MALIGNANCY IN PLANTAR ULCERS IN LEPROSY

Sir,

Carcinomatous transformation in trophic ulcers, though not very frequent, is a potential danger to leprosy patients. After retirement from active service with TLM in India and Bhutan I (RGR) had several short-term assignments in Bhutan, Bangladesh, and Tanzania. On these occasions I came across six cases of malignancy in plantar ulcers 3 of which were proved histopathologically squamous cell carcinoma. The rest showed strong clinical evidence of malignancy though it was not possible to take a biopsy. In some cases it appeared that this possibility is not given sufficient consideration in diagnostic and therapeutic procedures and sometimes it is overlooked. This is not altogether surprising as in some of the newer textbooks on leprosy,[1] malignancy in plantar ulcers is not mentioned at all or only parenthetically.

Condensed histories of 2 of the cases are given below:

Case 1

A hill farmer from West Bhutan, male, aged 63, with lepromatous leprosy of 37 year's duration, who had received DDS monotherapy for 19 years. He was BI 4·3+, negative after 10 years. He was treated mostly as an out-patient, occasionally as an in-patient. He had had trophic ulcers on the left forefoot (MTH area) on and off for 20 years. Conventional treatment with immobilization and protective footwear was only partly successful under the constraints of village life.

In August 1986 he was admitted to hospital with a large cauliflower growth of uncertain duration, which covered the whole left forefoot. There was secondary infection with foul smelling pus and involvement of regional lymph nodes. There was no decisive improvement despite adequate treatment. On 27 August 1986 a biopsy was taken from the edge of the ulcer and lymph nodes. Six weeks later there was a spread of infection and acute haemorrhaging. On 21 October 1986 a below the knee amputation was performed. On 22 October 1986 the result of the biopsy was received: well differentiated squamous cell carcinoma; lymph nodes show no evidence of malignancy.

Postoperatively the patient developed necrosis of the skin covering the stump and then hepatitis. His death occurred on 5 November 1986.

Case 2

A farmer from Eastern Bangladesh, male, aged 59, with borderline–tuberculoid leprosy of 5 year's duration, who had received irregular multidrug therapy for 4 years. His BI was negative. Treated mostly as an out-patient in a village clinic, and occasionally as an in-patient for ulcer treatment in a central hospital—the ulcer was of the left heel and of 4 year's duration. In November 1988 the ulcer was covered with hypertrophic granulation tissue. A biopsy showed hyperplastic squamous epithelium, nonspecific infiltration of the dermis by acute and chronic inflammatory cells; no malignancy.

The patient went to another centre for excision of the ulcer and returned 2 months later. The ulcer had not healed. He received further irregular treatment at the village clinic.

On 2 March 1990 the patient was admitted to hospital with a plantar ulcer on the left heel, 5·5 × 4 cm, covered with sluggish red, partly fungating granulation tissue overhanging at the edges. On probing the rough surface of the calcaneus foul smelling pus was present. The lymph nodes were enlarged and tender. A biopsy showed well-differentiated squamous cell carcinoma infiltrating the dermis.

Discussion

Duration of the ulcer is an important factor in the aethiopathogenesis of cancer in trophic ulcers. Constant tissue irritation due to frequent traumatization and/or chronic infection with or without osteomyelitis needs time to have its effect. The great majority of leprosy patients receive domiciliary or village clinic treatment, where they are sometimes seen irregularly, and ulcers do not receive much attention. It is essential that plantar ulcers are checked regularly and carefully by the drug delivery team so that the beginning of malignant degeneration is not overlooked.

Even though this happens in only a small percentage, those who get it face a fatal outcome. Epidemiologically this does not matter. But besides trying to control the endemicity of leprosy we should continue to care for the afflicted individual. The size of the problem is difficult to estimate. In Bhutan, e.g. I know of 3 cases out of a total of about 6000 leprosy patients. Assuming that 12% of all patients have ulcers this means that among 720 ulcer patients 3 develop malignancy, i.e. 0·4%. Applied to India with 4 million leprosy patients this would mean 480,000 ulcer patients with approximately 2000 cases with malignant degeneration. Even if this guess is grossly off the mark it merits closer scrutiny.

Further it would be worthwhile to investigate the following:

1 Duration of disease, duration and type of treatment.
2 Type of disease—does immune deficiency play a role?
3 Quality of nervous damage—Superficial, deep sensation? Loss of autonomous function? Loss of motor function?
4 Duration and location of ulceration. Type of ulcer treatment.
5 Other aspects of aethiopathogenesis.
6 Development of secondaries.
7 Consequences regarding prevention and treatment.
Is wide excision still advisable, or rather below the knee amputation?
Are prosthesis available, and what of rehabilitation?
For prevention every plantar ulcer should receive early attention and consistent treatment. Better still is the prevention of disability. The best prevention, of course, is early detection and treatment of every leprosy patient before disability can develop.

The possibility of malignant degeneration should be thought of and taught at every level of staff dealing with leprosy patients.

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