Letters to the Editor

Extensive carbamazepine eruption with eosinophilia and pulmonary infiltrate

Sir,

Carbamazepine is a widely used anticonvulsant which not infrequently causes a rash. However, more severe reactions are less well recognized and may suggest glandular fever, other viral infections or lymphoma.1-6

A 15 year old girl developed erythoderma with oedema of the face and extremities 2 weeks after starting treatment with carbamazepine. One week after withdrawal of carbamazepine she was still unwell and had increasing dyspnoea and nonproductive cough. Abnormal findings were erythoderma rash with hypopigmentation and scaling, facial oedema, cervical lymphadenopathy, pyrexia of 40.2°C, tachycardia 145/min, tachypnoea 25/min, blood pressure 85/70 mm Hg and bilateral basal and mid-zone crepitations. Chest X-ray showed extensive bilateral basal shadowing, and blood gas estimation demonstrated severe hypoxia (PO2 5.8 kPa). The maximum white cell count was 32.9 x 10⁹/l and there was striking eosinophilia of 9.9 x 10⁹/l and occasional atypical lymphocytes on blood film. ESR was 15 mm/h. Liver enzymes were elevated. There was no evidence of bacterial or parasitic infection, and IgM antibodies to Epstein–Barr virus were not found. The antibody titre to measles was elevated at the same level (1/640) on two occasions 10 days apart. Histological examination of a skin biopsy showed moderate lymphohistiocytic infiltrate in the dermal papillae and around dermal blood vessels, with endothelial cell swelling and mild papillary oedema. These nonspecific features were consistent with the diagnosis of drug eruption. Subsequent patch testing using 20% carbamazepine (supplied by Ciba–Geigy Pharmaceuticals) produced macular erythema only at 72 hours, but there was no visible reaction in 10 control subjects (none of whom had been treated with anticonvulsants). Treatment was prednisolone 80 mg daily, reducing as all symptoms resolved over the following few weeks.

This patient has many features in common with previous reports of severe hypersensitivity reactions to carbamazepine.1-5 The diagnosis of rash due to carbamazepine was not doubted until the patient developed symptomatic pulmonary infiltrate, increasingly abnormal liver function, increasing leukocytosis and marked eosinophilia, at a time when her rash was decreasing. A viral infection was suspected but although the antibody titre to measles virus was elevated, this did not alter during the period of observation in hospital. Two previous patients have been reported in whom there was concurrent infection but, as in the present patient, the clinical picture was felt to be due to an adverse reaction to carbamazepine rather than due to the infection.1,2 Diagnostic difficulty is frequent as the clinical findings in patients with severe hypersensitivity reactions to carbamazepine have previously been reported to be similar to viral infections3 including glandular fever.4 A notable feature in our patient was marked oedema of hands, feet and face. As eosinophil mediators have a role in the development of cutaneous oedema,7 it may be relevant that the peripheral blood eosinophilia in our patient was greater than in most previously reported cases of carbamazepine reactions.

Patch testing is not generally considered useful for diagnosis of drug eruptions, but was positive in six of seven patients with carbamazepine reactions reported by Houwzerijl and colleagues.8 The macular erythema in the present patient would not be interpreted as a proven allergic reaction, although all controls were negative. The diagnosis must therefore rely on the constellation of clinical features described unless rechallenge is performed, and even that may not be conclusive.

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References

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