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Johne's Disease: A Review Article

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Abstract: **Johne's disease (Paratuberculosis) is a chronic enteritis of ruminants caused by Mycobacterium avium *subsp*. paratuberculosis (MAP). MAP is a subspecies of *M. avium*, effects wide range of animals including domestic cattle, sheep, goats, buffaloes, camelids and wild ruminants resulting in progressive and chronic enteritis known as Johne's disease . Clinically indected animals show watery diarrhea, emaciation and eventually death due to lack of effective treatment. Clinically as well as subclinically infected animals shed bacteria in feces and milk. Fecal-oral rout is the main rout of transmission. It is economically very important disease in livestock due to decrease in milk yield, working efficiency and culling of infected animals. Enlargement of mesenteric lymph node and transverse corrugation in intestine is characterstic finding in post-mortem. Strategies to control this disease include improved management practices, testing and culling and vaccination.**

Keywords: **Johne's disease, diarrhoea, corrugation.**

1. INTRODUCTION

Johne's disease (JD) is a chronic Infectious granulomatous enteritis of ruimanats characterized by progressive wasting and persistent diarrhea. Johne's disease gets its name from the veterinarian Dr. H.A. Johne (Germany) who isolated and identified the organism in 1894 in granulamatous lesions in the intestine of affected cattle that stained acid-fast indicating of mycobacterial organism. It mainly effects dairy cattle, Sheep and goat. But the disease have also been reported in horses, pigs, deer, alpaca, llama, rabbits, stoat, fox, and weasel (Greig *et al*., 1999).

The disease is caused by *Mycobacterium avium subsps. Paratuberculosis* (MAP) which is an a Aerobic, acid-fast and slow growing bacteria. MAP is highly resistant to environmental stresses like temperature and drying, and it can persist in farm soil for several years (Singh et al., 2013). MAP is also resistant to many common disinfectants but it can be killed by cresylic acid compounds and sodium orthopehylphenol.

2. TRANSMISSION

Johne's disease is a contagious infection. Affected animals shed organism in feces and milk (Hines et al., 2007; Seyyedin et al., 2010; Hasonova et al., 2009). Fecal-oral route is the main rout of transmission. Contaminated food, water, vehicles and other equipment may be a source of transmission from one herd to other. Male animals may carry MP in accessory reproductive organs and to some extent in semen. Though in- utero transmission of disease has been established, role of semen and embryo transfer in disease transmission has been reported (Khol et al., 2010). Calves may get infected by the colostrums from effected cow (Stabel, 2008); calves have been reported to shed microbe in feces at 5 months of age (Hasonova et al., 2009). Humans may get MAP from raw milk, meat and contact with animals (Eltholth et al., 2009; Alluwaimi, 2007).

3. EPIDEMIOLOGY

Johne's disease has worldwide distribution and it has been increasing range of animal species (Vansnick, 2004). Among ruminants, dairy cattle are most susceptible to disease. Co–infection of paratuberculosis with other diseases has been reported e.g. brucellosis (Singh et al., 2013a). In USA herd prevalence has been reported 91.1% (Lombard et al., 2013), in

Chile 28–100% (Kruze et al., 2013). Prevalence of Johne's disease in goats has been reported from all over the world with prevalence of 7.9% in Republic of Cyprus (Liapi et al., 2011), 76.9% USA (Manning et al., 2002), 74.3% Chile (Salgado et al., 2007), 62.9% France (Mercier et al., 2010), 79.4 %India (Singh et al., 2013) and 44.1% Argentina (Fiorentino et al., 2012). It has also been seen in ovine species (Sikandar et al., 2013). Abbas et al. (2011) tested samples in 3 semen production units in Punjab, Pakistan and found almost 20% positive breeding bulls and almost 33 % positive teaser bulls.

4. CLINICAL SIGNS

Johne's disease has Long and protracted incubation period which may extend even up to 2 years or more. Clinical signs usually first appear in young adulthood, but the disease can occur in animals at any age over 1–2 years and in dairy cattle is most frequently reported in the 3–5 year old age group. Effected animal have normal appetite but excessive thrust. As Johne's disease is a chronic disease so, mild and progressive signs are seen in animals. Milk production decreases in all lactating animals and body condition becomes poor. In effected cattle chronic watery diarrhoea leads to severe emaciation, dehydration and ultimately animal dies as effective treatment is not available. Progressively there is weight loss and emaciation. Reduced ruminal motility in effected goats has been reported (Lybeck et al., 2011). In sheep and goat diarrhoea is not a constant symptom but feces is soft and emaciation is there.

