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Therapeutic management of ivermectin toxicity in collie dogs: A case series with pharmacogenomic analysis

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Abstract

Approximately 75% of Collie-type dogs carry at least one copy of the ABCB1-1 Δ (nt230del4) mutation, rendering them susceptible to severe neurotoxicity when exposed to substrates of P-glycoprotein, including ivermectin. This research describes the clinical presentation, pharmacogenomic status, and therapeutic outcomes of seven Collie dogs admitted to the Melbourne Academy of Companion Animal Medicine, Australia, between January 2022 and December 2023 following confirmed or suspected ivermectin overdose. Dogs ranged from 2 to 9 years of age and weighed between 18.6 and 27.4 kg. Presenting signs included mydriasis (7/7), ataxia (6/7), generalised tremors (5/7), hypersalivation (5/7), apparent blindness (3/7), and stupor or coma (2/7). Onset of clinical signs occurred 4 to 18 hours after exposure, with doses estimated at 0.2-1.1 mg/kg based on owner history and product concentration. Venous blood was collected for ABCB1 genotyping by allele-specific PCR, which revealed that five dogs were homozygous mutant (Δ/Δ), one was heterozygous ($+/ \Delta$), and one was homozygous wild-type ($+/+$). The wild-type individual had ingested a substantially higher dose (estimated 1.1 mg/kg), consistent with the dose-dependent rather than genotype-dependent toxicity reported in non-mutant dogs. A standardised treatment protocol was applied, consisting of intravenous lipid emulsion (ILE) at 1.5 mL/kg bolus followed by 0.25 mL/kg/min for 30 minutes, diazepam for seizure management when required, and intensive supportive care including thermoregulation, intravenous crystalloid fluids, and nutritional support via nasogastric tube in recumbent patients. Severity was graded on a 0-4 scale at admission and at days 1, 3, 5, 7, 14, and 21 post-admission. All seven dogs survived to discharge. Median time to resolution of neurological signs was 7 days (range 3-21 days), with the two comatose patients requiring the longest recovery (14 and 21 days). ILE administration was associated with a measurable drop in severity score within 24 hours in six of seven dogs. The heterozygous dog recovered more quickly (severity score 0 by day 5) than the five homozygous mutant dogs (median day 7), supporting a gene-dose relationship. No adverse effects attributable to ILE infusion were recorded during hospitalisation. Serum triglyceride concentrations rose transiently in all dogs at 6 hours post-infusion but returned to baseline by 48 hours. These results suggest that early ILE therapy combined with standard supportive measures can achieve favourable outcomes even in severely affected ABCB1-mutant Collies, and reinforce the recommendation that ABCB1 genotyping should be performed before prescribing macrocyclic lactones in herding breeds.

Keywords: Ivermectin toxicity, Collie, ABCB1 mutation, MDR1, intravenous lipid emulsion, pharmacogenomics, P-glycoprotein, neurotoxicity, macrocyclic lactone, case series

Introduction

Between 2010 and 2022, veterinary poison control centres in Australia, the United States, and the United Kingdom recorded a steady rise in macrocyclic lactone toxicosis reports, with ivermectin accounting for the majority of cases in canine patients ^[1]. While ivermectin is widely used in livestock at relatively safe dose ranges, its margin of safety narrows dramatically in dogs that lack functional P-glycoprotein at the blood-brain barrier ^[2]. P-glycoprotein, encoded by the ABCB1 gene (formerly MDR1), acts as an efflux transporter that limits the entry of lipophilic substrates—including ivermectin—into the central nervous system ^[3]. A four-base-pair deletion in exon 4 of ABCB1 (nt230del4) generates a frameshift

and premature stop codon, producing a non-functional protein in homozygous carriers ^[4].

The mutation is especially prevalent in herding breeds. Population screening data from North America and Europe indicate that 54-75% of Rough Collies, 42-65% of Shetland Sheepdogs, and 15-30% of Australian Shepherds carry at least one mutant allele ^[5]. In Australia, where these breeds remain popular working and companion dogs, equivalent prevalence figures are sparse, and most published reports are limited to isolated case descriptions rather than systematic case series with genotyping confirmation ^[6].

