Left Displaced Abomasum (LDA) in Dairy Cows

What it is, where it comes from and what you can do to prevent it

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Summary

Left displaced abomasum (LDA) is a frequent and costly disease. It may be expected to occur in 3% - 5% of cows within one month after calving. Each case will produce costs of more than US$ 600. It has a complex pathogenesis which is largely related to feed and feed management but is intrinsically linked to metabolic diseases of the transition period. While LDA may be corrected in individual animals with fair chances of economic success, it is necessary to monitor the condition on a herd basis: An incidence of greater than 3% should lead to active measures against. It is difficult to predict LDA in individual animals: Metabolic parameters are not specific enough, while sensor technology can so far not predict it early enough. Preventing LDA is mainly related to the general principles of preventing metabolic disease but must focus specifically on feed management.

What does "LDA" mean?

Ruminants have a very different digestive system from monogastrics: A complex of "forestomaches", most notably the rumen, serves as a fermentation chamber for plant material. After the forestomaches, the ingested feedstuff passes through the abomasum. The abomasum is the equivalent to the stomach of monogastrics and is situated close to the ventral abdominal wall. An "Abomasal Displacement" means that the abomasum leaves this position: In certain situations, the abomasum may become atonic and flaccid and accumulate excess fermentation gas. Filled with this excess gas the abomasum may ascend along the abdominal wall, drawn by this gas, to the left side of the abdomen; more rarely it may ascend to the right site. This is called "Left Displaced Abomasum" (LDA) or, less frequently, "Right Displaced Abomasum" (RDA). The displacement usually occurs in early postpartum dairy cows. A displaced abomasum effectively blocks the passage of a normal ingestion. Clinical signs of LDA therefore are reduced dry matter intake, decreased faecal output and possibly a general depression. The degree of symptoms may vary. In cases of RDA, the ascend of the abomasum usually involves a "volvulus", i.e. a turn of the organ, leading to a blockade of blood flow and aggravating the symptoms. (Smith 2002).

How common is LDA?

Like a lot of metabolic diseases, LDA tends to be underdiagnosed and under-recorded, i.e. the condition is either not recognized or a diagnosis is not recorded by the farm (Pryce *et al*. 2016). This makes a precise evaluation of disease incidence difficult with only a few reports covering a larger number of herds and animals.

The US National Animal Health Monitoring system reported an LDA incidence of around 3.5% with 2.5% in larger herds (>500 cows) and 4.8% in smaller herds (<500 cows). 50% of these cases were diagnosed within two weeks after calving, 80% were diagnosed within the first month of lactation. (Caixeta *et al.* 2018). This magnitude of incidence is recurring in most publications. In a prospective study covering 1044 cows from 20 herds a total of 53 cases, representing an incidence of 5.1%. The median time from calving to diagnosis in this study was 11 days. (LeBlanc *et al*. 2005). (Pinedo *et al.* 2020) analysed health records of 11729 cows from different areas of the United States. While the occurrence of LDA in primiparous animals was negligible (prevalence <1%), the authors found the incidence influenced by season and geography. While in the warm season the incidence may be as high 3.9%, this would decrease to 2.2% in the cold season. The lowest incidence reported was 1.85% for the Northern region of the US during the cool season.

It may be concluded that an incidence of 3% per year should be set as an alarm value, requiring action, while well-managed herds may reach an incidence of 1% (Caixeta *et al*. 2018).

What is the pathogenesis of LDA?

