Arch. Inst. Razi, 1977,29,71-78

# FIRST REPORT OF GEELDIKKOP IN SHEEP IN IRAN

By

AMJADI, A.R., AHOURAI, P. and BAHARSEFAT, M.

## INTRODUCTION:

Tribulosis so called Geeldikkop (Thick Yellow Head) in South Africa named for Photodynamic Dermatitis accompanied by icterus, occurs in sheep and goats which graze Tribulus terresteris (7) in certain period of the year. A number of steroidal saponins has been isolated from the plant(4).The toxic factor is apparently not always presented, although it is believed that the plant in the preflowering, flowering or fruiting stages can be toxic (9), but the wilting is the important factor leading to development of toxicity (12). Prior to wilting the plant is regarded as a good sheep fodder. The plant is highly toxic for sheep and goats under certain condition of growth and climate and according to Steyn (17),half a million of sheep and goats were lost due to grazing the T.terresteris in South Africa (4).

Theiler (18), in 1918, showed that the plant is responsible for Geeldikkop, later, although some workers have relegated the plant to a secondry causal role, but Tonder, in 1972, (19), could successfully reproduce the disease in sheep by feeding T. terresteris. In some experiments, ligating of bile-ducts of sheep (11) and guinea pigs (1), had produced both photosensitization and icterus.

Phyloerythrin, a degraded product of chlorophyll, has been known as a sensitizing substance in some animals in certain condition (13); when the liver is damaged the amount of this pigment increases in blood stream resulting in photosensitization.

As seleniferous soil is correlate with areas in which the occurrence of Geeldikkop is high, it has been considered that subtoxic amount of selenium in

the diet predisposes the animals to photosensitization by Tribulus (3).

In experimental cases which have been done in South Africa (2–17) and Australia (5–6), on Geeldikkop, the situation was complicated by presence of high nitrate in the plant which consequently resulted in nitrate poisoning in cattle and sheep.

The symptoms of Geeldikkop could be reproduced by feeding other plants too (10,14 & 16).

The object of this communication is to report the first case of Geeldikkop in sheep in Iran due to grazing Tribulus terresteris.

**THE SUBJECT:**

In July 1975, a sick sheep of native breed (Sangsari) aged 14–16 months was submitted to the Pathology Department of State Razi Institute for diagnosis of the disease.The anima! was from a flock of 700 sheep and 40 goats that were kept in an area near Saveh 180 Km. far South West of Tehran for grazing. The flock had moved to the area two months previously, where the pasture was overgrazed and 80 % of the fodder plants consisted of T. terresteris.

The morbidity rate in the flock ranged 8–9% and mortality rate was 50 % of infected animals. None of the goats were showing any clinical symptoms. The clinical manifestation in the diseased sheep was rather the same. The sick animals were keeping their head in upward position constantly and scratchig their face, ear and nose against any object that they could find on their way, also they hit their head and face by their hind legs. The animals were showing photophobia and had rather to stay in shade. Their faces, ears, eyelids, lips and noses were swollen, edematous and painful in palpation. Acute dermatitis and scab formation on the skin surface due to the serum oozing were noted. Cutaneous necrosis were presented on face, nose, lips, ears and also around the articular joints (Fig. 1). Loss of lip and ear were noted in some sheep.

The affected animals showed an increase of body temperature up to 39.5C° visible mucous membrane as well as bare parts of the skin were showing jaundice. Lachrimation and nasal discharge were presented. Animals showed anorexia and dyspnea which might have been due to heavy nostril edema. The urine was intensely yellow in color. Keratitis was marked in both eyes and observed in all cases. Cornea and lens underwent opacity in some cases.The cornea was ruptured and the eye ball was sunken (Fig. 2).