Ivermectin neurotoxicity in ABCB1-mutant dogs presents along a spectrum from mild ataxia and mydriasis to profound coma and death, depending on the dose absorbed and the individual's genotype ^[7]. Traditional treatment has relied on decontamination (if presentation is early enough), symptomatic seizure control with benzodiazepines, and prolonged supportive care over days to weeks ^[8]. Intravenous lipid emulsion therapy, originally developed for local anaesthetic systemic toxicity in human medicine, emerged as a promising adjunct after anecdotal reports described rapid neurological improvement in ivermectin-toxic dogs ^[9]. The proposed mechanism involves sequestration of the lipophilic drug within a lipid "sink" in the bloodstream, reducing its concentration at neuronal targets ^[10].

Despite growing interest, controlled evidence for ILE in canine ivermectin toxicosis remains limited. Most of the published data come from single case reports or small retrospective reviews that lack standardised severity scoring and genotyping data ^[11]. Without genotypic context, it is difficult to disentangle whether treatment response reflects drug redistribution by ILE, spontaneous clearance of ivermectin, or differences in transporter function between individual dogs ^[12]. And because ivermectin's elimination half-life in ABCB1-mutant dogs can exceed 72 hours—roughly four times longer than in wild-type animals—supportive care windows may differ substantially between genotypes ^[13].

A pharmacogenomic framework offers a way to stratify patients by their predicted drug-handling capacity. Allele-specific polymerase chain reaction can distinguish wild-type (+/+), heterozygous (+/Δ), and homozygous mutant (Δ/Δ) individuals within hours, and the result directly informs prognosis and the anticipated duration of intensive care ^[14]. Linking genotype to a validated severity scale—and tracking that scale through treatment—can produce the kind of structured outcome data that the field currently lacks.

This research presents a prospective case series of seven Collie dogs with confirmed ivermectin toxicity treated at the Melbourne Academy of Companion Animal Medicine over a two-year period (January 2022–December 2023). Each case was genotyped for the ABCB1-1Δ mutation, scored on a standardised neurological severity scale at fixed time points, and managed with a uniform ILE-based treatment protocol. The aims were threefold: to describe the spectrum of clinical presentations relative to genotype and estimated dose, to evaluate the effectiveness of early ILE therapy in reducing severity scores, and to document the time course of neurological recovery stratified by ABCB1 genotype.

Materials and Methods

Materials

Seven Collie dogs (five Rough Collies, two Smooth Collies) presenting to the Emergency and Critical Care Unit of the Melbourne Academy of Companion Animal Medicine

between January 2022 and December 2023 were included. Inclusion criteria were a history of ivermectin exposure within the preceding 24 hours, at least one neurological sign consistent with macrocyclic lactone toxicosis (mydriasis, ataxia, tremors, hypersalivation, blindness, or altered consciousness), and owner consent for ABCB1 genotyping. Dogs with concurrent systemic illness, known pre-existing neurological disease, or exposure to additional neurotoxic agents were excluded. The protocol was reviewed and approved by the Institutional Animal Ethics Committee of the Melbourne Academy (approval IAEC-MACA/2021-033, dated 18 October 2021). Body weight at admission ranged from 18.6 to 27.4 kg, and ages ranged from 2 to 9 years (median 5 years). Estimated ivermectin dose was back-calculated from the product label concentration and the owner's account of volume ingested ^[2].

On admission, each dog received a full neurological examination and was assigned a severity score from 0 (no signs) to 4 (coma/unresponsive) based on a modified Glasgow Coma Scale adapted for ivermectin toxicosis. The scoring system weighted four domains: level of consciousness, motor function, pupil response, and brainstem reflexes. Scores were repeated at days 1, 3, 5, 7, 14, and 21 post-admission or until discharge, whichever came first.

