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Reversible transient lesion of the corpus callosum secondary to meningoencephalitis

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CASE STUDY

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ABSTRACT

A 40-year-old male, presented with right-sided weakness, ataxia, expressive dysphasia and was treated for cerebrovascular accident (CVA). However, he became increasingly confused, disorientated and reported worsening headache along with fevers, photophobia and neck stiffness. On further history, the patient's family reported the patient as being more confused and incoherent rather than having true ataxia and expressive dysphasia. CSF results were consistent with viral meningoencephalitis and MRI revealed a crescent shaped splenial lesion. He was treated empirically with ceftriaxone and acyclovir. On follow up, he remained symptom free and MRI showed regression of the splenial lesion. Reversible transient splenial lesions of the corpus callosum secondary to viral meningoencephalitis are rarely encountered. This case highlights the importance of exploring different aetiologies of splenial lesions to avoid misdiagnoses with more commonly seen conditions such as CVA.

Key Words
Reversible transient splenial lesion, meningoencephalitis, MERS, MRI, CVA

Implications for Practice:

1. What is known about this subject?
Reversible transient splenial lesion of the corpus callosum are rarely encountered on MRI findings and do not present with typical findings of hemispheric disconnection.

2. What new information is offered in this case study?
Meningoencephalitis can present as a rare cause of transient lesions of the corpus callosum.

3. What are the implications for research, policy, or practice?
Clinicians should remain vigilant when encountering a splenial lesion of the corpus callosum and look into different aetiologies to avoid misdiagnosing patients with more common conditions such as CVA.

Background

Reversible splenial lesion with transient alteration in intensity on MRI has been described in a handful of cases in the world. It is also known as mild encephalitis/encephalopathy with a reversible isolated splenium of corpus callosum lesion (MERS). There are a number of aetiologies that have been described, both neurologic and non-neurologic. This phenomenon often resolves on its own once the underlying aetiology is managed. Due to the rarity of the condition, its clinical picture can often be initially misdiagnosed as more commonly seen conditions such as CVA and migraine.

We report on a case where a 40-year-old male was initially thought to have a CVA but became a diagnostic dilemma due to his evolving clinical picture.

Case details

A 40-year-old male presented with right sided weakness, ataxia and expressive dysphasia. This was associated with a throbbing occipital headache. His background history included working as a glazier, a 13-pack year smoking

history, with no medical or surgical history of significance. He reported drinking alcohol socially and denied illicit drug use. On examination, there was reduced power on the right side with the rest of the neurological examination being unremarkable, including no ataxia or speech disturbance. In addition, there were no signs of hemispheric disconnection such as alexia, agraphia, apraxia or hemineglect. CT scan showed no acute haemorrhagic event. MRI scan showed a T2 hyperintensity and restricted diffusion on DWI involving the splenium of the corpus callosum and he was diagnosed with likely recent splenial infarct.

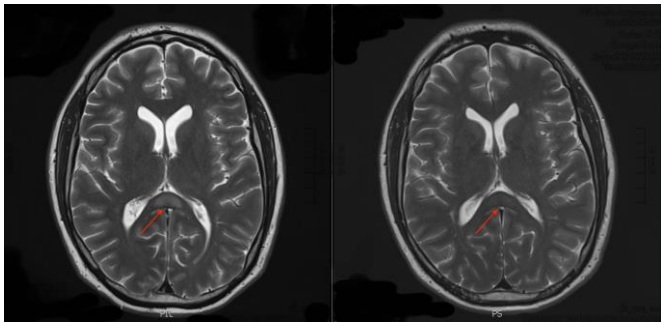
Two days into his admission, the patient developed photophobia and neck stiffness associated with a worsening headache. Altered mental status is noted with patient becoming increasingly confused, disorientated and incoherent throughout the admission. Right-sided weakness had resolved but he developed left-sided paraesthesia. On further history, the patient had flu-like symptoms with subjective high-grade temperatures one week earlier. Upon clarification, the patient's family reported patient as being increasingly incoherent, confused and disorientated rather than having true expressive dysphasia or ataxia. The onset of symptoms was thought to be more progressive rather than acute in the week leading up to presentation. On examination, he was febrile and neurological exam showed equal power bilaterally but with reduced sensation on the left side. No cerebellar signs were elicited. He also demonstrated nuchal rigidity and photophobia.

