

PATHOLOGICAL ASPECT OF LEPTOSPIROSIS IN SHEEP AND GOATS IN IRAN (*)

by

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INTRODUCTION

Leptospirosis is an infectious disease occurring in various species of animals and man. The causative agents are spirochetes of the genus *Leptospira*.

The disease in sheep and goats has been reported from many countries. Hartley (1952) described first case of The disease in sheep in New Zealand (5); In the same year Khudaiberdiev reported the disease from Russia, (8). In 1953 Hoeden described the disease in goats in Israel, (6). There are subsequent reports from: Western Australia in 1953, (14), United States in 1953, (1), Turkey in 1957, (4), Italy in 1958, (15), Hungary in 1959, (2) Argentina in 1961, (17), and Portugal in 1964, (3).

Since 1957 the disease has been studied serologically in Iran, (9,10,11, 12,13), and *L. grippotyphosa* was isolated from sheep in 1961, (12). A pathological survey was done on Leptospirosis in sheep and goats during 1968-1972. The object of this communication is to report gross and histopathological changes associated with this disease.

THE SUBJECT:

In 1968 a severe outbreak of leptospirosis occurred in the Kermanshah region in the west, later in Caspian sea and Gorgan regions in the northern part of the country. Also we received several specimens from scattered infected areas throughout the country. Several hundreds of sheep and goats died during these outbreaks. Numerous sick and dead animals were submitted to the Path. Dept. at the Razi Institute for diagnosis and pathological investigations. The submitted animals were postmortemed and tissues from liver, kidneys lungs as well as other organs were collected in formol saline for histopathological examination. Tissues were processed by the paraffin embedding method. Sec-

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tions were cut 5 microns in thickness and two batches of histoslides were prepared, one stained by H & E and the other by the silver impregnation method. Blood and urine were collected for serological tests and laboratory investigation.

CLINICAL SIGNS:

The infected animals were depressed and showed elevation in body temperatures of 40–41.5°C. The sick animals snuffled and held their heads down. Visible mucous membranes were pale, yellow in color with some petechial haemorrhages in the eye lids mucosa. The yellowish color of the mucosa and their paleness indicating severe anemia and jaundice. Heart rate was increased and dyspnea was prominent in sick animals. Abortion and haemoglobinuria were noted in affected herds. Morbidity rate was recorded as 90–100% in both species and mortality rate averaged about 18% in sheep and 42% in goats. Death occurred within 2–3 days. The animals that were treated with combined streptomycin and penicillin during early stage of the disease survived.

NECROPSY FINDINGS:

Several sheep and goats were postmortemed during outbreaks and the gross and histopathological changes were approximately the same in all cases, with some exceptions. Postmortem examination revealed;

Severe haemolytic anemia and jaundice. Icterus was mild in sheep but more prominent in goats. Slight edema with numerous ecchymotic haemorrhages were noted in the subcutis and serous membranes. The trachea was filled with a white frothy exudate and the tracheal mucosa showed mild, yellowish oedema. The lungs were pale and showed intralobular oedema that was stained by bilirubin and widened the septa; The liver was enlarged and friable. There were numerous subcapsular ecchymotic haemorrhages which were scattered throughout. The kidneys were swollen, enlarged and dark brown in colour with rough surfaces. Numerous subcapsular patchy greyish foci (2–7 mm in diameter) were present in the cortex (Fig. 1,2). These patches appeared as wedge shaped streaks in the cut surface, they commenced from beneath the capsul in the cortical region and extended down to the medulla. They were more prominent in the cortex than in the medulla. Urinary bladders were filled with dark brown urine.

HISTOLOGICAL FINDINGS:

Lungs showed alveolar and intralobular oedema with slight fibrinous deposition. Centrilobular necrosis which may have resulted from severe ana-

mia and anoxia was prominent in the liver. The sinusoidal endothelium was separated from apposition to the parenchyma by a proteineous oedema. There was separation of liver cells which disrupted the liver cell columns. The affected liver cells often had granular eosinophilic cytoplasms and in some instances they showed medium to severe fatty changes. The Kupffer cells were hyperplastic and contained an excessive amount of haemosiderine and there was a diffuse but mild cellular infiltration in the portal triads. (Fig. 3,4).

