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HAEMORRHAGIC INFARCTION DUE TO TRANSVERSE SINUS THROMBOSIS MIMICKING CEREBRAL ABSCESSES

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Cerebral venous sinus thrombosis is an uncommon condition which remains a diagnostic challenge for the clinician and radiologist. The wide spectrum of clinical and radiological manifestations can result in delayed or misdiagnosis. The authors present the case of a 50-year-old woman with headaches following an episode of mastoiditis. CT imaging revealed temporal ring-enhancing lesions which were thought to represent cerebral abscess formation and the patient proceeded to image-guided aspiration. MR venography, biopsy and histological examination confirmed the diagnosis of haemorrhagic infarction secondary to transverse sinus thrombosis.

KEY WORDS: **cerebral venous sinus thrombosis, cerebral abscess, venography, mastoiditis**

Cerebral venous sinus thrombosis remains a challenging condition due to its variability in clinical and radiological manifestation and relative rarity. Presenting features can range from headache to neurological deficit, seizures and coma2. Women are more commonly affected than men, and recognised associations include pregnancy and use of the hormonal contraceptive pill3. Magnetic resonance venography has superceded invasive angiography as the gold standard investigation, and is more sensitive than CT alone. Haemorrhagic infarction due to elevated venous and capillary pressure is estimated to occur in 10-50% of cases, and usually occurs in the adjacent cortex and white matter6. We present a histologically confirmed case of haemorrhagic infarction due to transverse sinus thrombosis, with ring-enhancing lesions on CT scanning which were initially thought to represent cerebral abscesses.

History and Examination

A 50-year-old woman presented to her GP with a 3-day history of pain behind the left ear following an upper respiratory tract infection. On eliciting tenderness of the mastoid, the GP made a clinical diagnosis of mastoiditis and prescribed a course of oral antibiotics. As the patient's symptoms persisted, a second course of antibiotics were prescribed. The patient reported improvement in her ear pain but over the following weeks she developed progressively severe headaches which were noted to be worse in the morning. An outpatient CT scan of the brain was performed, which revealed an area of subcortical hypodensity in the left temporal region. (Figures 1a and 1b).

In view of the history of an infective process as well as

the characteristic CT findings, contrast-enhanced CT and CT venography were performed, which revealed left temporal ring-enhancing lesions (Figures 2a and 2b) and a filling defect in the left transverse venous sinus (Figure 2c).

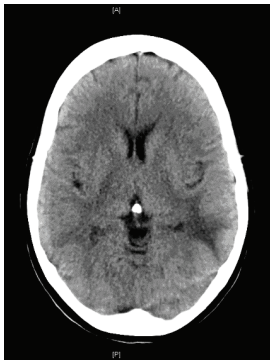

Figure 1a

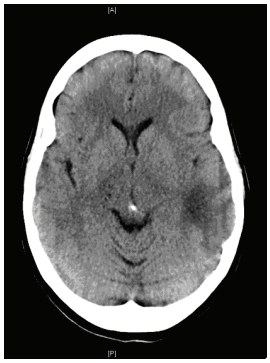

Fig ure 1b

Figures 1a and b. CT images. Non-contrast enhanced CT images reveal left posterior temporal hypodensity suggestive of transverse sinus thrombosis.

The CT imaging findings and patient history were consistent with a diagnosis of cerebral abscess formation and transverse sinus thrombosis as a consequence of mastoiditis. No elevation of serum inflammatory markers was found, but this was considered to be a result of recent antibiotic treatment. In order to isolate the infective organism the patient was prepared for burr hole aspiration of the abscesses.

Operation

To facilitate image-guided aspiration of the ring-enhancing lesions using the Stealth neuro-navigation system (Medtronic SNT, Tennessee), a pre-operative Stealth sequence MRI scan was performed, which once again confirmed the presence of ring-enhancing lesions (Figure 3).

Intra-operatively no pus was found, but haemorrhagic and necrotic material within a pseudo-capsule was excised and sent for micriobiological, cytological and histological assessment. Gram staining revealed only scanty white cells and no organisms. Cytological examination also confirmed the absence of tumour cells.

Postoperative course

The absence of pus or tumour cells on initial micros-

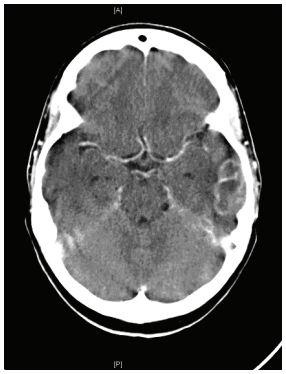

Figure 2a

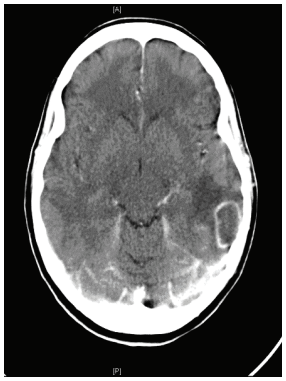

Figure 2b

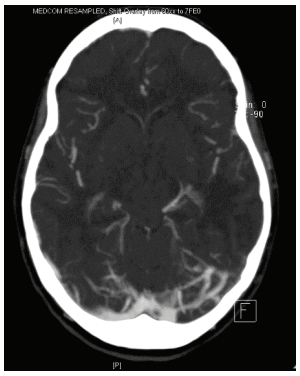

Figure 2c

Figures 2a, 2b and 2c. CT imaging. Contrast-enhanced CT scanning revealed left temporal ring–enhancing lesions, and CT venography confirmed a filling defect in the left transverse sinus extending into the sigmoid sinus and jugular bulb.

