In the winter of 2002, a 29-year-old man was admitted to our intensive care unit. He had become unwell and confused over the preceding 2 days and had a grand mal seizure. On admission, he was febrile, tachycardic, hypoxaemic, and had a Glasgow Coma Scale score of 9/10. We intubated and ventilated him. Blood tests showed leucocytosis (21 × 10⁹/L, mainly polymorphonuclear leucocytes), serum creatinine 0·18 mmol/L, raised transaminases, and a creatine kinase of 1865 U/L. We did a lumbar puncture; opening pressure was 270 mm H₂O, and the cerebrospinal fluid (CSF) had no white cells, 4 red cells/μL, glucose 4·6 mmol/L, and protein 0·87 g/L. We did PCR for influenza and herpes simplex viruses and screened for salicylates, amphetamines, and cocaine; these tests were negative. There was no carbon monoxide in the blood. Brain CT showed no abnormalities, but MRI showed bilateral, symmetric lesions in the thalami and hyperintense changes on T2-weighted images in the midbrain, anterior pons, and medulla tegmentum; there were asymmetrical changes in the frontal cortices, left hippocampus and uncus, and right temporal cortex (figure A, B). No contrast enhancement was seen and the cerebral veins appeared patent. A throat swab taken on admission cultured influenza A/Moscow/10/99 (H3N2) virus, a prevalent strain in New Zealand.

On the basis of our patient’s presentation, culture results, and the MRI showing such widespread vasogenic and cytotoxic oedema, we diagnosed acute necrotising encephalopathy, secondary to influenza. Despite treatment with acyclovir, oeslamivir, and methylprednisolone, the patient remained unwell and required ventilation for a further 3 weeks. Complement fixation testing confirmed acute influenza A infection by a four-fold increase in antibody titre. Repeat MRI showed that the thalamic lesions which had been hypointense on T1 had become hyperintense (a sign of petechial haemorrhage), while some of the T2 signal abnormalities had partially resolved (figure C, D). Neurological recovery was protracted and when last seen, in May, 2003, he was staying in a rehabilitation home and was coherent but remained tetraparetic and dysarthric.

Acute necrotising encephalopathy was first described in a series of Japanese children who presented sporadically between 1979 and 1995. All the children developed coma and seizures following a short febrile illness; mortality was 28% with neurological sequelae in 63% of survivors. 148 Japanese patients developed this complication during the 1998–99 winter influenza epidemic; 130 were infected with virus, a prevalent strain in New Zealand.

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References