**NECROPSY FINDINGS:**

The pathological aspect and gross lesions were approximately similar

in all posted animals. But, individual variations were observed in extent and severity of the lesions. The characteristic lesions were dermatitis, Keratitis and marked jaundice which developed all over the body. Subcutaneous tissues were edematous and yellow in color which exaggerated at head, face, neck and around the articular joints. Lungs were stained with yellow edema and in some cases they were mottled by patchy grey hepatization that were scattering throughout the lungs. Liver was moderately enlarged and brownish yellow in color. Numerous subcapsular and parenchymal patchy grey foci were scattered throughout the liver. Bile ducts were prominent and gall bladder was distended and filled with thick jelatinous bile. Kidneys were enlarged and focal disseminated discoloration were noted in cortex region at cut surface.

## HISTOLOGICAL FINDINGS:

*Kidneys:* The vesseles were engorged and filled with red blood cells. Renal capsules were thickened due to proliferation of fibroblasts. There were adhisions of the inner layer of the capsules to the cortical surface by fibroblasts invasion in between.

The lining epithelial cells of proximal convoluted tubules underwent severe degeneration and necrosis and the tubules contained albuminous casts. The necrotic epithelial lining cells were so intimitely mixed with proteinaceous content of the lumen that, together formed a dense, homogeneous mass which was limited by the epithelial basement membrane and interestitial tissues (Fig. 3). The adjacent tubules to the proximal convoluted, were unijured and they had healthy appearance. The glumeruli were presenting slight changes. Medular region was intact and did not show any alteration.

*Liver:* Engorgement of the vesseles in liver indicated moderate congestion of the tissue. The hepatic lobules had lost their natural structures and they were presenting homogeneous mass. The sinosoids were disappeared due to dissociation of hepatic cords, (Fig. 4). Hepatic cells were showing mild fatty degeneration, coagulative necrosis with eosinophylic cytoplasm and pyknotic nuclei. Some of the cells were damaged severely and disintegrated or disappeared, while the others were enlarged. Hepatic cells contained enormous amount of pinpointed brownish yellow pigments which considered as bile pigments. The triads were presenting mild fibrosis with periportal and perivascular infiltration of mononuclear cells predominantly of lymphocytes. Hyperplasia of the bile ducts was also noted in these areas.

Histological studies on eye revealed marked thickness of the cornea due to severe edema.

## DISCUSSION :

Tribulus terresteris is a plant of self growing in deserted areas. In Iran, the plant spreads on dry soil of waste lands, road sides, deserted areas and in over grazed pastures with dry climate which mostly are located in high altitude, 1000 – 1500 m. from sea level, (Figs. 5 & 6).

Attention should be paid to the prevalence of the plant and its toxication in this country. Jaundice and photosensitization are important clinical signs of the disease. There are numerous causative agents that could produce jaundice in animals, among which blood parasites are the commonest. So there is possibility that the correct diagnosis of the disease might have been confused and neglected in the past in Iran. Keratitis that accomanied with other changes in this disease may be considered as pathogonomic sign for differentiation and identification of the disease.

The disease has been considered as hepatogeneous photosensitization, so the active principale of the plant produces damages both in liver and kidneys. Phylloerythrin which is a degradation product of Chlorophyll, normally is absorbed and excreted via bile. When excretory capacity of liver is deranged, due to toxic substance, phylloerrythrin accumulates in the blood and sensitizes the animal to light.

In our case the disease subsided by transfering the herd from infected pasture. The affected animals were kept in shade and they showed very good responce to antihistamine drugs (8).

## SUMMARY:

Marked clinical symptoms of Geeldikkop were diagnosed in sheep in Iran. Clinical symptoms revealed photosensitization dermatitis which accompanied with icterus and keratitis. Histologically there were severe damages to the liver and kidneys. The morbidity rate ranged 8 – 9 % and mortality rate recorded 50 % of infected animals. None of goats were showing any clinical sign of the disease.

