Cervical hyperextension in a lamb with nutritional myo-degenerescence secondary to vitamin E deficiency

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SUMMARY
A female 25 day old Lacaune lamb exhibited cervical hyper-extension, standing difficulty, tachypnoea and tachycardia although it was alert and had a normal appetite. Serum CK and LDH activities were markedly elevated whereas the blood selenium concentration was within the usual values and the serum vitamin E concentration was dramatically depressed. Heart, thigh and vertebral muscles were pale and cellular degenerescence was evidenced by histology. The white muscle disease was diagnosed and probably only resulted from a severe vitamin E deficiency.

Keywords: Lamb, cervical hyperextension, white muscle disease, selenium, vitamin E, deficiency.

RÉSUMÉ
Hyper-extension cervicale chez un agneau présentant une myo-dégénérescence secondaire à une carence en vitamine E
Une agnelle Lacaune âgée de 25 jours présentait une hyper-extension de la nuque, des difficultés à se tenir debout, une tachypnée associée à une tachycardie bien qu’elle fût alerte et eût un appétit correct. Des activités sériques de la CK et de la LDH fortement augmentées et une concentration sérique en vitamine E très fortement diminuée (indétectable) ont été observées alors que la concentration sanguine en sélénium était incluse dans les valeurs usuelles. Le cœur, les muscles vertébraux et ceux de la cuisse étaient pâles et des lésions histologiques de dégénérescence cellulaire ont été mises en évidence. La maladie des muscles blancs a donc été diagnostiquée et semble résulter d’une carence sévère et isolée en vitamine E.

Mots clés : Agneau, hyper-extension cervicale, maladie des muscles blancs, sélénium, vitamine E, carence.

Introduction
Nutritional myo-degenerescence (white muscle disease-WMD) results from a selenium and/or vitamin E deficiency and affects different animal species, as sheep, goats, cattle, horses, swine, poultry, rabbits, fish, laboratory and exotic animals, monkeys and even marsupials [10, 14]. In sheep, the congenital form affects stillborn or weak neonatal lambs which die soon after birth whereas the delayed form affects lambs aged from 3 weeks to 4 months [14, 25]. The main locomotor clinical signs range from mild stiffness to recumbency and reluctance to move eventually combined with respiratory distress [23, 25]. In necropsy, symmetrical muscular degenerescence, especially of the thigh and shoulder muscles, as well as of the myocardium, is usually found. The deep cervical muscles are also affected, although their lesions are commonly subclinical and generally do not cause functional disturbance [26]. The majority of the reported cases resulted from a combined selenium and vitamin E deficiency [2, 3]. The nutritional myo-degenerescence attributed to a pure vitamin E deficiency (the selenium concentrations were in normal ranges) has been rarely reported, both in natural [11, 15] and in experimental diseases [24]. This clinical case describes a case of white muscle disease in a lamb that exhibited a normal whole blood selenium concentration but was deficient in vitamin E. Furthermore, this animal presented a severe focal myopathy of the cervical muscles, clinically evidenced by a marked opistotonus as the most prominent sign.

Clinical case
A female 25 days old lamb from a dairy sheep flock of 200 Lacaune sheep was submitted to the Clinic of Farm Animals, because it was reluctant to walk for 3 days. The lamb had a normal appetite, was suckling and had free access to alfalfa hay and concentrates, while its dam consumed also alfalfa hay and concentrates for milk-producing sheep.

During the clinical examination, it was noted that the animal was alert and not febrile, exhibited tachypnoea and tachycardia, while thoracic auscultation was normal. A paresis of the hind limbs, a cervical hyper-extension and a generalized muscular atrophy (figure 1) were also observed. The haematological profile (Table I) was normal, although serum CK
CERVICAL HYPEREXTENSION IN LAMB WITH VITAMIN E DEFICIENCY

and LDH activities were significantly increased (5 000 U/L and 2 000 U/L respectively) compared to usual values in lambs [12, 22]. The serum vitamin E concentration (determined by fluorometry) was below the detection limit (< 0.1 mg/L) (usual values: 0.8 – 3.0 mg/L) while the whole blood selenium concentration was 180 µg/L (fluorometric determination) (usual values: 120-500 µg/L) [8, 15, 16, 20].

After euthanasia by intravenous administration of pentobarbital sodium, the gross examination revealed a moderate body condition and a pale coloration of various muscles, especially of heart, thigh and the vertebral muscles, leading to the suspicion of the white muscle disease (WMD). Beginner enteritis was also noticed but parasitological examination of the intestinal content was negative for coccidium, cryptosporidium, trematodes, nematodes and cestodes. No macroscopic lesion was observed in brain and in cerebellum. The histopathological examination of injured muscles revealed a myo-degenerescence with reduction of the fibre diameter coupled to fibre splitting and confirmed the white muscle disease suspicion (figure 2). No histological lesion was recorded in the central nervous system.

The WMD was diagnosed based on the clinical, pathological and biochemical findings. According to the flock owner and to the local veterinarian, this disease has been diagnosed in 4 other lambs which have been successfully treated with a commercial selenium-vitamin E preparation. The dietary mean contents of selenium and vitamin E were 0.2 mg/ kg of dry matter and 3 mg/ kg of dry matter respectively.

