World Applied Sciences Journal 7 (1): 19-23, 2009
ISSN 1818-4952

Outbreak of Maedi-Visna in Sheep and Goats in Palestine

[1]W. Hananeh and [2]S. Barhoom

[1]Department of Pathology and Animal Health,
Pathology Laboratory, Faculty of Veterinary Medicine,
Jordan University of Science and Technology, Irbid 22110, P.O. Box 3030, Jordan
[2]Clinical Studies, Faculty of Veterinary Medicine, Nablus-Palestine P.O. Box 7

Abstract: Clinical and laboratory studies in an outbreak of Maedi-visna disease in a flock of sheep and goats were conducted. The clinical findings of the disease were loss of wool, dyspnea and coughing, progressive condition loss, exercise intolerance and chronic mastitis. Nervous signs were developed later and were characterized by gait abnormalities of the hind limbs, ataxia, tremor of facial muscles and recumbency. A definitive diagnosis was made by characteristic clinical history, gross and histopathological lesions and detection of seropositive animals by ELISA.

Key words: Outbreak % Small ruminants % Maedi-visna % Palestine

INTRODUCTION

Maedi-visna (MV) or ovine progressive pneumonia (OPP) is a widespread viral disease belongs to small ruminant lentivirus (SRLV) which is a member of the genus lentivirus of the family retroviridae. This virus causes chronic encephalitis, slowly progressive pneumonia, arthritis and mastitis [1]. The name of the disease is derived from two Icelandic words; maedi ("dyspnea") for the progressive pneumonia and visna ("wasting") for the encephalitis [2]. Visna was firstly described and reported in Iceland in 1935 [3], while maedi was reported later in 1939 and was caused by the same virus that causes visna [4]. Although, Maedi visna virus (MVV) infects sheep mainly, goats also can be infected with the same virus [5-8]. In addition goats have their own virus that is closely related MVV and termed caprine arthritis encephalitis virus (CAEV). Phylogenetic analyses comparing nucleotide sequences of MVV and CAEV showed clear indications of the existence and epidemological importance of cross-species transmission between sheep and goats without demonstrating clearly that one virus had emerged from the other [9].

The disease have a long incubation period of over than 2-years. It has a protracted clinical course, lasting 6 months to several years, unless terminated by intercurrent disease [10]. The disease is transmitted by ingestion of infected colostrums, milk and aerosol route between animals of all ages in close contact [11]. A definitive diagnosis of disease is made at necropsy when characteristic lesions are found grossly and histopathologically in conjunction with a supportive clinical history of disease and seropositive test results [12]. This study deals for the first time with an outbreak of MV in a flock of sheep (East-Friesian Breed) and goats (Saanen breed) recently encountered in Palestine where clinical, pathological and serological studies were conducted.

MATERIALS AND METHODS

An outbreak of Maedi-Visna occurred in a private farm of East-Friesian sheep and Saanen goats. The disease appeared in Tulkarem Governorate, North Palestine. Complete clinical examination was performed on the affected animals in December 2005 and one recumbent 3 year old ewe was euthanized and subjected to thorough post-mortem examination. Specimens from the medulla oblongata, anterior spinal cord, midbrain and affected lungs were collected and fixed in 10% neutral buffered formalin for routine histopathological examination according to Gamble and Bancroft [13].

Corresponding Author: W. Hananeh, Department of Pathology and Animal Health,
Pathology Laboratory, Faculty of Veterinary Medicine,
Jordan University of Science and Technology, Irbid 22110, P.O. Box 3030, Jordan

The farm had 200 animals; 60 goats and 140 sheep. Ages of animals varied from 2 to 5 years old. Thirty animals; 10 goats and 20 sheep showed one or more of the following clinical signs; loss of wool, dry cough, labored breathing, dyspnea, nasal discharges, loss of condition despite the good appetite and chronic mastitis. those animals were selected for serological examinations. The rest of the flock was clinically sound.

Serological Tests: The clinically affected thirty animals, 20 sheep and 10 goats including the recumbent ewe from the flock were bled and sera separated and investigated at Kimron Veterinary Institute (KVI), Bet Dagon-the reference laboratory for Palestine Ministry of Agriculture-by Enzyme Linked Immunosorbent assay (ELISA) using a commercial whole virus antigen.

