MRI OF CEREBRAL CORTICAL VENOUS THROMBOSIS

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INTRODUCTION

Though stroke is most commonly related to atherosclerosis in the adult population, the causes of acute stroke in young patients are frequently due to rarer pathologies, for which treatments may differ. MRI has considerably improved the non-invasive visualisation of cerebral arterial and venous lesions and depiction of brain parenchymal changes. This case report illustrates the role of MRI in the assessment of the cerebral venous thrombosis.

CASE REPORT

A previously well 33-year-old woman presented with one week's history of intermittent throbbing headache associated with stuttering expressive and receptive dysphasia, each episode lasting less than three hours. She was referred to hospital as an emergency following an episode of dysphasia, which persisted. There were no previous illnesses. The patient was on the oral contraceptive pill (OCP).

Apart from her dysphasia, the rest of the clinical examination was normal. There were no other focal neurological signs. She had a microcytic anaemia of 66gm/dl (normal range 115-165g/dl), subsequently shown to be a combined nutritional deficiency of iron and vitamin B_{12}. Her blood biochemistry was normal. At lumbar puncture the opening pressure was 23cm CSF, with normal glucose and protein, two white cells and 19 red cells per cubic centimetre.

An MR brain scan showed vasogenic oedema and swelling within the white matter of the left temporal lobe, with high signal within the Sylvian vein on the cortical surface (Figure 1). The appearances confirmed the diagnosis of focal cerebral ischaemia secondary to cortical venous thrombosis.

The patient was advised to stop the OCP, and recovered with no further active treatment. Anticardiolipin antibody, antithrombin III, protein C, protein S, the lupus anticoagulant and Factor V Leiden were all normal. An MR brain scan two months later was also normal.

DISCUSSION

Thrombosis of the intracranial cerebral veins and venous sinuses is increasingly recognised as a result of heightened clinical awareness and also because of improvements in the quality and availability of brain MR scanning.

Various predisposing factors may increase the risk of cerebral venous thrombosis. In approximately 70% of the cases, it is due to acquired or inherited coagulation disorders, the most common inherited coagulation defect being Factor V Leiden mutation.[1] Causes of sluggish venous flow also predispose to thrombosis, including hyperviscosity syndromes and dehydration, particularly in premature infants, and diabetic ketoacidosis in adults. Damage to the vessel wall secondary to infection (e.g. mastoiditis), infiltration (e.g.meningioma) or trauma accounts for a further 10% of cases. The role of the OCP in cerebral venous thrombosis is probably multifactorial. In about 20% of cases, no obvious cause can be found. Usually a combination of factors contribute to the thrombotic event.

The clinical features vary depending on whether thrombosis affects predominantly the central deep veins, the cortical venous system, the major venous sinuses or combinations of these. The commonest syndrome is that of isolated involvement of one or more of the major dural sinuses, particularly the superior saggital sinus. This is associated with signs and symptoms of raised intracranial pressure (headache and papilloedema), occasionally cranial nerve palsies (particularly the VIth nerve) and transient visual field disturbances. Dural sinus thrombosis without cortical vein thrombosis does not usually result in parenchymal infarction, unless very extensive. Involvement of the cortical veins in isolation is very rare,[2] but usually results in one or more areas of venous infarction, with an associated neurological deficit, and a high incidence of associated seizures.[3] A depressed conscious level is a recognised feature, it may result from extensive or multiple cortical venous infarcts, or from brainstem compression as a result of swelling of the affected brain. Involvement of the central deep venous system, particularly the great vein of Galen, leads to deep coma and disturbances of eye movements and pupillary reflexes. Thrombosis of cerebellar veins, the draining transverse sinuses, or the sigmoid sinuses results in signs and symptoms of headache, vertigo, vomiting, ataxia and a more gradual onset of impaired consciousness.

The imaging features are frequently missed or misinterpreted, resulting in delayed diagnosis. Patients usually undergo CT first, and the changes are frequently misinterpreted as arterial infarcts, primary intracerebral hemorrhages, or tumours. A number of features on CT favour a diagnosis of venous over arterial infarction. These include: a well-demarcated hypo-intensity appearing early after the onset of symptoms (more rapidly than with arterial infarction); the presence of multiple lesions; prominent swelling of the lesion and also of the brain beyond the visible hypodensity, together with haemorrhage in the central part of the lesion.[4] Haemorrhage into venous infarction is frequent, usually central and 'finger-like'. With improvements in CT technology, involvement of major venous sinuses is increasingly accurately identified, although isolated cortical venous thrombosis is obscured by the high density of the skull vault on CT. High density may be seen in the venous sinus on a non-contrast scan due to clotted blood. The sinus fails to enhance following contrast, but is outlined by normal enhancement of the sinus wall and adjacent veins (the 'empty delta sign'). By using very thin slices with rapid

