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Abstract

Anaphylaxis is a potentially life-threatening inflammatory and immune reaction to exposure of an allergen, or antigen. Patients may experience anaphylaxis after exposure to insect stings, medications, or blood products. Anaphylactic patients experience distributive shock causing potent vasodilation and require treatment with vasopressors, such as epinephrine. They may also develop complications such as arrhythmias, hemoperitoneum, coagulopathy, severe gastrointestinal signs, hypoglycemia, and respiratory distress. Treatment of anaphylaxis involves blood pressure management, downregulation of histamine production, and nursing care. Mortality in anaphylaxis is generally low, and while anaphylactic patients require intense treatment and nursing care, they often recover well.

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EMERGENCY MEDICINE/CRITICAL CARE

Understanding and Managing Anaphylaxis

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Anaphylaxis is a potentially life-threatening allergic reaction. In veterinary patients, insect sting, medication, or blood product exposures are common causes of anaphylaxis. Clinical signs may differ in dogs and cats, but hypotension caused by global vasodilation is commonly present in both species. Treatment is supportive, and veterinary nurses play a key role in monitoring and caring for these patients. With timely, aggressive treatment, patients often make a full recovery.

PATHOPHYSIOLOGY OF ANAPHYLAXIS

Simply put, anaphylaxis is an overreaction of the immune system to a specific antigen. Antigens are proteins that declare the identity of their source (e.g., allergens, viruses, bacteria). In a normal immune response, detection of a foreign antigen triggers an inflammatory response that ends with the destruction and removal of the intruder; however, in hypersensitivity reactions, including anaphylaxis, the resulting inflammation is excessive, causing damage to the body itself.

Immunoglobulins

Initial exposure to an antigen creates a strong T-cell response. T cells are white blood cells (WBCs) that can attack infected cells directly or promote inflammation by releasing cytokines and stimulating B cells (another type of WBC) to produce antibodies. Antibodies bind to antigens, thereby neutralizing them or flagging them for destruction by other cells. B cells can produce 5 major types of antibodies, also known as immunoglobulins (Ig): IgM, IgG, IgA, IgD, and IgE (**BOX 1**).[1] The 2 immunoglobins that play a role in anaphylaxis are IgE and IgG.[2]

IgE

IgE is responsible for most hypersensitivity reactions seen in anaphylaxis; however, IgE reactions never come from primary antigen

Take-Home Points

- Describe the immune system and the role of the immunoglobulin E response in hypersensitivity reactions.
- Describe presenting clinical signs and secondary complications of anaphylaxis in dogs and cats.
- Demonstrate a clear understanding of distributive shock and the challenges of treating and providing nursing care for these patients.
- Explain the use of diagnostic imaging and hematology/chemistry monitoring in anaphylactic patients.

BOX 1

Role of Immunoglobulins in the Immune Response[1]

IgA Initiates secretion of mucus to protect against pathogens

IgD Regulates B-cell activation and differentiation into plasma cells

IgE Initiates hypersensitivity reactions and has anthelmintic activity

IgG Triggers the complement system in the immune response

IgM Binds to bacterial surfaces and activates complement cascade

exposures. Instead, the initial exposure causes IgE to bind to mast cells and basophils in large amounts. Subsequent exposures to the same or similar antigens can cause mast cell degranulation, causing release of histamine that triggers a hypersensitivity reaction.[3]

IgG

IgG can trigger an anaphylactic reaction independent of histamine release by binding to monocytes, macrophages, and neutrophils. IgG hypersensitivity reactions do not require a primary and secondary exposure to an antigen; rather, a patient may have a reaction on the first exposure.[4] IgG hypersensitivity reactions are also called anaphylactoid reactions because although they produce the same clinical signs as IgE anaphylaxis reactions, they do not involve histamine.[4] Treatment for both IgE and IgG reactions is the same.[4]

Histamine

Histamine is a proinflammatory mediator that is stored in mast cell granules. When released, it binds to histamine receptors (**BOX 2**), stimulating the release of more proinflammatory mediators in the form of cytokines and chemokines.[5] Histamine receptors are concentrated in the gastrointestinal tract, lungs, and vasculature of the portal system in the liver. The location of the histamine receptors most affected during anaphylaxis is considered the "shock organ." Shock organs drive the primary clinical signs of anaphylaxis and differ by species (see **VASODILATION AND SHOCK** section).

