysis catheter placement using the same methodology as case 1. The HP treatment was performed using the same type of cHP device as case 2 on a standalone HP machine (PuriFi Pump; CytoSorbents Corp; VetResQ). The total extracorporeal circuit volume approximated 41% of the dog's blood volume based on a blood volume of 80 mL/kg in dogs. As a result, blood products (packed RBC and FFP) were primed in the circuit considering the high extracorporeal circuit volume. Minimum return pressure was noted at the beginning of the treatment, and the machine stopped running intermittently. This was attributed to the height of the table being much lower than the machine and the patient's small size. Approximately 40 minutes into HP treatment, it was suspected that the intermittent interruptions in blood circulation through the machine led to clotting in the cHP device. Therefore, a new circuit and cHP device were replaced. Blood products (packed RBC and FFP) were again primed in the circuit before connecting to the dog. The dog was bradycardic while recovering from sedation. However, after starting HP, the dog became tachycardic and appeared agitated. After changing to the new circuit and cHP device, the dog started shaking, coughing,

retching, and then regurgitated. Venous blood gas was then performed, and severe hypocalcemia (ionized calcium, 0.34 mmol/L; reference interval, 1.18 to 1.35 mmol/L) was noted, which we suspect was due to citrate toxicity. Calcium gluconate 10% was given IV, and the ionized calcium level was returned to normal limits (1.33 mmol/L) after HP treatment. Twenty-three times the blood volume was processed in approximately 90 minutes. Blood samples were collected for serum meloxicam concentration before and upon completion of HP and when the cHP device was replaced during treatment. During hospitalization, the dog developed diarrhea and had suspected gastrointestinal bleeding that improved with supportive care. Recheck kidney values were within normal limits (BUN, 14 mg/dL, reference interval, 11 to 27 mg/dL; creatinine, 0.4 mg/dL, reference interval, 0.5 to 1.4 mg/dL) before discharge.

Case 4

A 5-month-old male intact Poodle mix dog weighing 9 kg was referred to North Carolina State University for ingestion of 51.6 mg/kg of meloxicam approximately 21 hours before presentation. His physical examination and bloodwork (BUN, 10 mg/dL, reference interval, 11 to 27 mg/dL; creatinine, 0.3 mg/dL, reference interval, 0.5 to 1.4 mg/dL) were unremarkable on presentation. Due to high-dose meloxicam ingestion, HP was recommended. The dog was sedated for dialysis catheter placement using the same methodology as case 1. The HP treatment was performed using the same type of cHP device and machine as case 3. Blood samples for serum meloxicam concentration were collected on presentation, before and upon completion of HP, and during hospitalization (1 and 12 hours post-cHP). The HP treatment was uneventful, and 26 times the blood volume was processed over 2 hours. During hospitalization, the dog developed hematochezia that resolved with supportive care before discharge.

Case 5

A 1-year-old male neutered Beagle weighing 8 kg was referred to North Carolina State University for vomiting after ingestion of 168.75 mg/kg of meloxicam approximately 12 hours before presentation. Activated charcoal, intralipid therapy, and IV fluids were given before the referral. On presentation, the dog had unremarkable physical examination and bloodwork (BUN, 20 mg/dL, reference interval, 7 to 27 mg/dL; creatinine, 1.1 mg/dL, reference interval, 0.5 to 1.8 mg/dL). Hemoperfusion was recommended considering a high dose of meloxicam ingestion. The dog was sedated for dialysis catheter placement using the same methodology as case 1. The HP treatment was performed using the same type of cHP device and machine as case 3. Due to the high dose of meloxicam ingestion, the cHP device was replaced approximately 1 hour into the HP treatment. The HP treatment was uneventful, and 32 times the blood volume was processed over 2.2 hours. Blood samples for serum meloxicam concentration were collected before and upon completion of HP and during hospitalization (4 and 8 hours post-cHP). While in the hospital, the dog developed regurgitation that resolved with supportive care before discharge.

