

Left displacement of the abomasum in dairy cattle: recent developments in epidemiological and etiological aspects

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Abstract – The research with respect to displacement of the abomasum (DA) in dairy cattle is reviewed. Evaluated articles describe epidemiological and experimental studies. The occurrence is elevated with regard to breed, gender, age, concurrent diseases, environmental aspects and production levels as contributing factors and emphasis is placed on the effects of nutrition and metabolism. Reviewing the experimental work, distinction is made between the research into gas production in the abomasum and hypomotility of the abomasum, since both represent presumed pathways in the development of DA. Although the different fields of research have positive contributions to the understanding of the pathogenesis of DA, contradictions in the different studies are present. This is partly due to extrapolation of results from sheep to cows, or because of a low number of cows in the experiments. Finally, general suggestions are made for further research in the field of the pathogenesis of DA.

abomasal displacements / risk factors / etiology / review

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1. INTRODUCTION

Since the first report of displacement of the abomasum (DA) in a cow in 1950 [2], this disorder in dairy cattle nowadays has become more common. The DA is characterised by the abomasum filled with gas floating in the dorsal part of the abdomen. This state can result in anorexia and signs of colic, accompanied by a drop in milk yield, discomfort of the cow and, in some cases, death [15]. After (non-) surgical correction of the position of the abomasum the production can be disappointing, which may result in culling of the cow. The total estimated economic loss of a case of DA is between US \$250 to \$450 [1]. Geishauser et al., have calculated the annual loss in North America due to DA as up to 220 million dollar [23]. The incidence of DA varies, depending on the country, from 0 to 7 percent per year [5, 35]. There is however a large variation at herd level within a country [63, 69]. Some herds seldom have a case of DA, while in other farms the incidence can be 20% [13, 31, 34]. When the herd-incidence is high, DA can result in considerable economic losses.

In the pathogenesis of the DA the accumulation of gas in the abomasum is crucial.

The underlying hypothetical cause of this accumulation is a combination of two pathways: an increased production of gas in the abomasum and a hypomotility of the abomasum [15]. The gas accumulated in the abomasum consists mainly of methane (70%), and carbondioxide [15, 59]. In a normal functioning abomasum the gas production is equal to the clearance in oral or aboral direction. When motility of the abomasum is inadequate accumulation of gas may occur [3, 15, 19]. The vagus nerve plays a predominant role in abomasal motility [10, 11, 19, 22, 54]. Besides the effect of the vagal nerve, large amounts of volatile fatty acids (VFA) in the rumen and abomasum [4, 24, 50, 60, 67], endotoxins [62, 67, 68], metabolic alkalosis [50] and low blood calcium levels [42] are mentioned as plausible causes for a decreased motility. Kuiper and Breukink reported periodical inactivity of the abomasum, mainly the corpus [36]. They suggested a relation with the laying position of the cow, or a day-night rhythm, since the inactive periods occurred mostly at night-time. In a postpartum cow one or both mechanisms, hypomotility and gas production, can play a role, resulting in accumulation of gas and buoyancy of the abomasum. In Figure 1 a flowchart is presented. In this

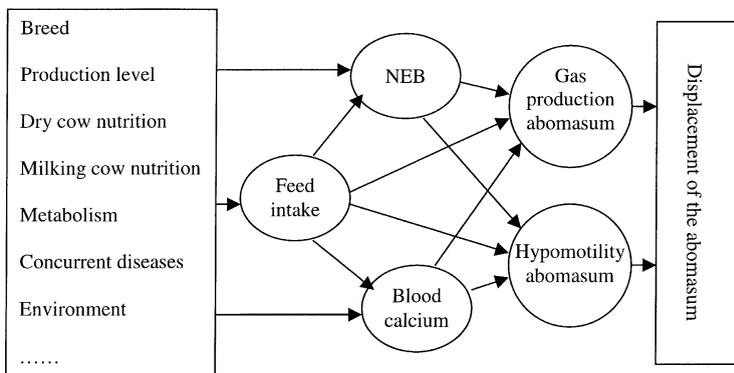


Figure 1. Flowchart of risk factors of DA (in left text box), related to etiologic factors (ovals). The pathogenic pathway (circles) of the etiologic factors resulting in displacement of the abomasum are also presented.

chart epidemiological aspects relate to etiological factors. These etiological factors are linked with the two pathogenic pathways (hypomotility and gas production) that lead to DA.

