COBALT DEFICIENCY IN PASTURED SHEEP IN THE SOUTH-WESTERN CAPE PROVINCE

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ABSTRACT

Annually recurrent illthrift and mortalities in a small flock of pastured sheep at the Regional Veterinary Laboratory, Stellenbosch were cured by the supplementation of cobalt. The similarities between acute cobalt deficiency and ovine white liver disease are discussed.

Key words: Cobalt deficiency, sheep, illthrift, white liver disease

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Routine daily inspection of a small flock of South African Mutton Merino/Dorper crossbred sheep kept on mixed dryland pasture consisting mainly of kikuyu (*Pennisetum clandestinum*) grass at the Stellenbosch Regional Veterinary Laboratory revealed unexpected loss in body condition in ewes, as well as illthrift and mortality in lambs. A commercial protein/mineral sheep block (Rumevite Sheep Block, Rumevite/Agricura Animal Production) containing inter alia 8 mg cobalt per kg, was available ad lib. The consumption of these blocks was lowest during the spring months when the quality and quantity of the grazing was best, and highest in autumn when poor grazing prevailed.

Since 1976 it had annually been noticed, paradoxically, that the sheep were in best condition during autumn when the grazing was dry and of poor quality, and in poorest condition when they were on the lush spring grazing. Ewes were usually in excellent physical condition when lambing, between April and July, and the lambs usually grew well during the first 6-8 weeks after birth. Lambs born in the latter part of the period were affected at a younger age than those born earlier in the lambing season. By October/November most lambs were unthrifty and pot-bellied. From 1977 to 1979, 22 out of 54 lambs born, died between the ages of 1 to 7 months.

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Most lambs wasted away gradually. Some, however, died within 2 d of first showing signs of stiffness and lethargy, and a small number died acutely while in good condition and without having shown any clinical signs of disease. In addition to the poor condition, drooping upper eyelids, a serious ocular discharge as well as a mild diarrhoea and anaemia were seen clinically in most subacute and chronic cases. Recumbency, nystagmus and icterus were seen occasionally, but photosensitivity was not observed. From about February each year, all the sheep began to improve. This improvement was more marked in adult than in young sheep.

Post mortem investigations on 22 lambs that died or were euthanased in extremis, revealed that 14 were emaciated while the condition of the remainder varied from reasonable to good. Other changes were non-specific; the most important of which was a slightly enlarged, pale, sometimes yellowish, friable liver (found in 14 cases). The changes in the liver were more prominent in the acute cases. Pulmonary oedema and congestion were also more often seen in acute cases.

Chronic cases manifested ascites and a mild hydrothorax which was usually associated with localised atelectasis and pneumonia. Most chronic cases were anaemic while a slight icterus was seen in 2 of the acute cases. No blood parasites were observed on examination of peripheral blood smears.

Organs from 21 of the 22 lambs were examined histopathologically. One of the lambs was in an advanced stage of decomposition. Moderate to very severe fatty changes (n=18), mild to moderate bile duct proliferation (n=20), mild peribilary fibrosis (n=5), a brownish-grey pigment in the cytoplasm of the Von Kupffer cells (n=11) and mild to moderate splenic haemosiderosis (n=9) were observed in liver specimens. A moderate nephrosis was found in 2 cases. Copper, iron, zinc and manganese concentrations in the liver specimens assessed by atomic absorption, and serum concentrations of phosphorus and magnesium were found to be within the normal range.

As cobalt deficiency was considered to be a possible cause of the illthrift and since laboratory assays for cobalt or cyanocobalamin were unavailable to us at that stage, it was decided to determine the effect of cobalt supplementation administered to a group of the ewes and their lambs.

A flock of sheep (n=30) was divided into control (5 ewes, 5 young ewes, 6 lambs) and treatment (5 ewes, 9 lambs) groups. The mean body mass per lamb (23,65 kg) was approximately the same in each group. All animals grazed together. For a period of approximately 3 months, the treatment group was dosed with cobalt chloride at a dosage of 7 mg of cobalt per ewe and 5 mg per lamb per week. A protein/mineral sheep block (Rumevite Sheep Block) was available ad lib. After the initial period of 3 months, all animals were supplemented with cobalt. For the first 6 months, 7 mg of cobalt was given per os weekly. Thereafter all lambs were given 2 ml of hydroxycobalamin (Neocytamin injection, 1000 mcg per ml. Milvet Ethicals) subcutaenously at the ages of 2 weeks and 2 months. All sheep older than 4-5 months, received one "Cobalt Heavy Pill for Sheep" (Top Brand, Adelaide+Wallaroo Fertilizers Ltd. Australia) per os by means of a special dosing gun.