There were marked degenerative changes in the cortical epithelium of the renal tubules. The changes varying in severity from hydropic swelling to necrosis and desquamation. Desquamated epithelium produced granular and cellular casts. In addition, there were hyaline and albuminous casts that might have resulted from haemoglobinuria and direct bleeding into the tubules, (Fig. 5). Numerous biliary pigments were evident in the urinary epithelial cells and they appeared as dark brownish granular pigments. Most of the renal corpuscles showed marked changes, Bowman's capsules proliferated and were approximately 10 times thicker than normal, while the glomeruli were collapsed, shrunken and looked like they were under pressure, (Fig. 6). The interstitial tissues of the kidneys were distended by oedema and there was severe mononuclear infiltration, predominantly of lymphocyte and plasma cells. These interstitial reactions produced numerous streaks which appeared in the cortical region, extending through the medulla to the pelvic area.

Histological examination of silver impregnation (Levaditi's) stained preparations revealed single and clumped organisms in the urinary tubules, (Fig. 7).

The causative agent was identified serologically as *L. grippotyphosa* in the Kermanshah region.

In a serological survey which was done previously in various parts of Iran, positive reactions for *L. pomona*, *hyos* and *ictero haemorrhagica* and *grippotyphosa* were detected among sheep and goats in northern and central areas of the country.

DISCUSSION

Single individual cases as well as outbreaks of leptospirosis have been observed in sheep and goats in Iran. Outbreaks of Leptospirosis depend upon a favourable meteorological environment, since survival and spread of leptospires depend largely upon suitable conditions of soil and water in the contaminated areas, (7,16). Optimal climatological factors such as frequent rains, flood and mild weather existed for outbreaks of leptospirosis in the Kermanshah,

Caspian Sea and Gorgan regions. Several hundreds of sheep and goats died from severe acute leptospirosis caused by *L. grippotyphosa* L. pomon and *Icterohemorrhagica*. These organisms were identified serologically in these areas.

Dogs showed characteristic symptoms of the disease in the infected areas.

Icterus in humans has been noted, these cases were treated under the care of physicians symptomatically without isolation of the organisms or serological tests.

S U M M A R Y

Several outbreaks of leptospirosis in sheep and goats were diagnosed pathologically in Iran. The gross and histopathological examination revealed; icterus was more prominent in goats than in sheep. Subcapsular hemorrhages with necrotic foci were approximately constant changes in the liver. Numerous streaks were noted in the kidneys, they started in the cortex, extended through the medullary region to the pelvic area. Microscopically these were identified as a non suppurative interstitial nephritis. Proteineous casts were presented in most of the proximal and collecting urinary tubules. Special stain on histo slides revealed, individual and clumped organisms in the urinary tubules. The causative agents were identified serologically as *Leptospira grippotyphosa* in the Kermanshah outbreak.

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REFERENCES

- 1- Beamer, P.D. and Hardenbrook, H.
Studies on Leptospirosis in domestic animals.
Leptospirosis in sheep. Vet. Med., 1953, 48, 365.
- 2- Bokori, J. et al. *Leptospira pomona infection in sheep in Hungary.*
Abstracted in Vet. Bul., 1959, 29, 487.
- 3) Fraga de Azeved, J. & Palmerio, J.M., first case of ovine leptospirosis in Portugal *Bol. Pecuar*, 1964, 32, (3), 113.
- 4- Hakioghly, F., *Leptospirosis histologically confirmed in sheep and goats in Turkey,* *zbl. allg. Path. Anat.* 1956, 95, 525
(*Abst. in Vet. Bull.* 1957, 27, 169)

