

HAIR FOLLICLE ATROPHY PREVENTS SPEEDY RECOVERY FROM SEVERE SARCOPTIC MANGE

SIR: Severe cases of sarcoptic mange in goats results in thickening of the skin, production of scabs and an extensive hair loss throughout the body (Zamri-Saad *et al.*, 1990). These are due to the feeding activities of the mite in the skin resulting in microscopic changes described earlier in goats and pigs (Sharma-Deorani and Chaudhuri, 1965; Sheahan, 1975). This paper describes the microscopic changes that prevents speedy recovery of sarcoptic mange lesions following ivermectin treatment.

Eight adult local kambing kacang goats with various degrees of sarcoptic skin lesions were used. The goats were divided into four groups based on the severity of their skin lesions as described earlier for pigs (Martineau *et al.*, 1984). Three subcutaneous ivermectin injections at two weekly intervals at the rate of 0.4 mg/kg were given to all goats. Prior to and at weekly intervals following the ivermectin treatment, 0.5 cm diameter of skin biopsy specimens were obtained from the edge of the mange lesions of each goat. Samples were immediately fixed in 10% formalin, sectioned at 5 µm and stained with hematoxylin and eosin.

Four goats had mild to moderate skin lesions and the remaining four were showing severe lesions. The encrustations which characterised the lesions varied in their thickness; severe cases had the thickest crust with focal accumulations of pyknotic mononuclear leucocytes. Mites were observed frequently beneath the crusts. Mild and moderate cases had thinner crusts and occasionally foci of pyknotic mononuclear leucocytes but mites were not observed.

All affected goats had similar degree of thickened epidermis with obvious parakeratosis and rete-ridge formations. Several sections from the severe cases had mite tunnels that were lined by flattened parakeratotic cells in the epidermis; some tunnels communicated with the dermis. There was scanty infiltration of lymphocytes and macrophages in the dermis which appeared to be more prominent in the severe cases. Many atrophied hair follicles which were markedly dilated and were lined by flattened and atrophied follicular cells were observed in the severe cases. Occasionally there were necrotic debris in the lumen of the affected hair follicles. The hair follicles of the mild and moderate cases showed no abnormalities.

There were marked microscopic improvements in the sarcoptic skin lesions following each ivermectin treatment particularly the severe cases. After 8 weeks of treatment, the crust and epidermis became thinner and the leucocytic infiltrations were observed only

around some major blood vessels of the dermis. The atrophied hair follicles, however, remained although other microscopic changes had regressed to almost normal.

The microscopic changes of scabietic skin observed in this study are similar to those observed earlier (Sharma-Deorani and Chaudhuri, 1965; Sheahan, 1975). One of the important microscopic changes is the atrophied hair follicles which was believed to develop as a result of blocked follicular canals (Sharma-Deorani and Chaudhuri, 1965). The follicular atrophic lesions remained even after the ivermectin treatment probably due to the longer period of time required for the removal of old hair root and recovery of the atrophied follicles before new hair regrowth can take place (Calhoun and Stinson, 1981). This results in a slower rate of hair regrowth, which may take more than 8 weeks after ivermectin treatment as observed earlier in the severe cases (Zamri-Saad *et al.*, 1990).

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