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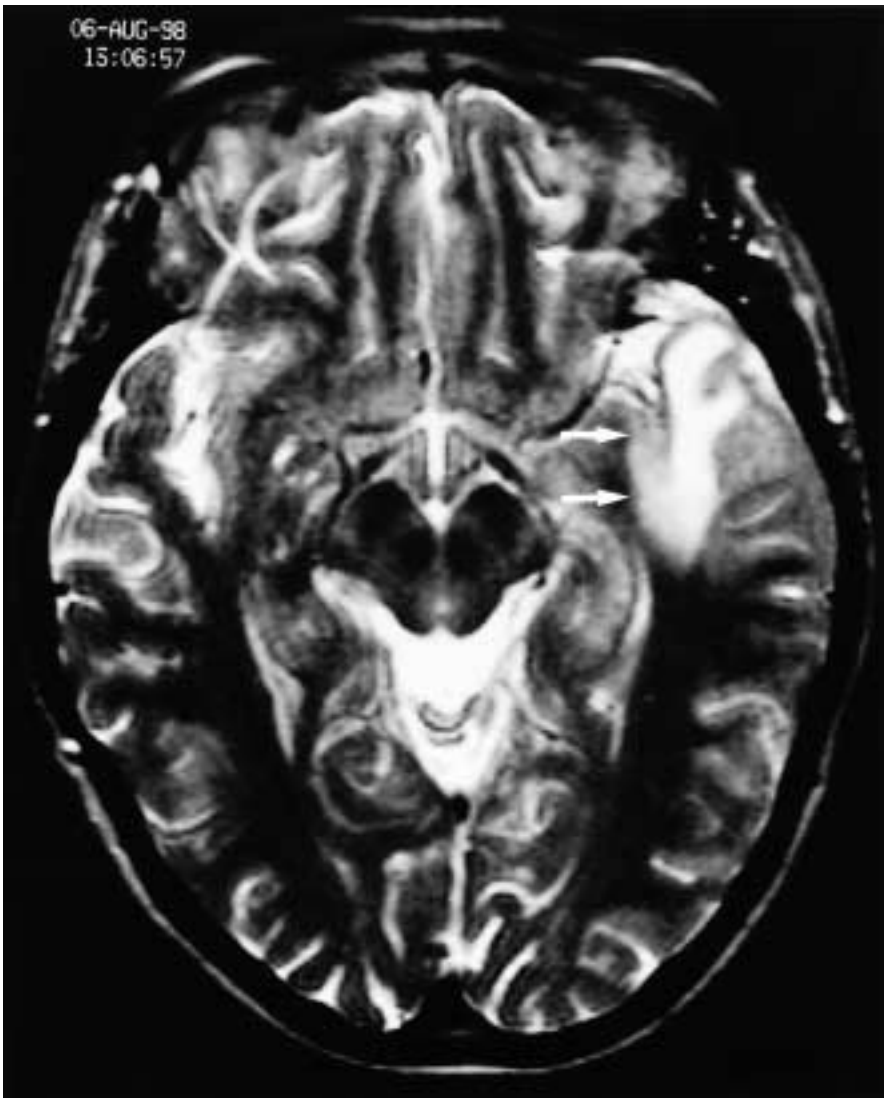

FIGURE 1A
T2 axial scan of temporal lobes shows grey and white matter high signal change and swelling in the left temporal lobe (arrows).

spiral CT scanning during contrast enhancement, a 'CT-venogram' can be obtained; this is a useful alternative means of identifying sinus thrombosis when MRI is not available.

The appearances of intracranial venous thrombosis on MR depend on the interval from the onset of thrombosis.[5] In the acute stage (days one to five) the thrombus appears strongly hypointense in T2-weighted images, and may not be distinguishable from normal flow on this sequence. On proton-density weighted images, the thrombus appears hyperintense relative to other tissues and is easily distiguishable. In the subacute stage (up to day 15), the thrombus signal is strongly hyperintense, initially on T1-weighted images (as in this case) and subsequently also on T2-weighted images. After three to four weeks, the thrombus signal becomes isointense on T1-weighted images but on T2-weighted images it remains hyperintense, though often inhomogeneous. The cerebral parenchymal changes of venous infarction are the same on MRI as on CT, with prominent swelling and frequent haemorrhage in the centre of the infarct.

The differential diagnosis includes septic emboli, early cerebritis, and viral encephalitis. The lesions may also mimic haemorrhagic primary cerebral tumours, or lymphoma. The key diagnostic feature is the direct visualisation of thrombus within a vein or sinus. In cases where the diagnosis remains uncertain, a delayed scan (of a few weeks) usually clarifies the diagnosis.