- 5- Hartly, W.J., ovine leptospirosis,
Aust. Vet. J. 1952, 28, 169.
- 6- Hoeden, J. Leptospirosis among goats in Israel.
J. Comp. Path., 1953, 63, 101.
- 7- Karaseria, E.V., et al.
Some features of the ecology of Pathogenic leptospires in an area where the disease occurs among wild animals,
Zhurnal Mikrobiologii Epidemiologii i immunobiologgii, 1974, No. 5,36.
- 8- Khudaiberdiev, N.D. Leptospirosis in Karakul sheep,
Karakulevodistov izverovodistove, 1952,5,69.
- 9- Maghami, Gh. A brief report on the survey of Leptospirosis in Iran.
Arch. Inst. Razi. 1970, 22, 203.
- 10- Rafyi, A. & Maghami, Gh.
Sur la fréquence de leptospirose en Iran.
Bull. Soc. Path. enot. 1957,50,657.
- 11- Rafyi, A. & Maghami, Gh.
Sur la fréquence de leptospirose en Iran
Bull. Soc. Path. enot. 1959, 52, 592.
- 12- Rafyi, A. & Maghami, Gh.
Sur la fréquence de leptospirose en Iran.
Bull. Soc. Path. enot. 1961, 54, 179.
- 13- Rafyi, A. & Maghami, Gh. and Niak, Ap.
Leptospirose ovine et caprine
Arch. Inst. Razi, 1968, 20, 25.
- 14- Seddon, H.R. Disease of Domestic animals in Australia, Part 5.
Bacteria disease, P.319, Service publication No.9, Div. of Vet. Hygiene, Austral a, 1953.
- 15- Sessi, P. Incidence of leptospirosis of sheep and goats in Tuscany, Italy, Ann. Fac. Med. Vet. Pisa, 1958, 10, 117.
- 16- Sullivan, N.D., Leptospirosis in animals and man.
Aust. Vet. Jour. 50, 5, 216.
- 17- Vidal, J.M.
Isolation of *Leptospira autumnalis* from a sheep in Argentina, Rev. Invest. Gonad., 1961, 11, 39.

R E S U M E

Aspect Pathologique de Leptospirose chez les moutons et les chèvres en Iran.

Differentes éruptions de Leptospirose chez les moutons et les chèvres ont été diagnostiqués pathologiquement en Iran. L'examen des cadavres et les coupes histologiques ont révélés l'ictére qui a été très avancé chez les chèvres, l'hémorragie subcapsulaire avec les foyers nécrotiques étaient approximativement les changements constant du foie. De nombreuses raies ont été notées dans la zone corticale qui ont été étendues à la région médullaire jusqu'à la zone pelvique. L'examen microscopique a identifié une néphrite interstitielle non-supurative. Les calculs proteiniques se sont présentés dans le proximal et le tronc des tubules urinaires. La coloration spécifique des coupes histologiques montre la masse des organismes dans les tubules urinaires. L'agent causal sérologiquement identifié est *Leptospira grippotyphosa*.

Z U S A M M E N F A S S U N G

Pathologische Aspekt von Leptospirosis in der Schafe und Ziegen im Iran.

Mehrere Leptospirosen Ausbruch von der Schafe und Ziege sind in Iran diagnostiziert worden. Die makroskopische und mikroskopische Untersuchungsergebnisse sind:

Bei dem Ziege sind Ikterus mehr sichtlich als Schafe. Unterkapseläre Hamorrhages und nekrotisches sind die Daueränderung von Leberrinden. Mehrere Streifen sind in Nieren erkannt worden. Diese waren von Rindenschicht bis Nierenmark erstrecken worden. Die Streifen sind in Mikroskopische Untersuchung als interstitial nephritis anerkannt worden. Einige Eiweißablagerungsherden werden in Proximal Harnkanälchen festgestellt. Durch eine bestimmte Frblösung könnte man die Organismen in Harnkanälchen gut sehen. Die Krankheitursache sind durch serologische Untersuchung als *Leptospira grippotyphosa* identifiziert worden.



Fig. 1- Patchy greyish white spots in various size in the cortical surface of the kidney.

