

Abomasal Bloat and Abomasitis in Calves

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Abomasitis is a sporadic disorder of young calves, lambs, and goat kids. Most affected animals are under 3 weeks of age. This disease is characterized by a fairly rapid onset of abdominal distension, depressed attitude, and occasional signs of colic. Affected animals may be seen to grind their teeth and salivate. Diarrhea may or may not accompany these signs. Once abdominal distension is severe, if the flank of the calf is shaken by hand, a tinkling and splashing sound may be heard. At necropsy, a gas-filled and inflamed abomasum is commonly seen. The abomasum often contains foul-smelling, sour clots of milk, and bile reflux from the small intestine may impart a greenish color to the abomasal contents. Hemorrhage from the abomasal lining, however, may cause the contents to become rust-colored or even black. The inflammation evident in the abomasal wall is the finding that prompts many to term this disease “abomasitis.” Ulcers may be visible in the abomasal wall, and occasionally, these perforate to release abomasal contents into the abdominal cavity, resulting in peritonitis. Although there are not a lot of descriptions of abomasal bloat in the veterinary literature, the proportion of affected calves that die appears to be well over 50-60%. Near death, calves show signs of shock, compromised respiration from the pressure of the distended stomach, and dehydration.

The cause(s) of abomasal bloat and abomasitis are not fully understood, and there are quite a few factors that have been proposed to be involved. Bacterial infection of the abomasal wall, compromised immunity from inadequate colostrum, ingestion of foreign bodies such as hair and coarse plants, and vitamin / mineral deficiencies have been proposed as factors involved in the development of this disease. Specifically, vitamin E, selenium, and copper deficiencies have been implicated as causative factors. Bacteria such as *Clostridium perfringens* type A and species of *Sarcinia* have been identified in the abomasum of affected calves. More recently, *Salmonella typhimurium* was isolated from the abomasal wall of Midwestern veal calves with abomasitis. Of these bacteria, only *C. perfringens* type A has been shown to experimentally cause the disease in calves; in 1988, investigators at the veterinary college at Kansas State University were able to recreate the disease in dairy calves by infusing *C. perfringens* type A into their stomachs. Interestingly, these investigators were able to create the disease in calves that were not copper-deficient. Belgian investigators have also detected *C. perfringens* in the abomasums of affected calves. The ability of this organism to produce gas from fermentation of nutrients in the stomach is considered to contribute to dilation of the abomasum in affected animals. *Clostridium perfringens* type A is part of the normal bacterial flora of the gastrointestinal tract of cattle. The factors that trigger this agent to possibly act as a pathogenic bacteria remain uncertain; however, certain strains make large quantities of toxins. Further, the numbers of this organism in the gut have been shown to increase when cattle ingest large amounts of easily digestible carbohydrates and protein (such as milk or lush grass).

Cases of this disease in beef calves have occurred soon after management practices that cause delays in regular nursing patterns or changes in environment that interrupt normal nursing patterns (e.g. winter storms). In dairy calves, poor milk hygiene, intermittent feeding of large volumes of milk, and cool milk temperature are considered to be potential contributory factors.

The common thread seems to be ingestion of a larger-than-ideal milk meal. This may result in a slowing of stomach emptying, which may then enable the bacteria to ferment the milk and create gas. Given the multitude of contributory factors proposed to date, it is possible that this disease occurs when multiple factors occur together.

Treatment of this disease has mixed results. Early intervention is critical. At the CSU veterinary hospital, when presented with a calf with abomasal bloat, our first step is to prevent further suckling and to not tube the calf with anything that might trigger further bacterial growth and gas production in the stomach – in other words, no glucose-containing oral electrolytes, milk, or milk replacer is fed until the calf has recovered. Passage of a stomach tube while the calf is recumbent or standing often does not relieve the bloat because the gas is trapped in the fourth stomach (the abomasum) and the tube only enters the first two stomachs (the rumen and reticulum).

A different method for passing the stomach tube may relieve the bloat in affected calves. The length of tube necessary to reach the stomach is typically the length from the mouth to the last rib on the calf's side. Once the tube is in place, a helper can lift the front end of the calf up so that the calf is sitting or standing in a near vertical plane – this may cause the gas to percolate up into the rumen and reticulum. The tube should be gently moved back and forth several inches while the calf is held in this position. Gentle pressure or massage of the calf's flank may facilitate gas release. The holder should listen or smell for gas release, or place the free end of the tube in a cup of water to see the bubbles that indicate that gas is being released. Before the tube is removed, procaine penicillin (10 cc of 10,000 IU/ml solution) mixed in 2 cups of mineral oil should be administered into the tube, with a cup or two of warm water as a "chaser" to push the medication out of the tube and into the stomach. This step is intended to slow bacterial fermentation of milk nutrients into gas. Subcutaneous administration of procaine penicillin (4 cc of 10,000 IU/ml solution per 100 lbs of bodyweight, 1-2 times per day) is also recommended, as some invasion of the abomasal wall by bacteria may be occurring. Calves also appear to benefit from intravenous or subcutaneous fluids and an injection of 50-100 mg (1-2 ml) of intravenous or subcutaneous flunixin meglumine (Banamine[®]). Intramuscular administration of this product should be avoided as it causes considerable muscle damage. Consult your veterinarian for other treatment options such as abomasal decompression.

Because the cause(s) of abomasal bloat and abomasitis are not well proven, preventive measures are currently based on a "best guess" as to what is going on to set these calves up for this disease. Certainly, one must consider whether or not the nutrition of the pregnant cow could be involved. Protein, energy, vitamin, and trace mineral problems should be identified. Management steps to ensure adequate colostrum intake by newborn calves is a must. Because the fermentation of milk by bacteria seems to be the source of gas, one must consider environmental or management factors that may trigger changes in the volume of milk ingested by a hungry calf. Management practices that cause prolonged interruption of suckling must be made time-efficient in order to limit engorgement of the udder and subsequent ingestion by the neonate of a larger-than-normal milk meal. Sudden and severe changes in weather may cause dams and their offspring to seek shelter or remain recumbent for prolonged periods of time. In such instances, when the weather clears or daybreak occurs, the dams then stand up – and the hungry calf is then presented with an engorged udder. This can be remedied by providing multiple locales for

shelter and bedding, or simply encouraging dams to stand up and eat by providing hay (weather permitting). This practice may encourage the dams to stand, thereby enabling more frequent, lower-volume nursing than if the cattle were left to “sit the storm out.” Group penning, inadequate fiber diets, and ectoparasites may trigger hair chewing by calves, and hairballs that form in the abomasum may irritate the lining and trigger the disease.

Currently, there is no clear data on whether or not conventional vaccines that include the inactivated toxins from *C. perfringens* types C and D induce antibodies in the colostrum that protect the calf from disease caused by *C. perfringens* type A. The degree to which cross-protection against type A occurs may vary by the manufacturer of type C and D vaccines. Check with your veterinarian, as he or she may request data on cross-protection from the vaccine manufacturer. A commercial vaccine that induces antibodies against the major toxin of *C. perfringens* type A is currently available; however, its efficacy in prevention of abomasal bloat / abomasitis is currently unproven. This is a troublesome disease that can flare up without any apparent predisposing cause. Please feel free to contact members of the CSU beef team with questions about this or other health problems of neonatal calves.