Types of Hypersensitivity Reactions

There are 4 types of hypersensitivity reactions. Type I hypersensitivity reactions happen when an antigen binds with IgE that is already bound to mast cells and basophils, triggering cellular degranulation and subsequent release of histamine, along with several proinflammatory mediators such as heparin, protease, serotonin, and bradykinin. The arachidonic acid chain oxidizes and produces leukotrienes, which are responsible for the urticaria (hives), angioedema (facial swelling), and bronchoconstriction that can be seen in type I hypersensitivity reactions. Anaphylaxis is usually a severe type I hypersensitivity reaction.[2]

Type III hypersensitivity reactions are IgG reactions that are independent of IgE and histamine. There is no mast cell degranulation in type III reactions. Neutrophils and monocytes are recruited, and immune mediators such as platelet activation factor, serotonin, prostaglandins, and interleukins cause the reaction. Type III hypersensitivity reactions are more commonly secondary to drug administration (serum sickness).[2]

Type II hypersensitivity reactions are cytotoxic or autoimmune, such as in immune-mediated hemolytic anemia or immune-mediated thrombocytopenia. Type IV hypersensitivity reactions are delayed reactions, such as keratoconjunctivitis sicca or flea allergy dermatitis. Neither type II nor type IV hypersensitivity is considered an allergic reaction; rather, these cause immune-mediated destruction of the patient's own cells.[2]

BOX 2

Histamine Receptor Functions[5]

H1R Affects smooth muscle contraction and vascular permeability; activates cytokine and chemokine production; activates platelet activation factor; initiates vasodilation

H2R Increases gastric acid secretion, cardiac output, and heart rate

H3R Affects neurotransmission and homeostatic regulation of energy levels; inhibits norepinephrine

H4R Causes mast cell degranulation; activates cytokine, chemokine, and platelet activation factor production; promotes immunomodulation

Vasodilation and Shock

In anaphylaxis, excessive release of histamine and inflammatory mediators leads to global vasodilation, causing hypovolemia, which causes a drop in peripheral blood pressure and subsequent distributive shock. The vasodilation also causes vasculature permeability, which can lead to fluid shifting and third spacing, worsening hypovolemia.

In dogs, the shock organs include the gastrointestinal tract, liver, and portal veins; therefore, dogs experiencing anaphylaxis may present for vomiting, diarrhea, and collapse. Hepatic arterial vasodilation, increases in hepatic venous outflow pressure, and hepatic portal congestion lead to an elevation in alanine aminotransferase (ALT) and a "halo sign" around the gallbladder on abdominal ultrasonography (**FIGURE 1**).[2]

In cats, the lungs are the main shock organs, although the gastrointestinal tract and spleen are also involved. Anaphylaxis in cats also causes profound peripheral vasodilation and gastrointestinal signs.[2] Feline patients with anaphylaxis often present in respiratory distress due to bronchoconstriction. Hemoconcentration may also be present due to splenic contraction.

FIGURE 1. Gallbladder edema, or "halo sign," on abdominal ultrasonography. The edema around the gallbladder is caused by portal venous congestion. While gallbladder edema does not rule out other disease processes, in canine patients that present for acute vomiting, diarrhea, or collapse, a halo sign may be indicative of an anaphylactic reaction.

Disseminated Intravascular Coagulation

Global vasodilation and inflammation can also trigger systemic inflammatory response syndrome (SIRS). SIRS is recognized as global inflammation that is dysregulated by the immune system and can be seen in disease processes such as anaphylaxis, sepsis, burns, trauma, or severe pancreatitis.[6]

Four SIRS criteria have been proposed in veterinary medicine: changes in heart rate; tachypnea; fever; and changes in WBC count, with an increase in band neutrophils.[6] SIRS can initiate the clotting cascade and lead to disseminated intravascular coagulation (DIC).

