Garbage Toxicoses - Carrion Toxicoses - Bacterial Food Poisoning

Specific Agents | Major Species | Usual Time of Onset | Usual Duration (if survives)
--- | --- | --- | ---
Bacterial toxins (food poisoning; most garbage poisonings, most carrion toxicoses; bacteria-contaminated Christmas tree water) | Small animals | Minutes to hours | Days; very often lethal
Endotoxins and enterotoxins | All species | Minutes to hours | Days; often lethal
Staphylococcal enterotoxins | All species | Minutes to hours | Days; often lethal
Clostridial enterotoxins | Most species | Hours to days | Days; often lethal

Sources

- Waste food materials or dead animals contaminated with bacteria, bacterial toxins and sometimes other toxicants.
- Protein-rich garbage serves as a favorable substrate for the growth of microorganisms and the production of toxins.
- Palatable foreign refuse is often present, such as meat wrappers (especially strings) and other substances which can serve as foreign bodies.
- Problems are more common in warm seasons and regions, especially when less than desirable hygienic practices or roaming are permitted.
- Food considered unfit for human consumption but fed to the family dog is another source.
- High moisture dog food, left at a warm temperature for a sufficient time is another common cause.

Note:
- Botulism, one form of occasionally encountered food/garbage poisoning intoxication is discussed under Toxicants Causing Paralysis.
- Discarded foods from the refrigerator can also be a source of fungal toxins (see handout on Tremorgenic Mycotoxins).
- The primary problem arising from bone ingestion is the formation of "bone-stools" which comprise a concretion that causes a terminal obstruction of the rectum and constipation.

Toxic Principles, their Mechanisms of Action, and Associated Signs

- Enterotoxins.
  - Common enterotoxin producing bacteria associated with enterotoxicosis in domestic animals include *Streptococcus* spp., *Salmonella* spp., and *Bacillus* spp.
  - Enterotoxins are proteinaceous substances that produce their biological effects by sorption with the gastrointestinal epithelium.
  - Enterotoxins are strong irritants of the gastric epithelial cells and therefore act as comparatively potent emetics.
  - The enterotoxins also disturb the permeability of the gastrointestinal epithelial cells, causing fluid and electrolyte losses into
the lumen and interfering with the normal absorptive mechanisms of the gut.

- Resolution of the dysfunction occurs primarily by necrobiosis of the affected cells and replacement with new cells migrating from the crypts, a process which takes 3 - 5 days. In the interim, the intestinal erosion may result in hemorrhage into the intestinal tract.
- Initial irritation of the gastrointestinal tract may result in increased motility followed in 10 - 72 hours by dilation and stasis of the gut.

**Signs of Enterotoxicosis.**

- The first and often the only clinical sign of enterotoxicosis is vomiting, which generally begins within 15 - 180 minutes of exposure.
- Often, this process removes sufficient toxin to give complete remission of clinical signs. Because of the irritating properties of enterotoxins, however, vomiting and retching may persist, eventually requiring medical intervention.
- Diarrhea, often bloody may be observed 2 - 48 hours after exposure.
- Anterior abdominal pain, which is often present initially, may in a period of hours extend over the rest of the cavity.
- Stasis and dilation of the gut often follows with gas accumulation in the gut lumen, distention and accompanying pain.
- In addition, the stasis favors the growth (or decreased removal) of gram negative bacteria, which liberate endotoxins (discussed below).
- Often patients with stasis of the gut become critically ill and somewhat refractory to treatment.

