



Original Full Paper

Coccidiosis, Paratuberculosis and Enterotoxaemia in Saudi Goats

Khaled Radad, Sary Khalil

Department of Pathology, Faculty of Veterinary Medicine, Assiut University, Assiut, Egypt
Corresponding author: Dr. Khaled Radad, Ph.D., Department of Pathology, Faculty of Veterinary Medicine,
Assiut University, Assiut 71526, Egypt Tel.: +2-882-333938 Fax: +2-882-366503
Email: khaledradad@hotmail.com

Submitted August 15th 2011, Accepted October 10th 2011

Abstract

During the year 2010, 66 goats suffering from mucoid to hemorrhagic diarrhea were submitted to the Pathology Division, Diagnostic Veterinary Laboratory, Jeddah, Saudi Arabia. After postmortem examination, tissue specimens were obtained from liver, lungs, heart, brain, stomach, small and large intestine and mesenteric lymph nodes for microbiological and histopathological examinations. Data from Microbiology Laboratory revealed that goats had intestinal coccidiosis (34 cases), paratuberculosis (9 cases), enterotoxaemia (22 cases) and mycotic gastritis (1 case). In the present report, we focused on the pathological pictures of the above mentioned diseases in examined goats.

Key Words: goats, coccidiosis, paratuberculosis, enterotoxaemia, gastritis

Introduction

Coccidiosis is an economically important disease. It affects a wide range of animals including cattle, sheep, goats, horses, dogs and cats as well as different avian species. In goats and sheep, coccidiosis causes enteric disease resulting in diarrhea, inefficient weight gains and occasionally death (9). The disease is caused by some species of the genus *Eimeria*. Infection of goats with coccidiosis occurs through ingestion of sporulated oocysts. In the small intestine, sporulated oocysts release sporozoites which infect intestinal epithelial cells.

Paratuberculosis (Johne's disease) is a chronic infectious enteric disease that affects domestic and wild ruminants (13). The disease is caused by *Mycobacterium avium* subspecies *paratuberculosis* (17). Paratuberculosis is widely distributed in small ruminants and the first case in goats was reported in 1912 (18). In the following years, the disease was reported in several European countries such as England, Italy and France, in many Asian and African countries and in United States, Argentine and Canada in American continent (13). The infection usually occurs orally throughout pastures, water and litters

contaminated with the feces containing the microorganism. The bacteria multiply within macrophages and with time cause granulomatous inflammation in the intestine and in the draining lymph nodes (4).

Enterotoxaemia is an economically important disease of livestock caused by *Clostridium perfringens* exotoxins. *Clostridium perfringens* is an anaerobic gram-positive, rod-shaped bacterium that is classified into 5 toxinotypes (A, B, C, D and E) according to the production of 4 major toxins namely alpha (CPA), Beta (CPB), epsilon (ETX) and iota (ITX) (16). *Clostridium perfringens* is a normal inhabitant of the intestine of most animal species including humans. When the intestinal environment is altered by sudden changes in the diet or other factors, *Clostridium perfringens* proliferates and produces potent exotoxins that act locally or are absorbed into the general circulation leading to devastating effects on the host (20). Enterotoxaemia of sheep and goats occurs worldwide and is caused mainly by *Clostridium perfringens* type D (3). The disease is characterized mainly by respiratory and neurological signs in sheep and diarrhea and enterocolitis in goats (19).

Systemic mycoses in animals are usually sporadic infections and cause non-specific syndrome. Infections usually arise from the fungal habitat as a saprophyte in organic matter, commonly moldy hay or straw or moist feed such as beet pulpy or brewers' grains.

In the present report, histopathological examination of gastrointestinal tracts of 66 goats submitted to the Diagnostic Veterinary Laboratory, Jeddah, Saudi Arabia showed a number of pathological lesions consistent with coccidiosis, paratuberculosis, enterotoxaemia and mycotic gastritis.

Materials and Methods

In this study, 66 goats suffering from mucoid to hemorrhagic diarrhea were submitted to the Diagnostic Veterinary Laboratory, Jeddah, Saudi Arabia. After postmortem examination, tissue specimens were obtained from liver, lungs, heart, brain, stomach, small and large intestine and mesenteric lymph nodes for microbiological and histopathological examinations. For microbiological examination, fresh tissue specimens from stomach, small and large intestine, mesenteric lymph nodes as well as gastric and intestinal contents were sent to the Laboratory of Microbiology, Diagnostic Veterinary Laboratory, Jeddah, Saudi Arabia. For histopathological examination, tissue specimens were collected in 10% neutral buffered formalin. After 24 h of fixation, tissue specimens were routinely processed for histology, sectioned at 4 µm and stained with hematoxylin and eosin (HE) (2). Stained sections were examined under a light microscopy and photographed using a digital camera.

