Johne's Disease: A Serious Threat to Livestock Economy

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ABSTRACT

Johne's disease is a fatal infection that affects primarily the small intestine of ruminants. It is caused by *Mycobacterium avium* subspecies *paratuberculosis* (*M. avium* subsp. *paratuberculosis*). It is a chronic and contagious disease of ruminants characterized by chronic diarrhoea, progressive emaciation and thickening of the intestine (corrugations). It particularly affects cattle, sheep, goat and wild ruminant, but rare in other animals. It causes major economic loss to farmers and livestock industry in terms loss of production and animal death. Lack of knowledge about its transmission and prevention among the farmers and farm workers lead to spread of this disease to the healthy animals and human. This article reviews some information about clinical signs, transmission and prevention of Johnes' disease.

INTRODUCTION

Johne's disease (JD), also known as paratuberculosis, is chronic bacterial disease of the ruminants causing significant economic losses to farmers due to decline in productivity (Chakrabarti, 2012). It is caused by *Mycobacterium avium* sub-species *paratuberculosis*, an acid-fast organism. This disease is characterized by chronic diarrhoea and progressive emaciation. It usually affects cattle, sheep, goat and other ruminants. Among wild animals, it has been reported in deer. Horses and pigs are affected very rarely. Chronic diarrhoea is unremarkable in sheep, goats and laboratory animals, rather progressive emaciation is a prominent sign (Mallikarjunappa *et al.*, 2021). Major route of

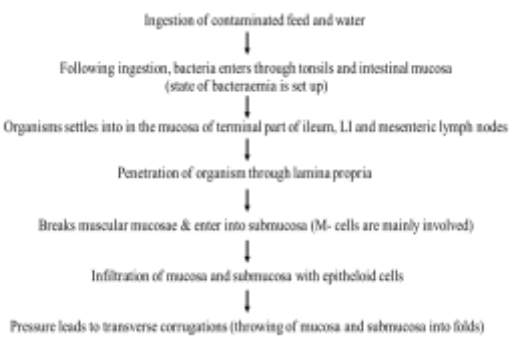
transmission of this infection is via faeces of infected animals. This infection mostly affects the small intestine but can spread to large intestine (colon and rectum). Therefore, bowel wash and rectal pinch are reliable methods for detection of this acid-fast bacilli. There are three strains, which are capable of producing disease in cattle i.e., the usual bovine strain and two sheep strain.

Transmission (Sweeney, 1996**)**

- Organisms are excreted through the faeces of infected animals, which contaminate feed and water, so infection mainly occurs by ingestion.
- Organisms persist without multiplication in pasture for long periods and such pastures are infective up to one year.
- The organisms have been isolated from genitalia and the semen of infected bulls. Foetal Infections occurs in cow with or without clinical signs.
- Infection can be transmitted through vertical route (in utero and infection through milk).

Pathogenesis (Sweeney, 1996; Mallikarjunappa *et al.*, 2021**)**

- Incubation period – up to two years or more.
- **Faeces from infected animals is the primary source of infection**



- Formation of symplasma stages in some parts of submucosa (syncytial masses of epithelioid cells, which have undergone necrosis, so not progress further). The villi become fused i.e., clubbed and get atrophied later. This disease is not of much zoonotic significance.

Symptoms (Chakrabarti, 2012**)**

- Clinical signs appear after several months.
- In cattle, chronic diarrhoea, which is neither offensive nor blood stained is observed.
- There is progressive emaciation and weakness inspite of good appetite and feeding.
- Drop in milk yield, absence of fever, sub-mandibular oedema are also observed.
- Hide bound condition and staring coat are also commonly seen.
- In sheep and goat, disease is manifested mainly by emaciation. Diarrhoea is not severe in these species.

Macroscopic lesions (Burnside & Rowley, 1994)

- Carcass is emaciated.
- Earliest lesions are seen in ileocaecal valve.
- Terminal part of ileum & large intestine is greatly thickened 2-20 times of which mucosa is folded & shows corrugation, which cannot be flattened or stretched out. The crests of corrugation show congestion.
- Mesenteric lymph nodes are enlarged.

- Fat depots are gelatinous in appearance (indicative of emaciated animal).
- Serous effusions in peritoneal and pleural cavity and intermandibular oedema may be noticed.

Microscopic lesions (Carrigan, 1990**)**

- Granulomatous enteritis characterized by presence of lymphocytes, macrophages, multinucleated epitheloid cells and giant cell in mucosa & submucosa of intestine.
- Intestinal villi and glands are atrophied.
- Mesenteric lymph node shows epitheloid granulation tissues.
- Impression smears or tissue sections stained by Ziehl-Neelsen (AF) will reveal large number of acid-fast, rod shaped organisms in cytoplasm of epitheloid cells.
- In later stages, arteriosclerosis in intestinal blood vessels can be seen.
- Lymphangitis of intestinal lymphatics can lead to knotty appearance.

Diagnosis (Chakrabarti, 2012**)**

- Clinical history, symptoms and lesions
- **Examination of rectal pinch:** insert the hand in to the rectum and with nail remove a small pinch of mucosa. Make the smear of this pinch, stain and examine for acid fast bacteria.
- **Intradermal Johnin** test in the neck area.
- Immunodiagnostic tests - ELISA, AGID, CFT
- Microscopic demonstration of epitheloid cells containing acid-fast bacilli in tissue sections of intestine
- At necropsy, the pathognomonic lesions in intestine are quite informative to assess the disease condition.
- A major advantage of faecal culture test is that it can identify cattle 1-3 years prior to the appearance of clinical signs. So, it is a 100% specific and 100% sensitive test.
- The Herrold's egg yolk medium with mycobactin often used in culture for the detection of this bacteria.

Treatment (Chakrabarti, 2012**)**

Treatment of this disease is not often indicated, but certain antimicrobials like isoniazid, rifampin, amikacin or their combination can be used. The animals treated with these antimicrobials should not be used for meat and milk consumption up to the period they are treated. Remission of clinical signs and reduction of faecal shedding often occurs after treatment, but the infection is rarely eliminated and require daily treatment. The condition relapses once the treatment is discontinued.

Prevention and Control (Chakrabarti, 2012**)**

Unaffected herds should be maintained closed. Herd replacements should be obtained from herds should be free from this disease. Newly imported animals should be screened. In infected farms, the potential source of infection should be identified and removed promptly. The important control measures must include maintenance of hygiene in above mentioned areas in farm, providing an uncontaminated source of colostrum and milk replacer, and calf housing should be free of contamination by faeces from infected cattle. Vaccination against these bacteria has limited effect on faecal shedding and onset of infection on heavy exposure. Therefore, vaccination does not

eliminate the need of good management and sanitation.

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