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Scientific Report

An occurrence of intestinal histoplasmosis in a goat flock

Babu Prasath, N.* and Selvaraj, J.

Department of Veterinary Pathology, Veterinary College and Research Institute, Tamil Nadu Veterinary and Animal Sciences University, Orathanadu, Thanjavur, Tamil Nadu, India

***Correspondence:** *N. Babu Prasath, Department of Veterinary Pathology, Veterinary College and Research Institute, Tamil Nadu Veterinary and Animal Sciences University, Orathanadu, Thanjavur, Tamil Nadu, India. E-mail: vetdrprasad@gmail.com*

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Abstract

Background: The paper describes the first documentary evidence of intestinal histoplasmosis in Indian goats. **Case description:** A goat flock in delta region of Tamil Nadu with a report of mortality of eight animals with the history of inappetence, constipation, tenesmus and hematochezia. Three goat carcasses were presented for necropsy. **Findings/treatment and outcome:** Postmortem examination showed mesenteric lymphadenomegaly with greyish yellow viscous contents. Ileum contained scattered dark brownish tubular concretions inside the lumen. Colon and caecum had scattered firm serosal and mucosal nodules with caseous exudate. Lumen contained viscous haemorrhagic content. Mucosa of the colon showed numerous tiny ulcers. Urinary bladder serosa showed a focal firm nodule with caseous exudate and irregular firm mass. Microscopically, lung revealed oval inclusions with clear halo in pulmonary macrophages. Caecum and colon showed ulcers with necrotic core. Necrotic border showed multinucleated giant cells and oval bodies with clear halo. Mesenteric lymph node and urinary bladder revealed pyogranulomatous inflammation with aggregation of macrophages filled with numerous tiny inclusions. Periodic acid-Schiff and Grocott's methanamine silver staining techniques and morphological features of the inclusion confirmed it as *Histoplasma capsulatum*. The case showed a resemblance with human colonic histoplasmosis. **Conclusion:** This report documents the first incidence of intestinal histoplasmosis in goats. Interestingly, the intestinal form of histoplasmosis is considered to be common in humans and it showed similarity with human disease in goats based on the gross and microscopic lesions.

Key words: Caseous lymphadenitis, Goats, Hemorrhagic-ulcerative colitis, Histoplasmosis, Pyogranulomatous cystitis

Introduction

Histoplasmosis is a granulomatous mycotic disease of humans and animals caused by dimorphic fungi *Histoplasma capsulatum* (Norton, 1994). *Histoplasma* spp. are opportunistic pathogens that primarily afflict immunocompromised individuals (Alsibai *et al*., 2020; Sumit *et al*., 2022; Sircar *et al*., 2023). The incidence of histoplasmosis has been reported to be high in humans and low in animals which might be related to scarcity of documentary evidence. It usually occurs as a pulmonary disease and is rarely seen as a disseminated form of multisystemic illness (Sumithra *et al*., 2013). Gastrointestinal histoplasmosis is a common form of disseminating disease and reported to be rare and infrequent (Sharma *et al*., 2017; Anderson *et al*., 2018). Colonic histoplasmosis is found to be a major life-threatening disease in humans (Psarros and Kauffman, 2007; Alsibai *et al*., 2020). The disease is characterized by pyogranulomatous lesions in the affected organs (Alsibai *et al*., 2020; Acharyya *et al*., 2021). To date, histoplasmosis is reported in a wide variety of animals, mostly companion animals including dogs, cats and also horses. Occurrence of histoplasmosis is under-reported in ruminants and found to be scanty (Bromel and Sykes, 2005; Schumacher *et al*., 2013; Fortin *et al*., 2017; Chavez-Peon Berle *et al*., 2020). It is noteworthy that despite the lack of authenticated documents on histoplasmosis in animals in India, it is considered to be endemic in East India and sporadic in South India involving human incidences (Vijayan *et al*., 2007; Sumithra *et al*., 2013; Bahr *et al*., 2015). This report forms the first documented evidence on intestinal histoplasmosis in a goat flock of India.

Case description

According to the owner of a goat flock of 10 adult animals aged 2 years at Mannargudi taluk, Tiruvarur district, Tamil Nadu, India eight goats died during a two week-period. Three dead goat carcasses were transferred to the Department of Veterinary Pathology, Veterinary College and Research Institute, Tamil Nadu Veterinary and Animal Sciences University, Orathanadu, Thanjavur for postmortem examination. Owner reported that the animals were purchased from a goat shandy of South

Tamil Nadu, with about 150 km and were transported by truck 2 weeks before the illness. Animals were housed in an open fencing system. Dead female goats had a history of inappetence, dullness and constipation with tenesmus for 5 days before death. A few animals of the affected flock had dark brownish black semifluid discharge while defecating occasionally. Systematic and detailed necropsy was carried out.

Samples such as heart blood smear and mesenteric lymph node impression smears were collected for cytology and tissue sample from affected organs were collected in 10% formalin for histopathology. Cytology smears were processed as per standard Romanowsky staining (Wright-Giemsa's method) and few impression smears from mesenteric lymph nodes were processed for Periodic acid-Schiff (PAS) staining. Tissues samples were processed as per standard paraffin embedding and tissue sections were stained with hematoxylin and eosin (H&E) staining technique (Bancroft and Layton, 2019). Duplicate tissue sections were stained with PAS (Layton and Bancroft, 2019) and Grocott's methanamine silver (GMS) staining protocol (Morris *et al*., 2019).

