PARATUBERCULOSIS IN A COW: A CASE STUDY

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Johne's disease, agranulomatous enteritis of ruminants is caused by *Mycobacterium avium* subsp. *paratuberculosis* (MAP) and manifested clinically by chronic diarrhoea and progressive emaciation. In cattle infection begins in very young animals but the signs of illness do not appear until the host is adult. In the terminal stage of disease, animals become cachetic, anaemic and too weak to rise (Manning and Collins, 2001).

One Brown Swiss cross bred cow belonging to the University Livestock farm, Mannuthy was presented with history of watery diarrhoea, weakness, emaciation and reduction in milk yield since few weeks. Appetite of the animal was normal and there was no response to the anti diarrheal treatment provided. On detailed clinical examination all the clinical data remained within the normal range. (Temp 101.8°F, pulse 72 / minute, respiration 25 / minute and mucous membranes were found to be pale). The dung and blood samples were collected. Endoparasitism was ruled out by parasitological examination. Single intradermal Johnin test was carried out and reading after 72 hours gave a negative result. Dung smear was made and examined for the presence of *Mycobacterium avium* subsp. *paratuberculosis* by Ziehl-Neelsen acid fast staining, but no organism could be detected. Day by day condition of the animal had gone worse and was on supportive therapy with Inj. Dextrose saline, Inj. Calcium boro gluconate(25%), Inj. Hivit *(Vit A 2000 iu, D_3 2000 iu, E 4 mg, Niacinamide 10mg, Thiamine 10mg, Pyridoxine 5 mg, Riboflavin 1 mg, D pantothenol 1 mg, D-biotin 10mcg, Vit B_{12} 10mcg and calcium 10 mg/ ml)* and Inj. Biotrim *(Sulphadiazine 200 mg, Trimethoprim 40 mg / ml)* intravenously. On the third day the animal was on lateral recumbency, too weak to rise and dehydrated with persistent diarrhoea and the animal died on the same day.

Post mortem examination revealed corrugation of ileum and endocardial mineralization. Mesenteric lymph nodes were normal in size without any enlargement or oedema. There was gelatinization of fat. No other gross lesions were visible. Smears from corrugated ileum and mesenteric lymph node revealed abundant acid fast organism. A piece of lymph node was subjected to IS*900* tissue PCR and paratuberculosis was confirmed at the Animal Biotechnology unit, Madras Veterinary College, Chennai.

The clinical signs were suggestive of bovine paratuberculosis. The animal did not respond to the standard antidiarrhoeal therapy. The faecal sample was negative on acid fast staining and the single intradermal Johnin test also gave a negative result.

Roy *et al.* (2004) opined that Johnin test is helpful in identifying the early stages of disease, but in later clinical stages the animal may not evoke enough response. In the early stages of paratuberculosis the cell mediated immune response predominates and then wanes with advancing disease (Austerman *et al.*, 2006). Negative result by single intradermal Johnin test in this case agrees with the observation of these workers.

On post mortem examination typical lesions were detected and the smear from various tissues also gave positive for the organism on acid fast staining (Fig.1)

Faecal sample examination was also negative on acid fast staining. Doyle (1956) was of the opinion that only 25 to 30 per cent cases could be diagnosed by examination of faecal samples. This finding indicates that a negative faecal sample result by light microscopy has no significant value in diagnosing paratuberculosis as opined by Hole and Maclay (1959). The organism may be absent or present in faecal sample at a level not detectable by light microscopy. Acid fast staining had limited sensitivity and as many as 10^6 bacteria per gram may be necessary for detection by light microscopy (Thoresen *et al.*, 1994).

Typical post mortem lesions were observed in this case. The classical intestinal change is diffuse thickening of mucosa, which is folded into transverse rugae. When well developed, the mucosal folds cannot be smoothed out by stretching (Jubb *et al.*, 1993). The gross lesion in Johne's disease is a chronic segmental thickening of ileum, caecum and proximal colon. Affected segments showed a variably thickened, rough, rugose

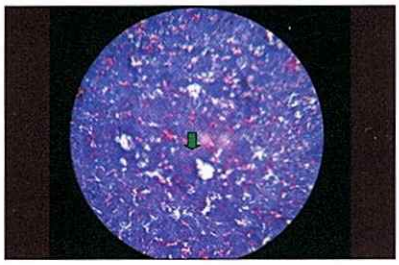

Fig 1. Clumps of acid fast bacilli in impression smear of intestine

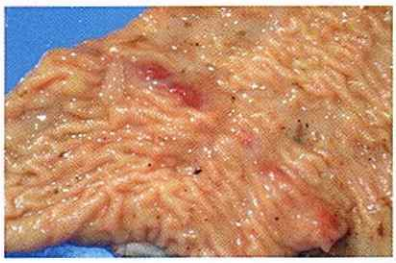

Fig 2. Thickened corrugated ileum with haemorrhagic spots

mucosa, often with multiple foci of ulceration (Fig.2). Secondary lesions in paratuberculosis include hepatic granulomas and endocardial and aortic mineralization (Kreeger, 1991). Aortic mineralization, when it occurs in association with the clinical signs and lesions of paratuberculosis, is specific for Johne's disease in cattle (McGavin and Zachary, 2007).

Jubb *et al.* (1993) reported that ileocaecal lymph node and affected segments of gut are candidate sites for culture to confirm a diagnosis of Johne's disease. Here the organism was present at a level detectable by light microscopy of acid fast stained impression smear and hence positive result was obtained. The disease was confirmed by IS*900* tissue PCR by amplification of 279 bp amplicons specific for MAP (Moss *et al.*, 1991).

Summary

A case of bovine paratuberculosis is presented which was tentatively diagnosed on the basis of symptoms and post mortem findings and subsequently confirmed by demonstration of acid fast organism in impression smear from intestine and IS*900* tissue PCR. Ziehl-Neelsen acid fast stained faecal smear examination by light microscopy and single intradermal Johnin test failed to detect the disease.

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