Abomasal Displacement, Etiology, Pathogenesis, Treatment and Prevention

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Introduction

Abomasal displacement has been diagnosed since 1898 when the first case of abomasal torsion in a calf was described (10). In 1928 and 1930 (79) the first abomasal torsions were described in adult cows and since then a gradually increasing number of abomasal torsions were recorded. In 1943 Emsbo (20) reported an increasing incidence of abomasal torsions in Denmark and gave a detailed description of the disease. The first case of left displaced abomasum (LDA) was published in 1950 (24), although Moore *et al* (46) stated in 1954 that they had seen their first case in 1948. The end of that first period of the history of abomasal displacement is marked by the detailed and comprehensive study of Dirksen (16) in 1962. Since then an avalanche of papers on abomasal displacement has been published.

The spate of papers has somewhat subsided. Apparently clinical researchers have lost interest in the subject. One could conclude from this fact all the questions about etiology, pathogenesis, therapy or prevention have been answered.

Nothing is less true, we know only a few answers.

This paper will deal with the history of abomasal displacement and will discuss the results of various studies that have been conducted to elucidate etiology, pathogenesis, treatment and prevention.

Finally, it will concentrate on new developments both in therapeutic approach as well as in possible preventive measures.

Definitions

Most workers investigating the etiology of the displaced abomasum (DA) do not distinguish between left displaced abomasum (LDA) or right displaced abomasum (RDA). Clinically the difference is obvious and RDA is frequently complicated by some form of torsion.

Wensfoort and van der Velden (85) have presented a concept based on a pendulum model and axes centered on the abomasum with which they were able to identify the various forms of abomasal displacement and torsion. With this concept, 25 combined displacements could be predicted. Of these 25 possibilities, 4 different types of positions were found during surgery in 217 cows with RDA. The positions of the LDA is according to the terms used a flexion of 180-270° to the left and can be compared clinically with the flexion 180-270° to the right without torsion or rotation. In both cases passage of food through the abomasum is still possible, a fact that is reflected in the clinical picture of the cows affected.

Although the incidence of LDA still is considerably greater than that of RDA, there are reports that the percentage of RDA is increasing (23, 55).

Incidence, Seasonal Occurrence, Breed and Age

Robertson (60) already in 1968 showed in his review that LDA was a world wide problem.

The incidence rate, without specification as to the direction of the displacement has been reported since 1961 where Pinsent (57) found an increased incidence over a period of 5 years (1955-1960). Robertson (60) saw an increase from 3% of the bovine admissions to the Large Animal Hospital of the University of Pennsylvania in 1960 to 30% in 1963. Coppock (12) found an incidence of 1.16% among the affected herds and .35% over the total lactation from all herds (267.844 lactations). Markusfeld (41) noted an increase of the incidence rate from 0. 05% over the period 1969-1974 to 1.9% in the period 1975-1976.

Hesselholt and Grymer (31) saw an increase of admissions to the clinic from 16% in 1967 to 24.3% in 1977 and reported an overall incidence of 0.12% - 1.9% in 96,400 cows in Denmark.

Sutherland (67) concluded from a 13 year survey in a population of 700 cows that the incidence of abomasal displacement had not increased.

Varden (78) reported an incidence of 0.2 - 0.4% over a 10 year period in a cow practice covering 2,400 dairy cows.

Erb and Grohn (21) found an incidence rate of 1.1% in Holstein cows in the first 21 days after calving. Dohoo and Martin (18) reported a lactational incidence rate of 1.4% from 2,875 lactation records of 2,008 cows. Robb *et*

Paper presented at the XVI World Buiatrics Congress, Salvador, Bahia, Brazil, August 13-17, 1990.

al. (59) reported a fairly high incidence of 4.36% in 50 dairy herds.

Pinsent (56) was the first to report a marked seasonal incidence after correction for seasonal calving patterns. The majority of the cases occurred between October and April, that is during the stabling season. This was confirmed by others (14, 42, 44, 59, 65). A few reports (18, 78) mentioned no seasonal pattern.