Results

Gross pathology

Data from Microbiology Laboratory revealed that 34 goats had intestinal coccidiosis (6 months – 3 years old), 9 goats had paratuberculosis (more than 2 years old), 22 goats had *Clostridium perfringens* type D enterotoxaemia (6 – 1.5 years old) and 1 goat had mycotic gastritis (1 year old).

Postmortem examination revealed that the intestinal mucosa was thickened with a nodular surface and focal areas of hemorrhage in goats suffered from

coccidiosis. Goats with paratuberculosis revealed marked emaciation. The intestinal mucosa particularly of the ileum appeared thickened and corrugated and the mesenteric lymph node was severely enlarged. On cut surface, mesenteric lymph nodes showed multiple foci of small granulomas with central caseation and little calcification. In case of enterotoxaemia, the intestinal wall appeared thin, eroded and hemorrhagic. The stomach of the goat with mycotic gastritis showed thickened wall, congestion, hemorrhage and necrotic mucosa (data not shown).

Histopathology

Histopathological examination of HE-stained sections of jejunum from coccidia-diagnosed goats revealed proliferative changes of intestinal villi (Figure 1A) and presence of different stages of coccidia in enterocytes (Figure 1B). Examination of mesenteric lymph node showed lymphocytic depletion (Figure 1C) and neutrophilic infiltration (Figure 1D).

In case of paratuberculosis, duodenal and ileal villi appeared broader and blunter and infiltrated with lymphocytes (Figure 2A). Moreover, the villi in the duodenum and ileum were intensively infiltrated with aggregates of epithelioid cells (Figure 2B,C). Mesenteric lymph nodes showed small to mid-sized granulomas formed mainly of epithelioid cells and central necrosis with little calcification (Figure 2D).

Histopathology of enterotoxaemia in goats consisted of diffuse congestion of mucosal and submucosal blood vessels throughout the gastrointestinal tract. In the mucosa of terminal jejunum and ileum, there were multiple areas of superficial to full-thickness coagulative necrosis, fibrin, cell debris, congestion and mono- and polymorphnuclear leukocytes (Figure 3A). There were also hemorrhages and thrombosis of mucosal and submucosal vessels which is a common finding in all examined goats (Figure 3A,B).

Mycotic gastritis is principally characterized by severe necrosis of the abomasal mucosa, hemorrhage, thrombosis, mono- and polymorphnuclear cellular infiltration and presence of branching and septated fungal hyphae surrounded by inflammatory cells (Figure 3C,D).

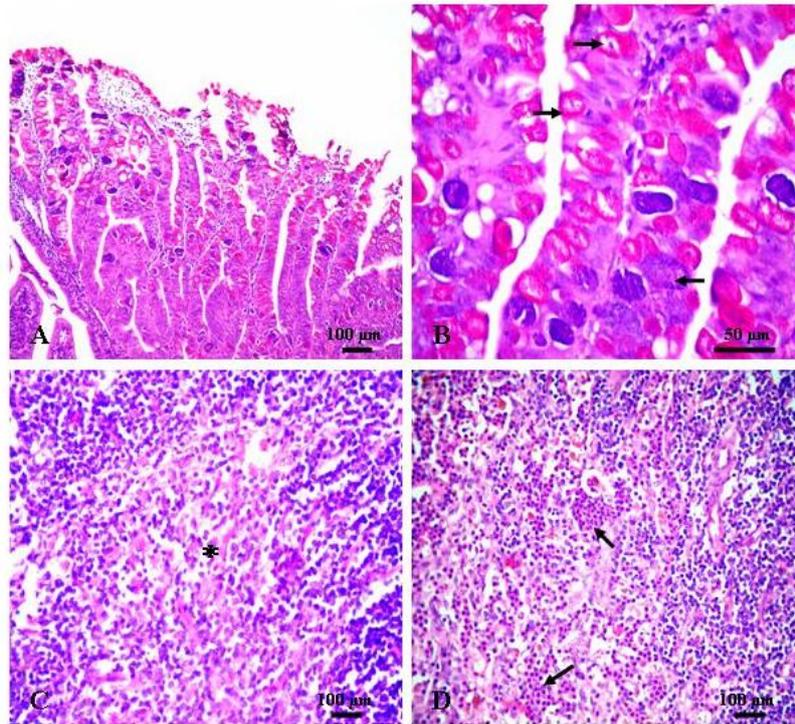


Figure 1. Representative micrographs for some histopathological changes as the result of coccidial infection in goats. A) Jejunum showing proliferative changes of the intestinal villi. B) Presence of different stages of coccidia in enterocytes (arrows). C) Lymphocytic depletion in a mesenteric lymph node (asterisks). D) Neutrophilic infiltration in a mesenteric lymph node (arrows).