RESULTS

Both sheep and goats were stabled in one pen. All animals were treated with broad spectrum antiparasitic drug ivermectin and vaccinated annually against the following enzootic infectious diseases: sheep pox, peste des petits ruminants and foot and mouth disease. The clinical signs of the disease started in a 3 year old ewe that developed respiratory distress and dyspnea during pregnancy. After parturition, there was a loss in condition and ataxia that started with the hind limbs and progressed to recumbency. There were tremors of facial muscles and generalized trembling as well. The other clinically affected animals showed the following clinical signs: loss of wool, dry cough, labored breathing, dyspnea, nasal discharges, loss of condition despite the good appetite and chronic mastitis.

Gross Pathology: Lungs were markedly expanded, firmer than normal on palpation and failed to collapse. Approximately 1/3rd of the anteroventral lung lobes were consolidated. Rib impressions were prominent on both lungs. The udder was markedly firm and enlarged. No other significant gross lesions were present.

Histopathlogy: Sections from both lungs exhibit similar changes. The alveolar spaces were markedly flooded with eosinophilic proteinaceous material. Diffusely the alveolar walls were extensively thickened with variable numbers of lymphocytes, plasma cells and fibroblasts. Multifocal areas of type II pneumocyte hyperplasia were prominent throughout the lung sections and multiple variably sized lymphocytic nodules were scattered throughout the pulmonary parenchyma primarily surrounding the airways and blood vessels (Figure 1).

Smooth muscles of the alveolar ducts and terminal bronchioles were moderately hyperplastic. Some sections showed large numbers of neutrophils infiltrating the alveolar spaces as well as the air ways accompanied with variable degrees of necrosis as seen in The brain lesions consisted of severe mononuclear cell infiltrates within the meninges and white matter of the cerebrum. Multifocal areas of variable degrees of demyelinations, prominent vasculature and gliosis were present primarily in the white matter as seen in Figure 2.

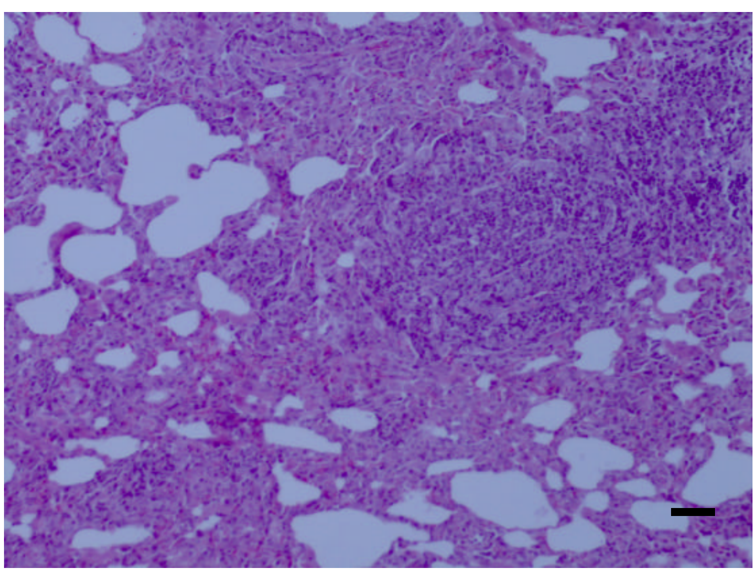

Fig. 1: Lung of sheep showed severe thickening of interstitium and prominent lymphocytic nodule within the pulmonary parenchyma. HE. Bar = 50 9 m.

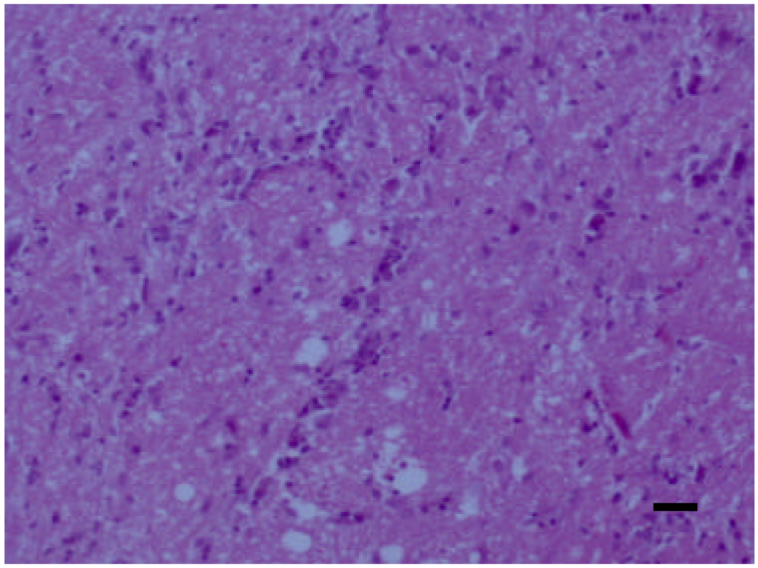

Fig. 2: Brain (cerebrum) of sheep with prominent demylination, vasculature and moderate mononuclear cell infiltrations. HE. Bar = 50 9 m.

