A Treatise on Left Abomasal Displacement in Dairy Cattle

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INTRODUCTION

The modern dairy cow has become a refined "biological machine." Hers is a background of production-oriented breeding and management practices that seek to maximize lactation capabilities. Such emphasis on these capabilities is not without consequence, however, and the dairy cow has acquired maladies that are uncommon in other members of the bovine species. This paper will review a problem that is more or less unique to dairy cows.

Though it is of lesser economic significance than the ubiquitous mastitis or reproductive dysfunction, left displacement of the abomasum is a prevalent disease in the worldwide dairy population. Veterinary diagnostic abilities as well as dairy management practices have markedly improved in the three decades since discovery of the left abomasal displacement (LDA).[1] These diagnostic improvements, coupled with the evolution of high-producing cows and their concomitant conformational changes, contribute to an increasing prevalence of LDA.[2]

Despite anatomical similarities of all adult bovidae, reports of LDA in non-dairy animals are few. It is the most common disease directly affecting the abomasum[3] and its treatment is the most common surgical procedure performed in a large California dairy practice.[4]

In LDA the abomasum moves ventrally and leftward, beneath the rumen. The buoyant nature of its contents then enables the abomasum to move dorsally where it eventually lodges between the left body wall and rumen.

The abomasum is attached to the rumen and the reticulum by bands of smooth muscle tissue, and communicates proximally with the omasum. Distally, the abomasum is attached to the cranial duodenum at the pylorus. The pyloric area connects to the liver via the lesser omentum. The greater omentum lies ventral to the pylorus and attaches along the abomasum's greater curvature. The nature of this attachment is such that the omentum provides no abomasal stabilization to prevent displacement.[5]

The leftward shift in abomasal position draws the omasum and cranial duodenum ventrad and to the left. Though this shift is unlikely to cause stricture of the affected segment of the digestive tract, sufficient impedence to flow exists, causing the clinical and metabolic changes so often seen in LDA.[2]

PATHOGENESIS

Theories abound regarding the etiology of LDA, yet despite thirty years and a myriad of studies it remains obscure. At best the disease can be described as having a multifactorial etiology.[2,3,6-11]

Genetic predisposition is cited as being a significant contributor by some authors[6,8] but is downplayed by others.[2,7] However, in light of today's selective breeding for production, it follows that cows having high production capabilities also must have the capacity to hold more feed. A more capacious abdominal cavity may, in turn, allow more room for abomasal displacement.[8,12]

A seasonal increase in the occurrence of LDA has been cited.[2,7] An increase in incidence was noted between October and April, and attributed to the practice of stabling cows during the winter months. Confinement and lack of exercise are the likely causes for the seasonality of LDA.

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Robertson[2] noted that 86% of LDA's were observed in the period from two weeks prior to parturition to two weeks following. He also states that 22% of LDA's occurred on the first day following calving. These observations correspond with those made by others[3,7,8,11,13,14] and it is safe to say that parturition plays a major role in the development of LDA.

The most plausible theory for the relationship between LDA and calving is of a mechanical nature. It is felt that the enlarging uterus displaces the rumen upward and pushes the abomasum cranially and to the left, creating an excellent opportunity for displacement. This theory is supported by a higher incidence of LDA in cows bearing twins.[8] At parturition the already semi-displaced abomasum becomes entrapped on the left by the rumen as the uterus empties.

Another significant co-factor in the production of LDA is a hypomotility or atony of the abomasum. Reduction or absence of the normal tone allows the accumulation of fluid and gas in the fundic portion of the viscus, an event that precedes displacement.[7]

Factors causing the hypo- or atonic state of the abomasum are numerous. Consequently, this area deserves more elaboration than the scope of this paper allows. This list includes: electrolyte disturbances;[8] abnormally high levels of volatile fatty acids;[15] and the presence of other disease concurrent with LDA.[2,7,8]

Volatile fatty acid (VFA) levels are a direct result of the feeding regimen. Therefore, a critical evaluation has been directed at the nutritional component of LDA. It is a common practice to increase the cow's energy intake late in the dry period to prepare her for the demands of early lactation following parturition.[16] This practice of "lead feeding" involves offering rations having high concentrate/low roughage content. Many feel that this practice predisposes to LDA.