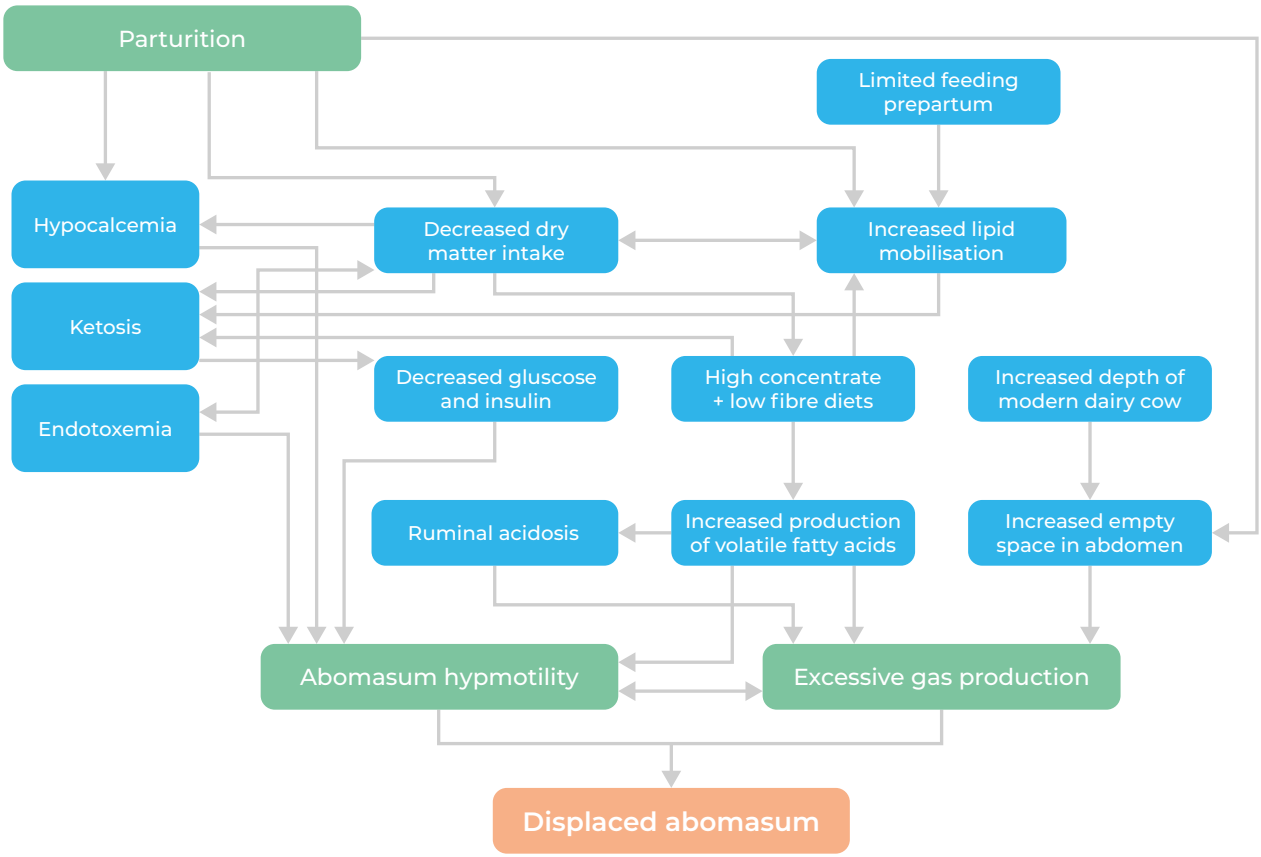
LDA is to be understood as a consequence of hypomotility of the abomasum. Muscular samples from cows with DA are less responsive to stimulation by electricity or neurotransmitters (Geishauser *et al*. 1998). The hypomotility leads to two synergistic mechanisms: Fermenting

ingesta in the organ produce gas. Neither gas or ingesta are removed by normal motility, leading to accumulation of gas and eventually excessive inflation of the organ (Caixeta *et al.* 2018).

The reason for this hypomotility is not completely understood but is certainly multifactorial. As said before, LDA is a condition almost exclusively found in the post-partum period and predominately in multiparous cows. This points to the pathogenesis of the condition: The hypomotility and consequently dislocation of the abomasum are intrinsically linked to the metabolic challenge dairy cows are facing during the transition period. Hyperketonemia, hypocalcaemia, hypokalaemia and endotoxemia may be present during the first weeks after calving and contribute to the occurrence of LDA (Caixeta *et al.* 2018). Hyperketonemia as pathway-disease has been repeatedly named as preceding LDA and, although the pathway is not quite clear, cows showing low plasma glucose and insulin have been shown to be likely to experience LDA (van Winden *et al.* 2003). Hypocalcaemia has been identified as a major precursor, with animals experiencing low plasma-calcium values being 4.8 more likely to develop LDA than normocalcaemic cows (Massey *et al.*). Hypokalaemia has retrospectively been shown to be present in cows with DA (Constable *et al.* 2013) but is unlikely to play a decisive role in pathogenesis (Caixeta *et al.* 2018). Endotoxemia may be a consequence of fat mobilization or ruminal fermentation disorders post-partum and has been shown to reduce contractility of smooth muscles and may therefore contribute to the occurrence of abomasal dislocation (Zebeli *et al.* 2011).