The research on abomasal displacements consists mainly of epidemiological surveys and experimental studies. The epidemiological research generated associations, risk factors and hypotheses. The experimental work is performed to test these and other hypotheses. The aim of this paper is to evaluate these efforts, with an emphasis on the developments in the last decade, and to suggest directions for further research.

2. EPIDEMIOLOGICAL FACTORS

2.1. Species, breed, gender, age and production level

The displacement of the abomasum as a disease is described in ruminants of the Western Hemisphere, sheep, goat and cattle, both male and female. With respect to the incidence of DA one should conclude that DA is mainly a disorder associated with cattle, in particular Holstein-Friesian, Jersey and Guernsey cows. Geishauser et al. [20] and Uribe et al. [61] reported a heritability of DA of 0.24 in German black-pied cattle and 0.28 in Holstein cows respectively. Van Dorp et al. [63] however, could not confirm this finding in Holstein cows.

The major risk period is the first month after calving, with an increasing risk with increasing age [7]. Other authors report that the first lactation is also a period with relative high risks for development of DA. This can be a result of a poor social and nutritional adaptation of the newly lactating heifer [33]. Lacasse et al. found a four times higher risk for DA in heifers that were fed ad libitum in the period of 1 to 1.5 years of age [37]. DA is associated with milk production: the higher the milk

yield the larger the risk of development of DA [17]. Other authors report that this relation is not always present [5, 53]. An explanation can be the findings of Detilleux et al.: DA cows have a 557-kg lower 305-day milk production than control cows and 30% of the milk loss occurred before DA diagnosis [14]. The general opinion is that cows that develop DA are high producing cows, but due to DA the current lactation period has a poor milk yield. Constable et al. conclude that, taking milk production into account, there is still an unexplained high incidence of DA in Jersey and Guernsey dairy cows [7]. An explanation can be that these breeds are more susceptible for the occurrence of hypocalcemia, which is discussed later.

2.2. Nutrition and metabolism

Cows developing a DA show depressed feed intake prior to DA [49]. This is in accordance with the remarks of Dirksen, who suggests that a decreased rumen filling enables the abomasum to move to the left, even before clinical presence [15]. This is reflected in the findings of Cameron et al. [5]. They report restricted roughage supply in late gestation as a risk factor for DA. There is an association between the amount as well as the quality of the roughage fed and DA [13, 31, 57]. Roughage of poor quality leads to a lowered feed intake with DA as a result [31]. Some authors recommended a fibre length of the roughage of minimal 1.3 to 2.5 centimetres [13, 57]. There is also an association between the kind of roughage fed and the occurrence of DA; the more maize silage is fed, the higher the probability of getting a DA (Van Winden, unpublished results). Parallel to these findings Cammack describes more frequent abomasal displacements and other disorders of the abomasum in herds that are fed a large proportion of maize silage [6]. The roughage component of the ration can not be regarded without looking at the concentrate part of the ration, since the

combination of both will result in the fermentation processes in the rumen. A proportion of at least 25% of roughage in the ration is a rule of thumb from nutritionists' point of view, where concentrates should be supplied three or four times daily [57]. Østergaard and Gröhn suggest that feeding concentrates together with roughage in a Total Mixed Ration (TMR) reduces the odds for DA [49]. Rumen fill, ration physical form and the amount of volatile fatty acid produced in the rumen are considered as some of the major causes for hypomotility of the abomasum resulting in the development of DA.

Nutrition as well as milk production and breed are confounded with metabolism. The modern high producing dairy cow has certain nutritional requirements to maintain the equilibrium of its metabolism. There are three factors concerning metabolism associated with the phenomenon of DA: hypocalcemia, metabolic alkalosis and negative energy balance (NEB). Several authors describe the association of hypocalcemia in postpartum dairy cows and DA [9, 45, 47, 53, 64]. Lowered calcium levels also in the second week of lactation are found in cows prior to DA [21]. A decreased contractility of the abomasal wall during hypocalcemia is the hypothesised cause of abomasal hypomotility.

Metabolic alkalosis is mentioned as a risk factor for DA [51]. Metabolic alkalosis can be a cause of hypocalcemia via a reduced sensibility of the receptors for parathyroid hormone (PTH). In Jersey and Guernsey cows there is a decreased number of vitamin D₃-receptors [29]. This can explain the relatively high incidence of DA in these breeds [7]. It seems likely that both the metabolic alkalosis as well as the Jersey- and Guernsey breed's susceptibility are based on the increased risk for hypocalcemia leading to an increased risk for DA. Hypocalcemia is the probable

pathway for the risk factors "breed" and "metabolic alkalosis".