As an inflammatory process, anaphylaxis also stimulates the coagulation cascade. Inflammatory mediators trigger the inflammatory cycle via factor VIIa, which then starts coagulation in the body. This process triggers DIC. In early-stage DIC, the body produces micro-clots that can lodge in small vessels of vital organs, where they block blood flow and can cause ischemia and organ damage. This may lead to increases in blood values (e.g., ALT, alkaline phosphatase, blood urea nitrogen, creatinine) or cause cardiac arrhythmias if the clots lodge in the vessels supplying the heart.[7]

Ongoing consumption of clotting factors and platelets eventually leads to late-onset DIC. In late-onset DIC, patients become hypocoagulable and thrombocytopenic. These patients may have hemoperitoneum and may be predisposed to spontaneous bleeding. Patients with late-onset DIC should be administered fresh frozen plasma.[7]

CAUSES OF ANAPHYLAXIS

The most common cause of anaphylaxis in veterinary patients is Hymenoptera envenomation.[2] This includes bites or stings from bees, wasps, and hornets. The venom contains protein antigens and enzymes that trigger IgE reactions. Patients presenting with signs of anaphylaxis should be thoroughly examined for stingers, which are often found on the lips, paw pads, tongue, or hard palate.

Vaccines can cause anaphylaxis, but it is rare. In 1 study, approximately 0.19% of dogs receiving vaccines experienced an adverse reaction; of these, most were mild or moderate.[8] Male small-breed dogs experienced the most reactions, and multiple vaccines given at the same time increased risk. Vaccine reactions

are due to serum, albumin, and stabilizers in the vaccine.

Some ocular medications, particularly ones that contain tetracycline and aminoglycosides, may cause anaphylaxis in feline patients.[2] Most affected cats show cardiovascular collapse and respiratory distress; however, survival rates are high.

Blood products may cause IgE or IgG anaphylactic reactions. Anaphylactic reactions are most common with plasma products, due to WBC degranulation and release of vasoactive mediators while stored.[9]

Morphine may cause histamine release, especially when given quickly intravenously. Buprenorphine has also been associated with anaphylaxis.[10]

Mast cell tumors can cause hypersensitivity reactions, especially when manipulated. Darier signs (erythema and wheal formation) caused by mast cell degranulation may be seen in these patients. Mast cell degranulation may also stimulate H2 receptors and cause profound gastrointestinal signs and shock. Patients that are having mast cell tumors removed may be dosed with diphenhydramine and famotidine prior to surgery to reduce the risk of anaphylaxis during tumor manipulation.[11]

TRIAGE OF ANAPHYLACTIC PATIENTS

Canine patients with anaphylaxis often present with vomiting, diarrhea, and/or collapse. Feline patients may present with respiratory distress in addition to gastrointestinal signs, collapse, and hypotension. Vasodilation and subsequent hypotension can progress to shock in both feline and canine patients.[2]

In some patients, anaphylaxis signs may be subtle and short-lived and seemingly resolve prior to or during presentation. These patients should still be monitored closely and, ideally, hospitalized in case they have a second delayed reaction (i.e., a biphasic reaction).[2]

On presentation, suspected anaphylactic patients should receive a full TPR (temperature, pulse, and respiration), in addition to blood pressure assessment. Patients with anaphylaxis may present with mild to moderate hypotension, pale gums, tachycardia with thready or weak pulses, and mild hypothermia. Feline patients may also have tachypnea or respiratory distress. Clinical signs and vital derangement may be mild or substantial. Urticaria and/or angioedema may or may not be present.

Abdominal-focused assessment with sonography for trauma (AFAST) may reveal gallbladder edema, which is often referred to as a "halo sign" (**FIGURE 1**). Up to 50% of anaphylactic patients also present with free fluid in the abdomen.[12] Patients with abdominal effusion rarely need transfusions or surgery. A retrospective study on anaphylaxis and hemoperitoneum showed that none of the patients required surgery or blood transfusions.[12]

After or during TPR and blood pressure measurements and AFAST, vascular access should be obtained. Typically a peripheral IV catheter is sufficient, although jugular or intraosseous catheters can be placed if a peripheral catheter is unobtainable. A minimum database of blood glucose, lactate, blood gas, packed cell volume/total solids, and a serum chemistry panel with ALT should be performed on a sample collected directly from the IV catheter, if possible.