Findings/treatment and outcome

Gross pathology

Externally, carcasses showed fair condition. Visible mucous membranes were pale. Cloudy viscous discharge was noticed from the nostrils and anus. Perineum was soiled with reddish brown sticky fecal material. Internally, subcutis was dry. Prescapular lymph nodes were bilaterally enlarged, on incision sero-sanguineous fluid oozed out and cut surface was brownish pink. On opening thoracic cavity, lungs did not collapse. Lungs were slightly heavy, voluminous and had glistening appearance. Mediastinal lymph nodes were enlarged and parenchyma was reddish brown. Left ventricle endocardium showed diffusely scattered prominent petechiae to irregular ecchymosis. Renal pelvis of both the kidneys showed multiple scattered petechiae. Liver was mottled. Diaphragmatic surface of right lobe of liver showed patchy (5 cm length × 3 cm diameter) dark brownish red slightly elevated areas. On incision, cut surface was dark reddish brown. Gall bladder was distended with turbid dark greenish bile. Gall bladder mucosa was thick and edematous. Mesenteric lymph node was enlarged with patchy reddish areas. On incision, thin clear fluid oozed out and a focal area which contained greyish yellow viscous exudate was observed (Fig. 1). Duodenal mucosa was slightly red and had irregular rough surface. Distal ileum was collapsed and contained dark brownish black, firm, tubular casts (3 mm length × 5 mm diameter) which were firm and tough to crush. Caecum contained dark reddish brown semiliquid and granular pasty contents with dry, firm, cake-like materials (Fig. 2) which were roughly rectangular with flat surfaces. Spiral colon contained dark brownish red granular pasty contents smearing the mucosa and firm dark brownish tubular casts (Fig. 3). Mucosa revealed multiple scattered diffuse ulcerative foci (1 mm). Urinary bladder near dorsal serosal surface of neck showed focal firm greenish black nodule. On incision, it contained cauliflower-head like gritty contents (Fig. 4) which could be lift-out easily and underneath tissue was surrounded by greyish green pasty contents.

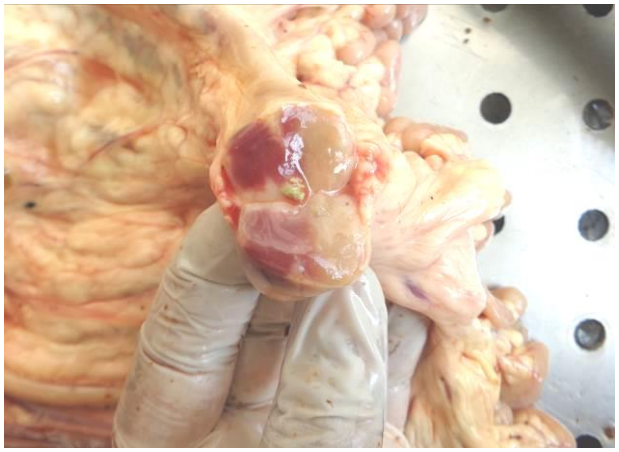

Fig. 1: Gross pathology of histoplasmosis in goats. Enlarged and oedematous mesenteric lymph node with greyish-yellow viscous contents

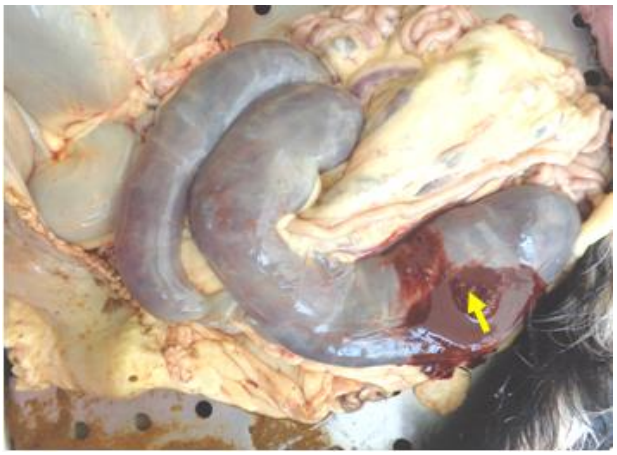

Fig. 2: Gross pathology of caecum representing histoplasmosis in goats. Haemorrhagic typhlitis with cake-like contents

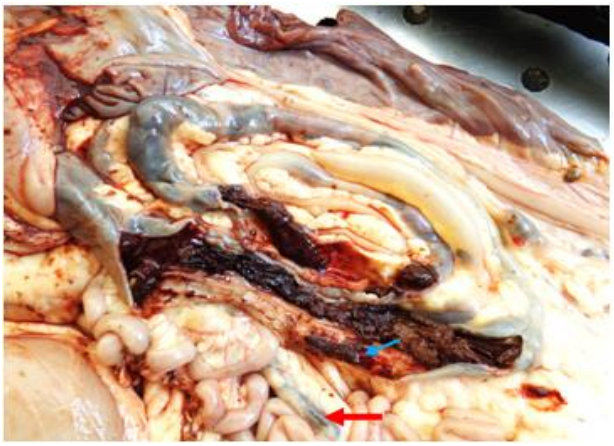

Fig. 3: Gross pathology of colon for histoplasmosis in goats. Haemorrhagic-ulcerative colitis and cylindrical concretions in ileum (broader arrow) and colon (thin arrow)

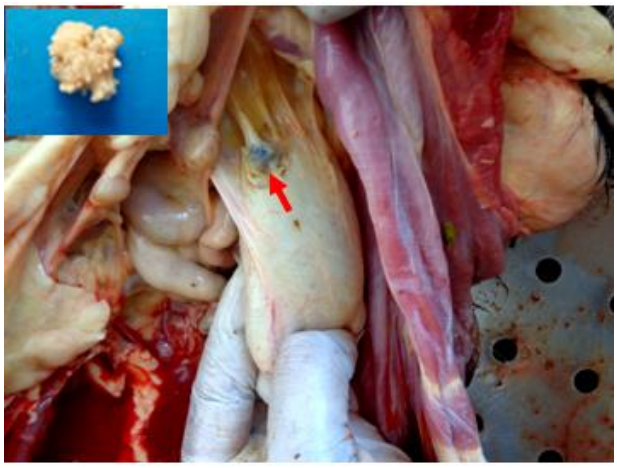

Fig. 4: Gross pathology of histoplasmosis in goats. Metastatic nodule in serosa of urinary bladder. Cauliflower head-like gritty content with in the nodule (Inset)