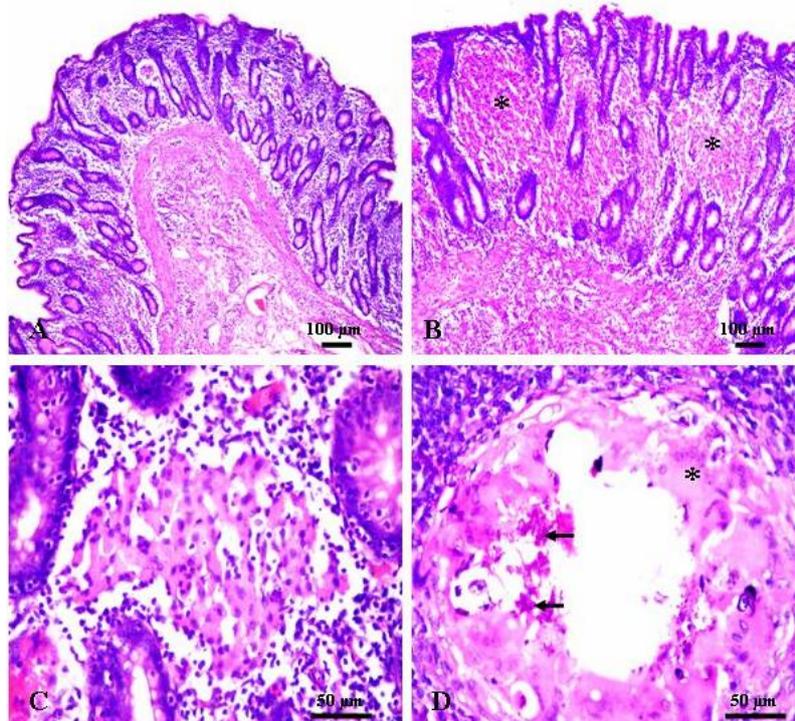


Figure 2. Representative micrographs for some histopathological changes as the result of paratuberculosis infection in goats. A) Ileum showing broader and blunter villi infiltrated with lymphocytes. B) Ileum showing intensive infiltration with aggregates of epithelioid cells (asterisks). C) Higher magnification showing epithelioid and lymphocytic cellular infiltration (asterisk). D) Mesenteric lymph node showing a small granuloma formed mainly from epithelioid cells (asterisk) and central caseous necrosis with little calcification (arrows).