In addition, a biomechanic influence may facilitate the dislocation of abomasum: After calving, the uterus dramatically decreases in size, creating space in the abdominal cavity. This may in turn facilitate the dislocation of an already inflated abomasum (P. D. Constable *et al.* 1992). The fact that dairy cows have gained in frame size in recent decades may therefore contribute to the occurrence of LDA in modern dairy cows (Becker *et al.* 2012).

An overview of the different influences leading to abomasal displacement is given by this chart from (Caixeta *et al.* 2018):



What are the risk factors of LDA?

As Abomasal Displacement is frequently understood because of Ketosis / Hyperketonemia and hypocalcaemia, respectively, the risk factors leading to these metabolic alterations also apply to DA. There are, however, certain risk factors that may contribute to the occurrence specifically:

Ration design. Several pathways facilitating DA through ration design have been suggested:

- The presence of excessive concentrates in a ration may subsequently lead to a higher presence of easily fermentable substrates in the abomasum as well. With an atonic abomasum, this may result in an excessive gas production facilitating the dislocation of the inflated organ (Shaver 1997; Cameron *et al*. 1998).
- By a deficiency of physically effective fibre a physiological fibre-mat will not form in the rumen. This will alter ruminal motility and thereby predisposing the abomasum to excessively produce gas (Shaver 1997)
- It has been postulated that the presence of concentrates will lead to higher osmolarity of the ruminal content, drawing water into the lumen, thereby increasing passage rate of undigested substrate into the abomasum. This will in turn lead to more fermentation producing the gas (T. Sarashina *et al*. 1990).

Feeding management:

- Feeding management and hygiene is repeatedly named as a predisposing factor for DA (Caixeta *et al*. 2018; Cameron *et al*. 1998). In a Swedish study, the cleaning of the feed alleys turned out to be the most relevant risk factor (Stengärde *et al*. 2012).
- The availability of feeding space, i.e., trough length, has been identified as a risk factor for development of DA. It is suggested that the increasing competition in overstocked pens with limited feed access changes the feed intake pattern and will negatively affect the health of the ruminal-abomasal complex(Cameron *et al*. 1998; Collings *et al*. 2011)

Body condition score (BCS):

- Although probably more linked to predisposing metabolic disease, it was shown that an excess loss of body condition after calving predisposes cows for developing DA (Gearhart *et al*. 1990)

Genetics:

- There is an ongoing discussion on whether DA is predisposed by genetics. Heritability is estimated differently between h^2=0,03 and h^2=0,53 (Zerbin *et al*. 2015). Although the increased frmae of modern dairy cows is believed to contribute to the likelihood to develop DA (see above: (Becker *et al*. 2012) , the genetic influence is generally believed to be low (Doll *et al*. 2009). A positive correlation between milk production traits and predisposal for DA is frequently discussed, however uncertain (Pryce *et al*. 2016)

What are the consequences of LDA?

There are various surgical methods for surgical correction of LDA. They range from open surgery to minimal invasive methods using endoscopy or the so called “blind stich”. The techniques have been extensively reviewed (Karvountzis 2016). The prognosis after surgical correction is usually good and cows return to milk production soon after. In a retrospective study from an Irish veterinary practice it was reported that from 54 cows operated, 76% returned to production, 72% of these animals being pregnant again 6 months after surgery (Sexton *et al*. 2007). In a more comprehensive study, the survival rate of 1411 cows operated at the Veterinary University of Hanover was examined. Of these, 38% were culled within one year with an average survival time of 138. The average survival time of all cows examined was 467 days (Ricken *et al*. 2005).

