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INVESTIGATION ON HEREDITARY CONGENITAL GOITRE IN GOATS

BY

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INTRODUCTION

Inherited goitre is recorded in Merino sheep (Rac, 1968 and Mayo & Mulhearn, 1969), crossbred saanen dwarf goats (Rijnberk et al., 1977); Boer goats (Van Jaarsveld, 1971); young and adult goats (Dawood and Muslih, 1988) and dead newly born Egyptian twin kids (Soliman et al. 1965).

Goitre possibly appears to be inherited as a recessive character in polled Dorset sheep (Davis, 1979). Adults may develop goitre, but it is usually the newborn that are most severely affected. Kalkus (1920); Welch (1928) Driscoll et al. (1978) and Omar et al. (1982) recorded such conditions in sheep, goats, cattle, pigs and horses. Congenital goitre has been observed in foals to mares on low iodine intake (Doige and McLaughin, 1981); but also to mares fed on excessive amount of iodine during pregnancy (Backer and Lindsey, 1968 and Driscoll et al., 1978). It is n known that goitre in goats is the cardinal manifestation of a secondary or conditioned iodine deficiency (Soliman et al., 1965). The outbreaks of goitre in lambs in Newzealand are frequent but sporadic, a fact which indicates a picture not associated with simple iodine deficiency (Cunningham, 1950). Dutt and Kehar (1959) investigated 1000 head of sheep and goats slaughtered at Bareilly abattoir and found

10 per cent incidence of goitre in goats without a single case in sheep. Most of these cases were in female goats. Similar condition in goats in Ambala District was recorded by Nasina (1964), where the water found to be totally deficient in iodine. The clinical picture of inherited goitre in goats is the same as for lambs (Rijnberk et al., 1977). It includes retardation of growth, sluggish behavior, rough haircoat and a thick scaly skin. The essential clinical signs of goitre in sheep are a high level of mortality and enalrgement of the thyroid above the normal size and varying greatly up to 222 grams. Baumann (1948) recorded the inherited goitre in nine newly born kids in Vienna, while Soliman et al. (1965) found the disease naturally occurring in a dead one in Egypt.

The aim of this work was to investigate the hereditary congenital goitre in goats from the view of the following: clinical signs of the cases, histopathology of goitrous gland and thyroid functions of both dams and their still living newly born kids.

MATERIALS AND METHODS

(a) Clinical Examination: of the 5 dams and their 5 still living newly born kids to any abnormality in their behavior, thyroid size, hair coat, body weight and musculoskeletal system.

(b) Gross Examination: of the affected thyroids of the 5 dead newly born kids was done after their removal and then investigated to any abnormality in the gland size, length and weight.

(c) Histopathological Examination: of the affected thyroid sections from these goitrous glands was done after secured in 10 per cent formol-saline and then stained with Haematoxyline and Eosin.

(d) Thyroid Functions: blood was obtained by jagular veinpuncture from the 5 dams and their still living 5 newly born kids, and the non-haemolysed serum samples were laboratory examined for the triiodothyronine (T_3), thyroxin (T_4) and protein bound iodine (PBI) by using Double Antibody Enzyme Immunoassay test kits method on a PYEUNICUM spectrophotometer. Similarly, samples were obtained from 5 recently parturient goats and their newly born kids.

RESULTS AND DISCUSSION

Clinical examination of the 5 dams shortly after delivery revealed that they were apparently healthy and have no perceptible goitre. Similarly, the 5 still living newly born kids were generally in a good health condition shortly after birth. Concerning the 5 dead newly born kids their examination showed an evident swelling protruding on both sides of the neck, oedema of the tissues around it and loss of hair on the head, neck and the rest of whole body (Fig. 1).

According to owners complaint, the kids died almost shortly after delivery. Gross examination of the swelling revealed that it was a marked en·argement of the thyroid gland. lobes were oval to elongated in shape, each 8-15 cm in length, 5-7 in width and 3-5 cm in thickness. The greatly enlarged glands weighted 100-224 gm and having a dark brown coloration, hard in consistency. Kelly (1983) found that, iodine deficiency, whether of primary or secondary origin, is generally recognized clinically in newly-born lambs by the birth of dead and weakly anmals in addition there may be palpable enlargement of the thyroid gland. Further abnormalities are found associated with goitre occur in the offspring of iodine deficient parents (Doxey, 1983).

