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Case Report

Clinical and gross pathological findings of Johne's disease in a calf: A case report

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ABSTRACT

Objective: This case report describes a clinical case of Johne's disease (JD) in a Friesian calf aging one and half years.
Materials and methods: Physical examination of the calf was carried out, history of the farm as well as samples for laboratory examinations were obtained. The laboratory examinations included hematological examinations by direct wet mount, hematocrit centrifugation technique and Giemsa stain, biochemical evaluation of serum, bacterial culture and isolation from feces and aspirate from the swollen jaw.
Results: Clinical history showed that the farm had a history of JD, and routine health screening revealed that one of the calves had clinical signs suggestive of Johne's disease. Physical examination of the calf revealed a lumpy jaw, enlarged bilateral pre-scapular and pre-femoral lymph nodes, while hematological and biochemical findings showed a normocytic normochromic anemia, severe leukocytosis with neutropilic left shift and lymphocytosis with hyperproteinemia characterized by hyperglobulinemia. Fecal bacterial tests showed the presence of acid fast bacilli. Based on the history and laboratory findings, the cow was diagnosed with JD. In order to ensure effective control measures, the calf was culled from the heard. Postmortem examination revealed inflamed pre-scapular and pre-femoral lymph nodes with the presence of paramphistomes in the rumen mucosa.
Conclusion: Since JD is a chronic disease that lingers in livestock farms, there is need for early identification and culling of infected animal in order to limit its devastation on the farm.

KEYWORDS

Acid fast, Bacilli, Calf, Gross pathology, Johne's disease

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INTRODUCTION

Johne's disease (JD) is a chronic irreversible wasting gastro-enteric disease of ruminants caused by *Mycobacterium avium subsp. paratuberculosis* (MAP) (Geraghty et al., 2014). The disease was first reported in cattle in 1895 in Europe and 1908 in the United States (Sweeney et al., 2012). The disease is mostly found in domestic ruminants such as cattle, buffaloes, sheep, goats and camels. JD affects about 40% of dairy cattle worldwide. The infected cattle remain as carriers for years and shed the organism via milk, colostrum and feces, and subsequently contaminate feed and water (Facciuolo et al., 2013). In addition, calves remain as asymptomatic carriers for a period of 2 to 5 years. In Malaysia, JD has been previously reported in a dairy farm in 2013 (Abdullah et al., 2013). However, sporadic cases JD have also been reported in dairy farms in Malaysia.

The JD is mostly manifested as subclinical form, which is suggestive of the involvement of other factors categorized as farm management variables, which play significant roles in changing the course of the disease from subclinical to clinical disease (Cetinkaya et al., 1997). The most common and routinely used diagnostic test for JD are bacterial examination in feces and ELISA (Espejo et al., 2015). There is no standardized or effective treatment for JD. The control of the causative pathogen in herds of cattle involves thorough screening, identification and removal of infected animals (Gowozdz, 2008). In addition, the institution of good hygienic practices and animal husbandry have been reported to reduce the prevalence of the disease (Facciuolo et al., 2013). It has been observed that complication of on-farm control measures is due to the persistence of MAP in the environment. This report describes a rare clinical case of JD in a calf based on laboratory and pathology findings.

Figure 1. Photograph of calf showing emaciation and lumpy jaw (arrow).

CLINICAL HISTORY

A Friesian calf aging one and half years was presented to the University Veterinary Hospital (UVH), Universiti Putra Malaysia (UPM) with a complaint of weight loss. The farm had previous history of JD. Physical examination of the calf showed the presence of lumpy jaw, enlarged bilateral pre-scapular and pre-femoral lymph nodes (**Figure 1** and **2**) and diarrhea. The mucus membrane was pink with a capillary refill time of 2 sec, while the body condition score was 2/5.

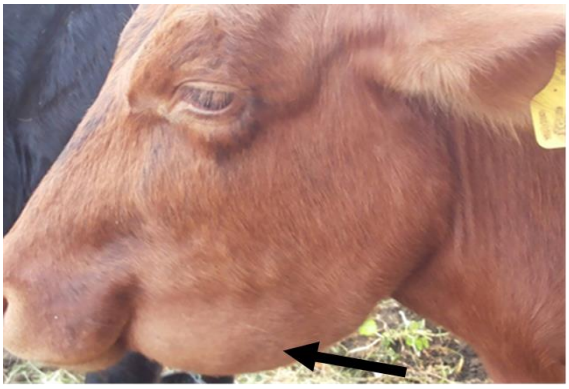

Figure 2. Calf showing enlarged jaw (lumpy jaw; arrow).

