Impaction in Dairy Animals

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Impaction in dairy animals refers to the failure of digestion, slow passage and accumulation of feed in one or more of the stomach compartments leading to constipation or absence of dung. In last few years this condition has emerged as one of the most common digestive disorder in dairy animals of Punjab. It is more common in pregnant or young cattle of less than 8 yr of age. Impaction is of two types.

Primary impaction – it refers of accumulation of poor quality roughages in any of the forestomach compartments. It is relatively rare condition and involves nearly all animals of the herd. For example feeding of hybrid napier bajra was associated with one of outbreak of impaction in dairy animals in Punjab. Sometimes ingestion of inanimate or indigestible substance like ropes a polyethene is associated with such impaction in sporadic cases in herd.

Secondary impaction – any inflammation of gut or associated organ which decease gut motility lead to secondary impaction. In a study carried out on forestomach disorders in GADVASU teaching hospital this kind or impaction has been observed as the most common cause for animals in our state. The major causes and management of this type of impactions are discussed below:

Penetrating foreign bodies: Nearly all agricultural operations like chaffing, cutting fodder, thrashing of wheat, grinding, mixing concentrates etc. are done mechanically in our state. This lead to spillage of broken sharp parts of machines, nails etc. in the fodder. Moreover our dairy animals are kept close to human inhabitation which allow accidental access of various hardware items in the fodder. Physical evidence of penetrating foreign bodies in more than 35% of the cases consisting of hair pins, nails, screws, electric wires, sharp broken parts of machines etc. was found in our study. To add to it is the indiscriminate eating habit of bovines which predispose them to ingestion of sharp foreign bodies. These bodies being heavier than fodder settle at floor of reticulo rumen

and the honey comb mucosa of reticulum is ideal for entrapping and holding them. These entrapped or free sharp penetrating bodies cause pain to animal during normal reticulo ruminal movements and thus there is cessation of these movement. Many a time the focal area of reticulum perforated by foreign body get necrosed and the foreign body fall back in the reticulum which relieves the animal of the symptoms of disease. At times the foreign bodies perforate through and through and deep in the wall of reticulum. This leads to spillage of ruminal contents loaded with wide array of microorganisms in peritoneum and result in peritonitis. Depending upon the immune status, the animal tries to either cordon off infection locally or it spread in whole of abdomen and become generalised. Inflammatory reaction of bovines is characteristically fibrino purulent which lead to warding off fluid at many sites in peritoneal cavity. The setting of peritoneal inflammation leads to pain cascade in neural rich peritoneum which ends up in reticulo ruminal stasis. The ingested feed & fodder gets accumulated in forestomach compartment, microbes die and fluid get absorbed and dryness of ingesta ensue.The stasis of forestomach compartment lead to their dilatation which resemble forestomach impaction many a time presented/ referred as reticulo ruminal, omasal or abamasal impaction. These impacted materials if not removed cause dryness or necrosis of leaves of omasum & it become difficult to restore their function even after removal of primary cause.

The peritoneal inflammation and absorption of toxins leads to toxaemia and cardiovascular compromise which proves fatal for the animal. At times the reparative local adhesions formed between diaphragm and reticulum contain infection and animal does not recover even after removal of penetrating foreign bodies .These case are referred to as adhesive peritonitis

Symptom of passing of scant faces or absence of dung is seen usually by the owner,some cases pass scant dung occasionally and with bigger size feed particles. There is progressive abdominal distension or tympany. Presence of pain in the start of problem exhibited by kicking at abdomen, restlessness and frequently getting up & lying down is observed frequently. The animal later on turns stoic, show arching of back or looking to abdomen on palpating it. Preference for eating of dry fodder or continuous standing for longer duration are some of other signs of pain shown by these animals occasionally. Regurgitation, recurrent tympany, only mucous or mucous coated

faeces, persistent anorexia, capricious appetite and thirst and dehydration are recorded in some of these animals

The clinical examination is an important tool for diagnosis of these cases in the field condition. Apart form general clinical examination, examination of reticulo rumen its consistency, motility, nature of contents, per rectal exam should be performed which form the basis of diagnosis of these case. Shape of abdomen, rectal examination for presence of distended intestinal loops and type of faeces make valuable point for confirmation of these cases. The hematology, radiography, ultrasongraphy and peritoneal fluid examination almost confirm these case.

The treatment should be stated as early as possible. A combination of antibiotics covering G+ve, G-ve and anaerobes are ideal for this. In our experience a combination of ceftriofur, enrofloxacin, ampicillin and metronidazole give good result. The supportive therapy in form of salines to check dehydration or metabolic acidosis vitamins supplements to boost immune system strength are good options. The antibiotic treatment is needed for 1-3 weeks depending upon the condition or severity of infection.

Abomaso duodenal ulceration (ADU): it is an important cause of forestomach impaction. These are many ascribed etiological factors responsible for ADU but abrasions caused by feed or fodder is the most accepted explanation. The type III type IV ulceration leads to leakage of gut contents into peritoneum which sets peritonitis. In addition the these the pain associated with non perforating type I & II abomasoduodenal ulceration lead to GIT stasis which may ends up in impaction. The clinical signs of ADU like absence of faces, progressive abdominal distension & tympanry resemble forestomach impaction. Tarry back faeces in some case of ADU is a prominent differentiating feature from other forestomach disorders. Examination of blood, positive occult fecal blood test, peritoneal fluid cytology for presence of leakage of gut contents almost confirm these cases. The treatment consist of injectable broad spectrum antibiotics, oral antacids like MgO for 1-2 week and avoiding use of ulcerogenic NSAIDS as analgesics.

Vagal indigestion: Vagal indigestion is a combination of diseases and usually sequele to aforementioned causes that results in ruminal distension and abdominal distension.

