Acute caprine fasciolosis: a case with unusual migration to lung

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Abstract Fasciolosis is an important parasitic disease of domestic ruminants and occurs worldwide as a result of infection with liver fluke species. This report describes the macroscopic and microscopic characteristics of acute fasciolosis in a goat with unusual migration to lung. A 10-month-old goat was presented with history of weakness and acute recumbency from 12 h ago. The clinicians didn't report clinical evidence of systemic disease. Hematological analysis showed no significant changes in blood parameters except a mild reduction in lymphocyte population and about 6 % eosinophilia and also normocytic normochromic anemia. A noticeable increase in the level of serum ALP, AST and also GLDH were observed. Moreover, total protein and albumin showed a slight decrease in value comparing to reference intervals. In macroscopic examination numerous short vermiform cords were noted on the liver surface and the surface had an uneven appearance. A large number of immature, wandering flukes were seen on the cut surface. Histopathologically, a wide range of hepatic lesions was found. The most important lesions were moderate to severe perihepatitis and haemorrhagic tracts on the hepatic surface. These lesions corresponded to migratory tunnels filled with blood, fibrin and cellular debris. However histopathological findings of lung revealed chronic suppurative bronchopneumonia, but this lesion is not only associated with larval migration.

Keywords Biochemical and hematological analysis · Fasciolosis · Goat · Histopathology

Introduction

Fasciolosis is an important parasitic disease of domestic ruminants and occurs worldwide as a result of infection with two liver fluke species: *Fasciola hepatica* and *F. gigantica* (Trematoda). *F. hepatica* is distributed throughout the world, mainly in temperate zones, while *F. gigantica* is found in tropical and subtropical regions of Africa and Asia. Thus, the two fasciolid species overlap in many African and Asian countries and sometimes in the same country, although in such cases the ecological requirements of the flukes and their snail intermediate host are distinct (Mas-Coma et al. 2005). The animal species most often affected are cattle and sheep; goats are less frequently infected, however, fasciolosis occurs as a major constraint for goat production in many areas of the world. Reported prevalence ranged from 3 % in Spain (Martinez et al. 1996) to 72 % in China (Wang et al. 1987; Wamae et al. 1998).

Clinically fasciolosis is often seen as a chronic wasting disease. In cattle, subacute or acute outbreaks occasionally occur, whereas in sheep, acute or subacute diseases are more frequent (Radostits et al. 2007). Goats can be considered a very receptive host for *F. hepatica* based on the recovery of mature flukes from primary and secondary infections, (Reddington et al. 1986) and although the infection usually evolves as a chronic disease, subacute and acute fasciolosis with high rates of mortality are not rare (Leathers et al. 1982). Although there are several papers about clinical signs, epidemiology and macroscopic and microscopic features of *F. hepatica* in goats, to the our knowledge, there is no document describing acute caprine

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fasciolosis caused by *F. gigantica*. The aim of this report is to describe the macroscopic and microscopic characteristics of acute fasciolosis in goat with unusual migration to lung.

Case presentation

A 10-month-old goat was referred to the School of Veterinary Medicine, Razi University, Iran with history of weakness and acute recumbency from 12 h ago. The clinicians didn't report clinical evidence of systemic disease.

Blood sample was collected from the jugular vein in vacutainers containing EDTA for hematological factors and into vacutainers without anticoagulant for biochemical analysis. Hematological analysis including total erythrocyte count, total leukocyte count, hematocrit value and hemoglobin concentration was determined using standard routine techniques. Biochemical analysis including serum total protein, albumin, GLDH, GGT, AST and ALP values was determined by Hitachi 919 (Japan) autoanalyser.

Results showed no significant changes in blood parameters except a mild reduction in lymphocyte population and about 6 % eosinophilia and also normocytic normochromic anemia. A noticeable increase in the level of serum ALP, AST and also GLDH were observed. Moreover, total protein and albumin showed a slight decrease in value comparing to reference intervals.

With the passing of time, the clinical signs worsened, and the goat died 24 h after the referral. The carcass was submitted for necropsy. Except liver and lung, no remarkable gross lesions were seen in the other organs. In macroscopic examination numerous short vermiform cords were noted on the liver surface and the surface had an uneven appearance. A large number of immature, wandering flukes were seen on the cut surface. Migrational tracks were distributed at random throughout all areas of

the livers, but were most common in the central and left lobes. Color progressively changed from brown-red in the smallest tracks to gray surrounded by a red zone of hyperemia in the larger ones (Fig. 1a). The gross appearance of lung showed irregular consolidation with lobular pattern. The cranial and middle lobes were the main affected areas. The pulmonary parenchyma was firm in texture and a few number of immature flukes were seen randomly (Fig. 1b). The cut surface of the consolidated lobules was moist and purulent exudate leaked from small airways.

Appropriate tissue samples of the left and right hepatic lobes and lung were fixed in 10 % neutral buffered formalin, embedded in paraffin, sectioned at 5 μ m, and stained with hematoxylin–eosin for light microscopic examination.

Histopathologically, a wide range of hepatic lesions was found. The most important lesions were moderate to severe perihepatitis and haemorrhagic tracts on the hepatic surface. These lesions corresponded to migratory tunnels filled with blood, fibrin and cellular debris. Migrational tracks were mainly composed of eosinophilic debris of disintegrated hepatocytes and infiltration and aggregation of numerous eosinophils and neutrophils, moderate macrophages with haemosiderin pigment and lymphocytes and less plasma cell (Fig. 2a). Often tissue elements surrounding the tracks were affected by a pronounced coagulative necrosis. The adjacent portal areas and the congested sinusoids were abundantly infiltrated by eosinophils, lymphocytes, and macrophages (Fig. 2b). Thrombosis of blood vessels was frequent in areas adjoining migrational tracks. Mural thrombi were more common in veins than in arteries. Thrombosis of central veins in migratory areas was relatively general. Arteritis and phlebitis were frequent in the migratory areas and not necessarily associated with thrombosis.