The provisional diagnosis at this stage favoured meningoencephalitis rather than CVA. His CTA and MRA showed no evidence of stenosis or irregularity to suggest vasospasm or vasculitis. There were no foci of demyelination demonstrated in the brainstem or cerebellar peduncles. In addition, both vasculitic and thrombophilia screens came back negative. A repeat MRI three days after admission showed the signal in the splenium of corpus callosum noted on T2 and DWI have reduced in intensity when compared to the initial scan. The patient subsequently underwent a lumbar puncture and the results were consistent with viral meningoencephalitis. Abnormal CSF results included elevated protein 1,900mg/L, WCC 472×10^6/L with 100 per cent mononuclear cells (94 per cent T-lymphocytes) and normal glucose 2.9mmol/L. The CSF sample consisted of normal IgG index with no oligoclonal bands detected.

This patient was reviewed by the infectious disease team and he was treated empirically on ceftriaxone and acyclovir and showed remarkable improvement and complete recovery within a week. The patient was followed up five weeks later and he had remained symptom free. His follow up MRI showed further regression of previous T2 hyperintensity in the splenium of the corpus callosum.

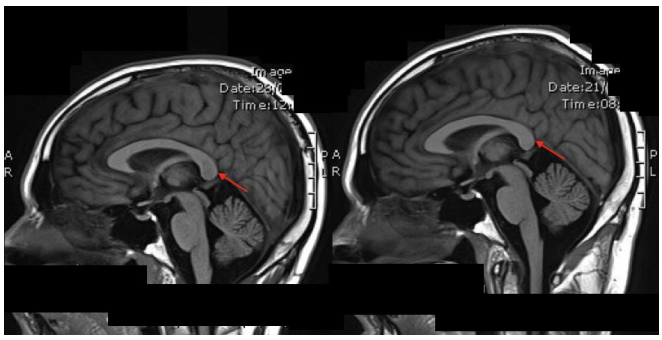
This case was discussed amongst different neurologists and neuroradiologists throughout the admission. Upon discussion, a diagnosis of reversible transient splenial lesion secondary to likely viral meningoencephalitis was made (Figures 1 and 2).

Figure 1: Axial T2



Axial T2-weighted MRI showed "boomerang" hyperintensity (left) involving the splenium of the corpus callosum. Repeat T2 MRI 5 weeks later showed regression (right) of previous intensity in the splenium of the corpus callosum.

Figure 2: Sagittal T1



Sagittal T1-weighted MRI showed hypointense lesion (left) of the splenium of the corpus callosum. Repeat T1 MRI 5 weeks later showed regression (right) of previous intensity in the splenium of the corpus callosum.

Discussion

Transient splenial lesion of corpus callosum differs in presentation to other causes of splenial lesion of corpus callosum. They do not demonstrate typical signs of hemispheric disconnection that one would expect, such as pseudo-neglect, alien hand syndrome, apraxia, alexia without agraphia, and visual apraxia.[1]

This patient was initially diagnosed with a likely splenial infarct after the splenial lesion was found on MRI. However, a splenial lesion found on imaging should not automatically trigger the diagnosis of CVA. There were several factors from the clinical history that pointed to a differential of encephalitis, particularly the preceding history of flu-like illness, fevers and altered mental status. More importantly, other clues from the clinical history include the more progressive nature of presentation which would favour the diagnosis of encephalitis rather than ischaemia where one would expect an acute onset of presentation. In addition, a splenial infarct can present with signs of hemispheric disconnection as mentioned earlier, and none of these were demonstrated on examination.[1]

