

Pathomorphological Study in Gastrointestinal Tract Disorders of Sheep

Amrender Nath Tiwari, K. K. Jakhar and Vikash Sharma*

Department of Veterinary Pathology, College of Veterinary Sciences

Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar-125 004, India

*Corresponding Author E-mail: sharmavikashjind@gmail.com

Received: 5.03.2018 | Revised: 3.04.2018 | Accepted: 7.04.2018

ABSTRACT

The present study was conducted to elucidate the clinic-pathological aspects of gastrointestinal tract (GIT) disorders in sheep. Study was conducted on 21 (6 healthy sheep and 15 diseased sheep) and 12 carcasses of sheep/lamb brought for postmortem to the Department of Veterinary Pathology, LUVAS, Hisar. This study revealed that incidence of GIT disorders was maximum in sheep of age group 2-6 months and overall incidence was more in females than male sheep/lambs. Major gross pathological changes noticed were congestion in abomasums, catarrhal and hemorrhagic enteritis, congestion in kidney, oedema and enlargement in lymph node, congestion and petechial haemorrhage in spleen and congested heart. In liver, most prominent change was congestion followed by necrotic foci, fibrosis and abscess in liver and in case of lungs, congestion, emphysema and consolidation were seen. Histopathologically, changes in abomasum were oedema, congestion and mononuclear cells infiltration in mucosa and submucosa. In intestine, there was oedema, mild congestion in mucosa and submucosa, haemorrhages in mucosa, desquamation of mucosal epithelium, goblet cell hyperplasia and replacement of glands by mononuclear cells. Liver revealed congestion of blood vessels, degenerative changes in hepatocytes including cloudy swelling, fatty changes, congestion in sinusoids, dilatation of sinusoids leading to atrophy of hepatocytes, leucocytic infiltration in portal triad area and bile duct hyperplasia along with fibroblast proliferation. In heart congestion and oedema were seen in myocardium along with leucocytic infiltration, sarcocystosis in cardiac muscle and hyalinization in myocardium along with mononuclear cell infiltration. In lungs changes including oedema, congestion, emphysema, serous inflammation, thickening of interlobular septa, fibrinous pleuritis and peribronchiolar lymphoid follicle formation were observed and In kidneys there were congestion, focal interstitial nephritis, hyaline degeneration, coagulative necrosis and infiltration of leucocytes predominantly mononuclear cells.

Key words: Pathological studies, Sheep and Gastrointestinal tract disorders.

INTRODUCTION

The domestication of animals was carried out since neolithic times. First goats and sheep,

second cattle and pigs and finally draft animal such as horses and asses were domesticated.

Cite this article: Tiwari, A.N., Jakhar, K.K. and Sharma, V., Pathomorphological Study in Gastrointestinal Tract Disorders of Sheep, *Int. J. Pure App. Biosci.* 6(2): 122-127 (2018). doi: <http://dx.doi.org/10.18782/2320-7051.6384>

Agriculture plays an important role in Indian economy and animal husbandry is an integral part of Indian agriculture. In India, cattle, buffalo, sheep, goat and horses are major domestic animals which are reared for milk, meat and draft purpose. Domesticated sheep belongs to the family bovidae, genus *Ovis* and species *aries*. The sheep are economically important livestock species, contributes greatly to the Indian economy, especially in arid, semi-arid and mountainous regions. These species play an important role in the livelihood of small and marginal farmers and economically weaker sections. In India, the sheep population is 71.558 million DAHD *et al*¹. In India, most of the sheep population is found in Rajasthan, Jammu and Kashmir, Gujarat, Haryana and Punjab. These states contain 1/3rd of total sheep population in India³. Sheep provides wool, leather and edible protein and enormous variety of useful by-products, manure (important source of organic fertilizer) and above all, are source of cash income, in time of need. A number of infectious, nutritional and metabolic disorders lead to heavy mortality in sheep inflicting great economic losses to poor farmers. Among these causes gastrointestinal tract disorders play a vital role^{4,9}, causing high mortality and morbidity affecting the profits in sheep production programmes.