Elevations in ALT are common in dogs with anaphylaxis, while cats are more likely to have hemoconcentration secondary to splenic contraction from hypoxia. Lactate may be elevated in both species. Coagulopathy (elevations in prothrombin and activated partial thromboplastin time) may be present in severely affected patients. Electrolyte derangements and hypoglycemia or hyperglycemia caused by shock and gastrointestinal effects may be seen. These patients also often have a metabolic acidosis.[2]

INITIAL STABILIZATION AND TREATMENT OF ANAPHYLACTIC PATIENTS

Quick recognition of anaphylaxis is important for stabilization and treatment. A brief but thorough history should be collected, including current medications. As steroids are sometimes used to treat allergic reaction/anaphylaxis, it is important to ask whether the patient is on steroids or NSAIDs, as combination or high doses of these drugs may cause the patient to develop gastrointestinal ulcers.[13]

An initial balanced crystalloid bolus (typically 5 to 15 mL/kg) is often administered through the IV catheter to correct hypovolemia and hypotension.

Epinephrine can be administered intramuscularly or intravenously. Epinephrine is an adrenergic agonist that promotes vasoconstriction to help combat the vasodilation and distributive shock in anaphylactic patients. It also has some inotropic effects and may help increase contractility; however, it is important to note that intravenous epinephrine can cause ventricular arrhythmias. While epinephrine does not directly antagonize histamine, it does block some production. Epinephrine can be given as an initial bolus of 0.01 mg/kg and then be run as a constant-rate infusion (CRI; 0.025 to 0.3 µg/kg/min).[13]

Histamine blockers may be administered; however, there is no general consensus on their effectiveness. H1 blockers (e.g., diphenhydramine, chlorphenamine) might inversely antagonize H1 receptors toward an inactive state, decreasing the systemic effects of histamine.[2] H1 blockers may not be as effective in anaphylactic patients and may slightly worsen hypotension. H2 blockers (e.g., famotidine, cimetidine) may help reduce acid production in the stomach.[5] There is limited literature on their efficacy, but they may help with gastrointestinal signs in anaphylactic patients.[13]

Steroids are not indicated in acute anaphylaxis cases. Steroids may worsen gastrointestinal signs, especially in the face of hypotension and hypoperfusion of the gastrointestinal tract. In stable patients, steroids may downregulate inflammatory mediators and the arachidonic acid cascade, which may help decrease release of inflammatory mediators.

Feline patients that present with respiratory distress should receive a bronchodilator, such as terbutaline or albuterol, to relieve bronchoconstriction caused by histamine release. However, bronchodilators may cause ventricular arrhythmias or sinus tachycardia; therefore, these patients should be monitored with telemetry or electrocardiography.[13] Patients with respiratory distress should also be monitored with oxygen support, such as in an oxygen cage.

Patients that develop coagulopathy or late-onset DIC may need fresh frozen plasma to help replace coagulation factors. Additionally, plasma helps with volume expansion in patients with hypovolemia. Fresh frozen plasma may also help replace albumin, which helps with hypoalbuminemia and aids in volume expansion.

NURSING CARE FOR ANAPHYLACTIC PATIENTS

Anaphylactic patients may be critically ill and require attentive and intensive nursing care. Aside from their potentially profound hypotension, anaphylactic patients may have complications such as arrhythmias, hypoglycemia, coagulopathy, nausea, diarrhea, and changes in mentation.

Comfort Care, Cleanliness, and Nutrition

Anaphylactic patients often have gastrointestinal signs such as nausea, vomiting, and diarrhea. Diarrhea may cause scald and skin ulceration if left on patients. Nursing care such as changing bedding frequently and as needed and wiping or rinsing feces from skin and fur helps keep patients clean and comfortable. Patients with longhaired tails may benefit from tail wraps to stay clean (**FIGURE 2**). Tail wraps should be changed if they become soiled or wet, as this can also cause ulceration of the skin.

Large recumbent dogs may benefit from Foley urinary catheter placement for cleanliness. Patients that can walk should be walked frequently to allow urination and defecation. Anaphylactic patients often receive fluid boluses and may need to urinate more frequently during their initial hospital stay. Small dogs and cats that are recumbent should have regular bladder assessments, and if they are not urinating on their own, the veterinary nurse should consult with the case veterinarian to discuss whether bladder expression or

FIGURE 2. Patient cleanliness is imperative as part of good nursing care. Anaphylactic patients with diarrhea may benefit from a tail wrap to help keep feces out of the fur on the tail. The wrap should be changed immediately if soiled.

intermittent urinary catheter placement may be beneficial or if a Foley urinary catheter can be placed.