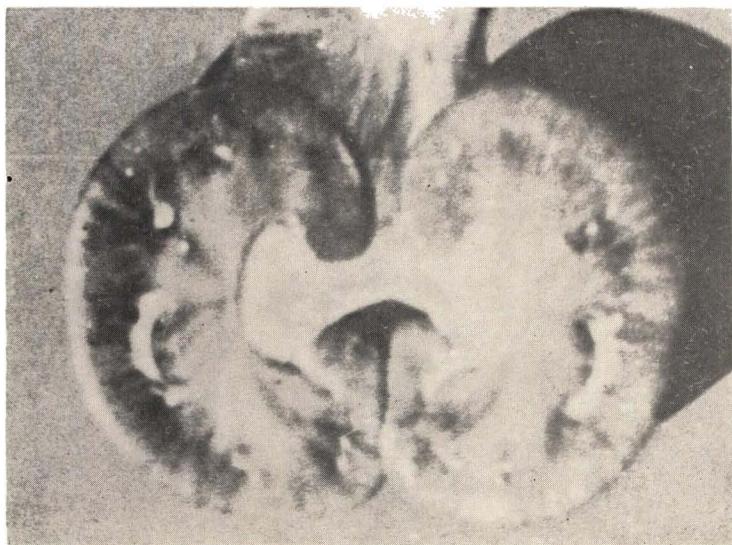


Fig. 2- Greyish white streaks in cut surface of the kidney; note the elongation of streaks from cortical to medullary region.



Fig. 3- Centrilobular necrosis; note the fatty changes of the hepatic cells.

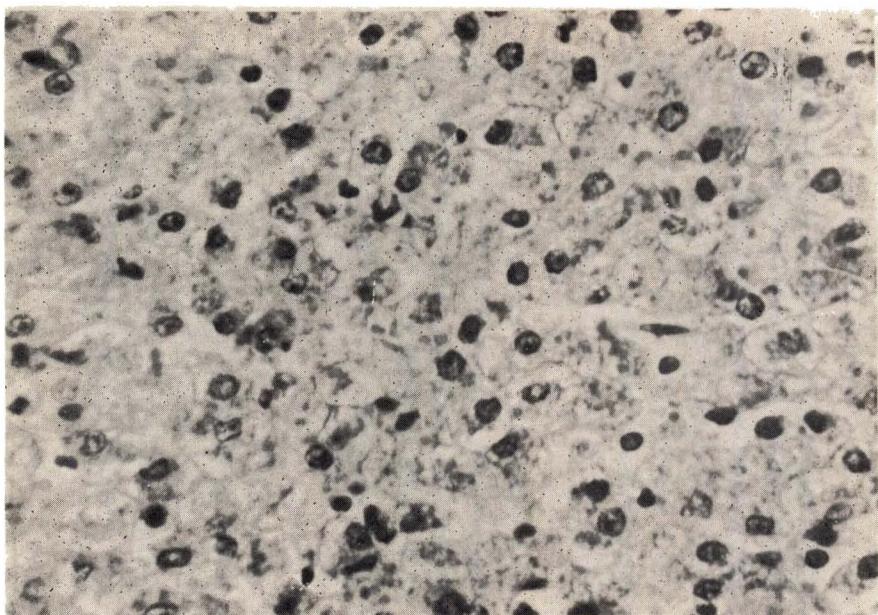


Fig. 4- Degeneration of hepatic cells; note dissociation of liver cells columns.

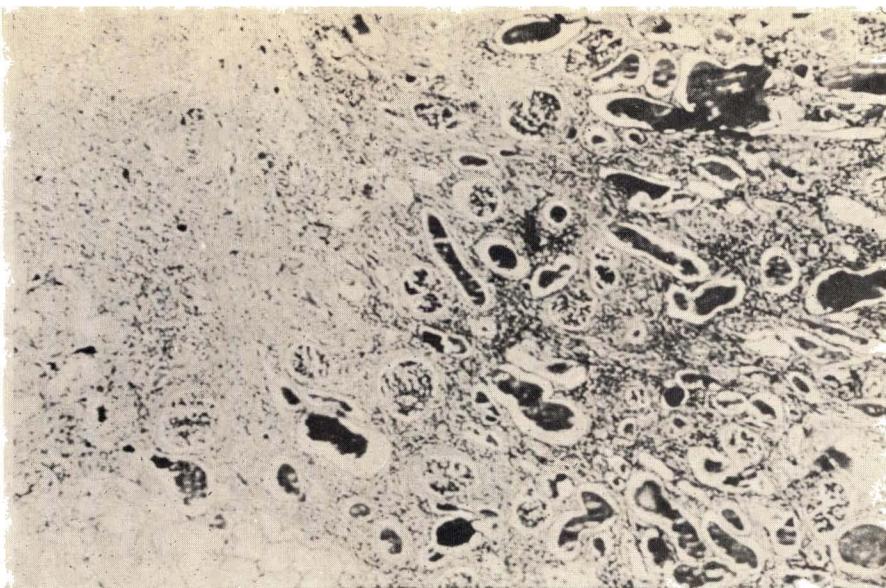


Fig. 5- Non suppurative interstitial nephritis; note the proteineous casts formation in urinary tubules.