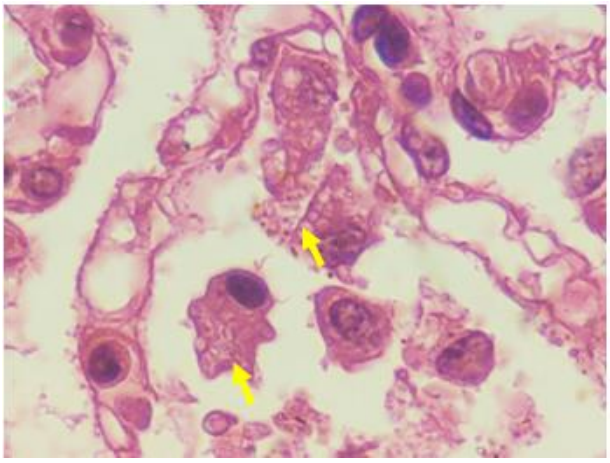

Fig. 5: Histopathological examination of lung for histoplasmosis in goats Pulmonary alveolar macrophages with *Histoplasma* spp. inclusions (H&E, ×400)

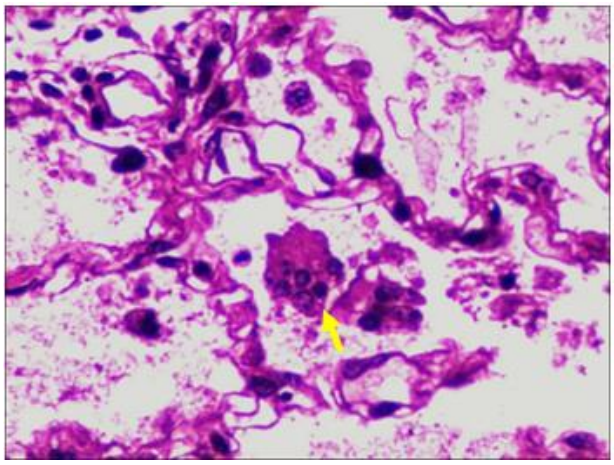

Fig. 6: Histopathological examination of lung for histoplasmosis in goats. Syncytium of alveolar macrophages within lumen of pulmonary alveoli (H&E, ×400)

Histopathology

Histopathological examination of lung revealed diffuse alveolar oedema. Alveolar wall was slightly thick due to congestion. Alveolar pneumocytes were eminent and contained oval shaped intracytoplasmic bodies with clear halo around (Fig. 5). Alveolar lumen contained multinucleated syncytial cells (Fig. 6) and few macrophages. Liver showed multifocal periportal haemorrhagic necrosis. Gall bladder mucosa was thick and oedematous. Mesenteric lymph nodes revealed granulomatous lymphadenitis characterized by aggregation of many epithelioid cells, macrophages and few lymphocytes, neutrophils and plasma cells. Epithelioid cells and macrophages contained multiple of tiny intracytoplasmic bodies that covered the cytoplasm and marginated the nucleus towards cell border. Colon revealed focal ulcer covered with necrotic mass characterized by fibrino-purulent exudate (Fig. 7) and surrounded by multinucleated giant cells and oval blue bodies with clear halo around (Fig. 8). Kidney revealed mild sub-acute interstitial nephritis along with scattered eosinophilic casts inside tubular lumen. Urinary bladder showed serosal granulomatous cystitis characterized by areas of necrosis bordered by neutrophils, macrophages,

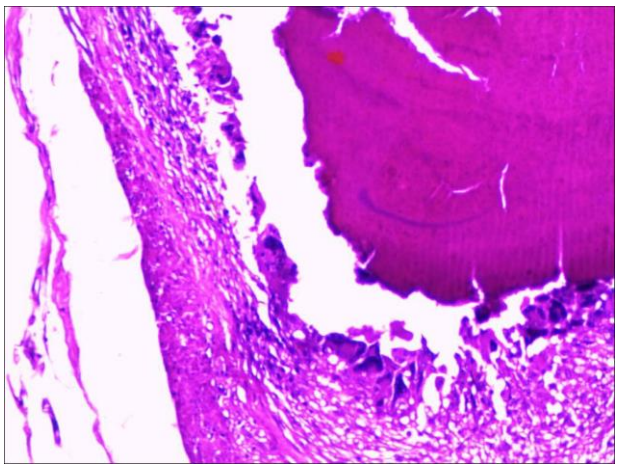

Fig. 7: Histopathological examination of colon for histoplasmosis in goats. Ulcerative and granulomatous colitis with giant cells (H&E, ×100)

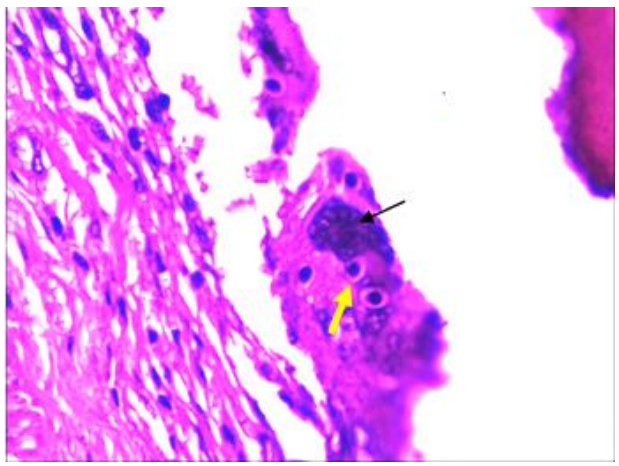

Fig. 8: Histopathological examination of colon for histoplasmosis in goats. Granulomatous colitis exhibiting giant cells (thin arrow) with *Histoplasma* spp. inclusions (thick arrow) (H&E, ×400)

epithelioid cells and giant cells. Epithelioid cells and macrophages contained tiny intracytoplasmic bodies. Urinary bladder nodular mass revealed hyalinised material at the center surrounded by fibrino-purulent content with multiple giant cells containing oval shaped blue bodies surrounded by clear halo. Urinary bladder serosa revealed granulomatous cystitis.

