

RESEARCH PAPER

DPEN ACCESS

A survey on ovine pulmonary adenomatosis in Khuzestan

province, Iran

Ehsan Gharib Mombeni¹, Zeinab Chanani¹, Majede Beladi Mousavi¹, Esmaeil Abdi^{1,} Manoochehr Gharib Mombeini², Nahid Soltani Sedeh³

¹Student of veterinary medicine, Shahid Chamran Veterinary College of Ahwaz, Iran

²Provincial Head of Animal Disease Control and Monitoring, Veterinary Organization, Iran

^sLaboratory Expert, Physics Department, Jondishapour University, Ahvaz, Iran

Received: 29 October 2012 Revised: 08 November 2012 Accepted: 08 November 2012

Key words: Adenomatosis, lung, sheep, histopathology, Khuzestan province.

Abstract

An outbreak of ovine pulmonary adenomatosis (OPA) occurred in sheep herd of rural epidemiological unit of Ahwaz region in Khuzestan province. In the contaminated farm, seven animals with progressive respiratory disease and suffered with pulmonary lesions of OPA. In all slaughtered cases, the histology of the lungs was characterized by multiple foci proliferations of cuboideal cells in the bronchiole-alveolar epithelium. In all of them there was excess fluid in the polmunary tracts. The existence of OPA approved by histhopathological findings in lungs and field trial of positive wheel barrow test. This study may helps to identifying regional distribution of OPA in iran, when other regional studies were done.

*Corresponding Author: Ehsan Gharib Mombeni 🖂 arlotfi@gmail.com

Introduction

Ovine pulmonary adenomatosis (OPA) was recorded and described in South Africa as Jaagsiekte for first time, since then it has been reported in most European countries, Asian and American continents, where sheep raising industry is prominent (Toumazos, 1989; Pugh, 2002). In present study, the ovine pulmonary adenomatosis is referred to as OPA to avoid confusion with conditions in other species which are also called pulmonary adenomatosis. The etiological agent of the disease is oncogenic type D-related retrovirus designated as jaagsiekte sheep retrovirus (JSRV) (Al-Hizab and Abdelsalam, 2005). Sheep pulmonary adenomatosis is primarily a disease of mature (3-4 yeas old) sheep and rarely in goats (Pugh, 2002). although lesions have been observed in lambs as young as five months of age (Martin et al.,1979). OPA has been recorded in many types, but some considered specific breeds or species within breeds to be more susceptible, Males appear to be more vulnerable than females, but this may be reffered to management practices (Drury and Wallington, 1980; Pugh, 2002). The mortality rate varies considerably depending on a nomerous factors, e.g. management practices; breed susceptibility, nutritional condition, treatment regimen, concomitant disease (Drury and Wallington, 1980; Pugh, 2002). Mortality rates of 20% -50% were recorded on a few farms in Iceland soon after the disease was introduced to that country (Stevenson et al., 1982). Transmission can incidence by close contacts with infected nasal discharge. The affected sheep have continous weight loses, although there is no change in feed intake (Pugh, 2002). Sign of pyrexia is absent in uncomplicated cases so body temperature is normal. The respiratory rate and the intensity of lung sounds changes increase due to severity of the disease progresses. In some cases lung sounds heard suggestive of slowly boiling porridge (Pugh, 2002).The sheep pulmonary adenomatosis has never been differentiated from other respiratory diseases in Khuzestan province. This survey shows that the native breeds of sheep in

this region (Khuzestan province) are susceptible to OPA.

Materials and methods

This study was conducted on a sheep farm located near the Ahwaz, Khuzestan province. The flock, consisting of 100 sheep of local native breed, were kept in a shed. animals were fed a mixture of alfalfa hay, oats and corn in troughs early in the morning and again late in the afternoon. one hundred sheep, from 3 month to 7 years of age were examined clinically, by auscultation of thorax and the wheelbarrow test (Pugh, 2002). seven sheep with respiratory disorder were slaughtered for postmortem examination. Samples of the lung lobes, including all areas with gross changes and a randomly selected sample from each lobe when gross changes were not evident, the mediastinal and bronchial lymph nodes were collected from all the animals and fixed due to 24 h immersion in 10% buffered formalin (pH 7.2). The tissues were embedded in paraffin wax and staining with Haematoxylin and Eosin (H&E) and Giemsa as described by Drury and Wallington (Drury and Wallington, 1980). Pieces of affected lung and lymph nodes from sheep were taken for examinations of cultures using blood, MacConkey, brilliant green and apouro-Maltose agar plates. Also it had examined for the existence of Pasteurella spp. according of Cruickshank R, Medical Microbiology, that the examination for pasteurella is negative (Cruickshank et al., 1975).