Bedding should be changed regularly to remove fur, feces, urine, and other soil and to help keep patients clean. Bedding should be well padded and comfortable. Large or thin patients require padding around bony parts, such as hips, elbows, and stifles, to help prevent decubital ulcers (pressure sores). Recumbent patients should have recumbency care performed every 3 to 6 hours to help prevent atelectasis as well as decubital ulcers.

Once patients are stable, nutritional support should be offered. Patients may require bland diets if they have continuous gastrointestinal signs; otherwise, palatable food should be offered. While hospital stays for anaphylaxis are normally relatively brief, the gastrointestinal distress on presentation, loss of coagulation factors, drops in albumin due to fluid resuscitation and vasodilation, and increase in metabolic demand due to shock make replacing lost protein and intake of calories vital to recovery in stable patients. These patients should be offered water by mouth once stable and mentally appropriate to drink.

Monitoring

Anaphylactic patients should have frequent and dedicated monitoring until they achieve cardiovascular stability. These patients may initially have dynamic changes in vitals, blood pressure, mentation, and cardiac rhythm.

Blood pressure monitoring is vital in anaphylactic patients. Oscillometric blood pressure monitoring may overestimate low blood pressure, which is common in patients with anaphylaxis. The gold standard for blood pressure monitoring is direct arterial blood pressure monitoring; however, this is not always financially feasible for clients or technically feasible for hospitals. Therefore, Doppler ultrasonography monitoring, while also not completely accurate, should be used in anaphylactic patients. Trending of blood pressure and monitoring for drops is more important than a single reading. Additionally, blood pressure monitoring can help guide vasopressor therapy, such as epinephrine CRIs.

Patients with anaphylaxis may develop sinus tachycardia or ventricular arrhythmias and should receive continuous electrocardiogram monitoring. In-hospital telemetry is advantageous since it allows for mobility with fewer cords and wires in which the patient may get tangled.

Full TPR should be assessed regularly in anaphylactic patients to watch for changes in hemodynamic status. Increases or sudden drops in heart rate may indicate hypovolemia or impending cardiac decompensation. Hypothermia is common in anaphylactic patients; therefore, these patients may require heat support. Feline patients or patients that have bolused fluid resuscitation or colloid therapy should be on respiratory watches and monitored at least every hour for worsening respiratory distress or fluid overload.

OUTCOME

Outcome is generally good for anaphylaxis patients with aggressive treatment. Mortality in anaphylaxis and severe anaphylaxis is less than 15% based on 1 study.[14] Negative prognostic indicators include elevated serum phosphorus, hypoglycemia, and coagulopathy. Early recognition of anaphylaxis and aggressive stabilization can help increase survival in patients with severe anaphylaxis.[14]

SUMMARY

Anaphylaxis is a potentially life-threatening immune reaction to an antigen, or allergen. It is typically a type I hypersensitivity reaction, with IgE activation and histamine release. The most common source of anaphylaxis is Hymenoptera (such as bees, hornets, or wasp) stings. Canine patients that present for anaphylaxis may present with vomiting, diarrhea, collapse, and potentially cardiovascular collapse. Feline patients may present for respiratory distress, gastrointestinal signs, and shock. Diagnostics for anaphylaxis may show gallbladder edema and elevations in ALT in canine patients, and feline patients may have hemoconcentration. Both species may have changes in lactate, coagulation factors, and other chemistry values such as glucose. Treatment of anaphylaxis aims to reverse the vasodilation from shock, treat bronchoconstriction, and potentially reduce histamine release or receptor activation. TVN

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1. **Which immunoglobin is responsible for most hypersensitivity reactions?**
   a. IgM
   b. IgG
   c. IgA
   d. IgE

2. **Most anaphylaxis is caused by which type of hypersensitivity reaction?**
   a. Type I
   b. Type II
   c. Type III
   d. Type IV

3. **The drug of choice to treat vasodilation caused by anaphylaxis is:**
   a. Dobutamine
   b. Epinephrine
   c. Pimobendan
   d. Dexamethasone sodium phosphate

4. **What is not considered part of appropriate nursing care for anaphylactic patients?**
   a. Recumbency care in recumbent patients
   b. Tail wrap for patients with profuse diarrhea
   c. Nutritional support
   d. Coupage

5. **What is the most common cause of anaphylaxis in veterinary patients?**
   a. Insect stings
   b. Blood products
   c. Vaccines
   d. Eye drops