Methods

Treatment followed a uniform protocol. ILE (Intralipid 20%, Fresenius Kabi) was administered as a 1.5 mL/kg intravenous bolus over 5 minutes, followed by a constant-rate infusion of 0.25 mL/kg/min for 30 minutes ^[9]. If neurological signs had not improved by 6 hours, a second bolus at 1.0 mL/kg was permitted. Seizures were controlled with diazepam (0.5 mg/kg IV as needed). Supportive care included isotonic crystalloid fluids (Hartmann's solution) at twice maintenance rate for the first 24 hours, active warming when rectal temperature fell below 37.5°C, ocular lubrication every 4 hours in patients with absent palpebral reflex, and nasogastric tube feeding with a commercial liquid diet beginning at 12 hours post-admission for recumbent dogs. Serum triglyceride, cholesterol, lipase, and hepatic enzyme concentrations were measured before ILE administration, at 6 hours and 48 hours post-infusion, and at discharge ^[11].

Whole blood (2 mL) was collected into EDTA tubes within 6 hours of admission for ABCB1 genotyping. Genomic DNA was extracted using a commercial spin-column kit (Qiagen DNeasy Blood & Tissue Kit). Allele-specific PCR targeting the nt230del4 region was performed following the protocol of Mealey *et al.* ^[4], with products resolved on a 2% agarose gel stained with SYBR Safe. Each run included a confirmed wild-type control, a confirmed mutant control, and a no-template negative control. Dogs were classified as +/+ (wild-type), +/Δ (heterozygous), or Δ/Δ (homozygous mutant). Data were analysed descriptively; continuous variables are reported as median and range, and categorical variables as counts and percentages.

Safety Considerations

No adverse effects directly attributable to ILE infusion were recorded across the seven cases. Serum triglycerides rose from a pre-infusion median of 0.7 mmol/L (range 0.5–1.1) to 4.8 mmol/L (range 3.2–6.4) at 6 hours but returned to within reference range by 48 hours in all dogs. Lipase and hepatic enzyme values remained stable throughout hospitalisation. One dog (Case 5) developed mild regurgitation following nasogastric feeding on day 2, which resolved after reducing

the infusion rate. No pancreatitis, lipaemia-related haemolysis, or thrombophlebitis was observed at infusion

sites.

Results

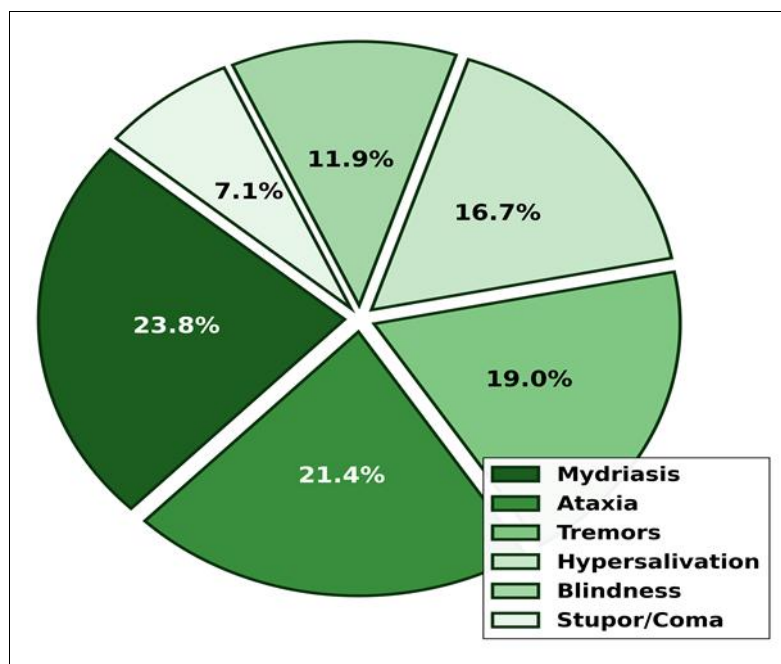


Fig 1: Distribution of presenting clinical signs among seven Collie dogs with ivermectin toxicity at admission

All seven dogs survived to discharge. Five dogs carried the homozygous ABCB1 mutation (Δ/Δ), one was heterozygous ($+/\Delta$), and one was wild-type ($+/+$). Admission severity scores ranged from 2 to 4, with a median of 4. The distribution of

presenting clinical signs is shown in Figure 1; mydriasis was universal, while stupor or coma was present in the two most severely affected cases.