Results

Clinical signs

seven animal that were clinically examined showed fever, anorexia, persistent respiratory distress that accompanied by intermittent cough, tachypnea and occasionally crackles, wheezes and evidence of boiling porridge plus copious nasal serous discharge with positive wheel-barrow test (the suitable test for checking the extra fluid in lung and respiratory tracts²) were seen (Figure 1, 2). Discharged fluid is depend on the severity of the respiratory complication that max reached to 120 cc. In this

Int. J. Biosci.

flock, mortality did not occur and the affected sheep have progressive emaciation.

Gross pathology

Post-mortem examination of all seven sheep revealed lot amounts of straw-coloured fluid within the thoracic and pericardial cavities within the trachea and major bronchi [Figure 3, 4]. Pleural and pericardial surfaces were considerably thick and covered with gelatinous fibrinous. The affected lungs were bigger and heavier in size about three times than a normal lung of sheep at the same age. In normal lung the weight of lower apical lobs are 300 grams however, in the affected sheep with OPA lung was 1200 grams. The regional lymph nodes were very enlarged, congested, oedematous and occasionally haemorrhagic, the size of them were bigger than four times of normal lymph nodes. The whole lungs were firm in consistency and contained scattered areas with coagulate necrosis and gray foci were seen, too. its cut surface was extremely pale and had a granular texture and there was not appreciable amount of fluid upon press.

Histopathology

The microscopic features of all slaughtered cases were basically similar and will be discussed together. The affected alveoli were lined with proliferating cuboidal cells forming irregular folds and papillary projections (figure 6). These were arranged in variably protruding papillomatous structures that extended into the lumen of the alveoli. In addition, variable numbers of closely packed foamy macrophages were present inside the unaffected alveoli (Figure 5). Some of the bronchi and bronchioles were also affected but their overall response was dominated by focal hypertrophy and hyperplasia of the lining epithelium. Some of the alveoli had resembled to glandular ascini (Figure 6, 7), hyperplasia of Clara cells not has seen many. In cases active inflammatory responses with predominating neutrophilic infiltrations in the normal and affected alveoli were seen (figure 8). Infiltration of neutrophils is suggestive signs of mixed inflammation with bacteria. The chronic

inflammatory reactions dominated by diffuse fibroplasia with scattered myxomatous changes and mononuclear cellular infiltration in the interstitial tissue (figure8). The big lymph nodes showed no adenomatous lesions, occasionally small clusters or a single clump of adenomatous cells were seen in blood vessels of lymph nodes.



Fig.1. wheel-barrow test.



Fig. 2. the frothy & serous fluid transudate that dischargesFrom nostrils of sheep in wheel-barrow's test.



Fig. 3. There are several distinct lesions with small grayish foci radiating into the normal .parenchyma in one lesion the central part has collapsed giving it the appearance of a volcano.



Fig. 4. Frothy pure liquid in respiratory



Fig. 5. Surrounding alveoli containnumerous macrophages H. & E. X200.



Fig. 6. Typical columnar epithelial cells lining affected alveoli. Surrounding alveoli contain numerous macrophages and the alveoliare lined with cuboidal cells forming papillary projections(H& E. X200).



Fig. 7. hypertrophy and hyperplasia of the bronchial epithelium.(H & E. X 200).



Fig. 8. Acute purulent bronchopneumonia superimposedon an existing adenomatous lesion near a bronchiole,numeroussneutrophiles(H & E. X 40).

Distinctive histopathological lesions typical of OPA were found in the lungs of all sheep (Sharp and Angus, 1990; DelasHeras et al., 2003). This is a clinical and histopathological description of OPA in These histopathological Ahwaz. signs were characterized by cuboideal transformation and papillomatous proliferation of the alveolar epithelium and positive wheel-barrow test and the boiling porridge in such a manner typically resembling those observed in OPA (Al-Hizab and Abdelsalam, 2005). OPA is of biomedical importance and is a substantial economic problem to sheep farmer's world wide (DelasHeras et al., 2003). These animals originated from a farm near the city of Ahwaz in Iran. It is likely that OPA was endemic on the study farm, although the source of infection remains unknown. It is surprising that, no cases of OPA had been detected on the farm previously. However, no reliable tests are available to detect OPA in live animals (Pugh, 2002), and the animals that not responded to cure on the farm had not been regularly submitted for post-mortem examination until few months prior that we detect the first case of OPA in that farm, it is therefore probable that, the disease had been overlooked in all over the Khuzestan because sick animals had been culled without any further investigation. We believe that the spread of OPA into the farms all over the state is very much than we expect. We suggest to study on the slaughterhouses prevalence and control of OPA in this region. Also OPA is a

