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CASE REPORT

Vertebral Absence in a Lamb with Vitamin A Deficiency

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ABSTRACT

A single female lamb aged 2 days was submitted to the Clinic of Farm Animals, Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece as it was unable to stand. At clinical examination, the lamb was found alert and was in sternal recumbency. External palpation across the spinal column revealed absence of the dorsal vertebral bodies. Hematology and blood biochemical profile were within the normal limits, except for serum vitamin A level that was below the detectable limit. Postmortem examination revealed absence of cervical, thoracic (1^{st} to 7^{th}), lumbar, sacral and coccygeal vertebrae while the 8^{th} -13^{th} thoracic vertebrae with the corresponding ribs appeared normal. Abdominal dissection showed swelling and congestion of kidneys and liver. Abnormal malformations were not observed in the urogenital and gastro-intestinal system of the lamb, as well as concomitant soft tissue defects. Liver copper was within normal range while liver vitamin A levels were below the detectable level.

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INTRODUCTION

Congenital malformations in sheep have been reported to be 0.2-2% in from Australia, New Zealand and United States, of these 50% are related to musculoskeletal system (Dennis, 1975; Dennis, 1993). These malformations can be result of genetic factors, infections, consumption of teratogenic plants or toxic agents (Sargison, 2008), administration of drugs during pregnancy (Navarro *et al.*, 1998) and nutritional deficiencies (Esfandiari and Dehghan, 2010). Vitamin A deficiency is reported to be a common cause of congenital malformations in rats, pigs, rabbits and cattle (Bulld *et al*., 1978). However, it has not been reported as a cause of congenital malformations in sheep.

Skeletal malformations, mainly abnormal segmentation and vertebral identity (extra or defective bodies or ribs), mishaped vertebral bodies have been reported in humans (Xia *et al*., 1999). Vertebral aplasia has been reported in calves (Newman *et al*., 1999), dogs and humans (Bulld *et al*., 1978). In lambs, perosomus elumbis is presented with lumbosacral vertebral aplasia (Dennis, 1974). However, vertebral aplasia has not been reported in lambs. This communication describes case of a lamb that was born with absence of vertebrae from vertebral column, and its vitamin A levels were found to be very low.

History: A single female lamb of 2 days old was submitted to the Clinic of Farm Animals, Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece as it was unable to stand, while its mother was healthy and had been dewormed with ivermectin before pregnancy. Adults were consuming straw and barley, while extra vitamins and minerals were not supplemented.

Clinicopathological examination and post-mortem findings: At clinical examination, the lamb was found alert, it was in sternal recumbency and unable to stand. External palpation across the spinal column revealed absence of the dorsal vertebral bodies. Hematology and blood biochemical profile were within the normal limits except CPK that was higher and serum vitamin A level was below detectable limit (Table 1). The animal was euthanized for welfare reasons. Postmortem examination revealed nonexistence of cervical, thoracic (1^{st} to 7^{th}), lumbar, sacral and coccygeal vertebrae and 8^{th} to 13^{th} thoracic vertebrae with the corresponding ribs normal (Fig.1). Abdominal dissection showed swelling and congestion of kidneys and liver. Abnormal malformations

were not observed in the urogenital and gastro-intestinal system of the lamb, as well as concominant soft tissue defects. Histopathological examination of brain and spinal cord did not reveal any lesion (Fig. 2). Examinations for viral infections (Bluetongue, BVD, Border disease) and toxoplasmosis were also negative. Liver copper was normal, while liver vitamin A levels were below the detectable level.

Table 1: Hematological and biochemical profile of the affected lamb

Parameter	Value	Reference values
PCV (%)	28	27-45[1]
Hb (g/dl)	9.5	9-15[1]
WBC (/µl)	7,100	4,000-12,000[1]
PLT (x 10^5/µl)	6	2.5-7.5[1]
ALP (U/l)	289	68-387[2]
AST (U/l)	65	60-280[2]
Total Bilirubin (mg/dl)	0.15	0.1-0.5[2]
BUN (mg/dl)	15	8-20[2]
Creatinine (mg/dl)	1.3	1.2-1.9[2]
CPK (U/l)	58	8-13[2]
Vitamin A	0	30-100 ng/ml[2]

[1]Schalm OW (1986) In: Schalm's Veterinary Haematology, 4th Ed, Lea and Febiger, Philadelphia, USA; [2]Pugh, DG. (2002) Sheep and Goat Medicine, Saunders, USA.