Clinical data and serum meloxicam analysis

For case 1, the blood samples obtained postcarbon device showed marked and constant removal

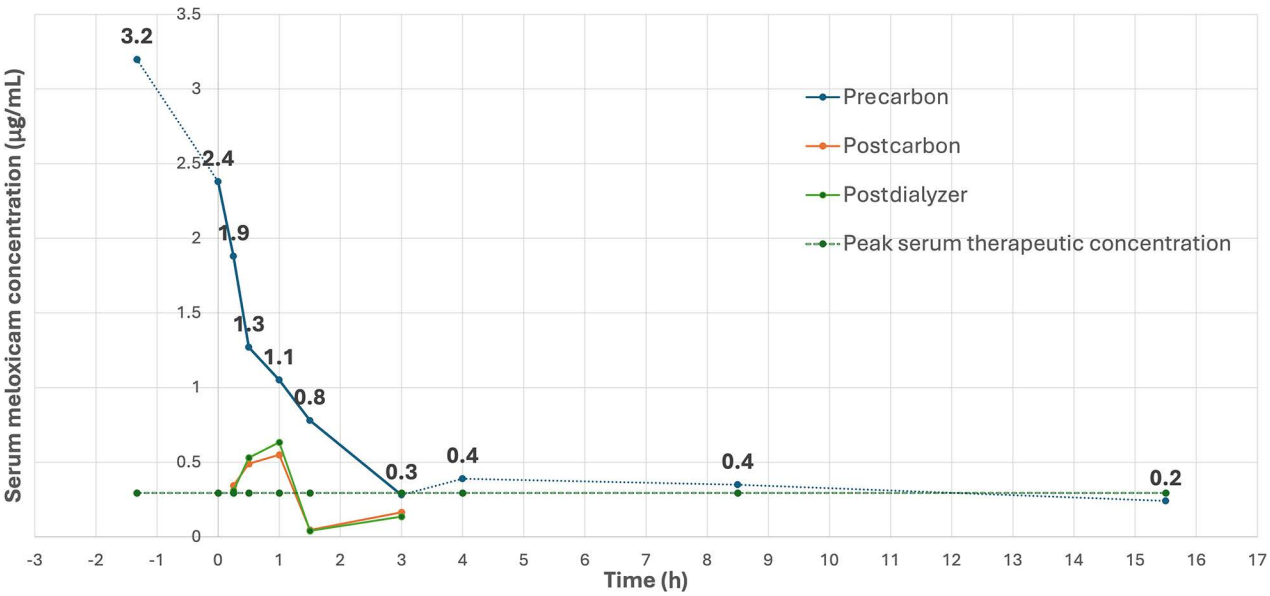

Figure 1—Serial serum meloxicam concentrations (micrograms per milliliter) during in-series carbon hemoperfusion and hemodialysis session in a 1.5-year-old dog with meloxicam toxicosis. Time 0 represents initiation of the session; negative time values represent samples collected on intake before the session. The solid blue line represents precarbon device serum meloxicam concentrations, the orange line represents postcarbon device concentrations, and the light green line represents postdialyzer concentrations. The dashed dark green horizontal line represents the reported peak serum meloxicam concentration following administration of the recommended dose.[22]

of meloxicam compared to precarbon samples at determined time points during ECTR, whereas meloxicam concentrations from postdialyzer samples were either similar or higher compared to postcarbon samples, with ER (percent) ranging from −8% to 13% **(Figure 1)**.