Cytology

Cytological examination of heart blood smear revealed oval shaped blue inclusion within the cytoplasm of the monocytes (Fig. 9) and neutrophils. Few oval shaped bodies were found extracellularly. Mesenteric lymph node impression (MLNI) smears revealed many epithelioid cells and macrophages with intracytoplasmic blue coloured oval bodies that were surrounded by clear halo capsule by Wright-Giemsa stain. Inclusions were suggestive of *H. capsulatum* which was confirmed by PAS and GMS staining. Periodic acid-Schiff staining of MLNI smear (Fig. 10) and tissue section of mesenteric lymph node (Fig. 11), urinary bladder and colon showed pink hued, variable sized oval bodies within the cytoplasm of the macrophages. Grocoot's methanamine silver staining of MLNI smear showed black coloured, tiny, oval bodies within the cytoplasm of macrophages (Figs. 12-14), urinary bladder and colon. Fibrocytes surrounding the inflammatory zone of urinary bladder serosa also revealed pink coloured intracytoplasmic inclusions on PAS staining.

Morphology of intracytoplasmic inclusions within the epitheloid cells and macrophages of colon, mesenteric lymph node and urinary bladder and in heart blood smears and lymph node impression smears were characterized by small to large, variably oval bodies with clear halo by Romanowsky stain, PAS and GMS in cytology and histochemistry. Based on the above morphological features, inclusions were identified as *H. capsulatum* causing histoplasmosis. The disease was diagnosed as "intestinal histoplasmosis" as the lesions entirely restricted to intestine especially colon with secondary metastasis to urinary bladder.

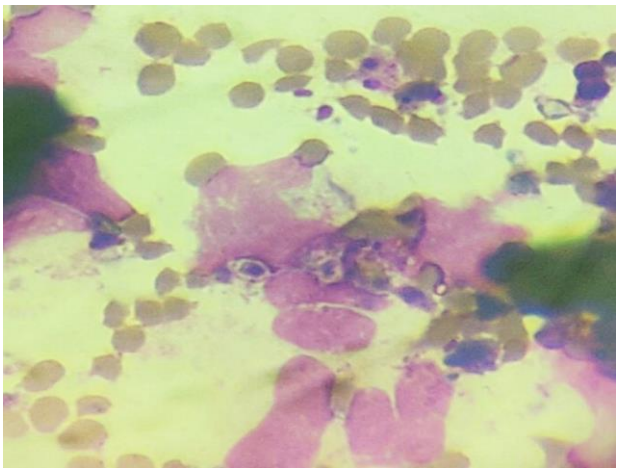

Fig. 9: Cytological examination of peripheral blood smear for histoplasmosis in goats. Intracytoplasmic *Histoplasma* spp. inclusions in blood monocyte (Wright-Giemsa stain, ×1000)

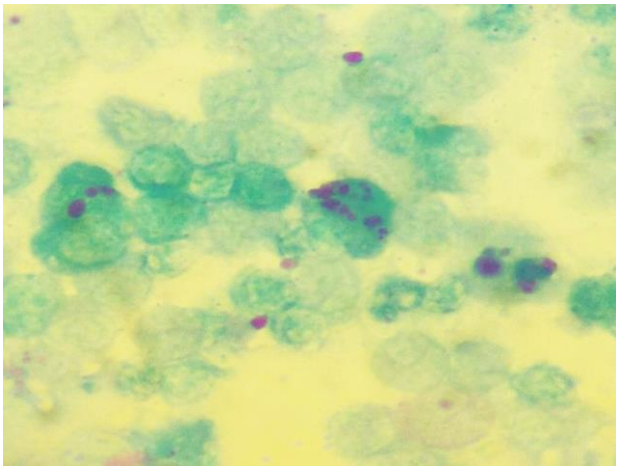

Fig. 10: Cytological examination of mesenteric lymphnode impression for histoplasmosis in goats. Intracytoplasmic *Histoplasma* spp. inclusions in histiocytes (Periodic acid-Schiff stain, ×1000)

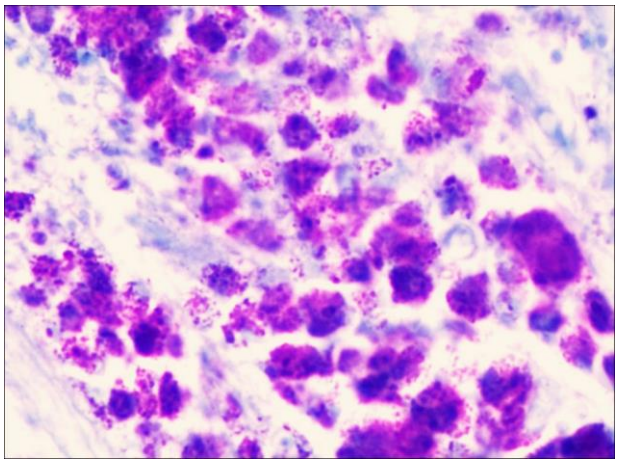

Fig. 11: Histochemical examination of mesenteric lymphnode for histoplasmosis in goats. Throng of histiocytes with tiny magenta coloured *Histoplasma* spp. inclusions (Periodic acid-Schiff stain, ×400)

