



## LETTERS TO THE EDITOR

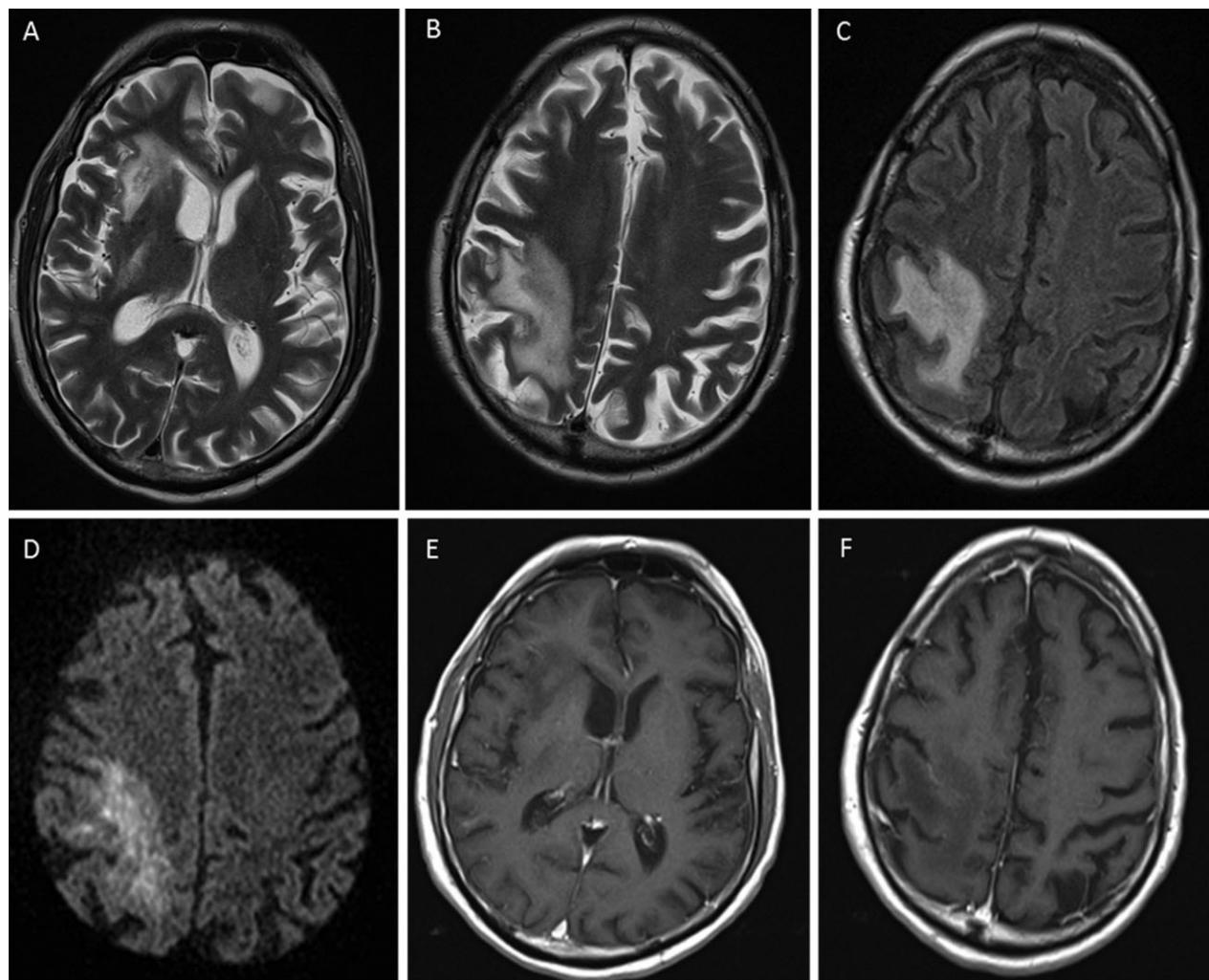
## Clinical-scientific notes

**Non-enhancing subcortical white matter lesions in central nervous system Listeriosis**

*Listeria monocytogenes* infection is a devastating disease, especially in high-risk groups, consisting of infants, pregnant women and immunosuppressed individuals.<sup>1</sup> Listeria is one of the most important organisms transmitted by the foodborne route.<sup>2</sup>

