Herd-Level Monitoring and Prevention of Displaced Abomasum in Dairy Cattle

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KEYWORDS

• Displaced abomasum • Herd health • Dairy cows • Transition period

KEY POINTS

- Displaced abomasum is a multifactorial disorder; for this reason, veterinarians should not monitor displace abomasum alone but in association with other diseases that can lead to its development.
- Monitoring, prevention, and treatment of infectious and metabolic disorders characteristic of early lactation (eg, ketosis, retained placenta, metritis, mastitis, hypocalcemia) decreases the incidence of displaced abomasa on dairy farms.
- When formulating diets to prevent displaced abomasa, several aspects are important: maintenance of proper levels of fiber, maximization of dry matter intake, and limiting the energy density of the diet.
- Facilities and cow comfort play a significant role in the development of displaced abomasum; for example, adequate stall design, heat abatement, bunk space, limiting pen moves, and separating animals from different parity groups.
- Delivering a consistent and properly processed ration daily is a key element to be considered when monitoring feed management in displaced abomasum prevention.

Displaced abomasum (DA) is characterized by the displacement of the abomasum from its normal position on the right ventral aspect of the abdomen to the right or left DA (LDA) side in cattle, with LDA being more frequently diagnosed than right DA. Abomasal displacement is a multifactorial disorder diagnosed almost exclusively in adult dairy cows. In a nationwide survey, it was determined that the DA incidence in United States dairy herds was approximately 3.5%, with occurrence varying from

Disclosure Statement: The authors have nothing to disclose.

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Vet Clin Food Anim ■ (2017) ■–■

https://doi.org/10.1016/j.cvfa.2017.10.002 **vetfood.theclinics.com**

2.5% in herds with 500 or more cows to 4.8% in herds having fewer than 500 cows.[1] Moreover, DA is known as an immediate postpartum condition with more than half of LDA cases being diagnosed within 2 weeks after parturition and 80% of the LDA cases occurring within the first month of lactation.[2]

Economic analyses have determined that the average cost per DA diagnosis is more than $700 when accounting for direct (eg, examination, correction, medication, discarded milk, death loss) and indirect (eg, future milk production loss, loss of body weight, decreased reproductive performance, increase risk of removal from the herd) costs.[3] Assuming the 3.5% incidence reported by the US Department of Agriculture survey with the estimated 9 million dairy cows in United States, it is reasonable to approximate that the annual losses to the dairy industry from DA exceed 150 million dollars. Thus, monitoring and managing dairy herds to decrease the incidence of DA is of extreme importance for the sustainability of dairy production and to improve animal health and wellbeing.

The diagnoses, medical therapy, methods for surgical correction, and posttreatment consequences of DA in an individual cow have been reported by various investigators.[4–8] However, the evaluation and management of DA at the herd level have not been explored as consistently. Therefore, the objectives of this review article are to discuss the possible nutritional and nonnutritional factors involved in DA development and provide suggestions on evaluation and management strategies at the herd level to decrease its occurrence.

NONNUTRITIONAL RISK FACTORS FOR DISPLACED ABOMASUM AND NUTRITIONAL RISK FACTORS FOR DISPLACED ABOMASUM

Disturbances of the Abomasal Motor Activity

Abomasal atony has been reported as a key factor in the development of DA. Moreover, it has been hypothesized that abomasal hypomotility occurs before the distention and displacement of the abomasum. Mechanistically, this abomasal stasis contributes to the inflation of this organ because the gas produced during digestion is not pushed out and cannot escape.

Geishauser and colleagues[9] demonstrated in vitro motility disorders in abomasal wall tissue collected from DA cases. During this experiment, tissue samples from DA cases presented normal spontaneous activity of smooth muscle in response to myogenic activity stimulus, whereas nerve-mediated contractile responses were diminished and sensitivity to acetylcholine was significantly decreased. Taken together, these findings suggest that the impaired emptying of the abomasum due to abomasal atony might be determined by a malfunction of the enteric nervous system in addition to an impaired cholinergic muscle responses. Further, decreased concentration of motility-stimulating neurotransmitters in the abomasal wall[10,11] and decreased electrical response activity measured via electromyography have been reported in association with LDA.[12] Further investigations are needed to compare the levels of such neurotransmitters between animals affected by DA and healthy counterparts within the same breed to confirm its relationship with DA development. Nonetheless, in addition to the neurologic factors associated with abomasal hypomotility, factors such as mineral imbalances, endotoxemia, decreased concentrations of glucose and insulin, and elevated β-hydroxybutyrate (BHB), among others, have been reported as possible factors associated with abomasal atony preceding the development of DA.

Calcium is extremely important for proper smooth muscle contractility and neuromuscular transmission. Hence, low-serum or plasma calcium concentration, hypocalcemia, has been implicated as among the main metabolic factors associated with the