Unnecessary Laparotomy for Abdominal Pain and Fever due to Clofazimine

Sir,

A 24-year-old Indian man was admitted to hospital in October 1974 with acute lymphadenitis and severe erythema nodosum leprosum (ENL), which were his first manifestations of lepromatous leprosy. He was treated with rifampicin for 6 weeks and dapsone; prednisone was used to control the ENL which was causing extensive skin ulceration. One unusual manifestation of his reaction was the appearance of haemorrhagic blisters on, and around the margins of, his palms and soles, which rendered his skin prone to ulceration from the slightest pressure or trauma. For this reason, prolonged immunosuppression was necessary and this could not be achieved with acceptable doses of corticosteroids. On 8 November 1974 treatment was started with clofazimine in a dose of 200 mg daily, which was gradually increased, reaching 500 mg daily on 25 December 1974, as it was still not possible to withdraw steroids. At the end of January 1975 he began to experience a series of fevers which were attributed to ENL, and on 29 January 1975 he complained for the first time of sudden, severe epigastric pain. There was marked epigastric tenderness, but a gastrografin meal failed to show an ulcer or a perforation. Two subsequent bouts of fever were not associated with other detectable evidence of reaction but extensive investigations failed to show a cause for them. The second bout from 8–15 March 1975 was accompanied by severe generalized abdominal pain and tenderness. It was though that he might have a paracolic abscess secondary to a perforated duodenal ulcer and laparotomy was performed on 16 March 1975.

Laparotomy showed the intraperitoneal fat to be stained red. The liver, stomach, duodenum, gall bladder, pancreas, spleen, kidneys and large bowel were normal and within the mesentery and about the aorta there were firm lymph nodes that were coloured black. Frozen sections showed chronic low grade inflammation with intracellular crystalline structures. Histology of the mesenteric lymph node showed non-specific inflammation with few reactive follicles but prominent sinus histiocytes. The brown pigment crystals seen in frozen sections had dissolved in processing.

Clofazimine was withdrawn and there were no further attacks of abdominal pain or of unaccountable fever. The ENL was adequately controlled with thalidomide.

It seems likely that this patient's abdominal pain was caused by lymphadenitis, produced by clofazimine in high dosage over a period of 11 weeks. His fever may have been due to clinically undetected leprosy reaction, for example in a deep lymph node, but one must also consider the possibility that it was due to clofazimine. The case emphasizes that this valuable drug should be used in high dosage for only a limited period of time, and under expert supervision, before an alternative such as thalidomide is introduced.

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