**Endotoxins.**

- Gram negative bacteria in garbage, offal and other decaying animal tissues can serve as a potent source of endotoxins.
- Endotoxins are lipopolysaccharide components of the cell walls of gram negative bacteria liberated upon bacteriolysis (lysis of the bacterial cells) before or after the ingestion of the offending substrate.
- Absorbed by the gastrointestinal epithelium and act through a number of mechanisms including: 1) activation of certain inflammatory mediators, such as proteases and other enzymes necessary for the activation of autocoids (histamines, kinins), 2) cardiovascular insult and shock associated with release of catecholamines, thromboxane and prostacyclin as well as secondary pooling of blood in capillary beds with resultant hypoperfusion, anaerobic metabolism and lactic acidosis, 3) alteration of the permeability of the gastrointestinal tract with loss of fluid into the lumen, 4) interference with gut and other endocrine metabolism including those which influence gastric emptying, 5) causing activation of pancreatic zymogens (may be an indirect result of pancreatic ischemia as a result of a drop in cardiac output during shock, rather than a direct effect) with secondary pancreatic autodigestion and acute pancreatitis, 6) competition with insulin for the same receptor, 7) activation of cascade reactions which activate complement and which initiate the clotting cascade, and 8) uncoupling of oxidative phosphorylation in the myocardium is reportedly produced which may contribute to a negative inotropic effect.
- The autocoids alter capillary permeability and increase hepatic portal resistance to blood flow with splanchic pooling of blood.
- The activation of clotting factors can result in disseminated intravascular coagulation.
- Erosions of the epithelium may occur with frank hemorrhage into the lumen.
- The net effect is one of profound circulatory shock, collapse and frequently death.

**Signs of Endotoxemia.**

- Fever, shivering.
- Vomiting, diarrhea, abdominal pain and tenderness.
- Abdominal distension.
- Semicommonousness.
- Shock, often with muddy mucous membranes.
- Often there will be gaseous gastrointestinal tract contents.
- Often the offending material may be present in the vomitus.
- Often the feces will be exceedingly foul smelling.
- Watery or bloody diarrhea may be seen.

**Diagnosis**

- Attempts to differentiate Enterotoxin induced disease from Endotoxin induced disease are generally impractical and do not greatly influence the choice of therapeutic approaches.
- Diagnoses are based upon an appropriate history of exposure with the typical clinical signs, especially gastrointestinal upset, with gas, foul smelling vomitus or diarrhea, abdominal pain and subsequent shock, and the associated increase in capillary refill time. The heart rate becomes rapid and the pulse has been described as thready.
- Radiographs (+/- barium) are indicated to rule out foreign bodies and intestinal displacement, including volvulus, torsion or intussusception.
- Rule out acute pancreatitis from other causes.
- Rule out acute hepatic or renal failure.
- With endotoxemia, a transient leukopenia, usually with a neutropenia followed by an increase in the leukocyte count with a predominance of polymorphonuclear granulocytes is observed.
- Fever is typical of endotoxemia, followed by hypothermia as the shock state progresses.
- The liver may be engorged and the resultant enlargement may make it palpable.
- Monitoring of blood glucose may be used to detect early endotoxic shock. In endotoxemia, inhibition of gluconeogenesis by endotoxins, depletion of hepatic glycogen, and increased peripheral use of glucose may result in hypoglycemia.

**Treatment**

- Treatment is also not greatly influenced by the differentiation of Enterotoxin vs. Endotoxin diagnosis.
- **Goals of therapy include:**
  - Limit absorption of the offending material.
  - Combat fluid and electrolyte imbalance.
  - Combat shock and secondary pancreatic hypoperfusion.
  - Control bacterial proliferation to prevent septicemia.
- **Specific approaches may include:**
  - The use of emetics for animals very recently exposed and who have no contraindications present such as severe weakness or semiconsciousness, as well as others.
  - The use of gastric or enterogastric lavage if large amounts of toxic materials are present (usually not necessary due to pronounced emesis and initial gastrointestinal motility: but may be of value if early stasis has ensued.
  - The vigorous use of activated charcoal administration.
  - The appropriate use of fluid and electrolyte therapy, as well as bicarbonate if indicated for metabolic acidosis.
  - Systemic parenteral chloramphenicol. Note the aminoglycosides are generally regarded as contraindicated because they may synergize with the endotoxins present in depressing the myocardium.
  - Heparin may be of value in very early instances of disseminated intravascular coagulation.
  - Corticosteroids may be life-saving in cases of severe circulatory shock.
  - Glucose solutions may be of benefit in promoting urinary output, while acting as a limited source of calories.
  - Supportive care may be needed for days after an episode of garbage toxicosis to prevent dehydration, electrolyte imbalance and secondary infection.
  - A frequently provided bland diet may be of benefit.
  - Some practitioners favor the use of cultured yogurt as a means of providing a "beneficial" flora.
  - Flunixin (Banamine) useful for its antiprostaglandin effects and possible direct antiendotoxin effects. To be effective must be given early in endotoxemic shock.