MATERIAL AND METHODS

19 carcasses of goats were received for the postmortem constituted the material of this study. A through post-mortem examination was conducted as early as possible taking usual aseptic precautions to avoid the extraneous contamination. All the tissues/organs of the carcasses were examined critically for the presence of gross pathological alterations and the lesions so detected were recorded. 3-5 mm size of organs showing lesions like abomasum, intestine, mesenteric lymph nodes, liver, spleen, heart, lungs and kidneys were collected in 10 percent buffered formalin for histopathological studies. The fixed tissues were washed in running tap water overnight, dehydrated in acetone, cleared in

benzene and embedded in paraffin wax (melting point 60-62 °C). Paraffin sections were cut at the thickness of 4-5µ and staining is done using Lily Mayer's haematoxylin and 2 percent water soluble eosin⁸.

RESULTS

Gross pathological changes

In abomasum there were congestion (5 cases) and necrotic foci on mucosa (2 cases), in intestine, major changes observed were catarrhal enteritis (5 cases) followed by congestion (4 cases) (Figure 1) and haemorrhagic enteritis (3 cases). Major gross changes in lymph node were enlargement and oedema (2 cases). In liver, most prominent change was congestion (5 cases) followed by necrotic foci (1 case), there was fibrosis of liver (3 cases) and enlargement of gall bladder due to thick bile. Spleen revealed congestion (4 cases) and petechial haemorrhages (2 cases) in parenchyma. In heart, major changes observed were hydropericardium (2 cases) followed by congestion (2 cases) and haemorrhages in endocardium (2 case). In lungs, most prominent change was congestion (5 cases) followed by consolidation (4 cases), and emphysema (2 cases). In kidneys, congestion (4 cases) and soft kidneys (3 cases) were the major changes observed. Pancreas revealed mild congestion (4 cases).

Histopathological changes

In abomasum, oedema, congestion and mononuclear cell infiltration in mucosa (Figure 2) and submucosa with tendency to lymphoid follicle formation were observed. Intestine revealed enteritis (Figure 3) with oedema, mild congestion in mucosa and submucosa and severe infiltration of mononuclear cells in mucosa. Other changes were goblet cell hyperplasia, necrosis and replacement of Lieberkuhan's by mononuclear cells and desquamation of mucosal epithelium. There was also depletion of lymphocytes in Payer's patches in large intestine. Mesenteric lymph nodes when examined

microscopically revealed depletion of lymphocytes in germinal centres of cortex. In the liver, major microscopic lesions were congestion in portal triad area with leucocytic infiltration with mild bile duct hyperplasia. Congestion of central vein with cloudy swelling and fatty changes in hepatocytes were also observed. Focal area of coagulative necrosis in parenchyma in periportal area and various stages of necrosis like pyknosis, karyorhexis and karyolysis were also noticed. Other changes were telangiectasis along with atrophy of hepatic cords. Necrosis in parenchyma and bile duct hyperplasia along with fibroblast proliferation were evidenced. Initiation of lymphoid follicle formation along with degenerative and necrotic changes in parenchyma was seen. Spleen revealed mild to severe depletion of lymphocytes in white pulp area and reticulo-endothelial cell hyperplasia in the around white pulp area. In heart, oedema and congestion were seen in myocardium along with leucocytic infiltration in some cases. There was presence of sarcocyst in myocardium. In lungs, histopathological lesions revealed oedema, congestion, emphysematous alveoli, serous inflammation, thickened interlobular septa due to serofibrinous exudate and infiltration of mononuclear cells in peribronchiolar area and hyperplasia bronchiolar epithelium. Pleuritis along with thickening of pleura and peribronchiolar lymphoid follicle formation along with serous inflammation were observed. In kidneys, there was congestion of intertubular blood vessels and atrophy of Bowman's capsule.

