SUCCESSFUL TREATMENT OF LEUKODERMA IN A BUFFALO HEIFER WITH PROLONGED ADMINISTRATION OF COPPER SULPHATE

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Case History, Clinical Examination and Diagnosis

On 22.8.97, a two years old Nili-Ravi buffalo heifer attended the outdoor clinics of Department of Clinical Medicine and Surgery for the treatment of white patches on the body. History revealed that the condition had started a month ago. Depigmented white patches appeared first on dorsal aspects of the body along the vertebral column, spreading later to involve much of the lateral and ventral thorax, abdomen, prescapular region and hind legs. About 40\% of the body surface was depigmented. The hair in the affected area had blanched to whitish color. A fresh green fodder of sorghum plus maize constituted the major component of the diet of the animal till late November. Subsequently the animal was maintained on berseem \((Trifolium alexandrinum)\) plus wheat straw. Pretreatment haematological profiles were as follows: ESR 20/lst hour; haemoglobin 13 g/dl; PCV 27\%; RBC count 4.75 millions/cmm; and WBC count 5800/cmm. A diagnosis of leukoderma was reached on the basis of typical clinical signs (Radostits \textit{et al.}, 1994; Smith, 1990).

Treatment and Treatment Response

The animal was administered 1 g of powdered copper sulphate in 250 ml of water daily for 15 days. No medication was undertaken for the next 15 days. This interrupted medication was continued for 7 months when a total of 6 rounds of treatment were completed. At around day 60 post initiation of treatment, the white patches started turning pinkish grey and black spots started appearing in the affected areas. There was rebounding of the signs when the owner suspended the treatment for one month. The complete resolution of the depigmented areas occurred in 7 months. The ventral patches were last to resume their normal color. The treatment was not associated with any side effect.

DISCUSSION

Leukoderma, also called acroderma or hypomelanosis, is an acquired defect of body pigmentation characterized by partial or total loss of melanin pigment from the skin. This condition may result from genetic abnormalities, trauma, inflammation, dietary imbalances, hormonal influences and immunologic disorders. In some instances, leukoderma is idiopathic (Moschella and Hurley, 1985). Dietary abnormalities, particularly molybdenum toxicity and copper deficiency are reportedly associated with faded or washed out coat colour in food animals (Radostits \textit{et al.}, 1994). Copper deficiency causes leukoderma since this element along with tyrosine (an amino acid) is required for the production of melanin. The copper deficiency in the subject of the present report might have resulted from molybdenum toxicity since soils in Punjab have been shown to carry excess levels of this element (Dhillon \textit{et al.}, 1992). The forages grown on such soils may accumulate molybdenum which may interfere with the absorption of copper from the gut (Radostits \textit{et al.}, 1994). Prolonged administration of copper sulphate might have affected a cure in the case reported here by alleviating/mitigating the state of this conditioned hypocuprosis. Procedure of alternate copper sulphate medication and no medication was adopted in view of the known cumulative effect of copper sulphate administration. The efficacy of copper sulphate in the treatment of leukoderma in buffalo is consistent with the observations of Dhillon \textit{et al.} (1991).

Our contention that the resolution of leukoderma was the result of copper administration and not due to self cure is supported by the rebounding of the signs when the administration of copper sulphate was stopped prematurely by the owner. Protracted oral administration of copper sulphate over months to affect a cure in leukoderma warrants testing the efficacy of parenteral preparations of copper (e.g. copper glycinate) to save time and labour.
REFERENCES