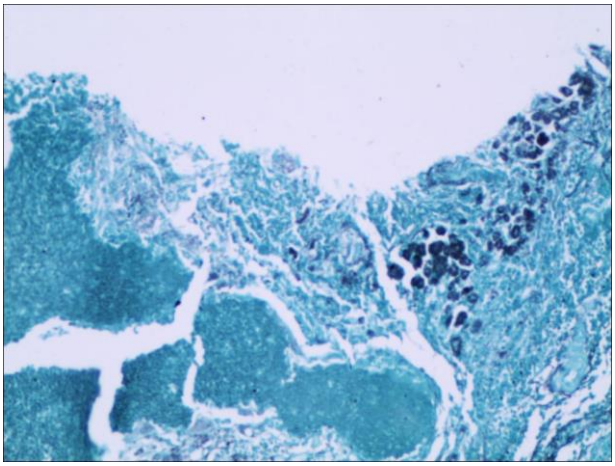

Fig. 12: Histochemical examination of mesenteric lymphnode for histoplasmosis in goats. Throng of histiocytes with black coloured intracytoplasmic *Histoplasma* spp. bodies around necrotic and granulomatous lymphadenitis (Grocott's methanamine silver stain, ×40)

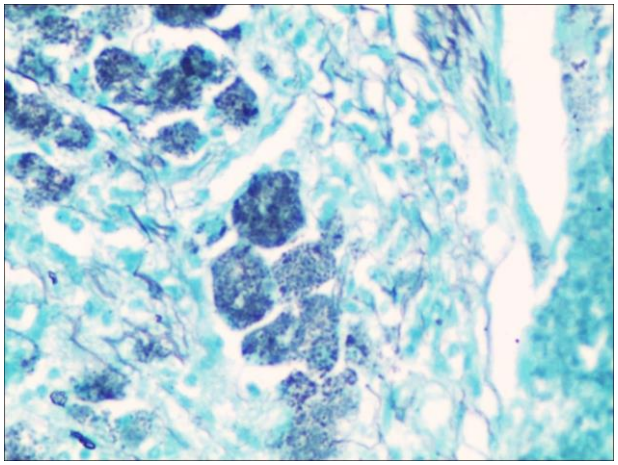

Fig. 13: Histochemical examination of mesenteric lymphnode impression for histoplasmosis in goats. Colossal aggregations of tiny black coloured intracytoplasmic *Histoplasma* spp. bodies having clear halo of capsule around in histiocytes beside the necrotic tissue (Grocott's methanamine silver stain, ×400)

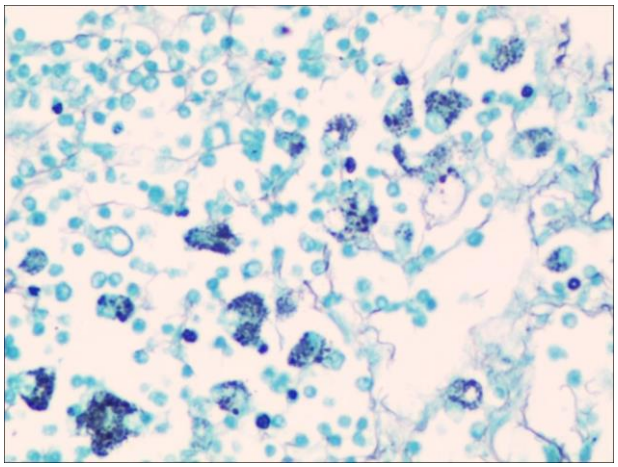

Fig. 14: Histochemical examination of mesenteric lymphnode impression for histoplasmosis in goats. Diffuesly scattered macrophages with tiny black coloured intracytoplasmic bodies of *Histoplasma* spp. with halo around (Grocott's methanamine silver stain, ×400)

Discussion

Histoplasmosis is an invasive mycotic disease caused by a dimorphic yeast *H. capsulatum*. The causative organism was first observed early in 1873 from horses suffering from epizootic lymphangitis (Guillot *et al*., 2018). Later in late 1904 to early 1906, the organism was identified in a human patient suffering from chronic lesions suspected to military tuberculosis by Samuel Darling who described the causative agent as a protozoan parasite and named it *H. capsulatum* by considering the disease similar to visceral leishmaniasis. Subsequently, in the year 1912, the same case was reevaluated and the causative agent was identified as yeast by a pathologist Henrique da Rocha-Lima who named the disease Histoplasmosis (Darling's disease) (Norton, 1994). The first animal case of histoplasmosis was reported in a dog during 1939 by De Monbreun (De Monbreun, 1939). Further, soil contaminated by bird excrements (1948) and guano of bats (1958) were proposed and proved to be a source for the disease by inhalation (Cano and Hajjeh, 2001). Subsequently, the disease became significant, occurring and reported in human beings and domestic and wild animals.

Animal histoplasmosis is reported in horses, dogs, cats, goats, rabbits and many more, including wild animals and rodents worldwide (Schumacher *et al*., 2013; Sumithra *et al*., 2013; Brandao *et al*., 2014; Fortin *et al*., 2017; Guillot *et al*., 2018; Schlemmer *et al*., 2019; Heilmann *et al*., 2021). As far as we know, the presented case in this study is the first report of naturally occurring histoplasmosis in goats in India.

