Unless the adverse effects of CMF are recognised and combated its potential benefit on survival will not be fully realised.

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Dapsone-induced optic atrophy and motor neuropathy

Blindness is one of the most serious side effects of treatment with drugs. We report a case of a young man who became blind and developed weakness of the legs after inappropriate use of dapsone. This is the first report of optic atrophy in association with dapsone.

Case report

A 20-year-old healthy Sudanese man was convinced that a lipoma on his shoulder was a lepromatous nodule. To cure his "leprosy" he took dapsone 600 mg daily for 10 days. Eleven days later he was admitted to hospital with vomiting, dizziness, and blurred vision. He looked ill and had cyanotic mucous membranes. No other physical abnormalities were noted. Dapsone poisoning was diagnosed, though tests for methaemoglobinaemia were not done. His condition improved when he was treated with fluid replacement and ascorbic acid. Two days later he was completely well and was discharged. He was told not to take dapsone and reassured that he did not have leprosy.

He returned three days later with blurred vision, generalised muscular pains, and difficulty in climbing stairs. He denied taking dapsone or any other drugs since discharge from hospital. Visual acuity was reduced in both eyes to counting fingers only. There was bilateral restriction of peripheral visual fields. Haemorrhages and exudates were present in both fundi, but the optic discs were normal. There were no other cranial nerve lesions. He had severe weakness in both legs and obvious foot drop on the left. Tendon reflexes were symmetrically decreased in the legs, but plantar reflexes were normal. There was no sensory deficit.

Results of laboratory tests on second admission were: haemoglobin concentration 14·8 g/dl, white blood cell count $5{\cdot}4 \times 10^9$/l (5400/mm^3), and erythrocyte sedimentation rate 3 mm in first h. No reticulocytes were seen and the red-cell sickling test gave a negative result. Results of cerebrospinal fluid examination and liver function tests were normal, as were fasting blood sugar concentration and concentrations of plasma urea and electrolytes.

Dapsone-induced motor neuropathy was diagnosed, and this drug was also considered as a cause of his blindness. A month after admission the visual loss persisted and both optic discs became pale, suggesting optic atrophy. Some of the exudates and haemorrhages, however, had cleared. Neither vision nor weakness showed any improvement during six months of follow-up.

Comment

The predominantly motor peripheral neuropathy, absence of sensory features, and clear drug history strongly incriminate dapsone. Dapsone-induced peripheral neuropathy is well recognised, though the mechanism is unknown and diagnosis is based largely on history and examination.[1] In common with some reported cases our patient has not shown improvement after stopping dapsone.[2]

Dapsone was stopped five days before the onset of blindness and weakness, though the dosage of dapsone was much higher than normal.

In our patient retinal haemorrhages and exudates were seen on the second admission. Pale optic discs suggesting optic atrophy developed gradually, without preceding optic neuritis or papillitis. Optic atrophy has not been reported in association with dapsone. Brain haemorrhages and thrombosed vessels, however, have been noted at necropsy in a fatal case of dapsone poisoning,[3] and peripheral gangrene has occurred after administration of high doses of dapsone.[4] We therefore suggest that a vascular process may have caused the optic atrophy.

The absence of cerebellar and long-tract signs, the development of optic atrophy without preceding optic neuritis, and the tropical environment make multiple sclerosis extremely unlikely. Repeated clinical and laboratory examinations have not detected parasitic or other infections.

We believe that dapsone should be included with disulfiram, chloroquine, and clioquinol[5] as a cause of peripheral nerve damage and optic atrophy, though the risk of optic atrophy may be related to large doses of dapsone.

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Rifampicin-associated pseudomembranous colitis

Pseudomembranous colitis has been associated with most antimicrobial agents except antituberculous drugs. Rifampicin may not be immune,[1] though the diagnostic criteria in the only case reported did not prove the involvement of *Clostridium difficile*. We report a further case of pseudomembranous colitis, apparently related to the administration of rifampicin.

Case report

A 60-year-old man, who had previously been given only phenoxymethylpenicillin, was admitted to hospital with tuberculous meningitis. He was treated with isoniazid, ethambutol, and streptomycin for one week and then with isoniazid, ethambutol, and rifampicin. After two weeks he was given nystatin, flucloxacillin, and gentamicin for one week for intercurrent infections and then dexamethasone 12 mg/day for one week, after which it was gradually withdrawn over about five weeks. Diarrhoea developed one week after nystatin, flucloxacillin, and gentamicin were stopped and soon after the initial dose of dexamethasone had been halved. The diarrhoea resolved after three days but recurred five weeks later, when dexamethasone was withdrawn. Pseudomembranous colitis was diagnosed and histologically confirmed after two weeks. Rifampicin was stopped, dexamethasone was restarted in low dosage, and he was given two one-week courses of vancomycin 2 g by mouth daily separated by one week, during which his diarrhoea returned. Sigmoidoscopic appearances were unchanged after one week but almost normal after two weeks. The diarrhoea settled within three weeks and did not return.