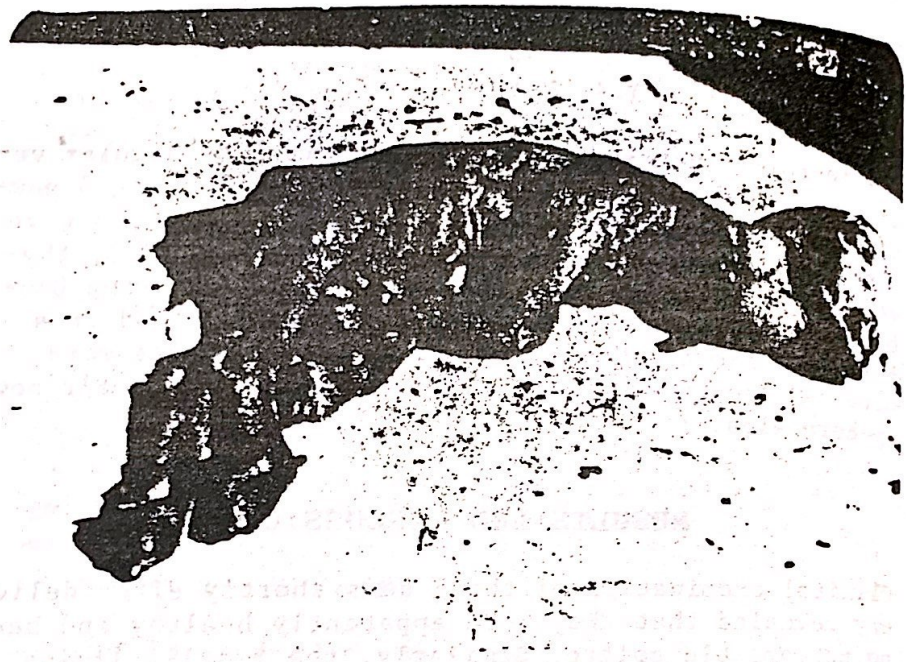

Fig. (1): Dead newly-born kid affected with simple goitre.

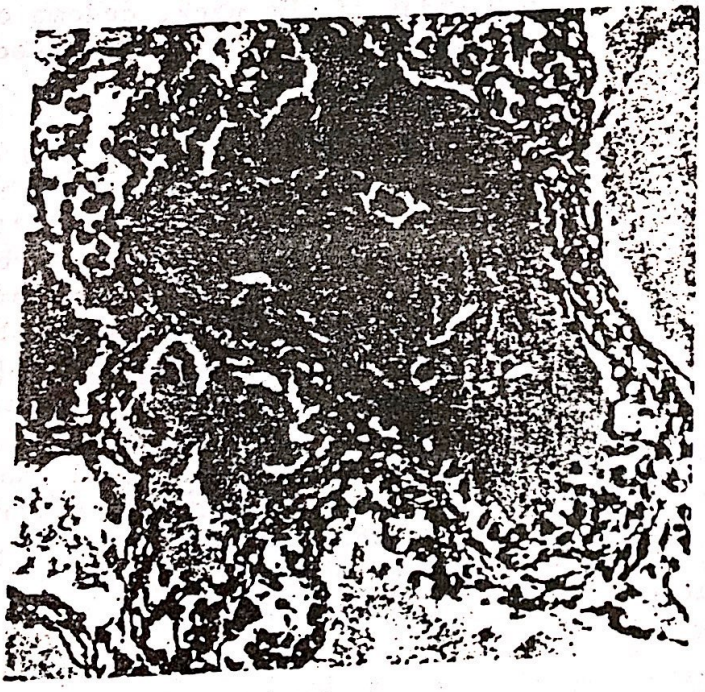

Fig. (2): Goitrous thyroid gland of newborn kid.

Histopathological study of sections prepared from various parts of both lobes of the affected thyroid showed that the general feature corresponds with that of simple goitre (Fig. 2), revealing marked hyperplasia of epithelial cells and degenerative changes in their nuclei. Colloid seemed to be depoted in some acini, while appeared normal in others. The cause of simple goitre is ordinarily a mild deficiency of iodine. The milder goitrogenic agents such as soybeans, are likely to produce this type of goitre (Jones and Hunt, 1983).

The results of thyroid function for the dams and their still living kids as sk in Table (1) suggested a hypofunction of the thyroid glands of the 5 dams. Soliman et al. (1965) found that although P.B.I. of the dam was beyond the normal value, that of the kids were normal and the occurrence of goitre in only one twin is supposed to be hereditary predeposition incited by the low iodine serum value in its dam.

Table (1): Results of thyroid functions of goat dams give birth to goitrous kids and of their still living kids.