As VFA levels increase with a higher proportion of ration concentrate, the rumen is also affected by the decreasing percentage of roughage. A minimum of 13–14% roughage is *required* for normal rumen function.[3] The ruminant stomachs are closely related anatomically and functionally, and disruption of function in one usually affects the others as well.[2,3,7,17] Robertson[2] feels that high concentrate levels can be fed if adequate roughage is made available to the animal.

Interestingly, beef animals are raised on high concentrate/low roughage diets and they enjoy freedom from LDA. However, they differ in conformational makeup, and are not expected to survive for several years on this feed regimen, unlike their dairy counterparts.[7]

Concurrent diseases are seen in a significant number of LDA cases. The presence of metritis, post-parturient hypocalcemia, ketosis, or retained placenta can contribute to abomasal hypomotility.[2,7,8,17,18]

An association between age, production, and LDA has been made. Four to seven year-old cows and high producers are most commonly afflicted.[2,7,8] Cows reach their productive peak between the second and fourth lactations. The dairyman gauges grain feeding by the cow's milk output. Therefore, high producing cows are most likely to be from four to seven years old, and because of their production levels, will receive more grain than their less-productive herdmates. The age and productivity association with LDA is most likely indirectly related to contemporary feeding practices.[2]

CLINICAL SIGNS

Clinically, LDA is not an especially difficult diagnosis to make, provided a good history is given and a thorough physical exam performed. LDA is highly suspect when the cow has recently calved, is anorectic, and shows a transitory response to treatment for indigestion or ketosis. Milk production commonly drops to between one-third and two-thirds normal. Affected cows will usually consume some hay but little or no grain or silage. The feces are usually reduced in volume and pasty in nature, though this has been demonstrated to be quite variable. With LDA, temperature, pulse, and respiration generally fall within normal ranges, unless complications exist. Likewise, the hemogram shows no appreciable change in the absence of concurrent disease.[13]

Affected cattle usually display a slab-sided appearance, especially on the left side. This occurs because the rumen is displaced medially by a distended abomasum along the left body wall. Occasionally the abomasum can be seen protruding into the left paralumbar fossa just caudal to the last rib.[11] Rumen movements are present but may be diminished in intensity and reduced in frequency due to pressure placed on the rumen by the abomasum. This is especially noted between the left eighth and twelfth intercostal spaces, where an LDA usually appears.

DIAGNOSIS

Diagnosis of LDA is accomplished quite easily if proper percussion and auscultation techniques are employed and if a "ping" is auscultable. Rarely an LDA may present which does not ping on percussion or may do so inconsistently.

The gas-filled abomasum, when percussed, gives a characteristic "ping" similar to the sound heard when snapping one's finger against an inflated inner tube.

A snap of the finger or a percussion instrument is used to strike the body wall six to eight inches from the stethoscope head. This distance relationship is important because percussion closer to the stethoscope may obscure the true ping.[19]

The area to be examined is an imaginary oval that extends from the elbow to the left paralumbar fossa. One should percuss around the stethoscope head, then move to a new area and repercuss until the entire area has been covered. A "ping" is most likely to occur between the ninth and thirteenth ribs.[20]

If diagnosis remains uncertain, paracentesis may be used for confirmation. A three-inch 16-gauge needle is used to aspirate from an area 10 centimeters ventral to the cranial line of dullness. A pH of 2–3.5 is indicative of abomasal contents.[21]

TREATMENT

Several techniques for the repair of LDA have been developed, including methods both surgical and nonsurgical. It is not the intent of this paper to detail each method, for there are excellent references which do so;[11,21,22] rather, an attempt to highlight currently accepted procedures will be made.