**REFERENCES:**


1 - BREMNER, D.P. (1974)
J. Com. Path., **84**, No 4, 555

2 - BROWN, J.M.M. (1959)
J.S. Afr. Vet. Med. Ass., **30**, 403

3 - BROWN, J.M.M. (1962)
Onder. J. Vet. Res., **29**, 111

4 - BOUGHTON, I.B. & al. (1935)
Texas Agr. Expt. Sta. Bull., 519

5 - CHIPPENDALE, G. & al. (1964)
Aust. Vet. J., **40**, 241

6 - HURST, E. (1942)
N.S.W. Poisonous Plants Comm. Sydney

7 - KINGSBURY, J.M. (1964)
Pois. Plants of U.S.A. and Canada Sec. ed.

8 - MEHNERT, O.H.J. (1962)
Tier. Umsh., 54

9 - QUIN, J.I. (1929)
Onder. J., **15**, 765

10 - QUIN, J.I. (1933)
Onder. J. Vet. Sci. Anim. Ind., **1**, 501

11 - QUIN J.I. (1933)
Onder. J. Vet. Sci. Anim. Ind., **1**, 505

12 - QUIN, J.I. (1935)
J.S. Afr. Vet. Med. Ass., **6**, 16

13 - RIMINGTON, C. & al. (1934)
Onder. J. Vet. Sci. Anim. Ind., **3**, 137

14 - RIMINGTON, C. & al. (1937)
Onder. J. Vet. Sci. Anim. Ind., **9**, 225

15 - STEYN, D.G. (1938)
J.S. Afr. Vet. Med., **33**, 155

16 - STEYN, D.G. (1928)
J.S. Afr. Vet. Med. Ass., **1**, 47

17 - STEYN, D.G. (1934)
The Toxicology of Plant in S. Africa

18 - THEILER, A. (1918)
7th. & 8th. Ann. Rept. Vet. Ser. Anim. Ind. Uni. S. Afr. p:155

19 - TONDER, E.M. & al. (1972)
J.S. Afr. Vet. Med. Ass., **43**, No 4, 363

Fig. 1. Photodynamic dermatitis, note the cutaneous necrosis around the articular joint with scab formation on the surface.

Fig. 2. Note sunken eye and cutaneous necrosis on the face.

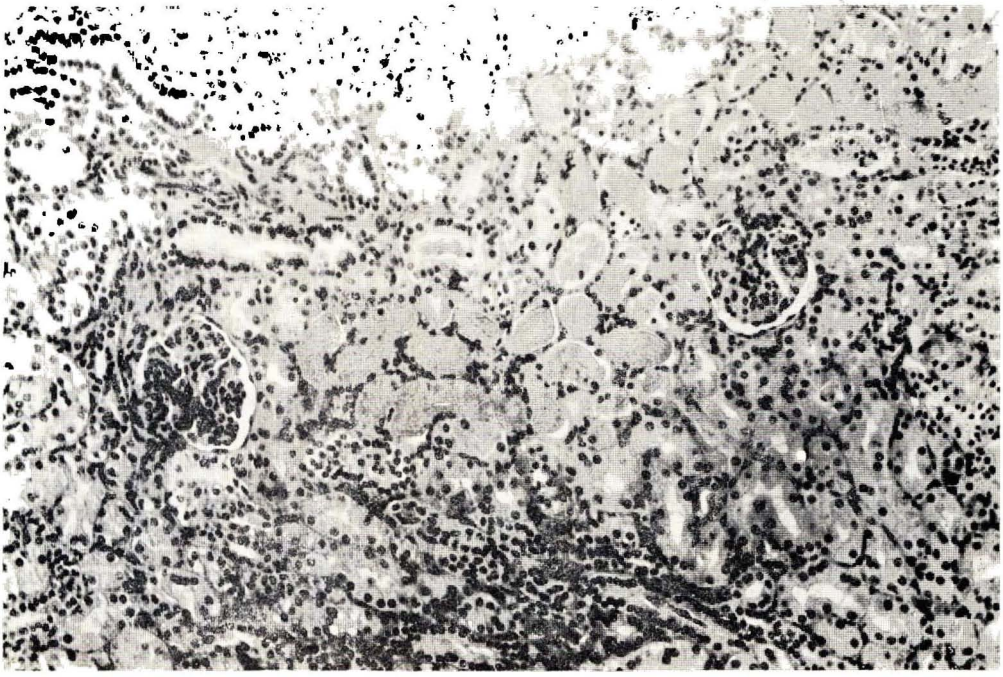

Fig. 3. Severe degeneration and necrosis of lining epithelial cells of proximal convoluted renal tubules. Note the homogeneous mass which is limited by epithelial basement membrane. X125

