Left Displacement of the Abomasum

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INTRODUCTION

Abomasal Displacement is one of the most common surgical conditions in dairy cattle, with left-sided abomasal displacements (LDA) representing approximately 85% of abomasal disease in lactating dairy cattle. LDAs occur most commonly during early or peak lactation, with a prevalence rate of 3-5% in high production dairy herds[8]. Although the exact cause of displaced abomasum has not been identified, the most common theory is abomasal hypomotility[1]. Many predisposing factors have been recognized, including late gestation, dystocia, and hypocalcemia[13]. An LDA can impact the individual cow's profit margin immensely, with the surgery and medical management alone costing between $250 and $400, not to mention the impact the disease has on milk production, reproductive efficiency, or mortality[2, 7, 13].

HISTORY & PRESENTATION

The most common presentation of LDAs are high producing, post-partum dairy cows within their first month of lactation that are between the ages of 4 and 7 years old[1,5,11]. 57% of LDA's will occur in the first 2 weeks of lactation, 80% within the first month, and approximately 85-91% within the first 6 weeks post-partum[8]. There is a trend for a LDA to present more frequently between January and March and less frequently during the fall[5]. While LDAs do occur in beef breeding bulls and calves occasionally, the disease is by far more prevalent in dairy cattle[1, 8]. Most frequent presenting complaints are sudden, moderate to severe decreases in milk production and decreased feed intake[13, 14]. Herd managers will typically report that the affected cow is no longer eating concentrates, but still has an affinity for forages[1]. Loose, scant feces that may be darker in color than normal also may be reported[8].

Upon physical examination, the animal will be depressed but vitals are typically within normal limits. The affected animal may exhibit decreased to absent rumen contractions[11]. Rectal exam is unremarkable, although an experienced practitioner may appreciate the rumen to be decreased in size and deviated more towards midline[1]. The disease is most commonly recognized by using auscultation and percussion to detect a high pitched "ping" (fluid/gas interface) on the left side of the cow's abdomen, usually between the 10th and 13th ribs[11]. It is important to remember that affected cows usually present with concurrent disease, such as mastitis, metritis, retained placenta, hypocalcemia, or ketosis[1, 11, 14].

PATHOPHYSIOLOGY

In the case of a LDA, the abomasum moves from its normal anatomic position and becomes trapped between the left abdominal wall and the rumen[1]. While the exact etiology is unknown, the most widely accepted theory is the combination of parturition and abomasal hypo-motility[5, 7, 8]. In late pregnancy, the distended uterus elevates the rumen off of the ventral body wall and pushes the abomasum into a more cranial and transverse position. When parturition occurs, uterine volume decreases and the rumen is allowed to fill, causing the abomasum to remain trapped on the left side of the abdomen[8].

Abomasal motility can be depressed by multiple factors, highlighted by electrolyte abnormalities, nutritional factors, and systemic disease such as in endotoxemia. Cattle diagnosed with hypocalcemia, or "milk fever", are 4.8 times more likely to develop a LDA[12]. In one study performed on 7 non-pregnant dairy cows, clinical hypocalcemia was induced to measure the rate and amplitude of ruminal and abomasal contractions[6]. The study showed that low ionized serum calcium levels <4.0 mg/dl decreased contractions by 50% of normal[6, 12, 14]. Hypocalcemia also

leads to other diseases that also predispose to a displaced abomasum, such as dystocia, retained placental membranes, metritis, and ketosis[3, 6].

In regards to nutrition, a sudden change from a dry cow diet high in roughage content to a lactating cow ration rich in concentrates also predisposes to abomasal hypo-motility and gas accumulation[3, 4, 12]. The increase in concentrates subsequently leads to a lower than normal rumen pH and excess volatile fatty acid (VFA) accumulation within the rumen, causing more VFAs to move into the abomasum than normal, thus increasing abomasal oncotic pressure and the water content within the abomasum[14]. Bicarbonate from the rumen then reacts with the excess water in the abomasum to form carbon dioxide gas, which combines with the normal inhabitant of the abomasum, methane, to further distend the abomasum[7].

DIFFERENTIAL DIAGNOSES

Left displaced abomasum can be diagnosed by the experienced practitioner rather quickly when combining the history, clinical signs, and physical exam findings, especially the “ping” between the 10th and 13th ribs on the left side of the abdomen[11,13]. However, the “ping” heard via simultaneous auscultation and percussion of the abdomen only leads to a presumptive diagnosis, as it is only indicative of a fluid-gas interface. Other conditions in which a “ping” can be auscultated and therefore must be differentiated from a LDA are: ruminal tympany, ruminal atony, pneumoperitoneum, peritonitis, and physometra[1, 8].