The clinical signs are usually gradual in onset and vary from gradual abdominal distension despite a poor appetite and decreased faecal output compared to herdmates on a similar ration. The nature and character of faeces typify vagal indigestion. The faecal consistency is thicker and more sticky than normal and contain 2-4 cm hay particles suggesting poor digestion and amount passed is decreased in proportion to what the cow eats. Extensive controversy exists in literature about the role of vagus in the pathogenesis of "vagal" indigestion. The question remains unanswered today but evidence suggest that vagal neuritis or injury to the vagus nerve is a minor factor in the development of vagal indigestion. The syndrome of vagal indigestion is classified into 4 types as type I called failure of eructation or free gas bloat, type II called Reticulo-omasal transport failure, type III called abomasal impaction and type IV called partial obstruction of the forestomachs. It is the omasal transport failure and abomasal impaction that is mostly observed after rumentomy in cases presented for impaction/stasis of GIT at university Vety. Clinics in the last 2-3 years.

Omasal transport failure may result from a number of causes all of which prevent or inhibit the transport of ingesta from the reticulum through the omasal canal into the abomasums. Common causes include space occupying lesions like lymphosarcoma, papilloma, squamous cell carcinoma, a large ulcer in the area, TRP, reticular and extra reticular abscesses. Localised peritonitis and adhesion between reticulum and diaphragm anterior to the omasum are most common cause of failure, owing rumenotomy the omasal orifice is found atonic and easily distensible. Abomasal impaction has been recorded due to feeding of very dry, coarse roughage such as wheat or oat straw and is accompanied by restricted access to water. The advanced pregnancy has been associated with majority of these cases. As the gravid uterus enlarges, it forces the abomasums further forward interfering with normal motility. If abomasal movement are already diminished, then a partial impaction may result. This impaction may in turn also lead to ruminal impaction.

Normally, as intraruminal pressure rises then is reflex opening of the cardia and other events involved in eructation follow. However, if the cardia is flooded with fluid or foam as in type I vagus indigestion called free gas bloat, the reflex cardial opening does not occur. Thus if animal is unable to clear cardia then more gas accumulate to the point of respiratory embarrassment and or death. Consistently long course of disease and

occasionaly bradycardia is characteristics of this malady. Conservative management of animals after correction of primary cause include providing all time access to water, feeding straw mixed with green fodder or concentrates. Regular exercise of animals is very helpful as it promotes colonic evacuation. **Primary Rumen impaction:** This occur most commonly due to poor quality roughage deficient in protein and digestible carbohydrate (late cut, highly lignified hay or straw) e.g. late bajra feeding, sole wheat straw feeding. These roughage cause decrease of microbial flora activity in the form of their decreased population and diminished metabolic and fermentative processes. Various other factors like antibiotic feeding that may lead to microflora inactivity, some inhibitory substances in plants, deficiency of specific mineral nutrients, prolonged anorexia etc. may lead to microfloral inactivity and this contribute to ruminal impaction. Due to decreased microbial digestion, the breakdown of ingested feed stuffs in prolonged. There is prolonged retention in the fore stomach due to failure to reduce the particle size of ingesta and thus gradual accumulation of undigested feed stuff. This lead to slow distension of reticulorumen which sometimes mimics vagal indigestion. Due to fore stomach distension there is weak contraction and moderate recurrent tympany. There is a loss of normal stratification of rumen contents due to decreased ruminal motility and fibrous components mixed with fluid settle on the floor of rumen. So there is decreased passage of ingesta from fore stomach and faeces are usually dry or absent and may contain indigested fibres. Secondarily, animal may develop ketosis, poor hair coat, emaciation or decreased production. When anorexia is the cause of loss of ruminal microflora animal may look gaunt, tucked up and lack of normal distention also induces rumen stasis. Treatment and management of true ruminal impaction depends upon severity of condition. Treatment of true rumen impaction consist in giving purgative, first oily than saline after hydrating the animals fully. Calcium preparations can be given subcutaneously which check the mild hypocalcaemia due to animal remaining off feed for long period and provide constant stimulus to the animal for muscle contraction. Vegetable oily 3-4 liter once, given orally helps to relieve the mild cases of ruminal impaction. It can be given on next day also. Saline purgative like magnesiuon sulphate sodium sulphate etc. can be given orally after hydrating the animal well. Once ruminal contents have become semisolid and rumen is hard and impacted and has lost the normal movements rumeotomy is the only alternative.

Peritonitis Apart from causes mentioned above the etiology of inflammation of peritoneum is so diverse that at times it become difficult to identify the cause. Various bacterial agents after becoming hematogenous may lead to peritonitis. Other causes include perforation of other abdominal viscera like uterus while doing AI or spontaneous idiopathic rupture of gravid uterus before or at time of delivery, penetrating abdominal injuries, chemical irritants like bile, urine etc. and other disease processes that allow transmural migration of bacteria (neoplasia, visceral ischemia, prolonged gut stasis). Animal exhibit generalized pain, stiffness of gait and guards its abdomen. There is abdominal distension either due to accumulation of peritoneal exudates or paralytic ileus and is accompanied by septicemia, toxemia, paralytic ileus, shock and adhesions. There is sequesteation of electrolytes and protein in abdominal cavity and atonic gut and therefore development of shock. Abdominal paracentensis post xiphoid and parallel to milk vein in abdominal region should be used in large animals to obtain fluid for examination.

Apart from these various diseases like right displacement of abomasus, left displacement of abomasums, intestinal obstruction, congestive heart failure, diaphragmatic hernia, caecal dilatation and torsion, diseases of liver(liver abscess) and pancreas etc. also possess similar signs and are considered while diagnosing cases of GIT stasis in ruminants.