DISCUSSION

In intestine, there was congestion followed by catarrhal and haemorrhagic enteritis. Simultaneous infection of rotavirus and *E. coli* may be the responsible for haemorrhagic enteritis. Rotavirus damages microvilli of small intestine which causes reduced starch digestion, results in promotion of excess bacterial (*E. coli*) growth simultaneously. It

induces an osmotic effect resulting in diarrhoea. In lambs, rotavirus enteritis is more severe when complicated by simultaneous infection with enterotoxigenic *E. coli*¹⁴. Congestion in abomasums, oedema and enlargement in lymph node were observed. In liver, most prominent change was congestion followed by necrotic foci, there was fibrosis of liver enlargement of gall bladder due to thick bile and abscess in liver. Spleen revealed congestion and petechial haemorrhages as the major change. These findings are agreement with Saleim *et al*¹³. In lungs, most prominent change was congestion followed by emphysema and consolidation. In heart, major changes observed were hydropericardium followed by congestion and in few cases mild haemorrhages were also revealed. Hydropericardium and ascites observed in sheep and goats can be correlated with heavy infection of blood sucking stomach worms (strongyles) resulting in excessive loss of serum albumin decreasing the osmotic pressure of plasma which forces fluid to move out of blood to tissues and accumulation of fluid in pericardial sac resulting in hydropericardium⁶. Kidneys revealed congestion as the major change. However, soft kidneys were revealed in sheep whereas in goats a case of subcapsular abscess was evidenced. Soft kidneys may be correlated with overeating of pasture results in accumulation of undigested starch which may enhance proliferation of *Clostridium perfringens* type D bacteria in rapidly growing lambs. This bacteria grows in feed in upper intestine secrete two toxins that are absorbed by the gut, alpha toxin damages gut lining and epsilon toxin absorbed by body and causes damage to brain, heart, kidneys and other tissues (www.fwi.co.uk/Articles/2005/.../Sudden-death-in-lambs). However in the present study no attempts were made to isolate anaerobic bacteria. Further studies are required to corroborate these findings.

Histopathological lesions in various visceral organs were in general, similar in both species. Major histopathological changes observed in abomasum were oedema,

congestion and leucocytic infiltration in mucosa and submucosa with tendency to lymphoid follicle formation. These findings are in agreement with earlier report of Hajimohammadi *et al*⁵. In intestine, major microscopic lesions were oedema, mild congestion in mucosa and submucosa and severe infiltration of mononuclear cells in mucosa. Other changes were goblet cell hyperplasia, necrosis and replacement of glands of Lieberkuhn's by mononuclear cells and desquamation of mucosal epithelium. These findings were in general, agreement with those reported by Panisup *et al*.¹⁰, Valente *et al*.¹⁷, Saleim *et al*.¹³ and Tariq *et al*.¹⁵. However, one confirmed case of Johne's disease among goats was reported which was characterized by degeneration of epithelial cells of intestine, degeneration of intestinal glands and infiltration of mononuclear cells like lymphocytes, macrophages, epitheloid cells and Langhan's type giant cells in mucosa¹¹. Mesenteric lymph nodes when examined microscopically revealed congestion in cortex and depletion of lymphocytes in germinal center of lymphoid follicle in cortex of lymph gland¹⁰. However, in one case of goat, impression smear of mesenteric lymph node revealed acid fast bacilli in foamy macrophages and histopathologically it also revealed giant cells¹¹. Histopathologically, liver revealed congestion of blood vessels, degenerative changes in hepatocytes including cloudy swelling, fatty changes, congestion in sinusoids, dilatation of sinusoids leading to atrophy of hepatocytes. There were focal areas of coagulative necrosis in parenchyma along with leucocytic infiltration in portal triad area

and initiation of lymphoid follicle formation in parenchyma and bile duct hyperplasia along with fibroblast proliferation which were similar to finding of Saleim *et al*.¹³ and Kumar *et al*.⁶. Lesions in heart included oedema and congestion in myocardium along with leucocytic infiltration and sarcocystosis. Similar lesions have been evidenced by Deepti *et al*.². In lungs, changes including oedema, congestion, emphysema, serous inflammation and thickening of interlobular septa were observed. Similar lesions have been reported by Panisup *et al*.¹⁰, Saleim *et al*.¹³ and Uzal *et al*.¹⁶. In kidneys, lesions were congestion, focal interstitial nephritis, hyaline degeneration, coagulative necrosis and infiltration of leucocytes predominantly mononuclear cells. Similar lesion had been evidenced by Saleim *et al*.¹³. Average gross lesion score of sheep was maximum for digestive system followed by respiratory system, kidney, heart and spleen.