The median ingested meloxicam dose was 16.7 mg/kg (range, 1.4 to 168.8 mg/kg). The median blood volume processed was 2 L/kg (range, 1.36 to 2.56 L/kg), equating to 25 times (range, 17 to 32 times) the estimated blood volume of 80 mL/kg in dogs. The median duration of ECTR was 2.2 hours (range, 1.5 to 2.6 hours; **Table 2**). All 5 dogs were discharged within 72 hours of hospitalization. The results of meloxicam concentration were available after the dogs were discharged. The data on serum meloxicam removal over time during ECTR are shown in **Figure 2**, and ER ranged from 86% to 96%. For 2 dogs (cases 1 and 4) who also had serum meloxicam concentrations measured on intake, the meloxicam concentrations at the beginning of ECTR were 25.6% and 47.5% lower than the measurements on intake. Cases 1, 4, and 5 had serum meloxicam concentrations measured during hospitalization, so

Table 2—Pharmacokinetic parameters of meloxicam during and after extracorporeal toxin removal (ECTR) treatment in 5 dogs with meloxicam toxicosis treated by hemoperfusion (cases 1 to 5), compared with 3 previously reported cases.

	Estimated ingested meloxicam dose (mg/kg)	ECTR modality	Blood volumes processed (PVE for TPE)	HP/TPE duration (h)	$t_{½}$ during ECTR (h)	$t_{½}$ post-ECTR (h)	Clearance during ECTR (mL/kg/min)	Clearance post-ECTR (mL/kg/min)	Elimination constant from ECTR (h^{-1})	Intrinsic elimination constant (h^{-1})
Case 1	1.4	HP/HD	17	2.6	1	16.6	3.81	0.22	0.68	0.04
Case 2	16.7	HP	25	2.5	0.6	ND	6.52	ND	1.24	ND
Case 3	6.5	HP	23	1.5	0.4	ND	10.42	ND	1.95	ND
Case 4	51.6	HP	26	2	0.7	5.1	5.19	0.72	0.84	0.14
Case 5	168.8	HP	32	2.2	0.4	7.5	8.84	0.49	1.56	0.09
Walton et al[11]	1	TPE	1.2	3.5	1.4	ND	2.63	ND	0.49	ND
Haire et al[9]	0.48	HP	5.5	1	1.2	19.8	3.13	0.19	0.55	0.04
Covo et al[10]	5.5	HP	15	3.45	1.1	ND	3.22	ND	0.6	ND

Cases 1 to 5 are dogs from the present report; the bottom 3 rows are previously published cases included for comparison. Blood volumes processed are reported as multiples of the dog's blood volume; for the therapeutic plasma exchange case, the value represents plasma volume exchanged. Elimination constants are assumed to be first-order rate constants.

HD = Hemodialysis. HP = Hemoperfusion. ND = Not determined (empty values in the original table are shown as ND). PVE = Plasma volume exchange. $t_{1/2}$ = Elimination half-life. TPE = Therapeutic plasma exchange.