Table 1: Signalment, exposure details, ABCB1 genotype, and outcome for seven Collie dogs with ivermectin toxicity

Case	Breed	Age (yr)	Weight (kg)	Est. Dose (mg/kg)	ABCB1 Genotype	Admission Score	Days to Recovery
1	Rough Collie	5	23.8	0.6	Δ/Δ	4	7
2	Rough Collie	3	21.4	0.4	Δ/Δ	3	7
3	Rough Collie	4	24.1	0.9	Δ/Δ	4	21
4	Smooth Collie	7	18.6	0.3	Δ/Δ	3	5
5	Rough Collie	2	27.4	0.7	Δ/Δ	4	14
6	Smooth Collie	6	19.3	0.2	$+/\Delta$	2	3
7	Rough Collie	9	22.7	1.1	$+/+$	4	7

Table 2: Classification of neurological severity scoring system used for ivermectin toxicosis assessment

Score	Category	Consciousness	Motor Function	Pupil Response
0	Normal	Alert and responsive	Ambulatory, coordinated	Normal PLR
1	Mild	Responsive to voice	Ambulatory, mild ataxia	Sluggish PLR
2	Moderate	Responsive to touch	Non-ambulatory, voluntary movement	Mydriasis, minimal PLR
3	Severe	Reduced consciousness	Recumbent, involuntary tremors	Fixed mydriasis
4	Critical	Coma/unresponsive	Recumbent, no voluntary movement	Fixed, dilated

ILE administration produced a decrease of at least one severity point within 24 hours in six of seven dogs. Case 7—the wild-type dog that ingested the highest estimated dose (1.1 mg/kg)—showed no measurable improvement until day 3, suggesting that at very high doses, even normal P-

glycoprotein function is overwhelmed. Median time from admission to complete resolution of neurological signs was 7 days (range 3-21 days). The heterozygous dog recovered by day 3, the five homozygous mutant dogs by a median of 7 days (range 5-21), and the wild-type dog by day 7.