DIAGNOSTIC APPROACH/CONSIDERATIONS

After making a presumptive diagnosis via history, clinical signs, and a left sided ping, a veterinarian may wish to support their diagnosis of a LDA before proceeding with an exploratory laparotomy. One diagnostic test to utilize in this situation is an abdominocentesis, also known as

the *Liptak* test. Insertion of the needle is performed over the area where the hyper-resonant ping is auscultated[10, 11, 13]. A fluid pH of <4.5 is indicative of abomasal fluid, indicating a LDA. A less invasive diagnostic tool is ultrasonography. The examiner would typically visualize an area of hypo-echogenicity between the left abdominal wall and the rumen, as well as mucosal folds within the abomasal lumen, seen as "sickle-shaped" structures[8].

As this disease is most commonly diagnosed in the field, the veterinarian may not have clinical pathology available at the time of presentation. If available, serum chemistry would be indicated. Findings would include hyponatremia, hypokalemia, and metabolic alkalosis, as evidenced by an increase in blood pH and an increase in bicarbonate. This is due to the sequestration of hydrochloric acid within the abomasal lumen, as abomasal outflow is partially obstructed[1, 8]. Since this is usually not available in an ambulatory setting, veterinarians typically are aware of this common abnormality and will treat accordingly following surgical correction of the LDA[11]. It is important to note that while a presumptive diagnosis is often adequate in this condition, an absolute definitive diagnosis is only made via exploratory laparotomy or necropsy.

TREATMENT

Surgical correction is the treatment of choice for a left displaced abomasum[2, 8, and 11]. The goal of any treatment of a LDA is successful replacement of the abomasum in its correct anatomical position and a complete return to full milk production[11, 13]. Once a diagnosis has been made, the practitioner has several different surgical techniques to choose from. All techniques have their individual advantages and disadvantages, which will be addressed, but ultimately choosing one particular technique is a clinician's preference[8, 11]. The three main surgical techniques that will be discussed are: right flank omentopexy, left flank abomasopexy, and right paramedian abomasopexy. Two other methods of correction are the roll & toggle and

laparoscopic techniques[8], however these methods are not of wide use due to the extremely poor success rate of the roll & toggle procedure (recurrence rate of >90%) and the expense and inconvenience of a laparoscopic procedure in a field setting[8,13].

The right flank omentopexy is regarded by many to be the surgical technique of choice for correction of a displaced abomasum, due to the fact that all 3 variations of a displaced abomasum can be corrected (LDA, RDA, AV) and the convenience in which this procedure can be performed[9]. This technique is excellent in a field setting because it is a standing procedure that does not require any assistance for the surgeon. It provides the opportunity for a complete exploratory celiotomy, even allowing evaluation of the cow's post-partum reproductive tract[8, 11]. The procedure itself is relatively simple, requiring the surgeon to first deflate the displaced abomasum and return it to its normal anatomical position on the right cranio-ventral aspect of the abdominal cavity; the omental tag ("the sow's ear") is located between the descending duodenum and the pylorus of the abomasum, and is incorporated into the closure of the incision[8]. The most distinct disadvantages to this procedure is that the omentum can be friable, resulting in failure of the pexy, and that anatomical fixation may not be ideal in some cases[8]. It is important to note that this is not a viable approach to the correction of a LDA in the case of severe adhesions, or if an abomasal ulcer perforation or peritonitis is suspected in the cranioventral abdomen, as in the case of traumatic reticulo-pericarditis (TRP, Hardware disease)[9]. However, this procedure has a great success rate and prognosis, with 86-90% of treated cattle returning to full milk production[8, 9].

While the right paramedian abomasopexy is not the most convenient procedure, it is widely accepted as the "gold standard" of abomasal displacement correction. In this technique, the animal is cast into dorsal recumbency and an incision is made approximately 8 cm behind the

xyphoid just to the right of midline and medial to the right cranial superficial epigastric vein (milk vein)[8]. This allows the most direct access to the greater surface area of the abomasum, providing the opportunity for a more ideal anatomical fixation that reduces the need for manipulation of the abomasum, thus decreasing the risk of iatrogenic omental or serosal trauma. This technique also allows the surgeon to concurrently treat abomasal ulcer perforation and it provides a more cosmetic appearance as the scar is on the ventrum of the animal[9]. However, this procedure requires additional restraint of the animal as well as additional assistance for the veterinarian performing the procedure. This approach provides minimal access to other abdominal structures and is contraindicated in cows with concurrent disease, such as pneumonia, hypotension, a distended rumen, a heavily gravid uterus, or a variety of musculoskeletal defects[8, 9]. This technique also has more complications than the right flank omentopexy, including incisional hemorrhage, dehiscence, herniation or fistulation, as well as recurrence of displacement. Relatively the same prognosis exists when comparing the two, with reported values of 83.5-95% of treated animals returning to full milk production[8, 9].