The present case describes the intestinal form of histoplasmosis in non-descript goats which were analogous to previous recorded report of intestinal histoplasmosis in humans (Sharma *et al*., 2017; Bhinder *et al*., 2018; Alsibai *et al*., 2020; Acharyya *et al*., 2021; Jimenez-Rivera *et al*., 2021), dogs (Schumacher *et al*., 2013; Chavez-Peon Berle *et al*., 2020) rabbits (Brandao *et al*., 2014) and reindeers (Fortin *et al*., 2017). Although, all parts of intestine were reported to be affected, distal ileum and colon are the most affected sites by the disease in humans (Psarros and Kauffman, 2007; Alsibai *et al*., 2020) and similar form is reported in dogs (Schumacher *et al*., 2013) which is in parallel to the present report. The present case described the haemorrhagic colitis with multiple ulcers and pyogranulomatous lesions which were in accordance with Alsibai *et al*. (2020) who diagnosed colonic histoplasmosis in 78 out of 202 human patient samples screened by cytology and histopathology. Psarros and Kauffman (2007) described the colonic histoplasmosis as the most life threatening form, as it may result in bowel perforation and haemorrhages. This explanation was obvious in the present study with the evidence of ulcerative colitis and haemorrhagic contents which may lead to perforations of the colon. Dark brownish casts observed in the distal ileum and colon of the present study mimics the description of Alsibai *et al*. (2020), who observed 2/5 cases with pseudotumoral fibro-inflammatory intestinal occlusion in the colon of the affected human patients. The cylindrical casts in the distal ileum and colon of the present study were firm and might be the result of concretions of the excreta with inflammatory exudate. The cylindrical casts on gross examination looked like tumorous mass and hence the term pseudotumoral fibro-inflammatory occlusion described earlier was used in the present study. Mesenteric lymphadenomegaly recorded in the present study was in accordance with histoplasmosis described in Nubian goat by Schlemmer *et al*. (2019). Enlargement of mesenteric lymphnodes warrants differentiation with intestinal form of tuberculosis was suggested by Acharyya *et al*., (2021) in humans was plausible for histoplasmosis in animals. Observation of caseous lymphadenomegaly and lesions of caecum and colon of histoplasmosis in small ruminants need to be listed in

differential diagnosis with the diseases caused by other similar mycotic diseases such as sporothricosis, blastomycosis and crytococcoses (Norton, 1994; Lamps *et al*., 2000).

Lesions of endocardium and liver were consistent with previous record of histoplasmosis in animals (Mitchell and Stark, 1980). Biliary obstruction as a consequence of histoplasmosis with hepatomegaly and hepatic lymphadenomegaly is described in humans (Cridge *et al*., 2019) that the fact that patients with disseminated histoplasmosis should be monitored for potential complications including hepatomegaly (Chavez-Peon Berle *et al*., 2020) is consistent with hepatomegaly and hepatic necrosis as observed in the present study.

Histologically, histoplasmosis is classified into 4 forms as 1) the tuberculoid form, 2) the anergic form, 3) the mixed form, and 4) the sequelae form (Alsibai *et al*., 2020). Generally, tuberculoid form corresponds to effective tissue response of the host enriched with activated macrophages and lymphocytes (CD4+ T lymphocytes). Pyogranulomatous lesions in the mesenteric lymph node, caecum, colon, urinary bladder and massive aggregations of histoplasma bodies in macrophages and appearance of multinucleated giant cells, epitheloid cells and lymphocytes were the distinct observations in the present study. Macroscopic and microscopic analogue of the lesions deliberately classifies the present case as tuberculoid form of intestinal histoplasmosis in non-descript goats. Cellular formula proposed by Alsibai *et al*. (2020) for classical histoplasmosis was 55-85% macrophages, 2-30% neutrophils, 8-18% lymphocytes and limited number of plasma cells and eosinophils. The above cellular formula was not consistent with the present study where epitheloid granuloma primarily composed of macrophages and lymphocytes with rare plasma cells. Periportal necrosis of liver observed in the present study was categorized under anergic form where little tissue response elaborated by the host.

Histoplasmosis is among the neglected and or underdiagnosed diseases of farm animals because of various reasons. First, it is reported primarily as an opportunistic pathogen and known to cause disease in immunocompromised or immunosuppressive patients. Further, there is lack of documentary evidence of histoplasmosis as a primary pathogen to cause mortality in farm animals. Histoplasmosis was well established as a cause of disease in horses, dogs, and cats but not in ruminants. Finally detection of the presence of *Histoplasma* spp. inclusions in lymph node cytology during conventional clinical examination was rare in ruminants and hence, decreased the chance of diagnosing the disease (Guillot *et al*., 2018; Schlemmer *et al*., 2019).

We attempted to reason out the possible chances for the present disease incidence with previous documented evidences. The reasons as follows, 1) change in environment and humid climate helps *Histoplasma* spp. to develop and survive in temperate and subtropical areas, 2) composite rearing of birds (backyard, commercial, and/or pet birds), movements of pigeons in big cities of India and movements of bats, extremely pose a chance for the etiological agent to flourish in nitrogen rich acidic soil contaminated with excrement of birds including chicken, pigeons and guano of bats at temperature between 22-29°C, 3) congenital immunoglobulin deficiency syndromes, 4) immunosuppression due to prolonged steroid usage, chronic inappetence, transportation for long distance, and 5) intensity of exposure to infective microconidia (Psarros and Kauffman, 2007; Schumacher *et al*., 2013; Sharma *et al*., 2017) were the possible sources for the infection with *Histoplasma* spp.

Although it was proposed to be the disease of immunocompromised patients, histoplasmosis is reported in immunocompetent dog in USA as a primary cause of disease (Heilmann *et al*., 2021). In the present study, it is possible that the animals might be immunosuppressed by the stress of transportation and exposure to new environment. The concentration of bird droppings in the farm habitat of these animals was unknown.