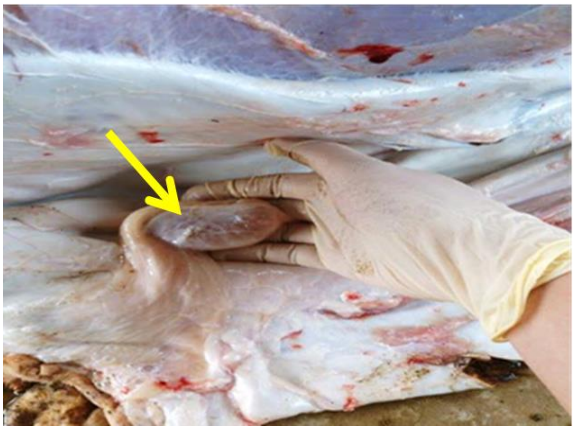

Figure 3. Enlarged and hyperemic pre-scapular lymph node (arrow).

Figure 4. Enlarged and hyperemic pre-femoral lymph node (arrow).

Figure 5. Rumen mucosa with paramphistomes.

DIAGNOSTIC WORK UP AND RESULTS

Whole blood was collected for hematology, direct wet mount, hematocrit centrifugation technique (HCT) and Giemsa stain. Serum was collected for biochemical evaluation, while fluid aspirate from the swollen jaw as well as fecal sample were collected for bacterial culture, isolation and identification**.** Hematological and biochemical findings showed normocytic normochromic anemia, severe leukocytosis with neutrophilic left shift and lymphocytosis with hyperproteinemia characterized by hyperglobulinemia (**Table 1**). Fecal culture revealed the presence of an acid fast bacilli. There were no bacterial growth from the fluid collected from the swollen jaw. From the laboratory findings, clinical examination findings and history of the farm, the calf was diagnosed with JD.

POSTMORTEM EXAMINATION

There was no therapeutic plan for this case, hence the calf was culled according to the guidelines described by Mueller (2015) in order to reduce the spread of the disease in the herd. At postmortem examination, the carcass was emaciated and there was an absence of omental fat in the viscera. The pre-scapular and pre-femoral lymph nodes were swollen and hyperemic (**Figure 3** and **4**). The rumen was full with ingesta and numerous para-amphistomes were seen attached to the rumen papillae (**Figure 5**). All other organs appeared to be apparently normal in size, consistency and texture.

DISCUSSION

JD poses a significant economic and public health implication to humans and the cattle industry (Gowozdz, 2008). This is due to the unavailability of accurate tests to diagnose different stages of the disease,which in turn makes the diagnosis, prevention and control of the disease difficult (Britton et al., 2015). The initial findings of this case report showed that even though the calf had a poor body condition score, it had a good appetite. This is suggestive of a classical case of JD in ruminants. In addition, clinical findings of lumpy jaw, enlarged bilateral prescapular and prefemoral lymph node and diarrhea were observed in this case. It is important to note that, at the earliest stage of JD, lesions are confined to the lining of the small intestines and the mesenteric lymph nodes. The lesions later spread to the jejunum, ileum, caecum, terminal end of the small intestine, colon and the mesenteric lymph node as the disease progresses (Sweeney et al., 2012). The manifestation of these gross lesions leads to malabsorption, protein leakage and subsequently severe weight loss. In clinical cases of JD, loss of proteins is reflected by lower protein and albumin concentrations in the serum, without changes in globulin concentration (Clarke, 1997). In advance cases, edema of the submandibular and other dependent areas may be seen (Clarke, 1997). In addition, rough and unthrifty hair coat, alopecia and in some cases with pigmentation. This was also reported in this case as the calf had lumpy jaw and unthrifty hair coat. However, in this case, there was increased total protein associated with hyperglobulinemia. This may be associated with active antibody production by the immune system in the form of immunoglobulins to fight the infection.