In the case of a chronic LDA where severe adhesions are expected, or when peritonitis or TRP is suspected, the left flank abomasopexy is a proper choice for surgical correction of the displaced abomasum[8]. This procedure allows visualization of the displaced abomasum and upon a successful procedure the anatomical fixation is ideal. The left flank approach does require assistance and certain veterinarians shorter in stature and arm length are at a disadvantage with this procedure. The left flank abomasopexy requires the surgeon to deflate the abomasum, pre-place suture in the sero-muscular layer of the abomasal wall, then pass the needle and suture to their assistant through the ventral abdominal wall approximately 3-4 cm to the right of midline and 4-5 cm caudal to the xiphoid. The assistant then pulls while the surgeon pushes the

abomasum into its proper position, and at this point the suture is tied tightly on the external surface of the ventrum[8, 11]. This technique does not allow a complete abdominal explore and the risk of small intestinal or omental entrapment resulting in an outflow obstruction is moderate, thus requiring the surgeon to be extra cautious before completing the abomasopexy[8]. The prognosis for return to function is similar to the other procedures discussed, with reported rates being between 85-90%[8, 11].

It is imperative for practitioners to remember that cattle presenting with LDAs either already have concurrent diseases present or will develop them in the near future, with the most common being ketosis and hypocalcemia[1,2]. Thus, oral propylene glycol is routinely administered to affected cattle, as well as a calcium product in the form of an oral paste or an intravenous fluid formulation[1, 11]. Fluid therapy is also vital to success in the form of return to normal milk production[2]. Typically dehydration and electrolyte imbalances are addressed with approximately 20 to 40 liters of isotonic fluids and a complete electrolyte package per 20 liters[11]. Flunixin meglumine or dexamethasone can also be administered in effort to increase abomasal motility and manage pain. Post-operative antibiotics are indicated as well, with careful consideration given to milk and meat withdrawal times[1, 2, 11]

MANAGEMENT

While surgical correction of a LDA is successful, it only adds to the financial impact this disease can cause; thus, management strategies preventing displaced abomasums must be implemented[4, 5, and 13]. Avoiding known risk factors is one practical management solution. However proper nutrition and bunk management are by far the best methods of prevention[3, 4, 14]. The most critical point during a dairy cow's production lifecycle in regards to a LDA is the transition period, which lasts from 2 to 4 weeks post-partum[7, 14]. During this time, progressive

dairy producers implement a “steam-up” ration, which gradually increases concentrate content to what it will be during lactation while also maintaining ample amounts of forages in the ration; they will also utilize anionic salt rations which creates a dietary cation-anion deficiency, or DCAD[3]. This strategy will induce a metabolic acidotic state in the transition cattle, preparing them for the increased need for calcium at the onset of lactation by stimulating the parathyroid glands to promote increases in ionized calcium through the action of parathyroid hormone (PTH) and the active form of vitamin D. These diets help prevent the common metabolic diseases such as hypocalcemia, ketosis, and hepatic lipidosis, all of which predispose cattle to a LDA[3, 4, 14].

EXPECTED OUTCOME AND PROGNOSIS

As a whole, the prognosis of a cow diagnosed with a left displaced abomasum is excellent, with the average successful treatment rate being approximately 90%[1, 8]. The goal of treatment is return to full milk production[11]. Prognosis of a LDA depends on multiple factors, including acute vs chronic duration of disease, the corrective procedure performed, and the existence of concurrent disease[2]. Important to note are the separate conditions of a right displaced abomasum and abomasal volvulus, as both of these have a much worse prognosis than a LDA[1,8]. A RDA’s prognosis is reported as good to poor, while an AV is always guarded[10]. Since a RDA can progress to an AV, it is important for the veterinarian to act promptly, as this is a true emergency[11]. Prognoses and complications of individual procedures have been previously discussed.

CONCLUSION

Left displaced abomasum is a costly disease for a dairy producer, but one that can be prevented through consistent and effective management practices. Surgical correction is the treatment of choice and the primary goal of treatment is return to full milk production. The most

common time for a LDA to occur is during the transition period when cows are switched from a high forage, low concentrate ration to a high concentrate, low forage ration; therefore, the majority of the research revolving around this disease pertains to the nutritional and metabolic factors that predispose cattle to this disease.

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