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Neuroimaging Highlight

Neurosarcoidosis-Induced Multiple Cerebral Microinfarcts

Charlotte Gallienne[1], Jian-Qiang Lu[2], Devin Hall[3] and Crystal Fong[1]

[1]Department of Radiology, McMaster University, Hamilton, Ontario, Canada, [2]Department of Pathology and Molecular Medicine/Neuropathology, McMaster University, Hamilton, Ontario, Canada and [3]Department of Medicine, McMaster University, Hamilton, Ontario, Canada

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A 58-year-old man with a history of coronary heart disease, ischemic cardiomyopathy, hypertension, and dyslipidemia presented with fatigue, left-sided headache, and mild confusion for 1 month. He was treated for presumed HSV encephalitis and improved after 3 weeks of acyclovir.

Two days following discharge, he returned with new onset ataxia and multiple falls. He was continued on acyclovir, and broad-spectrum coverage via vancomycin and ceftriaxone was initiated for suspected bacterial meningitis, given his unusual presentation. Despite these medications, his cognitive symptoms, namely confusion, short-term memory impairment, and disorientation in time, space, and person, progressively worsened. He developed paranoid behaviors and visual hallucinations. Cerebrospinal fluid examination revealed elevated protein (57.13 g/L), leukocytes (66×10^6/L), and glucose (5.2 mmol/L) with oligoclonal bands but was negative for malignant cells, HSV-1, HSV-2, VZV, enterovirus, fungi, cryptococcal antigen, acid-fast bacillus, and other bacteria. Electroencephalogram showed no seizure activity.

Initial contrast-enhanced MRI head demonstrated nodular leptomeningeal enhancement of the right temporal convexity sulci (Figure 1A, arrows), nonspecific FLAIR signal abnormality

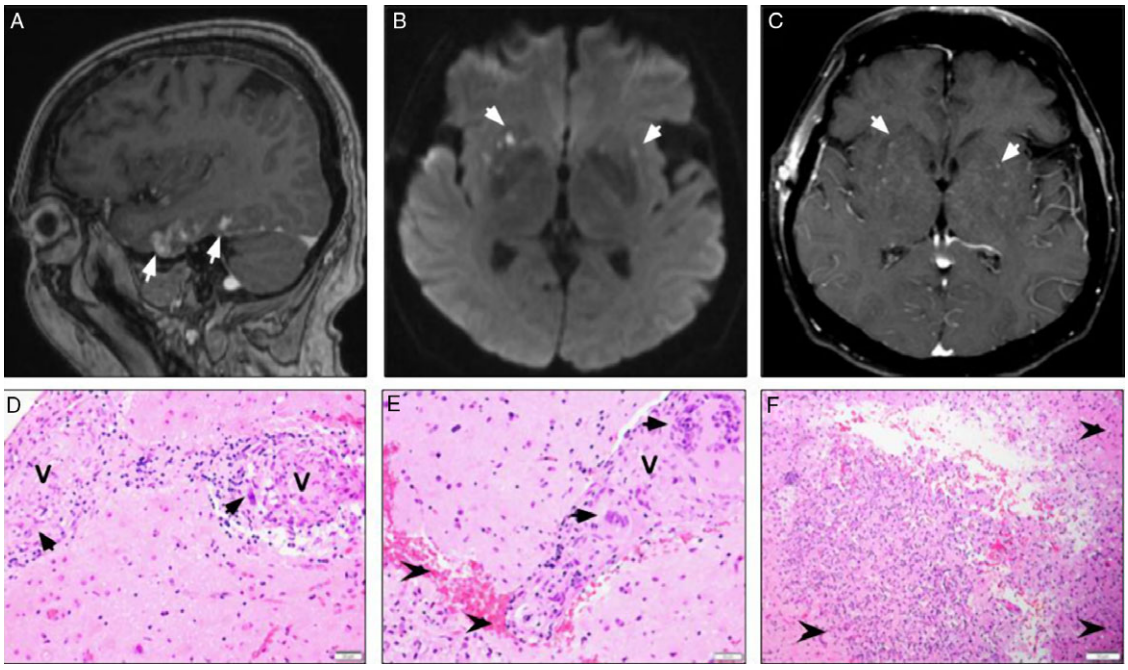

Figure 1: MRI and right temporal biopsy pathology images.

Corresponding author: Crystal Fong, Department of Radiology, McMaster University, Hamilton, ON, Canada. Email: crystal.fong@medportal.ca

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throughout the deep hemispheric structures bilaterally (not shown), and a few scattered punctate foci of restricted diffusion, not following any particular vascular distribution (not shown). At this time, differential considerations included a granulomatous process, based on the nodular leptomeningeal enhancement, or underlying vasculopathy, given the punctate micro-infarcts. Negative CSF analysis made tuberculosis, leptomeningeal carcinomatosis, or perivascular lymphoma less likely.

A right temporal biopsy exhibited multiple granulomas involving the leptomeningeal (Figure 1D) and cerebral (Figure 1E) blood vessels (marked with "V"), multinucleated cells in granulomas (arrows), and thrombosed cerebral vessels connected to subacute cerebral microinfarcts (E and F, arrowheads), suggestive of granulomatous inflammation causing multifocal cerebral infarcts.

Follow-up MRI 10 days post-biopsy confirmed the presence of progressive microinfarcts on diffusion-weighted imaging, now clearly involving a perivascular distribution of small vessels within the basal ganglia (Figure 1B, arrows) and central pons (not shown), with associated punctate perivascular enhancement on post-gadolinium T1-weighted imaging (Figure 1C, arrows). Ongoing leptomeningeal enhancement was seen in the right temporal lobe.

The patient was treated for a presumptive diagnosis of neurosarcoidosis with pulsed methylprednisone for 5 days followed by prednisone 60 mg daily but continued to have multiple falls. Methylprednisone was increased to 80 mg and subsequently to 140 mg daily to control his falls. Methotrexate was also initiated (10 mg weekly for 2 weeks, followed by 15 mg weekly). He showed marked cognitive improvement (MOCA performance changed from 16/30 to 22/30). Monthly infliximab infusions were initiated. After 5 months, his cognitive symptoms and leptomeningeal enhancement improved. Methylprednisone taper was initiated.

Our case has a few points of interest: diagnostic challenges given the atypical imaging and nonspecific clinical manifestation; the temporal leptomeningeal involvement as opposed to typical neurosarcoidosis involving the basal meninges[1]; multiple subacute to chronic microinfarcts rarely seen in neurosarcoidosis[2]; and the finding of neurosarcoidosis in the absence of other systemic involvement except mild lymphadenopathy within the mediastinum on imaging[1]. This case illustrates the diagnostic difficulty in atypical presentations of sarcoidosis, highlighting the need for vigilance in interpreting multiple seemingly unrelated findings on MRI.

Disclosures. The authors have no conflicts of interest to declare.

Statement of Authorship. Dr Charlotte Gallienne contributed to this case report by identifying the case and undertaking the chart review. Both Drs Charlotte Gallienne and Crystal Fong contributed to conceptualizing and drafting this manuscript and its intellectual content, as well as image acquisition and analysis. Dr Jian-Qiang Lu contributed by acquiring and reviewing the pathology slides, as well as conceptualization and drafting of this manuscript and its intellectual content. Dr Devin Hall contributed through review of the case, conceptualization and drafting of this manuscript and its intellectual content.

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