Letters to the Editor

Sarcoidosis and lymphoma: is there an association?

Sir,

I recently had occasion to review the literature on the association between sarcoidosis and lymphoma. This has been a topic for discussion in your journal in the recent past (Brennan *et al.*, 1983).

Due to the paucity of cases with both conditions, it is difficult to substantiate the theory that sarcoidosis predisposes to lymphoma (Romer, 1980, 1982). It has been proposed that it is the steroid treatment, rather than sarcoidosis itself, which is responsible for precipitating steroid sensitive lymphomas. This would not account for the predominance of Hodgkin's disease, as opposed to other lymphomas, in reported cases. It would support the average latent period of 2 years between discovery of the sarcoidosis and the lymphoma, but is less credible in those cases exhibiting a more prolonged interval. Up to 17 years has been previously recorded (Brincker, 1972). My interest was aroused by recently encountering a patient who developed a B cell lymphoma of the caecum 33 years after treatment for pulmonary sarcoidosis. This further strengthened my view that any such association between the two conditions is purely coincidental.

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Neuroleptic malignant syndrome successfully treated with amantidine

Sir,

The neuroleptic malignant syndrome occurs in

0.5–1% of patients exposed to neuroleptics, with a 20% to 30% mortality, and consists of hypertonicity of skeletal muscles, fluctuating consciousness, hyperthermia and instability of the autonomic nervous system (Szabadi, 1984).

We would like to report a case occurring in a 28 year old Chinese female, in whom the syndrome was unusually prolonged in spite of drug withdrawal, and who was successfully treated with amantidine.

The patient presented with pyrexia, generalized muscle rigidity, catatonia, brisk reflexes, and deterioration in conscious level. Ten days before admission, manic psychosis was diagnosed and she received haloperidol 2.5 mg i.m. twice, 10 mg t.i.d. orally for 3 days, and in the 4 days before admission, a total of 167.5 mg of haloperidol parenterally. She had a pyrexia of 38–39°C, sinus tachycardia (up to 144/min), marked pallor, diaphoresis and fluctuating blood pressure. No haematological, biochemical or microbiological abnormalities were present in blood or cerebrospinal fluid. Antinuclear factor was negative. Electroencephalogram (EEG) showed generalized delta waves.

No improvement was noted after stopping haloperidol and 3 weeks after admission, amantidine 100 mg b.i.d. was started. Within 24 hours, her conscious level became normal, the rigidity improved, the temperature fell to 37°C, and the pulse rate returned to normal. Amantidine was stopped after 17 days as she was asymptomatic. Within 24 hours, she became mentally withdrawn, the original symptoms returned and the generalized delta waves reappeared on the EEG. Amantidine was restarted and was eventually stopped 5 months later when the patient remained well.

Our patient's condition corresponds with that described in the literature but had an unusually prolonged course. The syndrome normally lasts for about 10 days (Buze & Baxter, 1985), but in our case it persisted for 3 weeks after neuroleptic withdrawal and relapsed after a 17 day course of amantidine. This may be related to the high total dose of haloperidol given (167.5 mg in 3 days) (Buze & Baxter, 1985), as the highest described in other cases was 40 mg in 3 days (Jalbut *et al.*, 1983).

Though the exact pathophysiological basis of the neuroleptic malignant syndrome is not known, many different drugs have been used (Buze & Baxter, 1985). We chose amantidine as it has few side effects and because of familiarity with its use. Amantidine, which increases synaptic dopamine availability (Stromberg