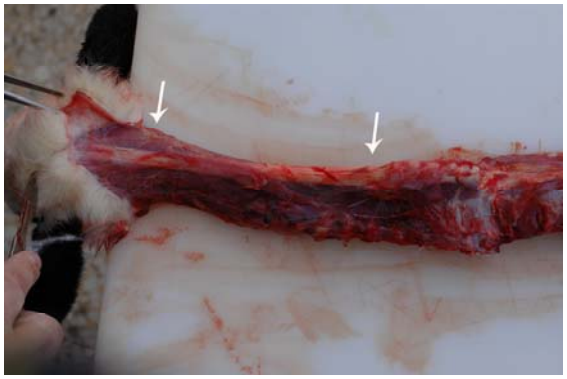

Fig. 1: Absence of cervical and thoracic (1st through 7th) vertebrae (double arrows).

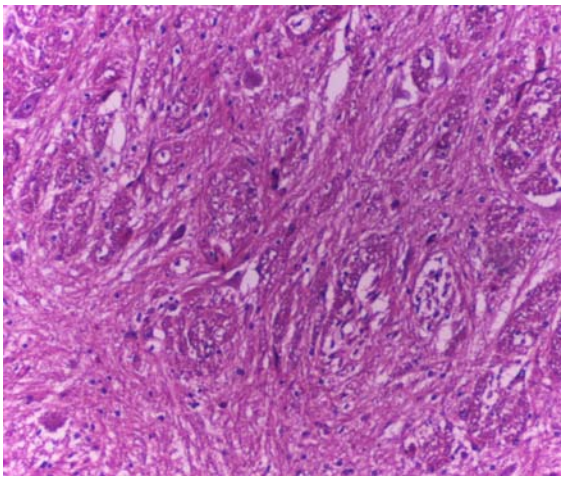

Fig. 2: Absence of histopathological lesions in spinal cord (cervical segment). HE X 200.

DISCUSSION

Vertebral aplasia has been reported in humans (Bulld *et al*., 1978; Al-Kaissi *et al*., 2011), dog (Carvallo *et al*., 2011) and neonatal calf (Newman *et al*., 1999). Cases of lumbar vertebral aplasia have been reported by the farmers (Dennis, 1974). However, cases of major vertebral column aplasia in lambs have not been reported in literature. Possible cause of this condition seems to be vitamin A deficiency in humans (Bulld *et al*., 1978). In animals, it is difficult to investigate the possible causes of congenital anomalies (Esfandiari and Dehghan, 2010).

In the present case, the affected animal had nervous dysfunction and was unable to stand. Differential diagnosis included white muscle disease, border disease, enzootic ataxia and inherited cerebral and cerebellar conditions. All the above conditions were excluded, because the macroscopical and histopathological findings of these diseases were not found. These findings are in agreement with previous clinical observations (Sargison, 2008). Also, perosomus elumbis, a rare congenital defect that is characterized by agenesis of the lumbosacral spinal cord and vertebrae, as well as soft tissue malformations could be taken into account for differential diagnosis (Thompson, 2005). In the present case, a larger area than lumbosacral was affected, while soft tissues were normal. Inherited bone malformation also could be the cause of this condition, but genetic etiology is difficult to prove (Thompson, 2005) and was not conducted in the present study. However, this condition occurred in this flock only once and according to the flockman, it was never observed previously. This probably excludes the inheritance for this condition.

Hypovitaminosis A seems to be a possible finding that could cause this condition. It is known that vitamin A deficiency can cause bone malformations in various animal species and humans (Bulld *et al*., 1978; McDowell, 2000) and probably caused vertebral aplasia or agenesis in the reported lamb. Vitamin A deficiency can cause bone malformations through both osteoblast and osteoclast malfunction, that leads to disorganized bone growth. Although vitamin A and carotenes were not measured in the feedstuffs, it can be presumed that vitamin A deficiency was the result of low carotene and vitamin A in the feedstuffs. It is known that straw and barley are poor in carotenes (McDowell, 2000), and it was reported that the animals were not supplied with extra vitamin A and/or carotenes.

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