Dirksen (12) reported that most cows had recently calved for the 3rd or 4th time. Most workers agree on the fact that the incidence increases through the 4th to 6th calving (30, 42, 44, 59, 69, 78).

Occasionally a higher incidence in first calf heifers is reported (8, 84).

Pinsent (56) again was the first to report that Channel Island breeds were at increased risk. This was confirmed by others (34, 42) but Robertson (60) found that the breed distribution of DA cases was similar to that of the whole population.

Most of Pinsent's (56) cases occurred during the first week post partum (64.3%) and 11.0% occurred from 1 - 4 weeks post partum.

Others (16, 22, 28, 46, 48, 78, 87) agree that during the period from 2 weeks ante partum to 2 weeks post partum the majority of the cases occur. The median day post partum of diagnosis in a Canadian study was day 8 (18) and in a New York study day 1 to day 15 (22).

Milk Production

It has been suggested early that a higher incidence of DA is to be expected in high yielding cows (12, 25, 26, 42, 44, 59). Later studies (59, 18) however found no differences in herd milk yield between high incidence and low incidence herds. Erb and Grohn (21) concluded from their studies that it is unlikely that a high cow milk yield is a risk factor for LDA.

Heritability

When tracing sire lines, higher rates of LDA were found among certain bull lines in two studies (43, 66).

Concurrent Diseases

Dirksen (16) found that in his clinical material a considerable amount of cows had a concurrent disease, 60% had ketosis. 10% had retained placenta and 8% had endometritis.

Hull and Wass (30) suggested that hypocalcaemia predisposed for DA, a suggestion later supported by Varden (78) who reported that 30% of 51 cows with DA had received treatment for parturient paresis before displacement was ascertained.

These findings, also have been reported by others, (14, 60, 78, 84) gained considerable interest in epidemiological studies using bivariate or multivariate analysis. In some of these studies the association between DA and milk fever has been ascertained (21) but in others such an association was denied (12, 41). The same opposing results were obtained in studies of other potential risk factors such as retained placenta (41, 18) and metritis (21, 41). Erb and Grohn (21) concluded that milk fever is a risk factor for LDA and that much of the association is mediated by retained placenta. Mastitis has been ruled out as a risk factor (21). The risk for DA in cows with ketosis was 39.04 times ($P < 0.01$) greater than in cows without ketosis. This relation was confirmed in other studies where clinical ketosis diagnosed prior to LDA was strongly related to LDA (13, 59).

Nutrition

It was soon after the first publication about DA that the suggestion was put forward that the occurrence of DA was caused by the increased use of concentrates in rations for dairy cows. Later this was specified by pointing out that steaming up (11, 42, 60), rations rich in protein (25) or rich in fat (29) were associated with a high incidence of DA. Nocek *et al.* (49) divided 289 cows into 3 groups during the dry period and fed all hay, 50% hay/50% corn silage or limited corn silage plus liquid protein supplement. They saw 3, 4.3 and 6.3% LDA respectively in these groups. The differences however were not significant.

Complete rations, especially those with a high grain percentage were used in herds experiencing a high incidence of DA (28, 44, 50). The avoidance of abrupt ration changes is a frequently given advice (60, 11).

Danish workers point out that in rations low on crude fibre (16% or less) there is excess risk for DA (27).

Robertson (60) however found no correlation between the amount of grain fed and the incidence of LDA within herds. Curtis (13) even found that higher energy intakes in the late dry period were preventive for LDA.

Also Robb *et al* (59) in their study of 50 dairy herds were not able to find differences in lead feeding, challenge feeding, dry matter, net energy or crude protein between high versus low incidence herds.

Housing and Other Husbandry Factors

It was suggested that the lack of free movement in tied stalls increased the incidence of DA but, studies of Martin (42) suggested that more cases occurred in herds using loose housing. It is shown that on pasture a DA can disappear spontaneously (34).

Other Factors Possibly Associated With the Occurrence of DA

Danish workers, headed by Poulson (37, 51, 52, 53, 54) have found that in high-yielding cows the acid-base balance changed in alkalotic direction during the year from summer to spring. They also showed that a change in the acid-base balance will induce an atony of the abomasum.

