

Selenium Responsive Dermatitis in Cattle

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A yearling Maine-Anjou heifer was admitted to the Iowa State University Veterinary Teaching Hospital for diagnosis and treatment of a clinical problem characterized by weight loss, extensive hair loss and multiple 2-5 cm skin lesions around the face, poll, ears, neck, back and rump regions. Less extensive lesions of similar appearance were also found on the legs and underline. Grossly, many of the lesions were ringworm-like in appearance with prominent, whitish, asbestos-like accumulations of scaley material on the surface. (Fig. 1)

History

The skin problem was first noted after the animal had been taken to a cattle show and later sold. The condition was first thought to be of minor significance and lesions were confined to the head region. Treatment with topical preparations was ineffective however, and the lesions gradually spread over the following two months to include most of the topline and sides of the animal.

Additional therapy with nutritional supplementation including zinc, vitamins A, D, E and B12 and surface application of Ivermectin had failed to bring about improvement.

Clinical Examination

On clinical examination the animal was found

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to be afebrile but somewhat inappetent and depressed. Some signs of pruritis were observed. Respiratory sounds and rumen function were determined to be normal but appetite and eliminations were slightly diminished. The hemogram was found to be normal except for a slight elevation in fibrinogen(600 mg/dl). The history and clinical findings in this case were similar to those observed in a bull of the same breed that had been admitted two years previously was found to have marginal serum selenium levels (60ppb). The bull had responded favorably to selenium therapy. It is known that dermatologic lesions are sometimes associated with selenium deficiency(1,2,3,4) and therefore was considered a differential diagnosis in this case.

Initial clinical chemistry of the heifer revealed slightly decreased serum magnesium (1.30 mg/dl), not thought to be clinically significant, and a decreased urea nitrogen (7 mg/dl). Other findings were normal except for the serum selenium which was markedly decreased (20 ppb), levels below 60 ppb being considered deficient. Serum electrophoresis gave near normal results with only slight increases in beta and gamma globulins.

Histopathology

Several skin biopsy samples were collected and submitted for histopathologic evaluation. The findings included hyperplastic dermatitis with parakeratotic hyperkeratosis. (Fig. 2) The hair follicles contained numerous mycotic arthroconidia and hyphae in hair shafts and follicular keratin. (Fig. 3) The fungal agent was later identified as *Trichophyton verucosum*.

Treatment

Initial therapy consisted of extensive grooming to remove the crusty superficial lesions and bathing with soap and water followed by topical application of common antifungal preparations. These included organic iodine and thiobendazole, the latter applied as a paste

containing 10% dimethyl sulfoxide (DMSO) solution.

Selenium was injected subcutaneously in 25 mg, 15 mg and 15 mg doses respectively over a 30 day time span. Serum selenium increased to 80 ppb following the first treatment and to 110 ppb following the second treatment. The third treatment was given at the time of dismissal. At that time the animal was improving as evidenced by regression of the skin lesions and regrowth of hair. The appetite was increasing and the physical demeanor was much improved. Weight gain which had been absent at the time of admission had improved to approximately 1.1 Kg/day.

Discussion

In the two cases under discussion it did not appear that either animal had been on a selenium deficient ration. It is likewise not established that selenium deficiency alone was responsible for the development of the dermatosis that was observed. It did appear however, that in both cases the condition was selenium responsive although other ancillary treatments were also used. No response was observed to other treatments that had been used prior to the administration of selenium.

It is not known if these two animals were related genetically, but both were of the same breed and from the same region. Less severe signs were also said to be present in half-siblings of the second animal, however, those cases were not available for study.

It is also known that similar clinical signs reoccurred in the first case (a three year old bull) after a period of approximately 18 months of being asymptomatic.

It does seem to be common knowledge that some animals become selenium deficient even though the ration would appear to contain adequate selenium¹. Indeed that information is implied on product literature that is supplied with injectable selenium compounds. It may be that selenium becomes unavailable due to complexing with other ingredients as is the case with magnesium for example. Furthermore, some animals may have an inherited or otherwise acquired inability to absorb selenium from certain kinds of feedstuffs.