Serological Tests: The results of sera investigation at KVI showed 10 (50%) sheep and 5(50%) goats were positive for presence of MVV antibodies out of examined 20 (50%) sheep and 10 goats, respectively. The slaughtered recumbent ewe showed positive result for the presence of MVV antibodies.

RESULTS AND DISCUSSION

Maedi-visna of sheep and caprine arthritis/ encephalitis (CAE) of goats are persistent slowly progressive viral infections caused by closely related lentivirus of retroviridae family in most of sheep-rearing countries of the world, with the notable exception of Australia and New Zealand [14]. Both viruses belong to SRLV. Several reports stated that MVV could transmit from sheep and infect goats under field conditions [5-8]. In this report 5 goats were serologically positive against MMV that give a good evidence that MVV could transmit naturally and infect goats. However, we could not state for sure that the clinical problems that appeared in the affected goats was due to MVV since demonstration of the virus within the clinically affected goats organs was not carried out but it is highly likely that the positive serological results was due to MMV infection in goats as well as in sheep. Because MV is characterized by long incubation period and clinical disease, the clinical MV cases occurs in adult animals however, clinical cases of nervous form of MV had been reported in 4 and 6 month old lambs. Usually, Maedi visna in sheep is manifested in two different chronic diseases; Maedi (respiratory form) and Visna (nervous form). However, occasionally both forms may coexist, as it is encountered in this study [15]. The encountered clinical findings in sheep and goats were characterized by coughing, dyspnea, loss of conditions that progressed to emaciation after lambing/kidding. Progressive ataxia was developed, started with hind limbs. Tremors of facial muscles and trembling were evident. All of the previously mentioned findings were in accordance with those previously reported [2]. The disease characterized by multisystemic infection affects mainly the lungs and central nervous system and less frequently mammary glands and joints. In addition, MVV was detected in different body organs and tissues including liver, brain [16] Kidneys [17], Ovine third eyelid [18] and Ovine ovaries [19]. Transmission is mainly via the ingestion of infected colostrums and milk or through respiratory contact, following infection, animals produce anti-viral antibodies, though seroconversion may occur any time from a few weeks to several months. The main cells infected in vivo are monocytes and macrophages, with the bone marrow serving as a reservoir of infection and the virus is disseminated to the target tissues via the blood stream inside monocytese [20].

The most convenient way to diagnose MV infection is to perform serology and the most widely used diagnostic test for detection antibodies is Enzyme linked Immunosorbent Assays (ELISAs) which recommended by OIE [12] because it is suitable for screening large numbers of samples and is more sensitive than agar gel immunodiffusion test (AGID). Also it is reported that ELISA is generally more sensitive technique than PCR due to the low viral load in the postseroconversion phase of infection [21]. In addition, Some infected animals may not be detected by PCR due to primer mispairing to target DNA sequences [22]. The economical losses from this outbreak were marked, since 15 out of 200 animals showed clinical signs and had needed to be culled. The extent of the infection within the rest of the farm could not be known at this time although the untested animals were apparently healthy. Control programs by testing of the animals and separation of the seropositve animals from the seronegative ones were advised. Otter and Boundy [23] reported the successful containment of an infected cohort of purchased sheep using enforced biosecurity practices. Examination of the rest of the animals was hindered because the owner of the farm had refused to check the serological status of the rest of the animals because of financial problems. A definitive diagnosis of disease was made clinically and pathologically where characteristic lesions are found in conjunction with a supportive history of the disease and seropositive test results.