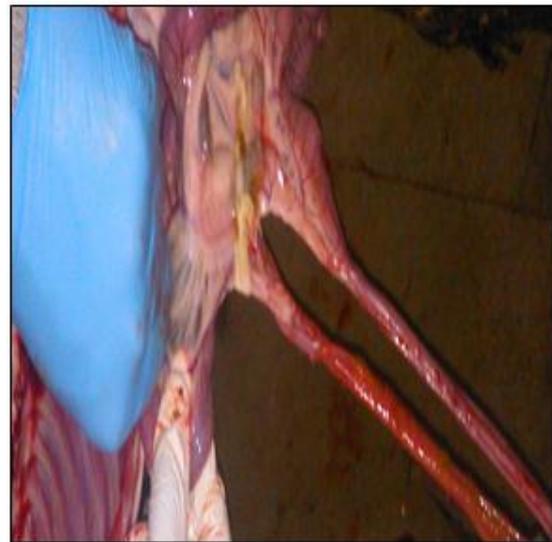


Figure 1: Intestine of a sheep showing congestion

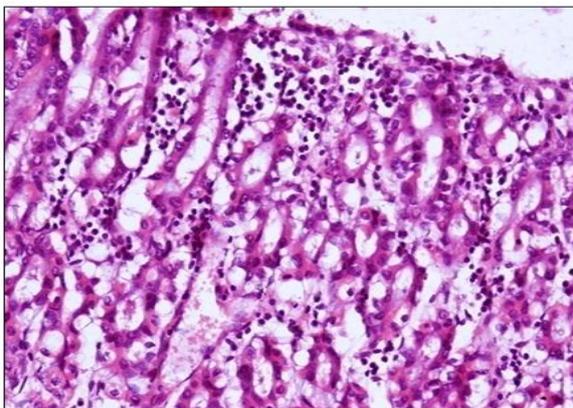


Figure 2 : Abomasitis showing infiltration of mononuclear cells in mucosa. H.&E. $\times 400$

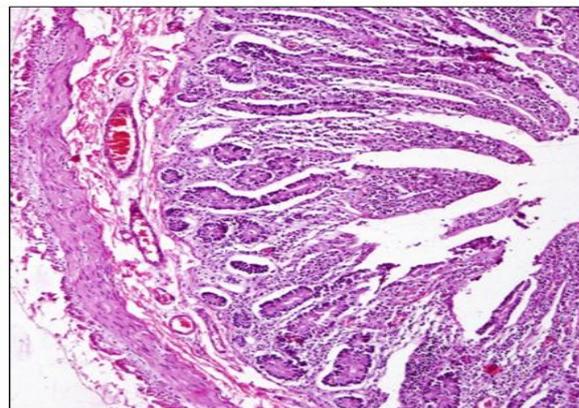


Figure 3: Intestine of a sheep: Enteritis showing congestion in submucosa and mononuclear cell infiltration in mucosa. H. & E. $\times 100$

CONCLUSIONS

GIT disorders in sheep causes pathomorphological effects in digestive system including abomasum, intestine, mesenteric lymph node and liver followed by lungs, kidneys, heart and spleen. Histopathological lesions included vascular changes, degenerative changes, growth changes, necrotic changes, inflammatory reaction along with infiltration of cells, growth disturbances, proliferative changes and lymphoid follicle formation in various organs.

Acknowledgments

The authors are highly thankful to Dean, College of Veterinary Science and Animal Husbandry, LLR University of Veterinary and Animal Sciences, Hisar, India, for providing necessary funds and facilities to carry out the investigation.