Another disturbance in metabolism is the NEB. Every postpartum dairy cow develops a NEB. However, not every cow experiences problems with it. Disease depends mostly on the depth and the duration of the NEB. A severe NEB has been regarded to result in an increased risk for DA [5, 9, 21, 26, 53]. Pathways mentioned are hypo- or hyperglycemic status, hyper-, or hypoinsulinemia in these cows [28].

2.3. Concurrent diseases and environmental aspects

Concurrent diseases, other than hypocalcemia and the NEB, consist of (endo-) metritis and lameness. (Endo-) metritis can have a risk attributive effect on the development of DA [8, 53]. Endotoxins and mediators of inflammation can be a direct cause of DA via motility disorders or indirectly via induction of hypocalcemia. Lameness as a herd problem is more often seen in herds with DA [40]. The explanation is reduced feed intake by lame cows, resulting in DA.

Environmental aspects comprise season, weather, and housing system and housing quality. Reports of occurrence of DA in different seasons are not concise, in general most cases occur in winter [5, 7, 8]. The hypothesized reason for this high incidence is the declining quality of the stored roughage over winter, with poor intake of roughage as a result. There is evidence that besides season weather conditions influence the incidence of DA. Rainfall, low temperature and strong wind increase the incidence of DA when cows are at pasture, probably via a reduced intake of roughage (Van der Post, unpublished results). No recent epidemiological reports are available about the effect of housing systems and housing quality, nor about the effects of walking exercise of the cows.

3. ETIOLOGICAL FACTORS

Recent epidemiological studies have three main subjects, which generated hypotheses: feed intake, negative energy balance and calcium related effects on the abomasal functioning, with respect to motility and production of gas.

3.1. Feed intake

Epidemiological research revealed a decreased feed intake prior to the development of DA. Okine and Mathison report that in cows with higher dry matter feed intake (DMI) the amount of large particles in the gastrointestinal tract was increased [48]. This increase in large particles was combined with an increased digestive flow. Diets low in fibre causing low rumen fill result in a decreased digestive flow in lactating cows [16]. Beside a change in amount of feed, the postpartum dairy cow experiences a change in the composition of the ration. In the dry period the diet consists mainly of roughage, while after calving the ration is rich in concentrates. A diet containing concentrates compared with a ration of only roughage resulted in a reduced myoelectrical activity of the abomasum in sheep [38]. In cattle a change from a roughage rich diet to a concentrate rich ration however, had no influence on abomasal myoelectrical activity or abomasal emptying [43].

Feed intake leads to production of volatile fatty acids (VFA). The inhibiting effects of VFA on the activity of the abomasum are controversial [4, 60]. Gregory and Miller showed a reduced activity of the abomasum when infused with VFA concentrations of more than 100 mmol/L [24]. One should consider that concentrations used are fivefold the normal abomasal contents [4]. According to Forbes and Barrio the inhibitory effect of VFA on the activity of the abomasum occurs through the osmotic pressure [18]. A high osmotic pressure results in a decreased motility of the abomasum and a reduced feed intake.

Whether interaction of the vagus nerve is the case is uncertain. Martens suggested that osmotic pressure has its effect through an overload of the abomasum with fluid [44]. An osmotic pressure higher than 341 in rumen contents results in a flux of water into the rumen. Also an increased osmotic pressure resulted in a decreased absorption of VFA by the rumen wall [39]. The only pathway of diminishing rumen volume is via a drain towards the abomasum, which leads to a distension of the abomasal wall [44]. When the abomasal wall is stretched too much, this can lead to decrease of motility via the vagus nerve or the nonadrenergic noncholinergic (NANC) system [22]. An impaired response of the abomasal muscles to acetylcholine was noticed in DA patients as a result of an inhibitory effect of nitric oxide (NO). Nitric oxide is part of the NANC system and has a relaxing effect of smooth muscles [19, 55].

