Lepr Rev (1983) **54**, 80

Abstracts

SEGGIE J & GELFAND M (1982) **Renal amyloidosis, a complication of tuberculoid leprosy and plasma cell dyscrasia: case reports of three patients.** *The Central African Journal of Medicine* **28** (5), 105–110

The authors describe 3 major categories of amyloidosis, the first associated with immunocytic dyscrasias, the second complicating chronic infections and certain neoplasms, and the third occurring as a heredofamilial disorder. Renal amyloidosis may occur in all 3. Case histories are given of 3 adult African males presenting with classical features of nephrotic syndrome secondary to renal amyloidosis.

The first patient had been diagnosed as a case of tuberculoid leprosy 7 years previously, and initially had received treatment with dapsone. On admission he had gross signs of neglected leprosy – damaged cranial nerves (5th and 7th), bilateral interstitial keratitis, anaesthesia and severe clawing of all 4 fingers of both hands, and extensive neuropathic ulceration of both feet. Stool examination revealed ova of *S. mansoni*. The second patient had Bence–Jones proteinuria, and bone marrow biopsy confirmed a plasma cell dyscrasia. A rectal snip contained ova of *S. mansoni* and *S. haematobium*. The third patient also had Bence–Jones proteinuria but the cause of the renal amyloidosis was not established. An immunocytic dyscrasia was suspected.

The authors question the view that amyloidosis is a singularly rare disease in Africans, for in their experience it accounts for 3% of adult African patients with nephrotic syndrome compared with a figure of 7% in the UK. They comment that amyloidosis is not known to complicate tuberculoid leprosy but has an incidence of 6–8% in the lepromatous type. *Reviewer's Comments.* In describing the first patient in this series there is a statement that 'the ravages of tuberculoid leprosy were all too readily apparent'. However, a diagnosis of tuberculoid leprosy cannot be sustained on the evidence, for deposition of leprosy bacilli in both corneae and widespread fibrotic damage to nerves supplying face, hands and feet, are pathognomonic of late-stage lepromatous leprosy. My conclusion is that this patient's leprosy was borderline-tuberculoid (BT) when he was seen 7 years previously, and in the absence of adequate treatment, or because of dapsone resistance, his disease downgraded to subpolar lepromatous (LLs). Hence, contrary to the title of this paper, renal amyloidosis occurred as a complication of lepromatous leprosy.

W H JOPLING

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