REFERENCES

1. DAHD, Department of Animal Husbandry, Dairing and Fishries. Annual report 2012-**13(3)**: 101-102 (2013).
2. Deepti, C., Katoch, R.C. Jaisal, S. and Mahajan, A., Enteropathogenicity of *Escherichia coli*. Strains isolated from diarrhoeic ruminants in Himachal Pradesh. *Indian J. Anim. Sci.* **69(7)**: 469-471 (1999).
3. Food and Agriculture Organization of The United Nations, www.faostat.fao.org (2010).
4. Green, L.E., Wyatt, J.M. and Morgan, K.L., Terminal ileitis in lambs. *Vet. Rec.* **127 (5)**: 119 (1990).
5. Hajimohammadi, A., Badiei, K., Mostaghni, K. and Pourjafar, M., Serum pepsinogen level and abomasal ulcerations in experimental abomasal displacement in sheep. *Veterinarni Medicina*, **55 (7)**: 311–317 (2010).
6. Kumar, S., Jakhar, K.K., Mishra S.K. and Purohit R., Pathology of gastrointestinal tract disorders in sheep. Paper abstracted at XXVIII Annual Conference of Indian Association of Veterinary Pathologists from 29th and 30th December at Department of Veterinary Pathology, Madras Veterinary College, Chennai. 63 (2011).
7. Kumar, S., Jakhar, K.K., Mishra S.K. and Purohit R., Pathology of gastrointestinal tract disorders in sheep. Paper abstracted at XXVIII Annual Conference of Indian Association of Veterinary Pathologists from 29th and 30th December at Department of Veterinary Pathology, Madras Veterinary College, Chennai. 63 (2011).
8. Luna, L.G., Manual of histologic staining methods of the Armed Forces Institute of Pathology. 3rd edn. Mc Graw Hill Book Company, New York (1968).
9. Paliwal, O.P., Krishna, L. and Kulshrestha, S.B., Enterotoxaemic jaundice in sheep. *Indian J. Anim. Sci.* **45(9)**: 656-659 (1975).
10. Panisup, A.S., Lamb mortality studies on the pathology and possible etiology, M.V.Sc. Thesis, CCS Haryana Agricultural University, Hisar, Haryana (India) (1974).
11. Reddy, G.B., Manjunath, G.B. and Shivasharanappa, N., Paratuberculosis is a chronic granulomatous disease caused by an acid fast *Mycobacterium avium subsp. paratuberculosis*. *Indian J. Vet. Pathology.* **36(2)**: 221- 223 (2012).
12. Saleim, R.S., Wafaa, A.F.E. and Nareman A., R. Bacteriological and immunological studies on *Escherichia coli* isolates recovered from diarrhoeic and contact apparently healthy sheep with histopathological changes encountered. *Vet. Med, J. Giza.* **52(2)**: 245-248 (2004).
13. Saleim, R.S., Wafaa, A.F.E. and Nareman A., R., Bacteriological and immunological studies on *Escherichia coli* isolates recovered from diarrhoeic and contact apparently healthy sheep with histopathological changes encountered. *Vet. Med, J. Giza.* **52(2)**: 245-248 (2004).
14. Snodgrass, D. R., Herring, A. J. and Campbell, I., Comparison of atypical rotavirus from calves, piglets, lambs and man. *J. Gen. Viro.* **65**: 909-914 (1984).

15. Tariq, K.A., Chishti, M.Z., Ahmad, F. and Shawl A., S., Epidemiology of gastrointestinal nematodes of sheep managed under traditional husbandry system in Kashmir valley. *Veterinary Parasitology*. **158(1-2)**: 138-143 (2008).
16. Uzal, F.A., Kelly, W.R., Morris, W.E., Bermudez, J. and Baison, M., The pathology of peracute experimental *Clostridium perfringens* type D enterotoxemia in sheep. *Journal of Veterinary Diagnostic Investigation*. **16(5)**: 403-411 (2004).
17. Valente, C., Kashari, Q., Furganti, G., Cardaras, P., Ciorba, A., Proccichiani, I. and Cenci, T., Diarrhoea in lambs: experimental infection with enterotoxigenic *Escherichia coli*. *Clinica Veterinaria*. **108(4)**: 278-285 (1985).