Upon inhalation of infective microconidia, disease takes two forms; 1) pulmonary or common form, and 2) disseminated or rare form (Sumithra *et al*., 2013). Disseminated form occurs once telemorph (sexual stage) in soil transforms into anamorph (asexual stage or yeast form) in alveolar macrophages. Organisms trap in mononuclear cell rich organs such as lymph nodes, liver and spleen by using integrin receptors. T cells defend against the pathogen, but organisms usually disseminate. Upon entering into circulation, organisms which are present in leucocytes disseminate to skin, bone marrow, brain, eye and intestine. Of which, intestinal form is infrequent and was reported to be high in humans and rare in animals. Distal ileum, caecum, colon and mesenteric lymph nodes were reported to be affected due to high lymphoid tissues (Lamps *et al*., 2000; Psarros and Kauffman, 2007). Ulcerative haemorrhagic colitis, pyogranulomatous and histiocytic lymphadenitis observed in the present study were in parallel with earlier findings (Lamps *et al*., 2000; Psarros and Kauffman, 2007; Sharma *et al*., 2017; Acharyya *et al*., 2021; Jimenez-Rivera *et al*., 2021). Ulcerative colitis might be due to invasive mycosis which was responsible for the haemorrhages. Concretions of haemorrhagic exudate with intestinal content has possibly altered the bowel movements and was responsible for constipation and hematochezia observed in the present study. Anemia evidenced by pale conjunctiva and erythrocyte morphological characters in blood smears might possibly be due to intestinal haemorrhage and bone marrow suppression. Histoplasmosis is reported to induce nonregenerative anemia (Chavez-Peon Berle *et al*., 2020). Malabsorption and anemia together contributed for death in the present study which was in accordance with previous documentary evidence in humans and animals (Fortin *et al*., 2017; Anderson *et al*., 2018; Chavez-Peon Berle *et al*., 2020; Jimenez-Rivera *et al*., 2021). Further, lesions in the spiral colon and caecum may have resulted in serosal adhesion and

pyogranulomatous cystitis.

Diagnosis of histoplasmosis is usually carried out by cytology, histopathology, culture, urine, serum assay, and molecular detection. An exceptionally, histoplasmosis is initially well diagnosed by rapid detection methods such as cytology, histopathology, and best be confirmed by PAS and/or GMS staining technique preferably (Alsibai *et al*., 2020). Although, tissue culture remains gold standard for diagnosis, is not recommended due to 1) delayed growth of organism which take as long as 4-6 weeks, and 2) laboratory culture is hazardous due to its zoonotic potential by easy aerosol spread. Molecular techniques can obviously confirm the disease, but histopathology was reported to be sensitive and rapid diagnostic method (Fortin *et al*., 2017; Alsibai *et al*., 2020; Jimenez-Rivera *et al*., 2021). In live animals, detection of antigen in urine (antigenuria) was more sensitive than serological detection using MVista *H. capsulatum* quantitative antigen ELISA in dogs (Psarros and Kauffman, 2007; Chavez-Peon Berle *et al*., 2020; Heilmann *et al*., 2021). Antigenuria was presumed to be sensitive for gastrointestinal histoplasmosis in dogs (Chavez-Peon Berle *et al*., 2020). A limitation of our study was that we did not assay urinary antigen. Appearance of *Histoplasma* spp. antigen in urine is by renal excretion from blood stream. Haemorrhagic lesions and interstitial nephritis in kidneys and metastatic cystitis in the present study increase the possibility of presence of *Histoplasma* spp. antigen in urine.

The disease is known to be caused by single organism *H. capsulatum* with three variants which includes *H. capsulatum* var *capsulatum*; *H. capsulatum* var *duboisii*, and *H. capsulatum* var *farciminosum*. Based on exclusion with the geographical occurrences and species involved by each variant of *H. capsulatum*, the present etiological variant was identified as *H. capsulatum* var *capsulatum* based on its morphological appearance. The morphological features elaborated in the present study were indistinguishable with earlier reports (Guillot *et al*., 2018; Schlemmer *et al*., 2019; Alsibai *et al*., 2020). Although, the similar oval morphology mimicked *Leishmania* spp., *Penicillium marneffei*, and *Candida glabrata*, the geographical occurrences and clinical manifestations by those organisms differ (Psarros and Kauffman, 2007). Hence, the present case was diagnosed purely based on the lesions and morphology of *H. capsulatum* in cytology, histopathology, histochemistry, and inflammatory cellular profile.

In conclusion, this report records the incidence of naturally occurring intestinal form of histoplasmosis in goats which mimics typical human colonic histoplasmosis. It documents the rarest mycotic disease causing mortality in farm animals and suggest that the clinician should consider histoplasmosis in the differential diagnosis for intestinal diseases of small ruminants. It also warrants that early diagnosis of intestinal histoplasmosis is important in the prevention of bowel perforation and hemorrhage.

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Conflict of interest

The authors declare that they have no conflict of interest.

References

Acharyya, BC; Acharyya, S and Chakrabarty, H (2021). Isolated gastrointestinal histoplasmosis: A rare diagnosis of pediatric chronic abdominal pain. Indian J. Pathol. Microbiol., 64: 827-830.

Alsibai, DK; Couppie, P; Blanchet, D; Adenis, A; Epelboin, L; Blaizot, R; Louvel, D; Djossou, F; Demar, M and Nacher, M (2020). Cytological and histopathological spectrum of histoplasmosis: 15 years of experience in French Guiana. Front. Cell Infect. Microbiol., 10: 591974.

Anderson, BR; Marriott, J; Bulathsinghala, C; Anjum, H and Surani, S (2018). Gastrointestinal histoplasmosis presenting as an acute abdomen with Jejunal perforation. Case Rep. Med., 2018: 8923972.

Bahr, NC; Antinori, S; Wheat, LJ and Sarosi, GA (2015). Histoplasmosis infections worldwide: thinking outside of the Ohio River valley. Curr. Trop. Med. Rep., 2: 70-80.

Bancroft, JD and Layton, C (2019). The hematoxylins and eosin. In: Suvarna, SK; Layton, C and Bancroft, JD (Eds.), *Bancroft's theory and practice of histological techniques*. (8th Edn.), UK, Elsevier. PP: 126-138.