At the end of the first month, the average mass per lamb in the treatment group was 3,3 kg more than that of the lambs in the control group. After the second month, the average mass of the lambs in the treatment group was 6,7 kg more and after another month, it increased to 14,8 kg per lamb.

Two of the 6 lambs in the control group became lethargic and emaciated and one died. The second one was euthanased in extremis. The clinical signs and

pathological changes seen at necropsy, were similar to those of the natural cases described earlier.

After supplementation of cobalt, the average mass of the surviving lambs in the control group increased from 28,8 to 38,7 kg over a period of 40 d. Since the cobalt/vitamin B_{12} supplementation was implemented, the illthrift and related mortality in the flock have disappeared completely. The mortality figure for lambs aged between 1 and 7 months was 22 out of 54 (40,7%) for the years 1977 to 1979. For 1980, the year of the experiment described in this report, it was 2 out of 15 (13,3%) and for the years 1981 to 1989, the mortality figure was 2 out of 308 (0,45%).

Although this small diagnostic trial has many scientific shortcomings, e.g. the small number of lambs per group, the method of dividing the lambs into the 2 groups, the fact that sex was not taken into account and the fact that twin lambs had to be in the same group, the dramatic response obtained by cobalt supplementation suggests strongly that cobalt deficiency was the primary cause of this illthrift problem and the related mortalities. The diagnosis is further supported by the improved condition of the lambs and the dramatically-reduced mortality figures after continuous cobalt supplementation was implemented.

The use of cobalt pellets is preferred, because the weekly dosage of cobalt to pastured sheep is unpractical, the intake of licks by sheep unreliable (especially along the coast), and the top-dressing of cobalt on pastures only economically justifiable in high production, intensive grazing systems[8]. Problems encountered with this method of cobalt supplementation are the loss of these pellets by the animal, and the development of an impervious coat, mostly calcium phosphate, encapsulating the pellet[8]. A steel screw administered with the pellet or 2 pellets together, has been used successfully to reduce the formation of this coating[8].

In our experience, the cobalt pills are not readily lost, as we have recovered all at slaughter, in some cases up to 5 years after administration. In about 30% of cases, however, the cobalt pellet had become coated with a hard, greyish-brown layer which can be expected to reduce or prevent the release of cobalt in the rumen. Contrary to reports in the literature, our attempts to prevent formation of this coating by administering a 10x12,5 mm grub screw with the pellet, was unsuccessful in many cases.

The following factors are possbily responsible for the seasonal occurrence of clinical signs of cobalt deficiency under our conditions in spring and early summer:

1. Soil contamination of the grazing is greater during late summer and autumn months, when the grazing is short and dry, than during winter and spring months, when the grazing is lush and fast-growing. This is important since most soils contain more cobalt than the plants growing on them and soil may constitute 10-25% of total dry matter intake in sheep when grazing is short and dry[6].
2. The cobalt content of fast-growing, lush grazing plants in spring is lower than that of plants during the dry summer and autumn months[2].
3. We have found that the intake of the protein/mineral supplement is always perceptibly higher during the dry months than during spring when the grazing is lush.

Ovine white liver disease (WLD) which has been described in New Zealand[1 2 3 6], Australia[6] and the United Kingdom[4 5], occurs mainly in late spring and in most cases affects young sheep of 3 to 6 months of age manifesting clinical signs of illthrift and mortality. Low concentrations of serum vitamin B_{12} and liver cobalt as well as pale fatty friable livers, are consistent findings in this condition. Histopathologically, parenchymal fatty change, bile duct proliferation and ceroid pigmentation are characteristic[6].

McLoughlin et al.[5] found lesions in the liver resembling ovine white liver disease in cobalt-deficient lambs and Mitchell et al.[6] agreed that the difference between WLD and cobalt deficiency might not be absolute. As our findings are similar to those of these authors, we are at this stage in agreement that it is not possible to distinguish between acute cobalt deficiency and white liver disease on the basis of these lesions.

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