5. PATHOLOGY

Early lesions occur in the walls of the small intestine and the draining mesenteric lymph nodes, and infection is confined to these sites at this stage. As the disease progresses, gross lesions occur in the ileum, jejunum, terminal small intestine, caecum and colon, and in the mesenteric lymph nodes. The intestinal lesions are responsible for a protein leak and a protein malabsorption syndrome, which lead to muscle wasting. Characterstic leions are found in intestine especially in the ileum. The wall of intestine become 2-20 times thickened. The mucosa of intestine is folded showing transverse corrugations. Histopathological examinations exhibit diffused granulomatous enteritis, accumulation of epithelioid giant cells and macrophage in submucosa and mucosa of intestine (Almujalli and Al–Ghamdi, 2012). Sheep and goats sometimes develop foci of caseation with calcification in the intestinal wall and lymph nodes.

6. DIAGNOSIS

Diagnosis of Johne's Disease is difficult due to long incubation period and lack of accurate tests which can predict the infection (Nielsen, 2008). Diagnosis is based on clinical signs, postmortem lesions, histopathology and diagnostic tests including direct test e.g. fecal smears, fecal culture and polymerase chain reaction (PCR) and indirect tests e.g. delayed–type hypersensitivity (DTH), interferon Assay, enzyme linked immuno–sorbent assay (ELISA), agar gel immunodiffusion (AGID), complement fixation test (CFT). In Indian conditions "serum ELISA" and "milk ELISA" are good screening tests, and "milk PCR" is "confirmatory test" for MAP infection (Garg *et al*., 2015).Combination of milk ELISA with milk PCR may be adopted as a model strategy for screening and diagnosis of Johne's disease in lactating/dairy cattle herds (Garg *et al*.,2015). The lymph analysis yielded significantly more positive results than the analysis using feces, blood or milk indicating higher sensitivity (Khol *et al*.,2014)

7. TREATMENT

There is no satisfactory treatment for Johne's disease . Combination of different drugs has been practiced as treatment measure, mostly with isoniazid, clofazimine and rifampin (Borody et al., 2007; St–Jean and Jernigan, 1991). Recently, lactic acid bacteria (LABATCC 334) have been used as probiotics for treatment of experimentally induced Johne's disease in mice (Cooney et al., 2014).

8. VACCINATION

Live-attenuated and heat-killed vaccines against johne's disease are available in some countries. The main advantage of vaccination is the prevention of clinical cases but vaccination may cause a reaction at the site of injection and may also interfere with eradication programmes based on immunological testing.

9. CONTROL PRACTICES

Developed countries around the world have adopted different programs for the control of Johne's disease as per their technical skills, financial capacities and administrative will. But Johne's disease is still a low priority disease in many developing and poor countries including India. Globally, there is lack of an 'international program' either for the control or eradicatation of this incurable disease of domestic livestock. Most of the developed countries have adopted rigorous practices (hygiene, management, vaccination) for the control and elimination of paratuberculosis infection from their herds and flocks and have succeeded in boosting per animal productivity and decreasing the production losses. India started policy in 1950s of testing of animals mainly cattle against Johne's disease and bovine tuberculosis since and provisions of segregation of positive and negative animals was made using Johnin and tuberculin. Since cattle cannot be slaughtered in India, the policy of maintaining Johne's disease infected (positive) animals in isolated herds was made but due to increase in the cost of maintenance and constrains in the funding, the program was not sustained. Presently, the Government of India does not have any program for the control of Johne's disease (Sohal, 2015).

10. ZOONOSIS

MAP can spread from clinically and sub clinically infected animals to man. Timms *et al*. 2012 reported that MAP is related to Crohn's disease in humans. But a very weak relationship has been observed between MAP and Crohn's disease in humans (Fawzy *et. al,2013)*. MAP has been reported to survive milk pasteurization (Grant *et al*., 2002) but USDA researchers found that it does not survive.

11. CONCLUSION

Prevention & control of Johne's disease is important as treatment is not available. The potential role of MAP in the etiology of Crohn's disease deserves substantial future investigation. The knowledge of how MAP causes disease still lags behind than for other pathogenic bacteria. Successful vaccine against MAP infection would be valuable and welcome development. Johne's disease control programs are the immediate requirements of the country in order to boost per animal productivity

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