		T_3 (ug/100 ml)	T_4 (ug/100 ml)	PBI (ug/100 ml)
Goats	Normal	239±29.24	5.79±199	3.47±1.50
	Give birth to Goitrous kids	204±20.80	4.23±1.35	1.99±1.12
Kids	Normal	249±31.80	5.74±0.52	8.10±0.20
	Born Goitrous kids	145±22.10	5.20±0.40	7.95±0.15

There is no evidence as to whether the cases are due to simple iodine deficiency or result of a conditioned one arising from goitrogenic factors could be deducted. This especially because, no accurate information could be collected of any particular food stuffs the dams were kept on. Devijlder et al. (1978) found that, although many explanations have been offered, the exact nature of the underlying defects remain unknown.

SUMMARY

Hereditary congenital goitre occurring only in one twin of each inbred breed of 5 goats is reported. The loss of hair on whole the body, oedema of tissues around the neck with enlarged thyroid gland in the dead newborn kids were cardinal sigs of hypothyroidism. Dams and their alive twin kids showed no perceptible clinical abnormality of their thyroids. Histopathological findings observed in sections of affected thyroids of the dead kids as well as, the thyroid functions of the still living kids and their dams suggested the view of hypofunction of the thyroid gland of the dams.

There is no evidence as to whether the cases are due to simple iodine deficiency or as a result of a conditioned one.

REFFERENCES

1. Backer, E.T. and Lindsey (1968): Determination of T_3 and T_4 in desicated thyroid, Schotanus and Jens N.V., Utercht., the Netherlands.

2. Baumann, R. (1948): Goitre among newly born kids Eien tierarztl. Mschr., 35: 585.

3. Cunnigham, I.J. (1950): Studies on iodine deficianey. Soil. Sci. Soc. Florida Proc., 10: 247.

4 . Davis, E.A. (1979): Inheritence of goitre in pooled Dorset sheep. N.Z. Veterinary Sci., 27: 126.

5 . Dawood, K.A. and Muslih, N.J. (1988): Congenital goitre occurrence and severity in different animals of Mosul area, Vet. Med. J. 36: 101-106.

6 . Devijlder, J.J.M.; Vanvoorthuizen, W.F.; Vandijk, J.E.; Rijnberk, A. and Tegelaers, W.H.H. (1978): Hereditary congenital goitre with thyroglobin. Deficiency in a breed of goats. Endo. 102, No. 4, 1214.

7 . Doige, A. and McLaughin, O. (1981): Cited from Kelly (1983): Veterinary Clinical Diagnosid. Baillliere Tindall, London, 3rd. Ed.

8 . Doxey, D.L. (1983): Clinical pathology and diagnotic procedures. Bailliere Tindall. London. 2nd. Edition.

9 . Driscoll, J. Hintz, H.F. and Schryver, H.F. (1978): Goitre in goats caused by excessive iodine. Amer. Vet. Med. Asso., 173, 858.

10. Dutt, B. and Kehar, N.B. (1959): Incidence of goitre in Brite. Vet. J. 115, 178. Goats and Sheep in India.

11. Jones, T.C. and Hunt, R.D. (1983): Vet. Pathology, Lea & Febiger, Philadelphia, 1602.

12. Kelly, W.R. (1983): Veterinary Clinical Diagnosis. Bailiere Tindall, London, 3rd Ed., 134.

13. Kalkus, J.W. (1920): A study of goitre and associated conditions in domestic animals. State Coll. Washington Agr. Expt. Sta. Bull., 156.

14. Mayo, G.M.E. and Mulhearn, C.T. (1969): Biochemical studies on gortre among merino sheep. Aust. J. Agric. Res., 20 533.

15. Nasina, M.A. (1964):Studies on iodine deficiency among goats in Ambaladistrict. Soil. Sci. Soc. Florida Proc, 18, 123.

16. Omar, M.S. Metwally, Z.A. and El-Meligy, A.A. (1982): Nodular goitre in kid., Res. Bull. No. 501, Fac. Agric. Zaga. Univ., Egypt.

17. Rac, R. (1968): Iodine deficiency among lambs. Res. Vet. Sci., 9: 209.

18. Rijnberk, A.J.; Vijlder, J.E., Van Dijk, T.J. Torna and Tegelaers (1977): Congenital defect in iodothyronine synthesis. Clinical aspect of iodine metablolism in goats with congenital goitre and hypothyroidism. Br. Vet. J. 133: 495.

19. Soliman, K.N. Abdel-Wahab, M.F. Shehata, H. Iskander, M. and Hosny, Z. (1965): Investigation on a naturally occurring goitre in a dead newly born twin kid J. Vet. Sci., U.A.R. 2: No. 2, 113.

20. Vanjaarsveld, P. (1971): Studies on goitre among goats J.S. Afr. Vet. Med. Ass., 42: 295.

21. Welch, H. (1928): Goitre among neonates and iodne deficiency Univ. Mot. Agr. Exp. Sta. Bull. 21, 21.