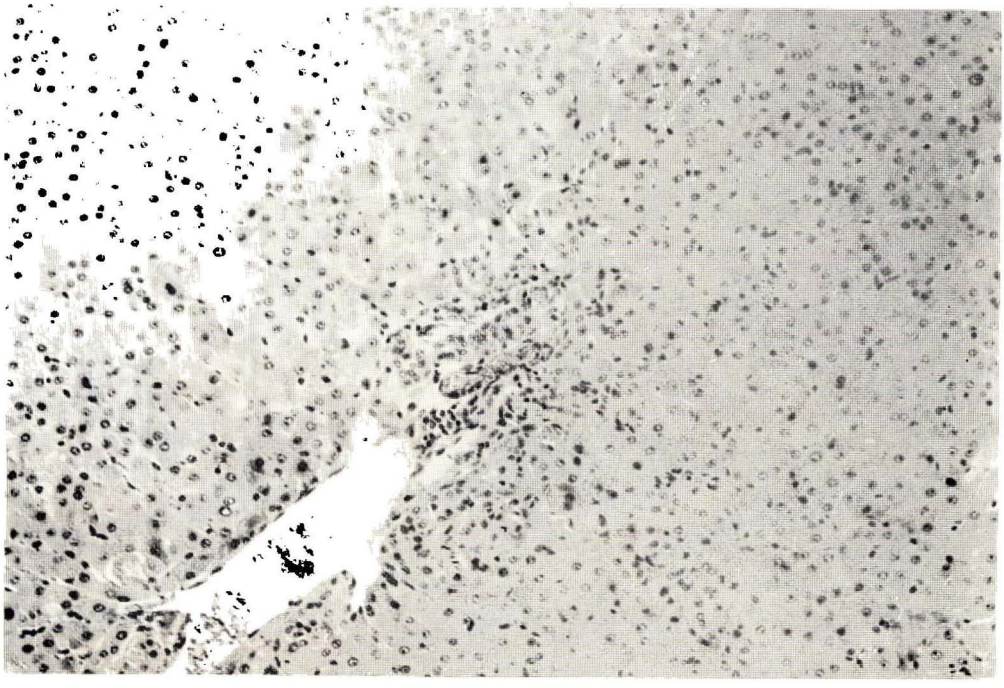

Fig. 4. A microphotograph of liver section. Note the periportal and perivascular mononuclear infiltration. X125