References

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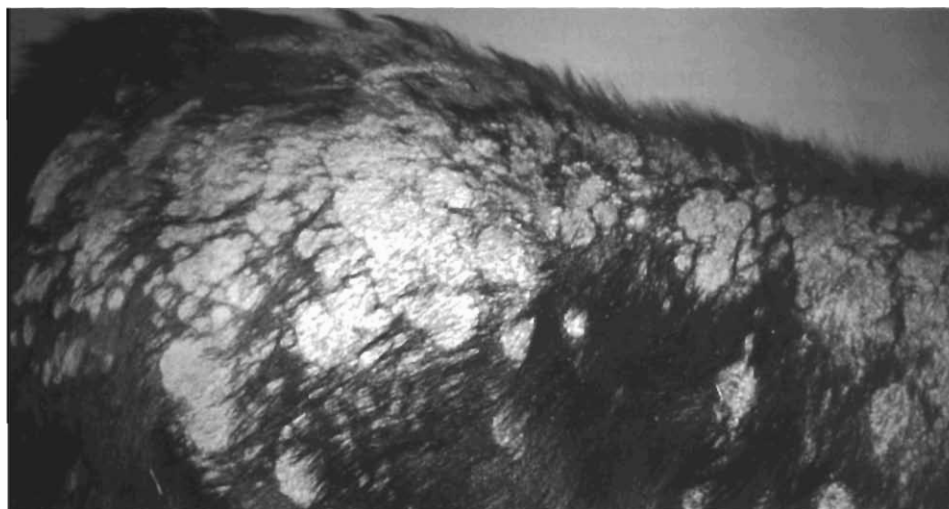


Fig. 1

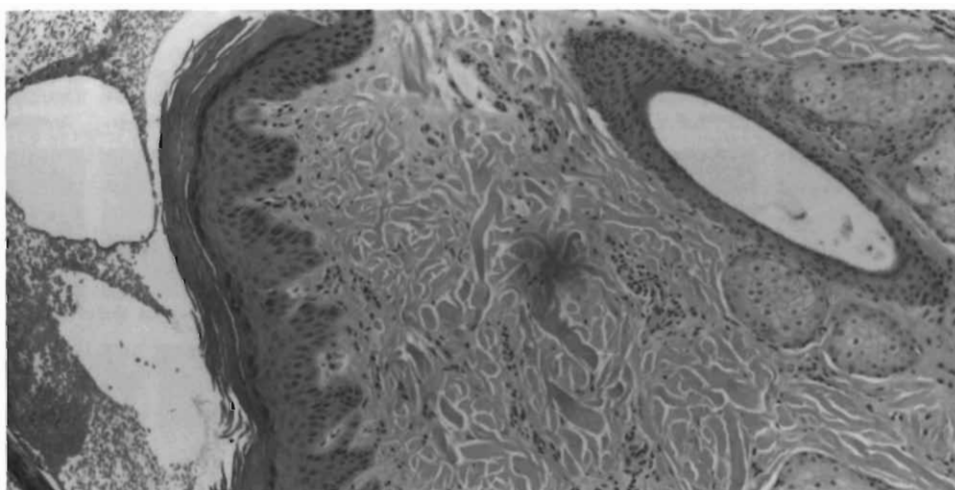


Fig. 2

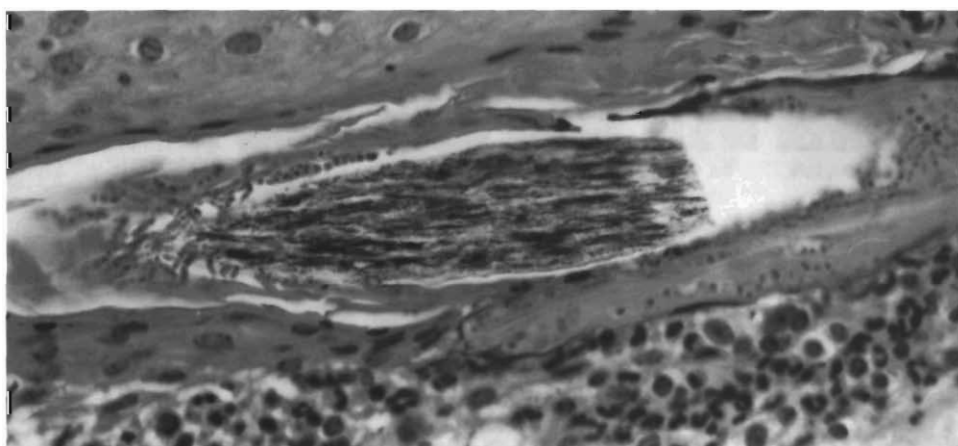


Fig. 3