Treatment is centred on the elimination of predisposing factors where possible. Anti-platelet agents are frequently used. Heparin may be of value, but the benefit is small. There are anecdotal reports of the use of endovascular thrombolysis, via a venous sinus or arterial route. In life-threatening cases this is said to result in dramatic improvement though the number of documented cases is small.

The diagnosis of cerebral venous thrombosis is often missed because it is not suspected clinically, and the features overlooked on CT. MR may help reduce the chance of overlooking key diagnostic features, but a high index of suspicion is required if this important cause of stroke in young patients is not to be misdiagnosed.

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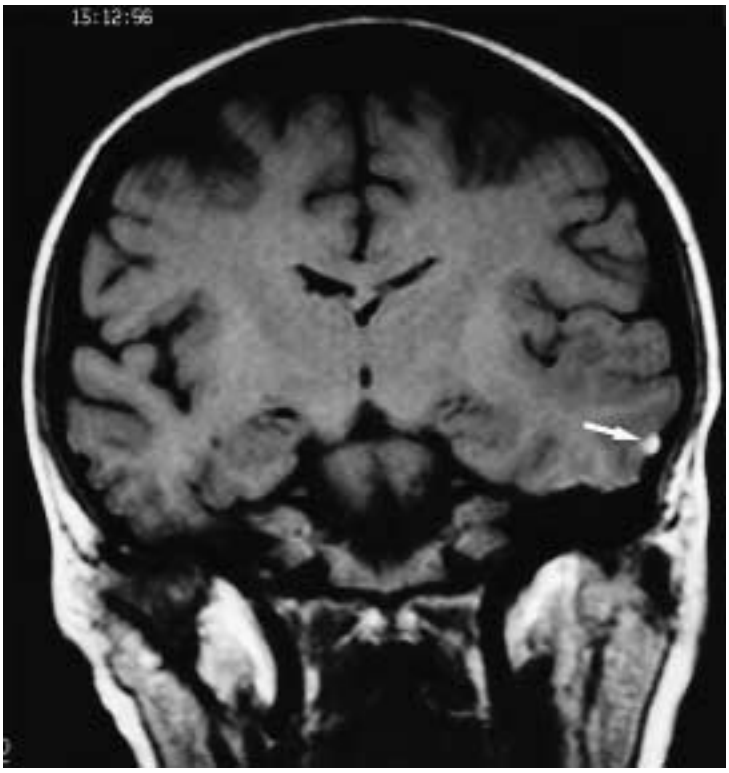

FIGURE 1B
Pre-contrast T1-coronal image of the temporal lobe shows a focus of high signal overlying the inferior temporal gyrus (white arrow). Similar changes on adjacent scans showed this to conform to the left Sylvian vein - the main cortical venous drainage of the temporal lobe.

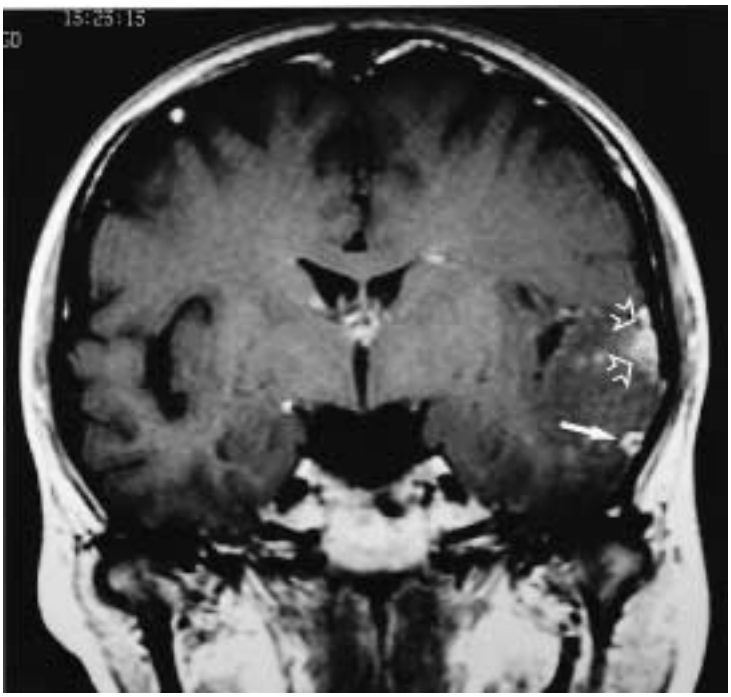

FIGURE 1C
Post-contrast scan of the same slice as 1B shows irregular cortical enhancement (open arrows) with a 'target' sign representing dural enhancement around the thrombosed cortical vein (white arrow).

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