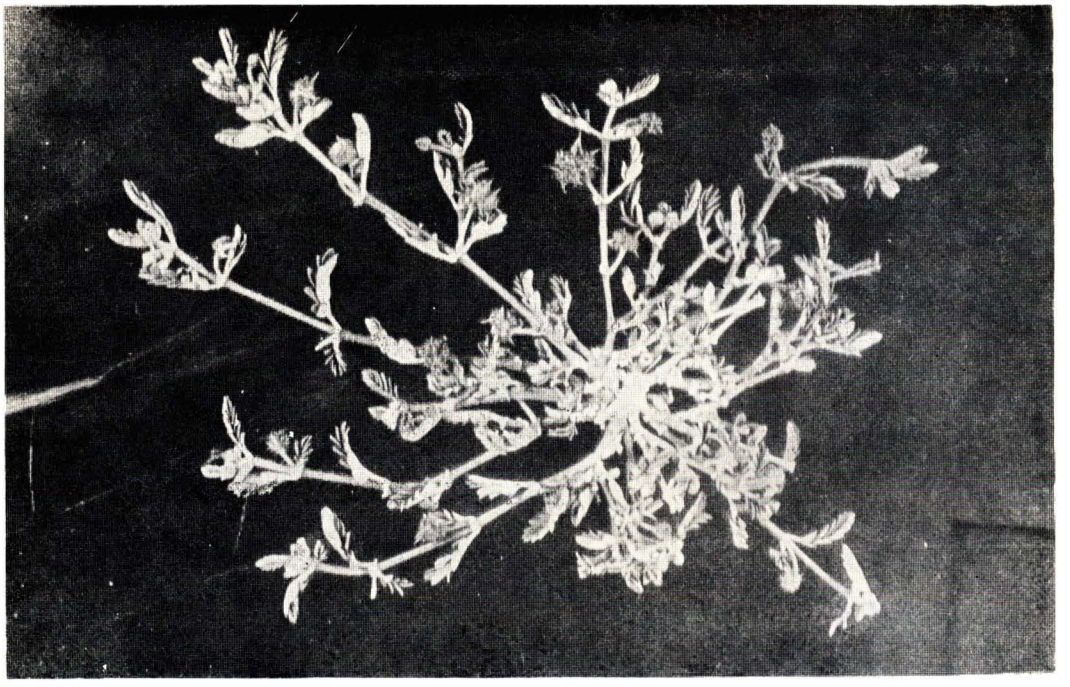

Fig. 5. Tribulus terresteris with recumbant stems.

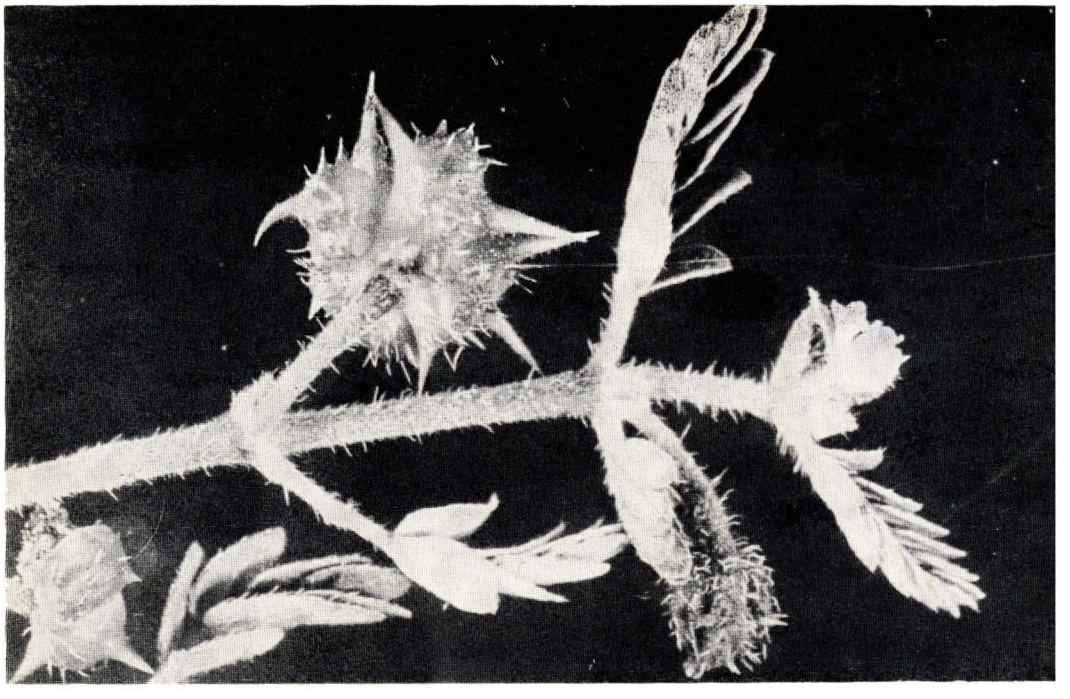

Fig. 6. A close up picture of fig. 5. Note small flowers and thorny fruits of the plant.