### TABLE I: Haematological and biochemical parameters measured in the female 25 day old Lacaune lamb presenting walking difficulties and cervical hyper-extension.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Observed values</th>
<th>Usual values [reference]</th>
</tr>
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<tbody>
<tr>
<td><strong>Haematology</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC (10^{12}/L)</td>
<td>10.5</td>
<td>9.5 - 10.7 [17]</td>
</tr>
<tr>
<td>Hb (g/L)</td>
<td>103</td>
<td>97 - 177 [17]</td>
</tr>
<tr>
<td>PVC (L/L)</td>
<td>0.300</td>
<td>0.243 - 0.498 [17]</td>
</tr>
<tr>
<td>Thrombocytes (10^{12}/L)</td>
<td>52</td>
<td>19 - 97 [17]</td>
</tr>
<tr>
<td>WBC (10^{9}/L)</td>
<td>8.1</td>
<td>4.9 - 15.9 [17]</td>
</tr>
<tr>
<td>Neutrophils (10^{9}/L)</td>
<td>3.2 (39%)</td>
<td>1.0 - 8.6 (20.7 - 54.3%) [17]</td>
</tr>
<tr>
<td>Lymphocytes (10^{9}/L)</td>
<td>4.7 (58%)</td>
<td>1.7 - 10.5 (33.7 - 66.0%) [17]</td>
</tr>
<tr>
<td>Monocytes (10^{9}/L)</td>
<td>0.2 (3%)</td>
<td>0.0 - 1.9 (0.0 - 11.8%) [17]</td>
</tr>
<tr>
<td><strong>Biochemistry</strong></td>
<td></td>
<td></td>
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<tr>
<td>CK (U/L)</td>
<td>5 000</td>
<td>20 - 227 [12]</td>
</tr>
<tr>
<td>LDH (U/L)</td>
<td>2 000</td>
<td>0 - 450 [12]</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td>90.0</td>
<td>88.4 - 123.5 [22]</td>
</tr>
<tr>
<td>BUN (mmol/L)</td>
<td>6.2</td>
<td>4.3 - 14.3 [22]</td>
</tr>
<tr>
<td>Se (µg/L)</td>
<td>180</td>
<td>120 - 500 [15, 16]</td>
</tr>
<tr>
<td>Vitamin E (mg/L)</td>
<td>ND</td>
<td>0.8 - 3.0 [8, 15, 16, 20]</td>
</tr>
</tbody>
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After euthanasia, the histopathological examination confirmed the white muscle disease (WMD) suspicion with variation in muscle fibre diameter (thin arrow), fibre splitting (thick arrow) and increased number of nuclei under the sarcolemma (short thick arrow). Haematoxylin-Eosin, X 240.
Discussion

The clinical findings were compatible with WMD, except for cervical hyper-extension that could be confused with opistotonus caused by CNS disease or intoxication. A cerebellar disease and secondary rigidity could lead to opistotonus with normal awareness, thoracic limb extension and flexion of the pelvic limbs up under the body [1, 13]. The primary or toxic cerebral disease was discarded because awareness and the cranial nerve function were normal and also because both brain and cerebellum were normal on histopathological examination. Although cervical muscles can be affected by nutritional myo-degenerescence [10, 26], clinical signs compatible with severe focal myopathy (cervical opistotonus or neck extension) were not seen to date.

The whole blood selenium concentration was within the normal reported limits, while vitamin E was severely depleted (no detectable concentration) [15, 16]. In this case, the white muscle disease can be only attributed to the vitamin E deficiency. This finding was uncommon compared to the results of previous studies conducted in Greece [4, 16]; a pure vitamin E deficiency is rarely reported as the sole cause of myodegeneration, both in field [11, 15] and experimental studies [24], and was particularly involved in the sub-capsular liver rupture in lambs [5]. However, this specific lesion was not found in the present clinical case.

Vitamin E deficiency could result from a low relevant concentration in the ewe milk as well as in dried alfalfa hay and concentrates that were consumed from the lambs and their mothers. It was reported that colostrum contained much more vitamin E than milk [7, 19]. According to different studies [6, 15, 18, 27], the stored feedstuffs have reduced α-tocopherol contents, and the esterified form of the vitamin E in the commercial concentrates is inadequate for the requirements of the rapidly growing lambs, since sufficient amounts are not added due to the high cost. Moreover, the concentration of polyunsaturated fatty acids (PUFA) in ewe’s milk could be a critical point for the white muscle disease clinical expression [11]. Indeed, a massive PUFA supply coupled to deficiency of antioxidants (such as selenium and vitamin E) can lead to occurrence of an oxidative stress and secondary membrane lipid peroxidation, leading to cellular degenerescence [9, 21]. As selenium and vitamin E concentrations can be partially each other compensate for mild deficiencies, only severe deficiencies generally induce the clinical disease [18, 27]. In this way, when vitamin E was severely depleted like in the present case, the normal blood selenium concentration is not sufficient for counteracting the primary deficiency and preventing the clinical expression of the WMD.

As a conclusion, as illustrated by the present clinical case, cervical hyper-extension can be a clinical symptom of the white muscle disease in lambs and the myo-degenerescence can be induced by a severe vitamin depletion not combined with selenium deficiency.

References