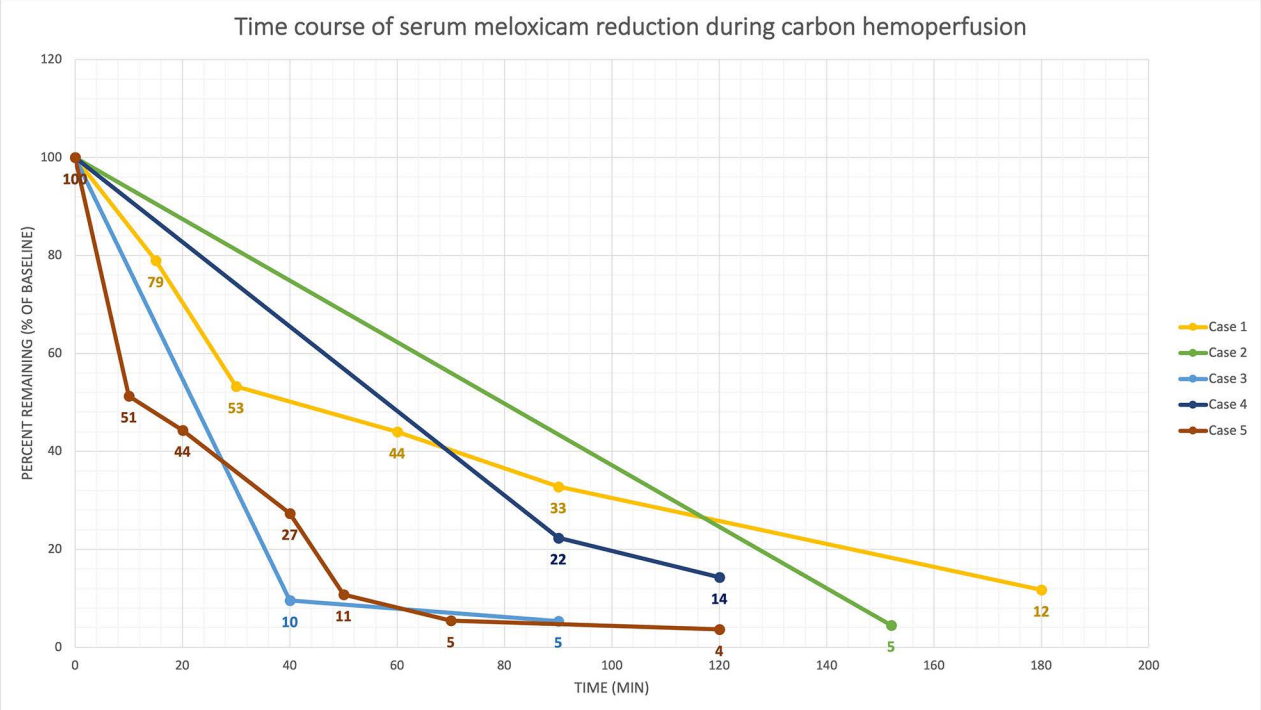

Figure 2—Time course of serum meloxicam reduction during carbon hemoperfusion in 5 dogs with meloxicam toxicosis. Case 1 was treated with in-series carbon hemoperfusion and hemodialysis. Cases 2 to 5 received standalone carbon hemoperfusion. Shown is the percentage of serum meloxicam concentration remaining over time during extracorporeal toxin removal. Time 0 represents initiation of the session; serum meloxicam concentration was defined as 100% for each dog at time 0.

k_{int}, $t_{1/2}$, and clearance after ECTR were calculated. The median k_{ect} from 5 dogs was 1.24 (range, 0.68 to 1.95); this was 13.8 times faster than k_{int} of 0.09 from 3 dogs (range, 0.04 to 0.14). For these 3 dogs (cases 1, 4, and 5), rebound phenomenon was also noted. Compared to the serum meloxicam concentration upon completion of ECTR, the measurements had an average 43.9% increase 1 hour after ECTR in 2 (cases 1 and 4) of these 3 dogs and approximately 140% and 65% increases at 4 and 8 hours after ECTR in the third dog (case 5), respectively. Twelve hours after ECTR, 2 dogs (cases 1 and 4) had lower meloxicam concentrations compared to measurements upon completion of ECTR.

Discussion

Extracorporeal toxin removal has gained popularity in veterinary medicine to remove exogenous toxins from medication overdose or accidental toxin ingestion.[5] There are 3 main modalities for ECTR in veterinary medicine, including HD, HP, and TPE. The application of these modalities is based on the pharmacokinetic properties of the toxin to maximize its removal. Nonsteroidal anti-inflammatory drugs were the most common toxicosis treated with ECTR in a retrospective study.[5] Previous studies also showed that charcoal HP,[16] in-series cHP and HD,[10] TPE,[11] and TPE combined with plasmaperfusion[15] treated dogs with NSAID toxicosis effectively. However, there is limited information regarding meloxicam kinetics during overdose and the efficacy of ECTR.[9–11] This case series reports the successful application of cHP in the management of meloxicam toxicosis in 5 dogs and reveals substantial reduction (86% to 96%) of serum meloxicam concentrations.