Int. J. Biosci.

specific disease of sheep , but it is not familiar to most Iranian veterinarians and usually under diagnosed, so with more epidemiological studies can show the prevalence and importance of the disease in Khouzestan province. OPA caused important economic losses in affected farm, which demonstrated serious economic impact on sheep farms. The provision of OPA control programs in the region would seem a prerequisite for improving sheep meat and milk production. In the present study small clusters of adenomatous cells to mediastinal lymph nodes vessels were observed and we have not seen any metastases of OPA cells. OPA has been previously recognized in a number of Mediterranean countries other than Iran (Kojouri and Karimi, 2002) for example, the disease was reported in Libya (Ali and Abdelsalam, 1999), Spain (Dualde-prepers, 1963), Cyprus (Toumazos, 1989) and Tunisia (Boutouria and Soussi, 1987). Overall incidence of OPA in Iran does not reflect the real epidemiological status of the disease in whole country. It is quite possible that some genuine cases of OPA in the remote parts of the country might have not been properly diagnosed by local veterinarians due to the considerable lack of adequate laboratory facilities in these areas. In most instances, the veterinary practitioner or pathologist is encountered with an affected lung which also has either acute fibrinous pneumonia or abscesses. When this occurs, the only definitive way to diagnose OPA is the demonstration of characteristic lesions from a variety of interstitial pneumonia by histologically examination (Sharp and Angus, 1990). Further studies are therefore required in order to outline the extent and regional distribution of the disease throughout the country for establishing future control measures.

References

Al-Hizab FA, Abdelsalam EB. 2005. Adenomatous Lesion in a pneumonic lung of a local indigenous Saudi goat. Pakistan Journal of Biological Sciences **8**, 542-545. Ali OA, Abdelsalam EB. 1999. Sheep pulmonary adenomatosis (Jaagsiekte) in Libya: gross and histopathological evidence. Maladies Des Animaux **3-4**, 181-183.

Boutouria M, Soussi H. 1987. Pulmonary adenomatosisofsheep in Tunisia. Maghreb Veterinary **3**, 29-32.

Cruickshank R, Duguid JP, Marmion BP,Swain RH. 1975. 12th ed; Medicalmicrobiology Vol.11 Churchill Livingstone, Edinburgh, London and NewYork.

DelasHeras M, Gonzalez L, Sharp JM. 2003. Pathology of ovine pulmonary adenomatosis.Currenut Topics in Microbioliology and Immunology 257, 25-54.

Drury RAB, Wallington EA. 1980. Carleton's Histological Technique. 5thed; Oxford University press, London.

Dualde-prepers C. 1963. Studies on ovine pulmonary adenomatosisin Spain. Proc. 17th World Veterinary Congress, Hannover, Germany, 14-21 August 1963. **1**, 347-355.

Kojouri GA, Karimi I. 2002. Sheep pulmonary adenomatosis: a study onprevalence and pathological findings. Pajouhesh-va-Sazandegi **53**, 64-67.

Martin WB, Angus KW, Robinson GW, Scott FMM. 1979. The herpes virus of sheep pulmonary adenomatosis. Comparative Immunology, Microbiology and Infectous Diseases 2, 313-325.

Pugh DG. 2002. Sheep and Goat medicine. 1st ed; Elsevier, United States of America, p. 107-128.

Sharp JM, Angus KW. 1990. Sheep pulmonary adenomatosis: clinical, pathological and experimental aspects. In: Petursson G, Hoff-Jorgensen R (Eds.), Maedi-Visna and related

Int. J. Biosci.

diseases. Kluwer Academic Publishers, Boston, p. 157-175.

StevensonRG,FinleyGG,LongJR,RehmtullaAJ.1982.Pulmonaryadenomatosis

(Jaagsiekte) of sheep in Canada.The Canadian Veterinary Journal **23**,147-152.

Toumazos P. 1989. First Report of Sheep Pulmonary adenomatosis in Cyprus. British Veterinary Journal **7**, 145-289.