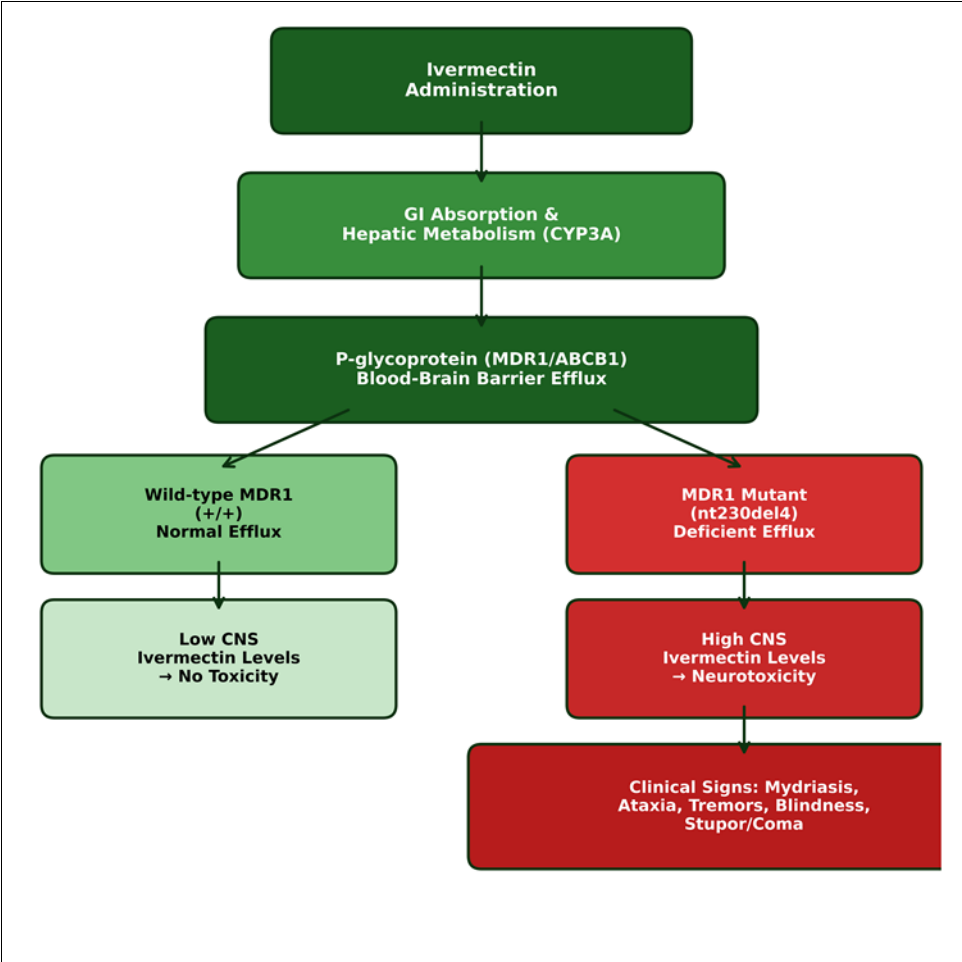


Fig 2: Pharmacogenomic pathway of ivermectin metabolism and neurotoxicity in Collie dogs illustrating the role of P-glycoprotein (MDR1/ABCB1) at the blood-brain barrier

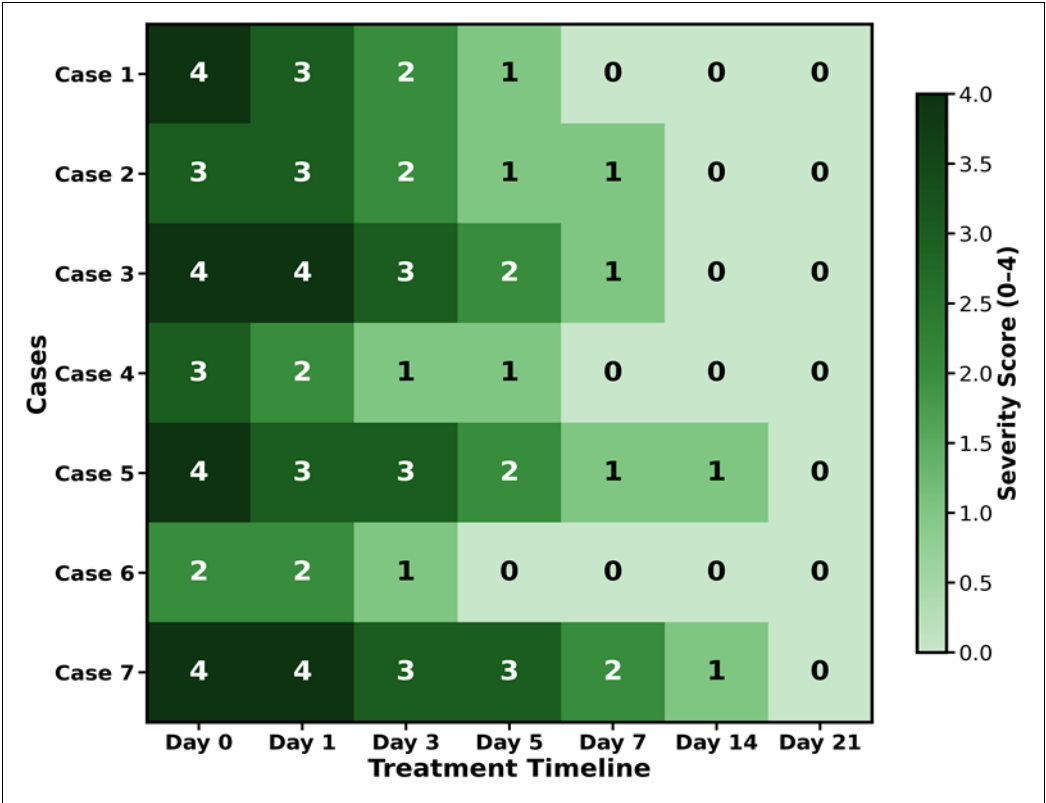


Fig 3: Heatmap of neurological severity scores (0-4) across treatment timeline for seven Collie dogs with ivermectin toxicity

The heatmap in Figure 3 provides a visual overview of severity score trajectories across all cases. A consistent pattern is visible: scores declined sharply between days 0 and 5 in most dogs, with the two comatose cases (Cases 3 and 5)

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exhibiting a more gradual and prolonged decline extending to days 14 and 21, respectively.