The changes in the acid-base balance in the alkalotic direction taking place during the winter and spring months occurred simultaneously with the increase in beet, beet-products and concentrate in the ration suggesting that it was dependent on the change in feeding.

In this respect it is remarkable that Markusfeld (41) found that the risk of DA in cows with aciduria was 6.17 times greater ($P < 0.01$) than for those with a normal urine reaction. Metabolic alkalosis in cows is often accompanied by paradoxic aciduria (38).

Pathogenesis of DA

Most workers agree on the theory that the factors responsible for displacement of the abomasum can be anatomical and mechanical. There normally may be some displacement of the abomasum before parturition. Lagerlof (40) found that at the end of the gestation period the abomasum is situated left of the midline. Jones (36) found that in 50 cows slaughtered while in dorsal recumbency the abomasum was situated right of midline in 50% of the cows. In 12% of the animals the abomasum was found on the midline and in 30% of the cases it was situated left of the midline.

Weaver (83) using the same procedure saw 61% right of the midline, 28% on the midline and 11% left of the midline. Begg and Whiteford (3) argued that the pregnant uterus forces the abomasum forward and lifts the rumen from the bottom of the abdomen. This brings the abomasum in a semi-displaced position. When, after calving the rumen returns to the bottom of the abdomen, the abomasum then is fixed in this semi-displaced position. Gas and ingesta then would accumulate and the subsequent abomasal dilatation would be followed by displacement.

Sack (62) confirmed that the position of the abomasum could vary in normal cows, but could not relate the position of the abomasum with the roughage/concentatio ratio in the ration.

The suggestion that anatomical factors play a role in the pathogenesis of DA is illustrated by Albert and Ramey (1) describing a patient with hydrops ascites due to chronic liver cirrhosis who developed an LDA. In this respect it is of interest that Markusfeld (41) found that the risk of DA for cows which had twins was 3.25 times greater ($P < 0.01$) than for those with a single calf.

On the other hand it is shown that in cows where a LDA was induced surgically, no changes could be found neither clinically nor hematologically (69).

Also the finding that an LDA can exist in cows for a long perod without clinical symptoms (2, 35) suggests that other factors than anatomical must be responsible for the disturbances shown by cows sufferng from DA. This is supported by the facts that DA is found in bulls, oxen, calves and yearlings and that concurrent diseases, metabolic disturbances and nutritional factors have been identified as risk factors for DA.

Most workers nowadays agree on the fact that abomasal hypomotility or atony are the primary conditions for the development of DA. As a consequence of this hyomotility, ingesta and gas are trapped in the abomasum, dilatation and dislocation follows. In the process of dislocation, anatomical factors may play a role. Swendson (68) suggested that the volume of the fore stomachs determines the direction of dislocation.

It is well known that the motility of the abomasum can be influenced by many factors. Svendson (68) following the suggestion that introduction of large amounts of concentrates had caused the increased incidence of DA examined abomasal contractions and abomasal gas production after feeding large quantities of concentrates. It was found that feeding large quantities of concentrates reduced the frequency of abomasal contractions and increased the production of gas in the abomasum. Infusion of ruminal contents from an animal fed a high concentrate diet into the abomasum of experimental cows reduced abomasal motility. The same was found for a solution of volatile fatty acids (VFA's) of a similar composition as found in ruminal fluid of a cow on a high concentrate diet. Bolton *et al.* (5) found that intra abomasal infusion acetic, propionic or bytyric acid was associated with a marked decrease in abomasal action potential activity and emptying rate. Butyric acid was the most effective followed by propionic and acetic acid. Ehrlein and Hill (19) however noticed no effects on abomasal motility when VFA's were infused in the abomasum. Twisselman (70) could not find an increase in the concentration of VFA's in abomasal fluid after feeding a high concentrate diet although the concentrations of VFA's in the rumen increased considerable. Breukink and De Ruyter (7) compared a high grain ration with an all hay ration and found that after feeding, a strong increase in ruminal VFA concentration occurred on the high grain ration and that no changes were seen after feeding hay. There were no changes in the concentration of VFA in the abomasum after feeding the two rations and also, between the rations, there was no difference in abomasal VFA concentration.