To the authors' knowledge, there is no study investigating the individual contributions of cHP and HD to serum meloxicam removal. From human studies,[17,18] there was no significant difference regarding meloxicam concentrations treated with HD. In-series charcoal HP and HD for carprofen toxicosis was performed, and the results suggested that carprofen was not effectively removed by HD.[19] However, a dog with ibuprofen toxicosis was treated with in-series charcoal HP and HD, and there was significant variation comparing the predialyzer and postdialyzer samples for ibuprofen.[20] Therefore, we performed in-series cHP and HD for case 1. We obtained precarbon, postcarbon, and postdialyzer samples for meloxicam concentrations at the predetermined time points. As the orange and green tracings show in Figure 1, meloxicam concentrations from postcarbon and postdialyzer had a similar trend. Meloxicam concentrations from postdialyzer samples were either similar to or higher than postcarbon samples. The data suggested that meloxicam was mainly removed from cHP but not from the high-flux dialyzer in the renal replacement therapy platform. Hemodialysis does not remove highly protein-bound toxins due to the diffusion mechanism, and our results support this principle. Based on the findings from the first case, we used a standalone HP platform to treat meloxicam toxicosis for cases 2 to 5. Although HD is not effective at removing meloxicam, using a renal replacement therapy platform to deliver HP remains a feasible option. The advantages provided by dialysate include warming the blood before returning to patients, providing glucose during treatment, and adjusting electrolyte and acid-base balance. Our study showed that standalone HP was delivered successfully and complications such as hypothermia or hypoglycemia were not observed, suggesting a standalone HP platform is safe even in small patients.

Currently, there is no established optimal ECTR modality for meloxicam toxicosis in dogs. Our results showed that k_{ect} was markedly higher than k_{int}, suggesting that cHP was efficient and useful for meloxicam elimination (Table 2). It was also noted that cHP markedly shortened meloxicam $t_{1/2}$ (0.4 to 1 hour vs 24 hours in healthy Beagles receiving oral meloxicam 0.2 mg/kg).[3] In addition, the median clearance of meloxicam during ECTR in our study was 6.52 mL/kg/min, approximately 37 times higher than the intrinsic clearance of meloxicam in dogs (0.175 mL/kg/min).[21] Based on Smith et al,[22] peak serum meloxicam concentrations reach 0.295 µg/mL approximately 4 hours after the recommended meloxicam dose was given (0.2 mg/kg, PO, once, followed by 0.1 mg/kg, PO, q 24 h). In our study, meloxicam concentrations at the beginning of ECTR were all above this range. Upon completion of ECTR, there was substantial meloxicam reduction in all 5 dogs, ranging from 86% to 96% reduction over 1.5 to 2.6 hours. Compared to the previous meloxicam toxicosis studies that treated with TPE, manual cHP, or in-series cHP and HD,[9–11] our results showed higher k_{ect} and clearance and shorter $t_{1/2}$ (Table 2). These findings can be explained because we processed a higher volume of blood.[9,10] In addition, older versions of cHP devices were used in the previous studies. AimaLojic Dublin cartridges were used in the current study, and AimaLojic Atlas cartridges were used in the previous studies.[9,10] Dublin cartridge removes toxin faster than Atlas, approximately 20% to 30% faster (Jeff Barnes, BS, AimaLojic Animal Health, unpublished data, 2026). It was also noted that the serum meloxicam concentrations were still higher than the peak therapeutic serum concentration (0.295 µg/mL) after ECTR. All dogs were hospitalized after ECTR, and they received either intralipid therapy, activated charcoal, or cholestyramine as part of supportive care. They were all discharged within 72 hours, highlighting that continued supportive care and decontamination is important even after ECTR.