In the histopathological examination of the lung, multiple areas of necrosis and occasionally sequestration foci

Fig. 1 Appearance of the liver and lung at initial examination. **a** Numerous short vermiform cords on the liver surface with uneven appearance and a large number of immature, wandering flukes on the

cut surface (arrows). **b** A few numbers of immature flukes in the pulmonary parenchyma (arrows)

Fig. 2 Histological sections of liver. **a** Fresh migrational track filled with blood, fibrin and cellular debris with severe inflammatory reaction around the migrational track (H&E; $\times 300$). **b** Portal area abundantly infiltrate by eosinophils, lymphocytes, and macrophages (H&E; $\times 700$)

were consistently observed within the pneumonic portions (Fig. 3a). Thickening of alveolar walls, mainly by lymphocytes and macrophage infiltration was evident. Cellular infiltration in the alveolar lumen, and occasionally walls, consisted of macrophages, lymphocytes, plasma cells and neutrophils (Fig. 3b). Varying degrees of bronchiolar lymphatic tissue hyperplasia (BALT) was another common finding. These findings were diagnosed as chronic suppurative bronchopneumonia.

Discussion

Fasciolosis, caused by *Fasciola* species, is a disease of herbivorous animals and has a worldwide distribution (Haseeb et al. 2002). *F. hepatica* may infest all domestic animals, including Equidae and many wildlife species, but chronically infected sheep are the most important source of

pasture contamination. A similar but larger fluke, *F. gigantica*, is restricted to warmer regions including parts of Africa and Asia. Human cases are usually associated with the ingestion of marsh plants such as watercress (Radostits et al. 2007). In developed countries, the incidence of Fasciola can reach up to 77 %. In tropical countries, fasciolosis is considered the single most important helminth infection of cattle, with reported prevalence of 30–90 % (Spithill et al. 1999). Acute fasciolosis in small ruminants most often occurs as sudden death without any previous clinical signs. Lack of appetite, ascites, abdominal haemorrhage, icterus, pallor and edema of mucosae and conjunctivae, weakness may be observed in sheep (Radostits et al. 2007). However, there are relatively few reports of epidemiology, clinical signs, and macroscopic and microscopic features of Fasciola in goats, but the occurrence of acute caprine fasciolosis with unusual migration is very rare and has not been reported yet.

Fig. 3 Histological sections of lung. **a** Multiple areas of necrosis and sequestration foci within the pneumonic portions (H&E; $\times 300$). **b** Cellular infiltration in the alveolar lumen and walls consist of macrophages, lymphocytes, plasma cells and neutrophils (H&E; $\times 3000$)

With respect to the relationship between infestation and blood parameter changes, the result found by Radostits et al. (2007) was nearly similar to this study. Even more, impaired liver function or increase excretion of enzymes may be attributed to larvae migration through hepatobiliary canaliculi which described by many authors in previous studies (Kaneko et al. 2008).

In this study, clinical signs and macroscopic characteristics were similar to other reports (Martinez-Moreno et al. 1999; Radostits et al. 2007). The pathological manifestations of classical form of fasciolosis were reported by several researchers in the world (Gajewska et al. 2005; Martinez-Moreno et al. 1999; Rushton and Murray 1977).

In the present study, pathologic changes to some extent were consistent with acute fasciolosis caused by *F. hepatica* described by other researchers (Martinez-Moreno et al. 1999). Histopathological findings revealed that a wide liver damage is caused by extensive migration of the immature flukes in the hepatic parenchyma.

Histopathologically, a clear cellular immune response was observed in the liver characterized by a marked infiltration of neutrophils, eosinophils, macrophages, lymphocytes and plasma cells, around migratory tracts as well as in portal areas. This is in agreement with other reports (Martinez-Moreno et al. 1999).

The cell mediated immune response to *F. hepatica* has been described in terms of in vitro proliferative response of peripheral blood lymphocytes (PBL) to fluke antigens and as the local cellular response occurring in the hepatic parenchyma (Meeusen et al. 1995).

In this study, accumulation of neutrophils and eosinophils were more prominent around the hemorrhagic tracts, whereas lymphocytes and plasma cells were scarce. In sheep experimentally infected with *F. hepatica*, fresh migratory tracts were not surrounded by leucocytic infiltrate (Meeusen et al. 1995) or this was scarce and mainly composed of neutrophils and eosinophils (Chauvin and Boulard 1996).

Aggregations of numerous lymphocytes and plasma cells, as well as proliferating fibrous connective tissue have been reported as a part of immune response in chronic fasciolosis (Martinez-Moreno et al. 1999).

In this case, thrombosis of blood vessels was prominently observed in areas adjoining migrational tracks. Generally, frequent thrombosis, significant hemorrhages, and extensive damage were associated mainly with the largest tracks.

However histopathological findings of lung revealed chronic suppurative bronchopneumonia, but this lesion is

not exactly associated with larval migration and some of inflammatory cells such as macrophages and lymphocytes have existed before the migration of parasites. It is thought that occurrence of larval migration to lung may be the result of aberrant migration of parasite to other organs specially lung (Eslami 2006).

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