As cows still may experience ruminal or abomasal atony post-surgery, the use of drugs promoting contractility may be considered. Interestingly, the macrolide erythromycin has been shown to effectively stimulate contractility of the abomasum (Constable *et al*. 2012). Due to its antibiotic nature, however, its use is limited and it can practically not be used to treat cows that have been found to be at risk for developing LDA.

The economic consequences of one case of LDA comprise the direct costs (treatment, milk withhold etc.) and indirect costs. These indirect costs are likely to be the greater share and comprise risk of culling, milk production loss in the lactation, reproductive losses, and others. In a stochastic simulation modelling the average cost of various diseases, LDA turned out to be the most costly disease with $432.48 in primiparous cows and $639.51 in multiparous cows (Liang *et al*. 2017). The total cost of displaced abomasum for the US dairy industry has been estimated to exceed 150 million US-$ per year (Caixeta *et al*. 2018). This points to the necessity to monitor, predict and finally prevent this condition.

How can cases of LDA be predicted?

Caixeta *et al*. (2018) concluded from an extensive review that only monitoring the incidence of DA was not sufficient to control the condition, as it was a multifactorial issue. The time delay from onset of causative conditions, such as hyperketonemia to development of the condition and, eventually, its diagnosis, make a control based on the diagnosis alone virtually

impossible. Prediction of LDA risk is so far based on two principles:

- **Metabolic analysis.** In their retrospective study, LeBlanc *et al*. (2005) describe the results of a thorough observation of different parameters including concentration of non-esterized fatty acids (NEFA), beta-hydroxy-butyrate, minerals and BCS. Between 0 and 6 d before calving, cows with NEFA concentration > or =0.5 mEq/L were 3.6 times more likely to develop LDA after calving, whereas other parameters examined allowed no prognosis on this condition. The sensitivity, specificity, and likelihood ratio (LR) were 46%, 82%, and 2.6, respectively. Up to 7 days after calving, retained fetal membranes (RFM), metritis, and increasing serum concentrations of BHBA and NEFA were associated with increased risk of subsequent LDA. However, considered separately, postpartum serum BHBA was a more sensitive and specific test than NEFA concentration. The odds of LDA were 8 times greater in cows with serum BHBA > or =1200 micromol/L (LR = 3.5). Cows with milk BHBA concentration > or =200 micromol/L were 3.4 times more likely to develop LDA.
- **Sensor technology.** Electronic monitoring of animals' activity and recording changes in behaviour, e.g., in ruminating, have been shown to reliably point to clinical disease, including LDA. However, the disadvantage is the proximity of changes recorded to manifestation of clinical disease (Abuelo *et al*. 2021). In their study, the researchers were not able to define rumination changes at drying off that would point to LDA post-partum, unlike with lameness or hyperketonaemia that could be predicted by rumination activity as early as drying off.

In conclusion, predicting LDA is possible in a very limited way only: Biochemistry may point to metabolic disease risk, but is rather unspecific when it comes to LDA. Sensor technology allows for early identification of animals developing the condition but is so far not able to identify animals early enough, e.g., at drying off.

How can LDA be prevented?

As pointed out earlier, LDA is related to metabolic disease with hyperketonemia currently understood as "pathway-condition" leading to all other post-partum diseases of the dairy cow. Preventing LDA therefore means to prevent metabolic disease in the first place. The specific prevention of LDA is described nevertheless and mainly relates to feed management and ration design (Caixeta *et al*. 2018). The prevention of LDA should start well before the onset of the new lactation and comprise the dry period, possibly the late previous lactation as well. The key elements are:

- Limiting energy intake in the dry period. This may be achieved by adding straw to the ration so to dilute the energy content.
- Providing physically effective fibre throughout. This aims at providing a normal ruminal fermentation and the formation of an intraruminal fibre-mat. This should limit the passage of easily fermentable substrates from the rumen into the abomasum.
- Control of TMR fed to late-lactation, dry period, and early lactation to provide a consistent feed quality throughout and minimize sorting behaviour.
- Provision of feed 24 hours a day and provision of feeding space to limit competition.
- Avoidance of overstocking
- Hygiene of all areas related to feed: Feedstuffs and feedbunks. Regular cleaning of feedbunks.
- In individual animals a calcium-supplementation (e.g., by orally administered products such as boli) to ensure adequate calcium levels.

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