If LDA were merely a displacement of the cow's fourth stomach, its repair would be straightforward. Complications are commonly present, as with concurrent disease conditions, and will affect prognosis and ultimate success. Wallace[9] noted that the presence of diarrhea in cows with LDA is a likely signalment of another ongoing disease process, and that the death rate in these cows was twice that in cows not showing diarrhea.

Nonsurgical therapy is deserving of mention because it is less expensive, less invasive, and less successful. The most commonly employed technique is that of casting and rolling the cow. Other less scientific methods include transporting over rough roads and simply walking the cow up a steep slope.[24]

Medical treatments include: large volumes of warm water given orally; smooth muscle stimulants such as cascara sagrada; and calcium borogluconate parenterally. Practitioners have reportedly given coffee grounds or instant coffee via stomach tube.[11,25,26]

With any of the above methods, recurrence is common, and as a result, surgical therapy is in vogue by virtue of the high success rate.

There are two objectives of surgical repair. First, the abomasum must be restored to its normal position, even if decompression is necessary. Secondly, the organ needs to be stabilized to prevent recurrence of the condition. Response to treatment is considered satisfactory if the cow returns to the herd and has good milk production.[5,9]

Four methods of surgical treatment are in common use today: the ventral closed-suture abomasopexy; the ventral paramedian abomasopexy; the left flank abomasopexy; and the right flank omentopexy. Each technique has its decided advantages and disadvantages; however, if performed properly and in cows not overwhelmed by concurrent disease, these techniques have 80–90% success rate.[22,26,27]

The ventral closed-suture abomasopexy (also called "blind stitch" or "roll-and-tack") technique is one that bridges the gap between being purely surgical or purely non-surgical. This method and its variants[28] are popular because they are quick, inexpensive and less traumatic than surgery. The cow is cast on her right side and rolled, as described by Braun.[24] The objective of rolling is to bring the gas-filled abomasum to the ventral midline via its natural buoyance in the abdomen. While in dorsal recumbency, the cow is percussed and auscultated to the right of ventral midline to confirm the abomasum's presence in its normal anatomical location. When the abomasum is in place, it is attached to the ventral body wall with a percutaneous stitch.

A four to six-inch upholstery needle armed with extra-heavy vetafil is thrust through the body wall and abomasum to secure it into place. Two adjacent stitches are placed through these structures in an area bounded by the xiphoid and umbilicus and between the ventral midline and the right "milk vein." This is the normal location for a nondisplaced abomasum. Two separate sutures are placed to ensure immobilization of the organ.

Properly done, the "blind stitch" has a good

success rate,[19,28] and is a boon to the practitioner whose clientele demand low-priced procedures, or whose preference is not to perform surgery. Essential to success, however, is being consistent in locating the abomasum on midline and penetrating it with suture material. Without proper stitch placement there is a good possibility of recurrence. Unfortunately, experience is the best guide for correct stitch placement. Also, the closed approach allows for no assessment of abomasal condition.

The ventral paramedian abomasopexy is similar to the "roll-and-tack" procedure in that it is performed with the cow in dorsal recumbancy. Unlike its "nonsurgical" counterpart, however, the abomasum is readily visualized in its normal position with this method of repair. Many times adhesions form between abomasum and body wall as a result of abomasal ulcers, and the operative techniques allow assessment and breakdown of these fibrinous attachments.

An incision is made in the same location as the "blind stitch" procedure's suture placement. With the abomasum in its normal position, repositioning involves little manipulation or deflation. The muscularis of the abomasum is then fixed to the peritoneum and transversus muscles with catgut suture. This procedure, like the other operative techniques, is highly rewarding if performed properly.[4,11]

Its disadvantages are recumbancy, restraint, and site of incision. Cows tolerate dorsal recumbancy poorly for long periods of time, and more manpower is required to put them in the recumbant position. A ventral midline incision is prone to the formation of seromas and hematomas due to gravity, and it is difficult to keep clean.[4,11] The greatest disadvantage of a ventral incision is that of dehiscence and subsequent herniation.