Hatanya *et al.* (29) found no effect on abomasal motility after introduction of VFA in the abomasum, but they found a decreased motility after the introduction of long chain fatty acids. They suggested that the introduction of considerable amounts of fat in the ration predisposed for abomasal hypomotility.

The role of hypocalemia in abomasal hypomotility was suggested by Hull and Wass (33) and Poulsen (51). Poulsen (54) stated that the combination of hypocalcemia and alkalosis in high producing cows can lead to serious hypomotility. The findings that cows suffering from DA show alkalosis can also be the consequence of a hypochloremia due to sequestration of chloride in an atonic abomasum (6). Vlaminck *et al.* (82) using broomsulfophthaleine (BSP) as a marker found that in cows with LDA the passage of contents through the gastrointestinal tract was significantly slower. After oral application of calcium carbonate (20 g/100 kg B.W.) in the presence of sodium to increase calcium absorption, the abomasal displacement disappeared in 11 out of 21 animals. However after the treatment was stopped a recurrence was observed frequently.

It is frequently suggested that abomasal motility is influenced directly or indirectly by mediators originating from concomitant disorders such as mastitis, retained placenta and endometritis.

It is shown that prostaglandin (PGE2 en PGF2α) has influence on the spontaneous motility of the antrum pyloricum of the bovine abomasum (72). Vlaminck *et al.* (81) found a small decrease in the rate of emptying of the abomasum when PGE1 and PGE2 was injected intramuscularly, PGF2α showed no effect. Repeated administration of prostaglandin synthesis inhibitors to animals suffering DA had no corrective effect. They concluded that their study did not support a possible role of prostaglandins in the origen of DA.

Studies of antral myo-electric activity, intraluminal pressure and duodenal flow revealed no significant relation between these parameters (80). This may be the consequence of the complexity of the interrelationship of abomasal, pyloric and duodenal activity.

In a study of the myo-electric activity pattern of the abomasal body in adult cows it was found that long lasting periods of inactivity exist, that occurred less than once per day but lasted sometimes several hours independent of whether the cow was standing or was lying down (39). If these periods of real inactivity are to be interpreted as periods of real mechanical inactivity, then they could play a role in the etiology of DA.

In recent years some interesting results have been obtained in studies regarding the relation of fatty infiltration of the liver and DA. Fatty liver is related to imparied fertility and increased incidence of diseases of the early post partum perod (58). Holtenius and Niskanen (32) found that cows with LDA had moderate to severe fatty changes of the liver. They suggested that the feeding regime prepartum might influence the concentrations of plasma lipids and the fatty infiltration in the liver. A high energy, low protein feeding especially seemed to predispose for accumulation of fat in the liver cells. The imbalance in the feeding before calving in their opinion may cause the metabolic disturbances resulting in fatty changes of the liver and may cause abomasal displacement.

In an extensive study Muylle *et al.* (47) showed that cows with DA had severe hepatic lipidosis and ketosis but they saw no glycogen depletion of the liver cells. In cows with DA but without hepatic lipidosis, glycogen depletion of liver cells were found. They found evidence of hepatic lipidosis as the main problems in 7 herds sufferng from a high incidence of parturient problems such as DA.

Van Di jk *et al.* (73) also found a higher incidence of fatty liver infiltration in cows suffering from DA compared with normal cows examined during the early postpartum period.

In another study Van Di jk *et al.* (74) studied blood and liver lipids before and after calving in 22 herds. The incidence of milk fever, retained placenta, endometritis, mastitis, ketosis, abomasal displacement and infertility was recorded in the 49 pluriparous cows involved in the study.

Sixteen cows (33%) were classified as having severe hepatic lipidosis (HL), 26% had moderate HL and 41% mild HL. Of all the cases of periparturient diseases 56% were found in cows with severe HL. The observed lower glucose and high NEFA and 3-hydroxybutyrate concentrations as well as a higher loss of body conditions in severe HL cows suggest a strong relationship between the degree of energy deficiency and that of the fat mobilization as well as between the degree of energy deficiency and the severity of hepatic lipidosis.