REFERENCES

1. Narayan, O. and J.E. Clements, 1989. Biology and pathogenesisof lentiviruses. J. Gen. Viro., 70: 1617-1639.
2. Marten, W.B. and I.D. Aitken, 2000. Diseases of sheep, third edition, Blackwell Science, Oxford, pp: 187-191.
3. Jones, T.C., R.D. Hunt and N.W. King, 1997. Veterinary pathology. sixth edition, Lippincott Williams and Wilkins,Baltimore, USA, pp: 331-334.
4. Gudnadottir, M., 1974. Visna-maedi in sheep. prog Med Virol., 18: 336-349.
5. Lamontagne, L., R. Roy, A. Girard and B.S. Samagh, 1983. seroepidemiological survey of maedi-visna virus infection in sheep and goat flocks in Quebec. Can J. Comp Med. 1983 July; 47(3): 309-315).
6. Pisoni, G., G. Bertoni, M. Puricelli, M. Maccalli and P. Moroni, 2007. Demonstration of coinfection with and recombination by caprine arthritis-encephalitis virus and maedi-visna virus in naturally infected goats. J. Virol. 2007 May, 81(10): 4948-55.
7. Shah, C., J.B. Huder, J. Böni, M. Schönmann, J. Mühlherr, H. Lutz and J. Schüpbach, 2004. Direct evidence for natural transmission of small-ruminant lentiviruses of subtype A4 from goats to sheep and vice versa. J. Virol., 78(14): 7518-22.
8. Gjerset, B., C.M. Jonassen and E. Rimstad, 2007. Natural transmission and comparative analysis of small ruminant lentiviruses in the Norwegian sheep and goat populations. Virus Res., 125(2): 153-61.
9. Roland, M., J. Mooney, S. Valas, G.F. Perrin and R.Z. Mamoun, 2002. Characterization of an Irish caprine lentivirus strain-SRLV phylogeny revisited. Virus Res., (85): 29-39.
10. Murphy, F.A., E.P. Gibbs, M.C. Hozinek and M.J. Studdert, 2003. Veterinary Virology, third edition, Academic press, New York, pp: 383-386.
11. Peterhans, E., T. Greenland, J. Badiola, G. Harkiss, G. Bertoni, B. Amorena, M. Eliaszewicz, R.A. Juste, R. Krassnig, J.P. Lafont, P. Lenihan, G. Pétursson, G. Pritchard, J. Thorley, C. Vitu, J.F. Mornex and M. Pépin, 2004. Routs of transmission and consequence of small ruminant lentivirus (SRLVs) infection and eradication schemes. Vet. Res., pp: 257-274.
12. World Organization for Animal Health [OIE], Manual of diagnostic tests and vaccines [online]. Paris: OIE; 2004. Caprine arthritis/encephalitis and maedi-visna. Available at: http: // www. oie. int/ eng/ normes/ mmanual/ A_00071.htm.
13. Gamble, M. and J.D. Bancroft, 2001. Theory and practice of histological techniques, fifth edition, Churchill Livingstone. Luna L.G. Manual of histologic staining methods of the Armed Force Institute of pathology. 3rd ed. Newyork, M.C Grow Hill Book company.
14. Geering, W.A., A.J. Forman and M.J. Nunn, 1995. Maedi-visna in exotic disease of animals Aust. Gov. publishing service, Canberra, pp: 163-168.
15. Watt, N.J., D.J. Roy, I. McConnell and T.J. King, 1990. A case of visna in the United Kingdom. Vet Rec (126): 600-601.
16. Brello, G.D., K. Angelopoulou, T. Poutahidis and I. Vlemmas, 2007. Detection of maedi-visna virus in the liver and heart of naturally infected sheep. J. Comp. pathol., 136(1): 27-35.
17. Angelopoulou, K., G.D. Brellou and I. Vlemmas, 2006. Detection of maedi-visna virus in the kidneys of naturally infected sheep. J. Comp. Pathol., 134 (4): 329-35.
18. Capucchio, M.T., E. Sanna, M.P. Sanna, S. Farigu, R. Minelli and F. Guarda, 2003. Maedi-visna virus detection in ovine third eyelids. J. Comp. Pathol., 129(1): 37-43.

19. Cortez Romero, C., F. Fieni, C. Roux, P. Russo, J.M. Guilbert, F. Guiguen, Y. Chebloune, M. Pepin, J.L. Pellerin, 2006. Detection of ovine lentivirus in the cumulus cells, but not in the oocytes or follicular fluid, of naturally infected sheep. Theriogenol., 66(5): 1131-9.
20. Clements, J.E. and M.C. Zink, 1996. Molecular biology and Pathogenesis of animal lentivirus infections. Clin. Microbiol. Rev., 9(1): 100-117.
21. de Andrés, D., D. Klein, N.J. Watt, E. Berriatua, S. Torsteinsdottir, B.A. Blacklaws and G.D. Harkiss, 2005. Diagnostic tests for small ruminant lentiviruses. Vet. Microbiol., 7(1-2): 49-62.
22. Karanikolaou, K., K. Angelopoulou, M. Papanastasopoulou, M. Koumpati-Artopiou, O. Papadopoulos and G. Koptopoulos, 2005. Detection of small ruminant lentiviruses by PCR and serology tests in field samples of animals from Greece Small Ruminant Res., 58: 181-187.
23. Otter, A. and T. Boundy, 2005. Establishment of a maedi-visna-free flock after the purchase of infected sheep. Vet Rec., 157(10): 282-4.