**Note** - Practitioners have long associated pancreatitis and malabsorption syndromes with repeated episodes of garbage toxicosis. Such repeated episodes must therefore be avoided.
Antibiotic-Induced Enterocolitis In the Horse, Guinea Pig, and Rabbit

<table>
<thead>
<tr>
<th>Major Species</th>
<th>Usual Time of Onset</th>
<th>Usual Duration (if survives)</th>
<th>Full Table for Other Mycotoxins, Bacterial Toxins, Zootoxins, and a Yet-To-Be Identified Agent that Affect the Gastrointestinal Tract</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horses, rabbits, guinea pigs, other posterior fermenters</td>
<td>Hours to days</td>
<td>Days; very often lethal</td>
<td></td>
</tr>
</tbody>
</table>

Sources

- Antibiotic contamination from feed mill - usually after mixing a batch of medicated feed for swine or cattle.
- Access to swine or bovine medicated feed.
- Use of wrong oral antibiotic in the horse, guinea pig or rabbit.
- Hamsters may also be susceptible.

Toxicity

- "Toxic" antibiotics in horse, guinea pig, and rabbit.
  - Lincomycin at 0.5 mg/kg BID.
  - Tetracycline.
  - Tylosin.
  - Neomycin.
  - Penicillin, oxytetracycline, streptomycin, and erythromycin have been implicated in the syndrome in guinea pig (chloramphenicol 30 - 60 mg/kg can be used)
  - Penicillin, ampicillin, vancomycin, erythromycin, cephalosporin, lincomycin, and gentamicin have been implicated in toxicity in hamster (chloramphenicol 50 mg/kg antibiotic of choice).

Mechanism of Action

The regular gut flora are killed and species of *Clostridium* bacteria overgrow with increased enterotoxin production. *Clostridium difficile* is among the most important toxigenic species. It produces two toxins that alter the intracellular protein known as "rho" to cause disorganization of the cytoskeleton of enterocytes.

Signs

- Anorexia.
- Nonresponsiveness and lethargy.
- Profuse diarrhea.
- Muddy mucous membranes.
- Tachycardia, dyspnea, and colic possible.
- Death.

Lesions

Acute catarrhal to hemorrhagic typhlitis and colitis; gastrointestinal mucous membranes very edematous.

Diagnosis

- Culture feces.
- Test feed for antibiotics.
- Differential.
  - Salmonella, Colitis-X.

Treatment

- Fluids and bicarbonate.
• Potassium added to fluids (slowly) as needed (monitor serum concentration).
• Activated charcoal.
• Flunixin meglumine.
• Normal gut flora.
• Antibiotics not secreted into gut.
• In preliminary studies, bacitracin has been shown to be useful in treatment and prevention of antibiotic-induced colitis in horses. Its use is not yet recommended in clinical practice.
• Steroids.
• An anecdotal report suggests that metronidazole plus yogurt (as a source of Lactobacillus organisms) is effective for antibiotic-treated rabbits experiencing significant gastrointestinal problems. The drug has reported activity against Clostridia and may therefore be a rational therapy.

References

Antibiotic-Induced Enterocolitis In the Horse, Guinea Pig, and Rabbit


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