The decision to repeat the MRI after the patient showed signs of meningoencephalitis proved to be a meaningful one. These radiological sequences have an important role in differentiating between ischaemia and other differentials. DWI imaging is particularly important in the evaluation of acute ischaemia as it is more sensitive than conventional T1/T2 sequences in detecting early small infarcts.[2] However, it can still be difficult to differentiate between an encephalitis and acute ischaemic stroke on DWI. Both of them have the characteristics of hyperintensity on DWI and reduced ADC values in the acute phase.[2] In acute ischaemia, the signal intensity on DWI increases during the first week and decreases following that, but the signal remains hyperintense for an extended period of time.[2] Restricted diffusion is usually less intense in encephalitis compared to infarction.[3] With regard to this case, the diffusion restriction noted in this patient's repeat MRI three days later has reduced significantly. Although it could have still represented an evolving infarct, it was more likely to be a transient splenium hyperintensity secondary to meningoencephalitis given the clinical findings. Having this initial control MRI to compare with allowed clinicians to define the progression of this lesion. As the patient's clinical condition became more apparent, it was becoming increasingly obvious this was a transient splenial hyperintensity rather than a splenial infact.

Other aetiologies of reversible transient splenial lesions have also been reported in literature, including epilepsy, antiepileptic drug usage, demyelination, hypoglycaemia, infections, adrenoleukodystrophy, AIDS dementia complex, lymphoma, ADEM, Marchiafava-Bignami disease.[1,4–6]

The typical appearance of transient splenial lesions on MRI is an ill-defined crescent shaped lesion extending across the splenium; also known as the "boomerang" sign.[1,4] It can also present as well circumscribed small oval lesions in the midline of the corpus callosum. Transient splenial lesions are visualized as hypointense lesions on T1-weighted images, hyperintense images on T2-weighted images and restricted diffusion of diffusion-weighted images. Bulakbasi et al. proposed that this transient signal is a result of intramyelinic oedema or inflammatory infiltrate of the splenium rather than a breakdown of the blood brain barrier or demyelination.[4]

Prognostic outcomes of reversible splenial lesions are dependent on its aetiology. In the case of viral meningoencephalitis, the prognosis is often promising. Bulakbasi et al. described five patients with transient splenial lesion secondary to influenza-associated encephalitis showed sudden-onset neurologic symptoms, including headache, new-onset convulsions, trigeminal neuralgia and left upper monoparesis.[4] All five patients reported prodromal flu-like episodes prior to the onset of symptoms. These patients demonstrated complete recovery with transient well-defined hyperintensities on MRI that completely disappeared on follow-up imaging. In addition, Tada et al. described 15 patients with reversible splenial lesion secondary to encephalitis recovered from their symptoms completely within one month.[5] The lesions had completely disappeared in all 15 patients at their follow-up MRI between three days and two months after the initial scan.

The presentation of acute disseminated encephalomyelitis (ADEM) can also resemble that of reversible transient splenial lesion. The difference lies within the quicker onset and recovery of neurological symptoms in reversible transient splenial lesion compared to ADEM. Takanashi et al. reported five patients with reversible splenial lesions developed neurological symptoms one-four days after onset of illness and showed completely recovery within 10 days.[6] In the case of ADEM, the symptoms can take weeks to months to manifest after the initial infection. When ADEM does involve the corpus callosum, the MRI findings are nearly always asymmetric in contrast to reversible transient splenial lesions which are often symmetrical. In addition, lesions in ADEM take months to disappear compared to these transient splenial lesions which can disappear within days to months.

Conclusion

Reversible transient splenial lesion is a significant but not non-specific clinical finding. This article highlights the rarity of this finding and it can often be misdiagnosed as CVA if it is not correlated with the clinical findings accurately. The

main differentials in this case are viral meningoencephalitis and CVA. The splenial lesion is mostly a confounding element in the differential diagnosis. When prompted with the aforementioned MRI findings, clinicians should be reminded to search for underlying aetiologies and commence appropriate therapy as soon as possible.

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PEER REVIEW

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CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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PATIENT CONSENT

The authors, *Yu A, Lee S,* declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.