The left flank abomasopexy is a procedure performed with the cow standing. The left paralumbar fossa is prepared and anesthetized as for all flank procedures.[23] Once inside the abdomen, the surgeon places several interlocking sutures in the muscularis of the abomasum along the greater curvature. Three to four feet of excess suture is left at both ends of this stitch for later use.

The organ is affixed to the ventral abdomen by pushing the suture ends through the body wall and skin from within. The surgeon attaches the long suture end to a straight intestinal needle and "guards" it with his index finger as it is carried to the ventral body wall. It is then thrust between the right "milk vein" and midline. This is performed separately with both suture ends, and they are tied externally. A large-caliber needle (12–14 gauge) attached to four feet of rubber hose is used next to deflate the abomasum. This allows its manipulation into the normal position.

This approach works to the advantage of patient and surgeon alike because it is performed with the animal standing. It also allows good assessment of accessible viscera. Abdominal adhesions are easiest to break down using this technique.

The danger of visceral puncture or improper suture placement (puncture of a milk vein, for example) does exist with this procedure. Surgeons lacking long arms or available help are at a disadvantage, as well. Both are limitations preventing proper stitch placement, the former from *within* and the latter from *without*. Also, the assistant runs the risk of being kicked.

The right flank omentopexy is performed through an incision in the right paralumbar fossa. The displaced abomasum is palpated by reaching behind the omentum and over the rumen once inside the abdomen. The dough-like rumen is easily differentiated from the gas-filled abomasum.

The abomasum is decompressed prior to manipulation. The danger of peritonitis is low if the decompression needle is inserted at a 45° angle or less to the abomasal surface. Following deflation it is important that the needle be guarded from leakage into the abdomen as it is retracted.

The flaccid abomasum can now be manipulated into the normal position by "rocking" the omasal-abomasal junction[22] or by pushing it downward from above.[26] Care should be taken not to apply excessive traction on the abomasum or its attachments.

Anchoring the abomasum is accomplished by suturing a fold of omentum three centimeters from the pylorus to the abdominal wall. For the sake of convenience this omentum can be incorporated into closure of the abdomen.

A variation of the omentopexy is to anchor the pylorus to the abdominal wall, alone or in conjunction with the omentum. Some feel that the pylorus is more secure to suture, and that is less apt to tear or stretch than omentum. Edwards[26] noted that recurrence of LDA following repair occurs most frequently due to sutures tearing through tissues or because of their

improper placement. It is the opinion of this author that pyloropexy provides the surgeon with both more stable fixation and peace of mind. Of all surgical methods described above, this approach is easiest to perform without assistance. More visceral structures are available to the surgeon via the right side incision, affording him an excellent opportunity to assess their condition.

The main drawback to use of a right-sided approach is that more skill and manipulation are demanded of the surgeon than with the others. Adhesions are least accessible via the right also. Proper suture placement is the keystone of success with this method.[22]

In conclusion, left displacement of the abomasum is a curious and frustrating disease. Most of its components—clinical picture, diagnosis, treatment, and expected response to treatment—are well understood. Like a puzzle, however, these component "pieces" make for an incomplete picture without the most important piece, the etiology.

Despite much work and much progress towards this end, the answer to the cause of LDA is shrouded in mystery. Contributing factors have been isolated, yet not enough is known of its causation to enact preventive measures.

The most significant co-factors in LDA occurrence seem to be recent parturition and hypomotility/atony of the abomasum. The former is likely a mechanical contribution, whereas the latter is deserving of more attention to determine its own causes.

All in all, LDA may well be, as one author stated, "a civilization disease."[2] Its multifactoral etiology relates not to individual causes but rather to demands placed on the cow by contemporary production practices.

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