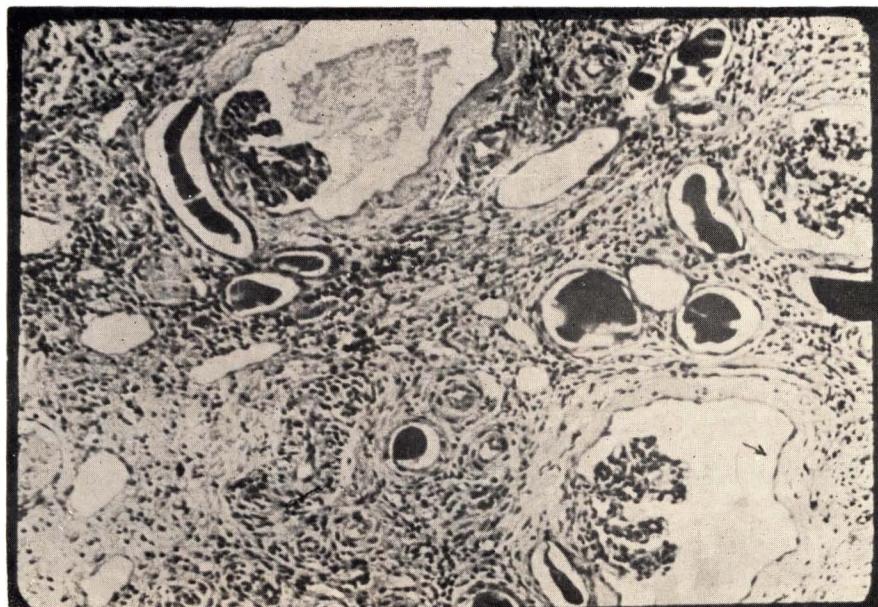


Fig. 6- Non suppurative interstitial nephritis; note the distention and proiferation of Bowman's capsul with shrunken glomeruli.

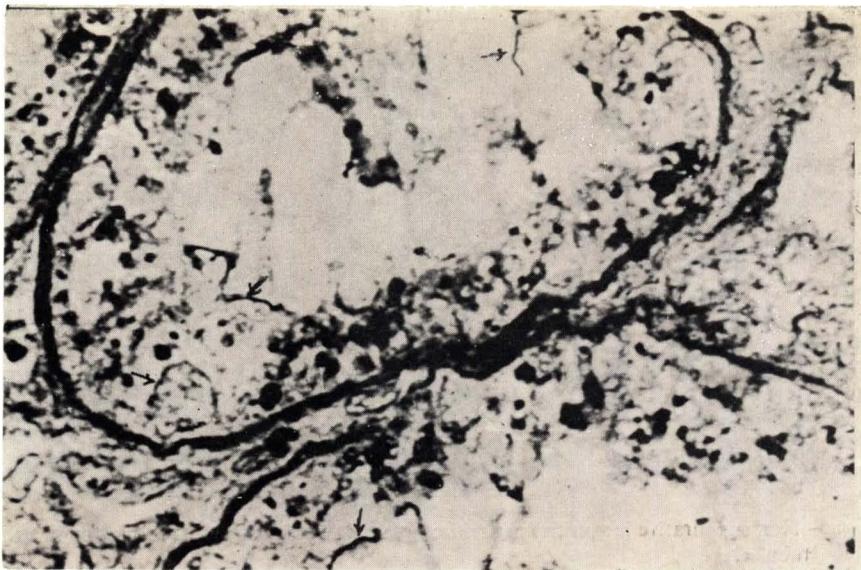


Fig. 7- Note the single and clumped organisms in the urinary tubules (silver impregnation Levaditi's stain).