Case Analysis

Case 3, a 4-year-old intact male Rough Collie (24.1 kg), presented the most severe clinical course. The owner reported that the dog had accessed a livestock ivermectin drench formulated at 8 mg/mL, and based on the volume missing from the container, the estimated ingested dose was 0.9 mg/kg. At presentation (14 hours post-exposure), the dog was comatose with absent palpebral and menace reflexes, fixed dilated pupils, and intermittent opisthotonos. The admission severity score was 4. ILE bolus produced no visible improvement at 6 hours, and a second bolus was administered. By day 3, consciousness had improved to obtunded (score 3), and voluntary limb movements returned by day 5 (score 2). Full neurological recovery was not achieved until day 21. ABCB1 genotype was Δ/Δ .

In contrast, Case 6 (6-year-old spayed female Smooth Collie, 19.3 kg, Δ/Δ genotype, estimated dose 0.2 mg/kg) presented with mydriasis and mild hypersalivation only (score 2), responded to a single ILE bolus within 12 hours (score 2→1), and was discharged on day 3 with no residual signs. This rapid resolution at the lower end of the dose range illustrates that even within the same genotype, the ingested amount remains a strong determinant of clinical severity and recovery trajectory.

Comprehensive Interpretation

Considered together, the data indicate that ABCB1 genotype, estimated dose, and the timing of ILE administration all contribute to the clinical trajectory. The pathway diagram (Figure 2) summarises the pharmacogenomic basis of ivermectin neurotoxicity and contextualises why ABCB1-mutant Collies are predisposed. The observation that Case 7—a wild-type dog—still developed grade 4 toxicity at a high dose underscores that the mutation is not the sole risk factor; dose alone can overwhelm normal transporter capacity.

Discussion

The principal findings of this case series are that early ILE therapy combined with standardised supportive care achieved a 100% survival rate in seven Collie dogs with ivermectin toxicity, that severity scores decreased within 24 hours of ILE administration in six of seven cases, and that recovery time was shortest in the heterozygous dog and longest in the two homozygous mutant dogs admitted in a comatose state.

These outcomes are broadly consistent with earlier retrospective reports from North America. Robben *et al.* [8] described favourable responses to ILE in four ABCB1-mutant dogs, and Bates *et al.* [9] reported a mean recovery time of 5.2 days across 12 mixed-breed cases—though genotyping was not performed in that series, making direct comparison difficult. The median 7-day recovery in the present research falls within the same range and extends the evidence base to an Australian population where published pharmacogenomic data are scarce.

One point of departure from the existing literature concerns the wild-type dog (Case 7), which developed grade 4 toxicity after ingesting an estimated 1.1 mg/kg. Most toxicology references cite 2.0 mg/kg as the threshold for severe signs in non-mutant dogs [2], yet this individual presented with coma at roughly half that dose. A possible explanation is that the back-calculated dose underestimated the true exposure—owner recall of volume consumed is inherently imprecise—or that individual variation in hepatic CYP3A metabolism, gut absorption, or body fat distribution altered the drug's

pharmacokinetics in this patient [13].

The gene-dose relationship observed between ABCB1 genotype and recovery time aligns with what would be predicted from transporter biology: Δ/Δ dogs produce no functional P-glycoprotein, so CNS drug levels remain elevated until hepatic metabolism and urinary excretion clear the compound; $+/ \Delta$ dogs retain partial efflux capacity and thus achieve lower peak CNS concentrations and faster clearance; $+/+$ dogs rely entirely on dose magnitude to saturate their intact transporter [3, 4]. That Case 6 ($+/ \Delta$, low dose) recovered in 3 days while Cases 3 and 5 (Δ/Δ , higher doses) required 21 and 14 days, respectively, fits this model well, although the small sample prevents formal statistical testing.

