RESEARCH NOTE: Heparinized Saline for Catheter Patency

Heparinized saline flushes are a common means of maintaining peripheral IV catheter patency in human and veterinary medicine. The assumed benefit is that heparin acts as an antithrombotic agent and its use would result in fewer blood clots, and therefore fewer occluded IV catheters; however, potential risks from IV heparin include allergic reactions, drug interactions, and inadvertent bleeding complications. This study examined whether heparinized saline (10 IU/mL) would be more effective at maintaining peripheral IV catheter patency than 0.9% sodium chloride.

Each of 30 healthy dogs had an 18-gauge, polyurethane IV catheter placed in either the right or left cephalic vein. Dogs were randomly assigned into 1 of 3 groups: a group receiving heparinized saline flushes (HS), a group receiving 0.9% sodium chloride flushes (S), and a control group receiving no catheter flushes during the study. No significant difference was found between the HS and S groups pertaining to maintenance of catheter patency; however, more S group dog catheters, from which aspiration of blood could not be performed compared to HS group dogs, suggested that heparinized saline flushes may be preferred for peripheral catheters used for serial blood collection. Catheter size and indwelling period may play a role in phlebitis and subsequent IV catheter occlusion; thus, limitations of this study included the use of only one catheter size (18-Ga) and a study period of only 42 hours.

High-Dose Insulin Therapy for Refractory Toxicosis

Diltiazem, a calcium-channel blocker (CCB), affects vascular smooth muscle, myocardial contractility, and AV nodal conduction. Toxicosis of CCBs can be fatal. The use of IV lipid emulsion (ILE) as an antidote for fat-soluble toxicants has gained popularity in humans when traditional therapies have failed. High-dose insulin (HDI) therapy has recently been shown to be superior to traditional therapies for CCB and beta-blocker toxicoses. This case report describes the use of ILE and HDI in the treatment of refractory diltiazem toxicosis in a 4-year-old Pomeranian, which was presented 2.5 hours postgastointestinal diltiazem at 79 mg/kg, with the reported canine LD₅₀ of 50 mg/kg. Bradycardia developed during initial evaluation, and blood pressure progressively decreased. Continuous ECG showed progression to second-degree AV blockade.

Treatment included IV fluids, charcoal, calcium gluconate, glucagon, and dopamine. Hypotension and bradycardia (with atrial standstill) progressed, and HDI and ILE were initiated. Regular insulin was dosed at 1 U/kg IV with concurrent CRI dextrose supplementation. ILE was given as a bolus of 20% lipid (1.5 mL/kg IV), followed by a CRI (0.25 mL/kg/hr IV). Blood pressure improved within 30 minutes, but severe bradycardia (35 bpm) persisted. HDI was continued with an insulin CRI (1 U/kg/hr). Significant improvement followed, HDI and ILE CRIs were weaned, and the patient was discharged 45 hours after admission. HDI and ILE are relatively new treatments in veterinary medicine but show promise for treatment of CCB toxicosis.

Commentary

For many in critical care and toxicology, ILE as a “lipid sink” has finally sunk in. ILE is a common antidote for fat-soluble drug toxicity. HDI therapy has been used effectively for toxicities such as severe beta-blocker and CCB poisoning. Conventional therapies (eg, atropine, dopamine, glucagon, calcium) often fail to reverse the toxic effects in severely poisoned patients. Moreover, prolonged use of catecholamines for refractory bradycardia and hypotension results in decreased cardiac output, decreased perfusion, and increased myocardial oxygen demand. HDI therapy has several benefits, including increased intracellular glucose transport, increased inotropy, and vasodilation. Adverse effects include hypoglycemia and hypokalemia as glucose and potassium are shifted from the extracellular to the intracellular space. While more research is needed, HDI therapy should be considered when conventional therapy is ineffective in severe poisoning cases.—Garret Puchtinger, VMD, DACVECC

Source