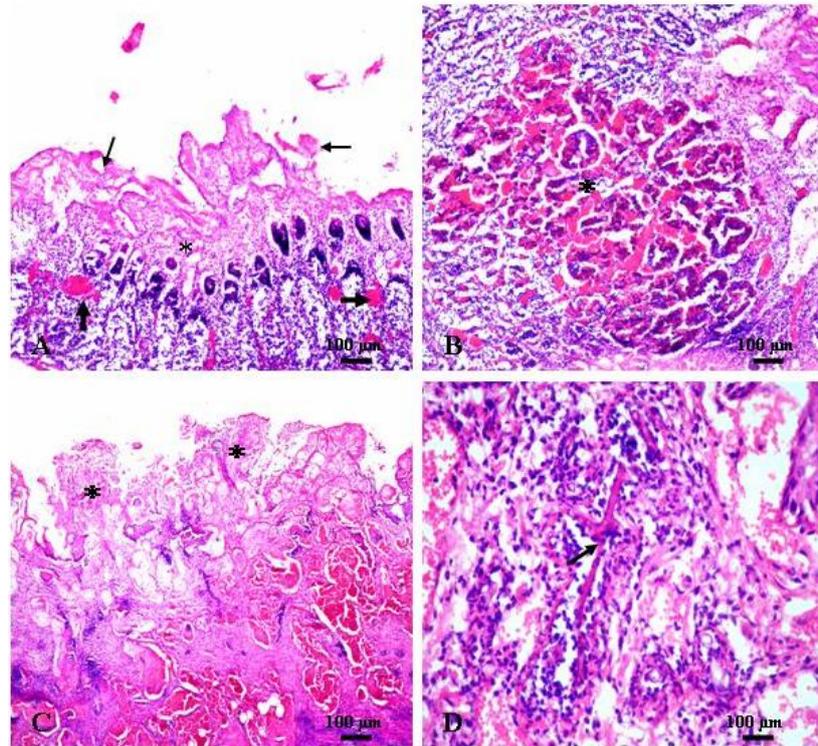


Figure 3. Representative micrographs for some histopathological changes of enterotoxaemia and mycotic gastritis in goats. A) Jejunum showing coagulative necrosis of the mucosa (asterisk), fibrin (thin arrows), cell debris, congestion (thick arrows) and mono- and polymorphnuclear leukocytes. B) Intestinal submucosa showing coagulative necrosis and hemorrhage (asterisk). C) Abomasum showing severe necrosis of mucosa (asterisks), hemorrhage and thrombosis. D) Abomasum showing a branching and septate fungal hyphae (arrow) surrounded by inflammatory cells.

Discussion

Sheep and goat farming in Saudi Arabia is based on an outdoor grazing system which in combination with periods of high stocking density and poor husbandry contributes to an increase in the incidence of infectious diseases in between livestock. In case of coccidiosis, such grazing system helps to deposit oocysts from either infected or carrier animals to the environment and vice versa to infect new animals. After ingestion of sporulated oocysts, sporozoites infect intestinal lining cells resulting in destruction of enterocytes with release of so many merozoites. Merozoites infect more intestinal cells causing further enterocytes destruction. Destruction of intestinal mucosa by coccidia results in hemorrhagic enteritis as seen in our report. Similarly, Hussein et al. (10) reported that coccidia were pathogenic to young camel calves and caused enteritis due to the destruction of intestinal mucosa by the giant schizonts. Histologically, intestinal mucosa of infected goats showed proliferation of intestinal villi and presence of different coccidial stages in enterocytes. Koudela and Bokova (11) and Dai et al. (7) reported mild subacute to chronic proliferative enteritis and presence of numerous developmental stages of the parasite in coccidia-

infected goats in Czech Republic and in experimentally infected goat with *Eimeria ninakohlyakimovae*, respectively. Moreover, mesenteric lymph nodes from infected goats were slightly enlarged and showed lymphocytic depletion and neutrophilic infiltration. No coccidian parasites were observed in lymph nodes and recruitment of neutrophils to lymph nodes seemed to be due to secondary infection. Coccidial schizonts were reported to be found in mesenteric lymph nodes of infected goats with *Eimeria apsheronica* (12).

In paratuberculosis-infected goats, there was intensive infiltration of jejunal and ileal mucosa with aggregates of epithelioid cells. Similarly, AL-Dubaib and Mahmoud (1) found infiltration of intestinal mucosa with epithelioid cells in goats infected with paratuberculosis in Qassim Region of Saudi Arabia. Moreover, Clarke (6) reported that diffuse epithelioid cell infiltration in the intestinal mucosa is the main microscopic lesion of paratuberculosis in ruminant animals. No granulomas had been observed in the intestinal tissues of examined goats. On the other hands, mesenteric lymph nodes of examined goats had microgranulomas consisted of aggregation of epithelioid cells and very few and small giant cells. Some of these granulomas showed central caseation and little calcification. In consistent with our findings, it

was shown that granulomatous lesions were more evident in mesenteric lymph nodes than intestinal tissue in deer experimentally infected with *Mycobacterium avium* subsp. *paratuberculosis* (5).

In enterotoxaemia-diagnosed goats, the lesions were characterized by presence of multiple areas of superficial to full-thickness coagulative necrosis in jejunum and ileum. *Clostridium perfringens* type D has been reported to affect mainly sheep and goats (3). In sheep, the disease is mainly characterized by respiratory and neurological lesions while in goats, the lesions are confined mainly to the intestine and they are in the form of enterocolitis (21). Fernandez Miyakawa and Uzal (8) reported that *Clostridium perfringens* type D toxin is more promptly absorbed by the ovine than the caprine intestine.

Mycotic abomasitis was seen in one of examined goats and was characterized by severe necrosis of the abomasal mucosa, thrombosis and presence of branching and septated fungal hyphae in the necrotic tissue. It was stated that systemic mycosis usually occurs as a sporadic problem in ruminants. Radostits et al. (15) mentioned that most cases of mycotic omasitis, rumenitis and enteritis were associated with diarrhea in adult ruminants. Migaki et al. (14) reported that mycotic infection of alimentary tract showed ulcers and necrosis of the mucosa in non-human primates.

In conclusion, our findings accentuate the importance of a careful histopathological examination in diagnosis of coccidiosis, paratuberculosis, enterotoxaemia and mycotic gastritis in goats.