In this case, the clinical history stated that the farm had a history of JD and it can be inferred that the calf might have be infected via contamination of feed with feces of shedders or from the dam through colostrum (Sweeney, 1996; Pant et al. 2014; Robins et al., 2015).

The pathognomonic signs of MAP in cattle include chronic and intermittent diarrhea to progressive weight loss from mild to severe, as was observed in this case (Clarke, 1997). This is consistent with the findings of this case report, because the calf was looking cachetic and with a body condition score of 2/5 (**Figure 1**). Other clinical manifestation includes fall in milk yield in the absence of pyrexia and toxemia in cows (Abendaño et al., 2013). In ruminant however, weight loss is as a result of malabsorption of protein and losses due to significant cellular infiltrate and edema that occur in the intestine (Salem et al., 2013). The development of the onset of clinical disease overwhelms the compensatory mechanisms of increased protein synthesis in the liver. The above findings classically describe the condition of the calf reported in this case.

The presence of *E. coli* and *Bacillus cereus* isolated from fecal sample connotes theirs presence as normal floras of the intestine, rather than the cause of the diarrhea as

Table 1. Results of laboratory investigation.

Parameters	Animal identification	
	T1403	**Reference values**
RBC × 10^{12}/L	5.65	5-10
Hb g/L	76.1	80-150
PCV L/L	0.25	0.24-0.46
WBC × 10^9/L	23.3	4.2-12
Band Neutrophils × 10^9/L	0.47	<0.2
Seg. Neutrophils × 10^9/L	6.99	0.6-4.0
Lymphocytes × 10^9/L	13.28	2.5-7.5
Total protein g/L	89.9	55-75
Albumin g/L	33.7	25-40
Globulin g/L	56.2	27-45
A:G	0.6	0.8-1.2
Direct wet mount	-	**n/a**
HCT	-	n/a
Giemsa stained blood film	Theileria 0.1%	n/a
Modified McMaster	Strongyle 400 e.p.g Coccidia 300 o.p.g	n/a
Acid fast Bacteria	+	n/a

Hb=Hemoglobin, RBC= Red Blood Cell, PCV= Packed Cell Volume, WBC= White Blood Cells, A:G= Albumin-Globulin Ratio, n/a=not applicable

observed in the clinical history. In addition, the diarrhea is not associated with salmonellosis since fecal examination for salmonella tested negative. Similar finding was also reported previously by Abdullah et al. (2013). The authors further opined that the presence of the *E coli* and *B. cereus* is as a result of overpopulation of normal floras precipitated by immune suppression due to the disease, which in this case is JD. Screening of this calf showed various hematobiochemical derangements from anemia to leukocytosis, hyperprotein-emia and hypoproteinemia. The calf that had positive acid fast bacilli during this evaluation had a severe leucoytosis with neutrophilic left shift and lymphocytosis, with hyperproteinemia characterized by hyperglobulinemia. The hyperglobulinemia suggest increased production of immunoglobulins by the immune system.

The calf was culled in order to protect other animals from contracting the disease. The post mortem examination showed the presence of inflamed lymph nodes and para-amphistomes on the rumen mucosa. Sweeney et al. (2012) and LeBlanc et al. (2006) opined that in order to effectively reduce the transmission of MAP, there is a need to employ measures aimed at preventing the introduction of the disease into the herd through sustained and frequent sanitation of stalls and calf hutches, restriction of the use of contaminated equipment in dairy farms, timely and adequate colostrum feeding and separation of calves from adult cows. However, these measures if effectively applied can only prevent the entry of infection, not the onset of clinical disease. It is important to note that, since JD is quite difficult to control and eradicate, culling of infected animals in a herd is one of the best control practices (Salem et al., 2013; Ritter et al., 2016).

CONCLUSION

JD is a common problem affecting dairy farms causing serious economic losses in farmed ruminants. There is paucity of information with regards to the accuracy of the diagnostic test as well as effective recommended treatment for JD. Hence, control and prevention is difficult. In addition, carrier and infected calves remain reservoirs of infection for a very long period of time and continue to shed the organism in the environment. The calf in this case was culled in order to prevent further contamination of farm.

CONFLICT OF INTEREST

None of the authors have any conflict of interest.

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