Bhinder, J; Mori, A; Cao, W and Malieckal, A (2018). A case of isolated gastrointestinal histoplasmosis. Cureus. 10: e2951.

Brandao, J; Woods, S; Fowlkes, N; Leissinger, M; Blair, R; Pucheu-Haston, C; Johnson, J; Elster Phillips, C and Tully, T (2014). Disseminated histoplasmosis (*Histoplasma capsulatum*) in a pet rabbit: case report and review of the literature. J. Vet. Diagn. Investig., 26: 158-162.

Bromel, C and Sykes, JE (2005). Histoplasmosis in dogs and cats. Clin. Tech. Small Anim. Pract., 20: 227-232.

Cano, MV and Hajjeh, RA (2001). The epidemiology of histoplasmosis: a review. Semin. Respir. Infect., 16: 109-118.

Chavez-Peon Berle, E; KuKanich, K and Biller, D (2020). Ultrasonographic findings of gastrointestinal histoplasmosis in dogs. Vet. Radiol. Ultrasound., 62: 108-115.

Cridge, H; Brinkman, E and Lathan, P (2019). Biliary dilatation: An unusual presentation of *Histoplasma capsulatum* in a dog. J. Am. Anim. Hosp. Assoc., 56: 285.

De Monbreun, WA (1939). The dog as a natural host for *Histoplasma capsulatum.* Am. J. Trop. Med. Hyg., 19: 565-587.

Fortin, JS; Calcutt, MJ; Nagy, DW and Kuroki, K (2017). Intestinal histoplasmosis in a captive reindeer (*Rangifer tarandus*), Missouri, USA. J. Zoo. Wildl. Med., 48: 925-928.

Guillot, J; Guerun, C and Chermette, R (2018). Histoplasmosis in animals. In: Seyedmousavi, S (Ed.), *Emerging and epizootic fungal infections in animals*. (1st Edn.), Cham, Switzerland, Springer International Publishing AG. PP: 115-128.

Heilmann, RM; Nabity, MB; Bryan, LK; Cook, AK and Scott, K (2021). Mucocutaneous nasal histoplasmosis in an immunocompetent dog. BMC Vet. Res., 17: 1-9.

Jimenez-Rivera, E; Davila-Ruiz, EO; García-Manzano, RA; Santos-Chab, GA; Aguirre-Trejo, JM; Fabián, JF and Tafoya-Ramirez, F (2021). Intestinal histoplasmosis in immunocompetent patient: An unusual cause of massive small intestine bleeding. Rev. Med. Hosp. Gen (Mex), 84: 123-128.

Lamps, LW; Molina, CP; West, AB; Haggitt, RC and Scott, MA (2000). The pathologic spectrum of gastrointestinal and hepatic histoplasmosis. Am. J. Clin. Pathol., 113: 64-72.

Layton, C and Bancroft, JD (2019). Carbohydrates. In: Suvarna, SK; Layton, C and Bancroft, JD (Ed.), *Bancroft's theory and practice of histological yechniques*. (8th Edn.), UK, Elsevier. PP: 181-184.

Mitchell, M and Stark, DR (1980). Disseminated canine histoplasmosis: A clinical survey of 24 cases in Texas. Can. Vet. J., 21: 95-100.

Morris, GB; Ridgway, EJ and Suvarna, SK (2019). Traditional stains and modern techniques for demonstrating microorganisms in histology. In: Suvarna, SK; Layton, C and Bancroft, JD (Eds.), *Bancroft's theory and practice of histological techniques*. (8th Edn.), UK, Elsevier. PP: 266-268.

Norton, SA (1994). Deep fungal skin diseases. In: James, WD (Ed.), *Military dermatology*. *Textbook of military medicine* - Part 3, *Disease and the environment*. (1st Edn.), Washington D.C., Department of the Army. PP: 1-40.

Psarros, G and Kauffman, CA (2007). Colonic histoplasmosis: a difficult diagnostic problem. Gastroenterol. Hepatol., 3: 461-463.

Schlemmer, SN; Fratzke, AP; Gibbons, P; Porter, BF; Mansell, J; Ploeg, RJ; Rodrigues Hoffmann, A; Older, CE and Clark, SD (2019). Histoplasmosis and multicentric lymphoma in a Nubian goat. J. Vet. Diagn. Invest., 31: 770-773.

Schumacher, LL; Love, BC; Ferrell, M; De Silva, U; Fernando, R and Ritchey, JW (2013). Canine intestinal histoplasmosis containing hyphal forms. J. Vet. Diagn. Invest., 25: 304-307.

Sharma, R; Lipi, L; Gajendra, S; Mohapatra, I; Goel, RK; Duggal, R; Mishra, SR and Gautam, D (2017). Gastrointestinal histoplasmosis: a case series. Int. J. Surg. Pathol., 25: 592-598.

Sircar, AD; Tran, MN; Vaidya, SA; Goldstein, EJC and Wheat, LJ (2023). Disseminated histoplasmosis in an immunosuppressed patient successfully treated with isavuconazole. BMJ Case Rep., 0: e253718.

Sumit, A; Kuldeep, AK; Syed Asif, H; Arun, N and Sharmila, D (2022). Disseminated histoplasmosis in HIV patients - Case series from a single tertiary care centre in India. AJIM., 10: 98-102.

Sumithra, TG; Chaturvedi, VK; Rai, AK; Sunita, SC; Siju, SJ; Remya, V and Susan, C (2013). A review and update on canine and feline histoplasmosis. Asian J. Anim. Res., 1: 14-26.

Vijayan, C; Suprakasan, S; Kumar, G; Nair, P and Jayapalan, S (2007). Primary mucocutaneous histoplasmosis presents as oral ulcer. Indian J. Dermatol. Venereol. Leprol., 73: 209-211.