According to International Renal Interest Society best practices consensus guidelines for HP for veterinary toxicities,[14] the goal of HP prescription is to provide sufficient clearance to eliminate toxin or mitigate ongoing toxic effects in a single session. Since there is no available recommendation for specific toxins, HP dose should be prescribed based on total blood volumes processed and length of HP. It is also recommended to prolong HP duration in massive intoxications.[14] A recent study[10] showed an 85% meloxicam reduction with 15 times the

blood volume processed over 207 minutes. Another study[9] showed a 44.4% reduction with 5.5 times the blood volume processed over 1 hour. In our study, the median blood volume processed was 25 times (range, 17 to 32 times) over 2.2 hours (range, 1.5 to 2.6 hours) with a median meloxicam reduction of 95% (range, 86 to 96%). All 5 dogs were discharged within 72 hours of hospitalization and had excellent short-term outcomes. Our results showed great variation in serum meloxicam concentrations at the beginning of ECTR. Even with the highest concentration of 1,175 times the peak therapeutic serum concentration, we noticed that a substantial 89% of meloxicam was removed approximately 1 hour into HP treatment. Currently, there are 2 commercial HP devices, cHP and a cross-linked polymer-based device, available in the United States at the time of writing this article. The efficacy of polymer-based HP for meloxicam toxicosis is undetermined. Future studies with a larger case number to investigate an optimal ECTR protocol for meloxicam toxicosis are required.

Meloxicam is an ideal drug for ECTR in toxicosis cases. Based on pharmacokinetics of solute removal, TPE decreases approximately 75% of the pretreatment levels of a solute with 1.4 times plasma volume exchange, with minimal additional clearance beyond 1.5 times plasma volume.[23] The efficiency of HP is based on surface area, pore size, and saturation of adsorbents.[24] In massive intoxication, prolonged HP duration is suggested, and the HP device can be replaced to maximize toxin removal.[14] In addition, there is no need to replace plasma with HP, therefore reducing the requirement for blood products and the risk of transfusion reaction. Hemoperfusion can be used alone or combined with renal replacement therapy or TPE platforms to maximize drug or toxin removal based on their characteristics.[15,25] As a result, we elected to use cHP on the standalone HP platform to treat meloxicam toxicosis for cases 2 to 5. Therapeutic plasma exchange was previously used in a dog with meloxicam toxicosis.[11] Those results showed 82% meloxicam reduction with 1.2 times the plasma volume exchange over 3.5 hours. Compared to our results, the k_{ect} in that study was 0.49 (vs 1.24), $t_{1/2}$ during ECTR was 1.4 hours (vs 0.6 hours), and clearance was 2.63 mL/kg/min (vs 6.52 mL/kg/min). The median meloxicam reduction in our study was 95% over 2.2 hours (Table 2). Our results showed higher k_{ect} and $t_{1/2}$ during ECTR, meloxicam reduction, and shorter treatment duration compared to TPE.[11] Manual cHP is a feasible option with small patients or lack of a standalone HP platform, but it could be less efficient due to the intermittent manner regarding drawing blood compared to the machine pump. The standalone HP platform also carries access, preadsorber, and return pressure monitoring, a rolling pump, and an air bubble trap; hence, it provides safety to detect clotting in the dialysis catheter or HP device and air in the circuit during treatment. Haire et al[9] performed manual cHP in a 3.5 kg cat, and the treatment shortened meloxicam $t_{1/2}$ (Table 2). Their data showed 5.5 times the blood volume processed over 1 hour with 44.4% meloxicam reduction. Compared to the smallest dog (case 3, 2.3 kg) in our study, we processed 23 times the blood volume over 1.5 hours and achieved 95% meloxicam reduction. Priming the circuit with blood product was performed in this case due to the high extracorporeal circuit volume. During the treatment, the dog developed gastrointestinal signs, and severe hypocalcemia was noted that we suspect from citrate toxicity. It is important to monitor ionized calcium during ECTR treatment if blood products are used considering the potential for citrate toxicity in a small-sized patient.