A 66-year-old, previously independent Caucasian man, was referred to the neurology service for acute onset left-sided hemiplegia and drowsiness 2 days after total colectomy. Four days earlier, he was admitted for a severe exacerbation of ulcerative colitis. There was no clinical response to the initial treatment with mesalazine, azathioprine, prednisone and a single dose of infliximab.

On clinical examination, he was conscious and drowsy with dense left-sided hemiplegia. Blood tests showed



**Figure 1** T2 and fluid-attenuation inversion recovery axial images (A–C) images shows a diffuse white matter high signal abnormality in the right subinsular region, posterior frontal and parietal lobes with sparing of the cortex. Restricted diffusion is identified (D). There is no enhancement in any of the described lesions on post-contrast images (E,F). There is effacement of sulci in the right posterior parietal region (F).

leucopenia of  $1.9 \times 10^9/L$  (neutrophils  $1.18 \times 10^9/L$ ) and anaemia with haemoglobin of 72 g/L. C-reactive protein was 129 mg/L. Computed tomography brain showed a hypodensity in the right fronto-parietal and subinsular region. Magnetic resonance imaging (MRI) brain revealed a diffuse high signal abnormality in the white matter of the right subinsular region, right parietal and posterior frontal lobe without perilesional oedema, with restricted diffusion, which were not typical of ischaemia, and did not demonstrate contrast enhancement (Fig. 1). Lumbar puncture showed clear cerebrospinal fluid (CSF) with  $37 \times 10^6/L$  white cells (polymorphs 4%, lymphocytes 96%), elevated protein (1000 mg/L) and low glucose (3.2 mmol/L; blood glucose 11 mmol/L). CSF Herpes Simplex virus 1 and 2, Cytomegalovirus, JC virus and Epstein Barr virus were negative. Blood and CSF culture grew *Listeria monocytogenes* sensitive to ampicillin. Antibiotic therapy with ampicillin at 2 g 4 hourly was initiated and planned for a period of 4 weeks. Two sets of blood cultures sent after 1 week of ampicillin showed no growth. The patient's sensorium improved with treatment. However, the dense left hemiplegia persisted. Unfortunately, the patient passed away 4 weeks later due to complications from aspiration pneumonia. On autopsy, cerebral tissue from the right fronto-parietal lobe showed *L. monocytogenes* on gram staining and culture.

Our patient was immunosuppressed and prone to developing *L. monocytogenes* meningoencephalitis and *Listeria* septicaemia. With *Listeria* septicaemia, cerebral parenchymal involvement can occur from cerebral invasion via endothelial cells of capillaries, and meningeal involvement can occur via epithelial cells of the choroid

plexus.<sup>3</sup> While cerebral abscesses, *Listeria* rhombencephalitis and *Listeria* encephalitis are well described in the literature, the imaging appearance of our patient's cerebral lesions was unique. The lesions involved the subcortical U fibres and white matter diffusely and did not enhance with contrast. There was minor perilesional oedema. There was no enhancement with contrast which is well described with *Listeria* abscesses and encephalitis, and there was no cortical involvement.<sup>4,5</sup>

The MRI findings of *Listeria* encephalitis in our patient were similar to progressive multifocal leucoencephalopathy with diffuse white matter lesions that do not enhance with contrast.<sup>6</sup> Another possibility is that our patient's lesion was an atypical subcortical early cerebritis stage of *Listeria* abscess formation. We acknowledge that the lack of enhancement of our patient's lesions could also have been due to chronic immunosuppression and ongoing steroid use. This case adds that involvement of only white matter with diffuse non-enhancing lesions is possible in central nervous system Listeriosis. Central nervous system Listeriosis should be considered in such patients with an appropriate clinical history, investigated and treated accordingly.

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