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MANAGEMENT OF ANAPHYLACTIC SHOCK

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INTRODUCTION

Anaphylaxis represents the most severe form of allergic reaction. It results from the immunologically induced release of mast cell and basophil mediators, such as histamines and leukotrienes, after exposure to a specific antigen in previously sensitized animals; these mediators target blood vessels and smooth muscle. It has a rapid onset with multiple organ-system involvement. If medical attention is delayed, death may occur from cardiovascular collapse and/or airway obstruction.

Examples of antigens known to cause anaphylaxis include drugs such as penicillins, asparaginase and cephalosporins, vaccines, snake venoms, food allergens, insects such as bees, wasps and hornets, and blood and blood product transfusions.

CLINICAL SIGNS

In an anaphylactic reaction a wide variety of clinical signs may be observed: restlessness, urticaria, pruritis, angioedema (particularly of the soft tissues of the head), upper airway swelling and stridor, upper airway obstruction, wheezing, bronchospasm, tachycardia, pale mucous membranes, vomiting, diarrhea, seizures and hypotensive shock.

The onset is usually sudden (within minutes to an hour after exposure to the offending antigen), and signs may last up to 24 hours. With treatment, one can expect resolution of clinical signs within hours, but a small proportion of anaphylactic reactions will follow a biphasic course (with a second phase seen in 6 to 12 hours in spite of successful initial treatment). Should anaphylaxis recur, treatment must be reinstated a second time.

There are species specific reactions. In dogs, the major organ system involved in acute anaphylaxis is the liver, specifically the hepatic veins, and the gastrointestinal tract. Dogs will show initial excitement followed by vomiting, defecation, and urination. Constriction of the hepatic vein causes portal hypertension and pooling of blood in the viscera, associated with signs of shock. Bowel edema and fluid translocation also occur, resulting in diarrhea (which may be hemorrhagic). Coma, seizures, hypovolemic shock and death may occur. In cats, the major ‘shock’ organs are the lung and the gastrointestinal tract. Cats show vigorous facial and head pruritis (scratching), followed by dyspnea, salivation, vomiting, incoordination and collapse.

MANAGEMENT

Anaphylaxis should be considered a medical emergency. Parenteral epinephrine is the recommended first line treatment in anaphylaxis. Stimulation of alpha adrenoceptors improves blood pressure and coronary perfusion; stimulation of beta-1 adrenoceptors has both positive inotropic and chronotropic cardiac effects; stimulation of beta-2 adrenoceptors causes and reduces the release of inflammatory mediators.

In anaphylactic shock, epinephrine is best administered either intramuscularly (IM) or as an intravenous (IV) constant rate infusion (CRI). Bolus administration of IV epinephrine has been associated with the induction of fatal cardiac arrhythmias and myocardial infarction. The intravenous route should be reserved for those with unresponsive anaphylaxis. The use of an IV CRI of epinephrine reduces the risk of these adverse effects, as lower doses of epinephrine are used. For most animals, only one dose of IM epinephrine is needed, although repeat doses may be given at 5-15 minute intervals until hemodynamic and respiratory status improves. An alternative to repeat IM dosing is intravenous CRI administration.

Stepwise approach to the management of acute anaphylaxis

1) Secure a patent airway and provide 100% Oxygen
2) For cardiovascular collapse: Use Epinephrine
   a) If peracute: 0.01 mg/kg of 1:1000 epinephrine administered IM, sublingually, endotracheally or SQ. Repeat every 15-20 minutes.
   b) If severe reaction: administer 0.1ml/kg of 1:10,000 epinephrine IV (prepare by taking 1 ml of 1:1000 (1mg/ml) and dilute in 9 mls saline. Look for improvement in systemic blood pressure and perfusion parameters. If there is a poor response repeat the epinephrine administration at ½ the dose – administer this IM. If still unresponsive, move to an epinephrine CRI.
3) Following IV catheter placement, provide an isotonic crystalloid fluid bolus at a ‘shock’ dose (90 ml/kg for dogs and 45-60 ml/kg for cats); Hetastarch may also be used at a dosage of 20 ml/kg rapid IV, followed by a CRI of 1-2 ml/kg/day.
4) Use an antihistamine such as diphenhydramine at a dosage of 0.5-2.0 mg/kg IM every 8 hours as necessary.
5) Once circulatory collapse is reversed, administer a rapidly acting steroid IV. Examples include prednisolone sodium succinate at a dosage of 8-15 mg/kg slow IV, or methylprednisolone sodium succinate at 2.0-20 mg/kg over 15-20 minutes, or dexamethasone sodium phosphate at 0.5-4.0 mg/kg slow IV.
   Steroids are not very useful during the crisis, but may control ongoing anaphylaxis due to persistent mediator release.
6) If life threatening hypotension persists, use an Epinephrine CRI: 4.0 mg epinephrine in 1 liter of saline; deliver at 1ml/kg/hr, or titrate to effect (can drip in to effect). Alternatively, a Dopamine CRI can be used at a dosage of 5-10 ug/kg/min.
7) If respiratory distress occurs, administer Aminophylline at 5 mg/kg over 10 minutes, administered IV, or Terbutaline at 0.01 mg/kg, administered sub-cutaneously.

References available from author upon request.