References

1. AL-DUBAIB MA., MAHMOUD OM. Paratuberculosis of goats at Qassim Region of central Saudi Arabia. Bulgarian Journal of Veterinary Medicine, 2008, 11, 65-9.
2. BANCROFT JD., STEVENS A. Theory and practice of histological technique. 3rd Edn. Edinburg, Churchill Livingstone, 1990.
3. BROWN C., BAKER DC., BAKER IK. Alimentary System in Jubb, Kennedy and Palmer's: Pathology of domestic animals. 5th Edn. Elsevier Saunders, Philadelphia, 2007.
4. CHIODINI RJ. Immunology: resistance to paratuberculosis. Vet. Clin. North Am. Food Anim. Pract., 1996, 12, 313-43.
5. CLARK RG., GRIFFIN JF., MACKINTOSH CG. Johne's disease caused by *Mycobacterium avium* subsp. *paratuberculosis* infection in red deer (*Cervus elaphus*): an histopathological grading system and comparison of paucibacillary and multibacillary disease. N. Z. Vet., 2010, 58, 90-7.
6. CLARKE CJ. The pathology and pathogenesis of paratuberculosis in ruminants and other species. Journal of Comparative Pathology, 1997, 116, 217-61.
7. DAI YB., LIU XY., LIU M., TAO JP. Pathogenic effects of the coccidium *Eimeria ninakohlyakimovae* in goats. Vet. Res. Commun., 2006, 30, 149-60.
8. FERNANDEZ MIYAKAWA ME., UZAL FA. The early effects of *Clostridium perfringens* type D epsilon toxin in ligated intestinal loops of goats and sheep. Vet. Res. Commun., 2003, 27, 231-41.
9. FOREYT WJ. Coccidiosis and cryptosporidiosis in sheep and goats. Vet. Clin. North Am. Food Anim. Pract., 1990, 6, 655-70.
10. HUSSEIN HS., KASIM AA., SHAWA YR. The prevalence and pathology of *Eimeria* infections in camels in Saudi Arabia. Journal of Comparative Pathology, 1987, 97, 293-97.
11. KOUDELA B., BOKIVA A. Coccidiosis in goats in the Czech Republic. Vet. Parasitol., 1998, 76, 261-67.
12. KANYARI PW. *Eimeria apsheronica* in the goat: endogenous development and host cellular response. Int. J. Parasitol., 1990, 20, 625-30.
13. KRUIZE J., SALGADO M., PAREDES E., MELLA A., COLLINS MT. Goat paratuberculosis in Chile: first isolation and confirmation of *Mycobacterium avium* subspecies *paratuberculosis* infection in a dairy goat. J. Vet. Diagn. Invest., 2006, 18, 476-79.
14. MIGAKI G., SCHMIDT RE., TOFT JD., KAUFMANN AF. Mycotic infections of the alimentary tract of nonhuman primates: a review. Vet. Pathol. Suppl., 1982, 7, 93-103.
15. RADOSTITIS OM., GAY CC., BLOOD DC., HINCHCLIFF KW. Veterinary Medicine: A textbook of the diseases of cattle, sheep, pigs, goats and horses. 9th Edn. London, WB Saunders, 2000.
16. ROOD JI. Virulence genes of *Clostridium perfringens*. Annu. Rev. Microbiol., 1998, 50, 333-60.
17. SING AV., SINGH SV., SINGH PK., SOHAL JS. Is *Mycobacterium avium* subsp. *paratuberculosis*, the cause of Johne's disease in animals, a good candidate for Crohn's disease in man? Indian J. Gastroenterol., 2010, 29, 53-8.
18. TWORT FW., INGRAM GLY. A method for isolating and cultivating *Mycobacterium enteritidis chronicae pseudotuberculosis bovis*, Johne, and some experiments on the preparation of a diagnostic vaccine for pseudotuberculous enteritis of bovines. Vet. J., 1912, 68, 353-65.
19. UZAL FA., KELLY WR. Enterotoxaemia in goats. Vet. Res. Comm., 1996, 20, 481-92.
20. UZAL FA., SONGER JG. Diagnosis of *Clostridium perfringens* intestinal infections in sheep and goats. J. Vet. Diagn. Invest., 2008, 20, 253-65.

21. UZAL FA., FISHER DJ., SAPUTO J., SAYEED S.,
MCCLANE BA., SONGER G., TRINH HT.,
FERNANDEZ MIYAKAWA ME., GARD S.
Ulcerative enterocolitis in two goats associated with
enterotoxin- and beta2 toxin-

positive Clostridiumperfringens type D. J. Vet.
Diagn. Invest., 2008, 20, 668-72.