Sarashina et al. concluded that the gas in the abomasum originates from rumen fluid. CO₂:CH₄ ratio in gas of the rumen is on average 2, whereas the ratio in abomasal gas is 0.4 [56]. Absorption of CO₂ via the abomasal wall can explain the shift in CO₂:CH₄ ratio. In cows with a higher amount of concentrates in the ration the CO₂:CH₄ ratio in both rumen and abomasal fluid increased due to a shift in metabolic products of the microbial flora [41, 56]. These findings are in accordance with previous work of Svendsen, who reported an increased amount of gas escaping from the abomasum in cows, that were fed a concentrate rich ration [60]. In this report a control diet of hay resulted in a production of 0.5 L gas per hour in the abomasum, whereas the concentrate rich diet resulted in more than 2 L of gas per hour. Another possible route of gas production is fermentation of contents in the abomasum. This is only possible in conditions of an elevated pH of the abomasal contents above 5.5. Van Winden et al. reported such a rise in abomasal pH in postpartum cows [65]. Besides a rise in pH, there was a large

variation in pH of abomasal contents in these cows in the second and third week after calving.

3.2. Negative energy balance

Dairy cows postpartum undergo a negative energy balance due to the fact that energy loss (milk) exceeds energy intake. During the early lactation glucose and insulin blood levels decrease, whereas ketone bodies and non-esterified fatty acid level in the blood increase [25, 58]. Patients with a displaced abomasum however, have often an elevated glucose and insulin level in the blood circulation [12, 30, 46]. Holtenius et al. report decreased abomasal motility in cows with elevated insulin, glucose, and glucagon levels as well as in cows with high insulin combined with low glucose levels [27, 28]. The authors mention difficulties in interpreting the results since the levels of glucagon, glucose and insulin are dependent on each other and the blood levels exceed normal conditions. The effect of high glucose levels can be mediated by a decreased vagal tone [18, 28]. Van Winden et al. found low levels of insulin and glucose in cows that later on developed DA [66]. The latter situation reflects the general metabolic characteristic of a cow in postpartum NEB. The elevated glucose and insulin levels found in DA cows are probably secondary to the disorder, whereas low glucose and insulin levels precede DA.

3.3. Calcium

Calcium is present in the blood circulation both in ionised form and bound to proteins. The sum of both is the total calcium concentration in the blood. This is used in the following text. Although, in general, rumen activity declines during a moderate hypocalcemia of 2 mmol/L [32], Madison and Troutt found that a calcium level below 1.2 mmol/L had a reducing effect on the abomasal motility [42]. They conclude that these levels are too low for cows

several weeks in milk and thus hypocalcemia can not be a major causative factor for decreased abomasal motility with respect to the development of DA. Another causative role for calcium can be the fact that calcium is a second messenger in the parietal cell of the abomasum. In man a reduced acid secretion in the stomach is reported during hypocalcemia [52].

4. CONCLUSION

One can, in general, agree that epidemiological and experimental research both have contributed to the insight into the pathogenesis of DA. However, there is little co-operation between both fields of research. Epidemiological studies generate hypotheses, which are seldom evaluated by experimental work. When experimental work does evaluate epidemiological findings, these epidemiological findings sometimes cannot be explained and even contradictions do occur. It is reported that calcium blood concentrations are 0.1 to 0.2 mmol/L lower in cows prior to DA, compared to healthy counterparts. However, these levels are higher than calcium levels that decrease abomasal motility (1.2 mmol/L). Contradictions occur in the glucose levels prior to DA: epidemiological studies report lower concentrations prior to DA, whereas high levels of glucose are associated with hypomotility of the abomasum in healthy cows. Another incongruent finding is the feed intake: feed intake is lower prior to DA, while high levels of VFA concentrations and osmotic pressure of the rumen contents, both a result of high levels of feed intake, are reported as reducing the abomasal motility. These incongruent findings are in most cases due to a low number of experimental animals or to extrapolation of sheep results to cows. Although costs should be taken into consideration, further research is preferably performed on cows in sufficient numbers (at least 20). These experiments

could generate advice for feeding practice in order to prevent high incidence of DA. During the experiments causative factors should be evaluated. Besides variables concerning the NEB, endotoxins and radicals could be evaluated. When discriminating variables are distracted an in vitro study can confirm the causative relation. With these in vitro studies emphasis should be made on the production of gas in the abomasum and the effects on the contractility of the abomasal wall. By monitoring milk, blood, rumen and abomasal fluid a predictor of DA could be distracted. This predictor might be used in dairy practice to identify animals that are at high risk for DA. The indicator should be easy to access and cheap.

During current dairy cow husbandry emphasis should be on the transition period of the postpartum dairy cow, since this is the period at risk for DA. By optimising the ration in the dry period (prevent obese cows), facilitate the adaptation process of the lactating cow (socially and nutritionally by adding dry cow two weeks before the estimated calving date) and optimise postpartum feed intake (prevent concurrent diseases), the problem regarding displacement of the abomasum could be reduced to a minimum.

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