In our study, 3 dogs (cases 1, 4, and 5) had serum meloxicam concentrations measured after ECTR during hospitalization, and a rebound phenomenon was noted. This was also noted in the previous meloxicam toxicosis case report[11] that was treated with TPE. Extracorporeal toxin removal only removes toxin in the intravascular compartment. Toxin rebound can be explained by a redistribution from extravascular compartments into the intravascular compartment if ECTR elimination is faster than this redistribution.[26] Rebound is more likely in patients with severe toxicosis, a large volume of distribution, or high lipophilicity, as these factors promote expanded multicompartmental distribution.[27,28] In our study, the rebound phenomenon was likely attributed to severe toxicosis and sufficient time for tissue distribution before initiation of ECTR. In our study, the rebound phenomenon was first noted at 1 and 4 hours after completing ECTR in cases 1, 4, and 5. Walton et al[11] found the rebound phenomenon 24 hours after TPE in a dog with meloxicam toxicosis. Although the 3 dogs in our study had meloxicam rebound after ECTR, they all recovered and were discharged within 72 hours of hospitalization. Future studies are required to assess this phenomenon to investigate the optimal protocol for meloxicam toxicosis.

Saturation of charcoal HP has been reported previously, which can result in progressive reduction in toxin removal.[29] A previous case report[20] using combined charcoal HP and HD to treat ibuprofen toxicosis in a dog. Those results showed postcharcoal samples had higher ibuprofen concentrations than precharcoal samples after 2 hours of HP. These findings suggested ibuprofen could leach out of the charcoal HP device due to saturation.[20] Saturation of hemoadsorbents could be from high toxin concentration at the beginning of treatment and cell debris or proteins adhering to the hemoadsorbents.[29] Compared to the legacy charcoal HP device, hemoadsorbents in cHP have a uniform spherical shape, a smooth external surface, and a larger surface area that could minimize saturation of the HP device.[24] However, at the time of this writing, real-time measurement of serum meloxicam concentration or predicting saturation of the cHP device during HP treatment is not feasible. As a result, the cHP device was replaced in case 5 approximately 50 minutes after HP treatment started considering an extremely high dose of meloxicam ingestion (168.8 mg/kg). In case 3, the cHP device was also replaced 40 minutes after HP treatment due to suspected clotting in the first device. Our data

revealed the greatest meloxicam reduction at the time of replacement, 90% and 89% in cases 3 and 5, respectively. Even with marked meloxicam reduction in these 2 cases, their serum meloxicam concentrations at the time of replacement were still over 126 and 9 times higher than the peak therapeutic serum concentration. Our results suggest that replacement of cHP device may be required in severe toxicosis to optimize toxin removal.

Our results also showed that serum meloxicam concentrations before ECTR were lower compared to intake. Two dogs (cases 1 and 4) had serum meloxicam concentrations measured on intake. Compared to intake, meloxicam serum concentrations before ECTR had 25.6% and 47.5% reduction over 100 and 60 minutes, respectively. This finding suggests ongoing tissue distribution of meloxicam before ECTR. Previous studies[30] documented meloxicam distribution $t_{1/2}$ of approximately 0.42 hours in sheep and goats, but there are no data available in dogs. The more time passes between toxin exposure and ECTR, the more toxin distributes to the tissues, causing organ damage and rendering ECTR less efficient as blood toxin concentrations decline. Therefore, timely intervention, such as gastrointestinal decontamination or ECTR, is essential to maximize drug removal and could potentially improve patient outcome. Intralipid therapy should also be considered before initiation of ECTR to confine toxin to the intravascular compartment and decrease toxin distribution to the tissues.

There are some limitations in our study. First, the sample size was small; thus, the optimal ECTR protocol for meloxicam toxicosis cannot be determined. Second, there was limited generalizability due to the clinical case nature, and standardization of the treatment protocol was not possible. On a similar note, the time points for serum meloxicam measurements were not standardized among these 5 dogs. Additionally, as the study was conducted in the clinical setting, there was no control group included.

In conclusion, cHP markedly reduced serum meloxicam concentrations in dogs with meloxicam toxicosis and had higher meloxicam clearance and shortened meloxicam $t_{1/2}$ compared with intrinsic elimination. Our results showed that dogs with meloxicam toxicosis that underwent timely cHP had excellent short-term outcomes. Carbon HP should be considered as an extracorporeal treatment option in dogs with high-dose meloxicam ingestion.

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Supplementary Materials

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