Van Meirhaeghe *et al.* (76) found that cows with DA had higher basal blood glucose levels than control cows independent of their ketotic status. They also found a much lower secretory response of insulin secretion in cows with DA. They suggested the presence of an insulin imsensitivity in these cows (75). The basal insulin levels observed in cows with DA were significantly higher than the expected values in the same post partum period of normal cows. Endogenous as well as exogenous insulin caused a marked decrease of abomasal emptying rate. They suggested that, since VFA's such as butyrate, iso-valerate and valerate induce a more pronounced insulin response than glucose, insulinogenic substances may be involved in the regulation of abomasal emptying and the disturbance in VFA production and/or metabolism may play a role in the pathogenesis of abomasal displacement

Insulin insensitivity caused by a decreased number of insulin receptors has been observed in obesity in man and sheep (77, 45).

These observations suggest that prevention of hepatic lipidosis may also prevent the occurence of DA.

Treatment

Displaced abomasum can be treated effectively by several surgical techniques (61).

The closed surgical techniques on blind-stitch aboma-

sopexy and the bar suture techniques are slightly less successful (63).

A non surgical treatment by rolling the cow is highly effective but has a high rate of recurrence and a much lower rate of recovery, independent on the additional treatment with calcium infusions, hypertonic saline infusions or oral administration of ammonium chloride (71).

Abomasal torsions or mesentenic torsions have been observed following LDA correction by casting and rolling (71, 64).

The decision which method to use or not to treat and sell the cow is largely dependent on the economic value of the cow.

It was concluded from a Dutch study (9) that surgical treatments had the highest expected monetary value in cows with a lactation value of 10% above herd average. A USA study (63) concluded that closed surgical techniques had expected monetary value close to surgical techniques. Rolling is preferred over selling but if an LDA recurs, surgical treatment or selling the cow is preferred over rolling.

Studies are under way to evaluate the possibilities of newly developed drugs that may influence abomasal motility and subsequent abomasal emptying. In this respect an abundance of information becomes available regarding the so-called pro-kinetics, substances that increase the motoric activity of the gastro-intestinal system such as dopamine-antagonists (domperidon, metoclopramide), cholinomimetics (carbachol, physostigmine) and drugs that stimulate the liberation of acetycholine at the neuromuscular junction such as cisapride.

These drugs and their analogues may become important in the treatment of LDA after correction by casting and rolling or even without that.

Prevention

From the study of the etiology of DA, a certain number of preventive measures can be drawn. Parturition, high milk production and aging are inevitable but feeding, management and housing can be controlled. Feeding a high energy diet in connection with low fibre has to be avoided. At least 1/3 of the ration has to exist of fibrous material with fibers of at least 5 mm length. The avoidance of abrupt ration changes is recommended frequently (50). Especially the feeding in late lactation and during the dry period has to gain more interest in order to avoid hepatic lipidosis. An energetic balance is important in this period. Corn silage, for instance may well be excluded from the ration.

It is not possible to prescribe a ration which will prevent DA under all circumstances but it is reasonable to believe that an energetically balanced diet containing sufficient fibrous material in late lactation and during the dry period is of great importance in the prevention of hepatic lipidosis and abomasal displacement. By changing the diet in such a way, Wenting (86) observed that in herds with a high incidence of hepatic lipidosis and post-parturient disorders the incidence of hepatic lipidosis decreased significantly and no DA occurred. One can expect that in the coming years more detailed information regarding this method of prevention of DA will become available.

Summary

The incidence of abomasal displacement continues to show a gradual increase. The need for preventive measures has therefore become a pressing matter. Successful prevention of abomasal displacement is based on adequate knowledge of the aetiology and pthogenesis. The present state of affairs is reviewed on the basis of the literature, personal experience and studies. Obviously, a large number of factors may play a role in the pathogenesis of abomasal displacement. Although knowledge of the aetiology of abomasal displacement is anything but complete, a number of (probably useful) preventive measures are suggested.

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