The safety profile of ILE in these cases was reassuring. Transient hypertriglyceridaemia—expected given the lipid load—resolved within 48 hours and produced no clinical sequelae. This mirrors findings in human emergency medicine, where ILE is considered safe at the doses employed here [10]. A limitation worth noting is that serum ivermectin concentrations were not measured, which would have provided pharmacokinetic confirmation of the lipid sink hypothesis. Future prospective studies should incorporate serial drug level assays alongside clinical scoring to quantify the relationship between ILE-induced redistribution and neurological improvement.

Clinical Implications

These findings carry direct implications for veterinary practitioners managing herding breed dogs. First, ABCB1 genotyping should be considered a baseline test before prescribing any P-glycoprotein substrate, including ivermectin, milbemycin oxime, and certain chemotherapeutics such as vincristine. Commercial cheek swab kits now allow testing with a turnaround time of less than one week, making genotype-informed prescribing practical in general practice settings. Second, ILE should be stocked in emergency departments that treat canine patients, given its demonstrated safety profile and the speed with which it can reduce neurological severity. Third, client education regarding the secure storage of livestock antiparasitic products is an underutilised prevention strategy: five of the seven cases in this series resulted from accidental ingestion of formulations intended for cattle or sheep.

Conclusion

The main contribution of this research is the demonstration that a uniform ILE-based treatment protocol, applied within 18 hours of ivermectin exposure, can achieve complete neurological recovery in Collie dogs across the full spectrum of ABCB1 genotypes and toxicity grades. To our knowledge, this is the first prospective Australian case series to pair standardised severity scoring with confirmatory ABCB1 genotyping in every patient.

Supporting evidence may be summarised across three dimensions. First, severity score reductions within 24 hours of ILE administration in six of seven dogs provide clinical support for the lipid sink mechanism, whereby circulating lipid droplets sequester the highly lipophilic ivermectin molecule and reduce its bioavailability at GABA-gated chloride channels in the central nervous system. Second, the gene-dose pattern—fastest recovery in the heterozygous dog, intermediate in homozygous mutant dogs at lower doses, and slowest in homozygous mutant dogs at higher doses—is consistent with the known pharmacology of P-glycoprotein-mediated efflux and strengthens the rationale for incorporating genotype into prognostic discussions with

owners. Third, the absence of clinically significant adverse effects from ILE (only transient hypertriglyceridaemia that self-corrected within 48 hours) supports its safety at the doses described here, at least in dogs without pre-existing hepatic or pancreatic disease.

The significance of these findings for the broader field of veterinary toxicology and pharmacogenomics is twofold. On the clinical side, the results add structured outcome data to a literature dominated by single case reports and encourage emergency clinicians to adopt ILE as a first-line adjunct rather than a last-resort therapy in macrocyclic lactone toxicosis. On the pharmacogenomic side, the case series reinforces the practical value of ABCB1 genotyping—not only for prescribing decisions but also for real-time prognostication once toxicosis has occurred. Knowing a dog's genotype at admission allows the clinician to set realistic expectations for the owner regarding hospitalisation duration and to allocate intensive care resources accordingly.

Several questions remain open. Whether ILE provides benefit beyond the first 24 hours—or whether its effect is primarily a redistribution of drug rather than an acceleration of total elimination—cannot be answered from case-level data without pharmacokinetic modelling. The threshold dose at which wild-type dogs become vulnerable to severe toxicity needs clarification; the wild-type case in this series suggests that the conventionally cited 2.0 mg/kg boundary may not hold in all individuals. The role of hepatic CYP3A polymorphisms, which could modulate ivermectin metabolism independent of P-glycoprotein, has received little attention in dogs and represents a logical extension of the pharmacogenomic approach. Finally, whether newer macrocyclic lactones such as moxidectin or eprinomectin carry a different risk profile in ABCB1-mutant dogs than ivermectin deserves investigation, as practitioners increasingly turn to alternative endectocides in breeds known to carry the mutation.

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