copy led to the patient undergoing formal MR imaging and venography. This more comprehensive study demonstrated low signal material within the walls of the lesions on T2 weighted images, which was suggestive of haemosiderin deposition and consistent with recent haemorrhage (Figure 4a). As this investigation was undertaken on the first post-operative day, sufficient time had not passed for haemosiderin deposition to be the result of bleeding due to surgery. This examination also better demonstrated thrombus within the left transverse sinus extending into the sigmoid sinus and proximal in-

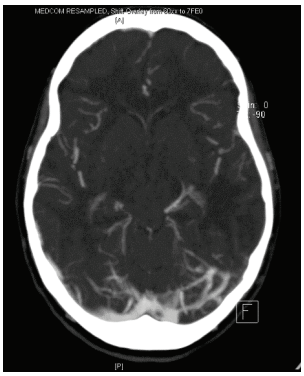

Figure 3. MR image. Stealth sequence MRI confirming the location of the ring-enhancing lesions and used for intra-operative neuro-navigation.

ternal jugular vein (Figures 4b and 4c). The patient was commenced on intravenous heparin, which was later converted to an oral anticoagulant. Her headaches resolved over a one week period and she was discharged home with no neurological deficits.

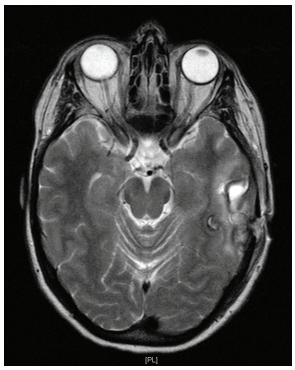

Figure 4a

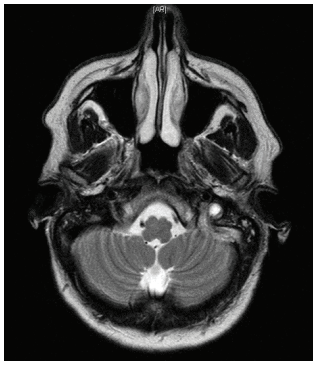

Figure 4b

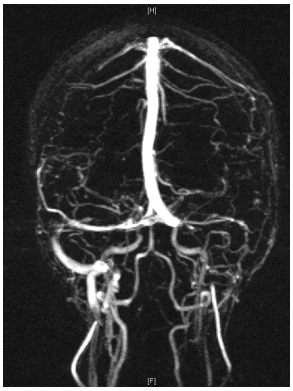

Figure 4c

Figures. 4a, 4b, and 4c. MR imaging. Formal MRI scanning revealed the presence of hemosiderin and extension of the thrombus as far as the proximal jugular vein.

Histopathological Findings

The excised tissue and capsule were processed and stained. A diagnosis of haemorrhagic venous infarction was confirmed. No evidence of infective or malignant disease was demonstrated.

Discussion

This case demonstrates some of the potential pitfalls in the diagnosis and management of patients with cerebral venous thrombosis as a result of the wide variety of clinical and radiological manifestations and their insidious onset. Haemorrhagic infarction is a relatively rare but potentially devastating outcome, and unilateral lobar haemorrhage associated with extensive oedema and mass effect has been reported7. In delayed presentations a haematoma is iso- or hypo-dense on CT scanning, and may also demonstrate ring-enhancement on administration of contrast thereby mimicking an infective or malignant process.

Although the clinical presentation of cerebral venous sinus thrombosis is highly variable, several typical symptom groups have been described1 – headache with papilloedema and visual disturbance; headache with progressive neurological deficit; and seizures often followed by a Todd's paresis. Thrombosis of the superior sagittal sinus classically presents with bilateral or alternating signs, and cavernous sinus thrombosis with chemosis, proptosis and ophthalmoplegia. A minority of patients present with a progressive coma as a consequence of thrombus progression into deep veins.

Univariate analysis has identified prognostic factors associated with poor outcome and these include papilloedema, reduced conscious level, coma, older age, intracerebral haemorrhage and involvement of the straight sinus3. A multivariate regression analysis in the same study identified haemorrhage as a statistically significant predictor of poor outcome.

MRI and MRV have replaced invasive venography as the investigation of choice, and have sufficient sensitivity to be utilised as the sole investigation for cerebral venous thrombosis8. However, it is likely that CT imaging will be the first-line investigation in most cases – which may be normal in 10 to 20% of patients

with proven thrombosis1. CT venography may become more widely used as CT technology improves and evidence emerges demonstrating its efficacy in visualising sinuses and smaller veins with low flow5.

It is arguable as to whether formal pre-operative MRI scanning and MR venography, as opposed to a limited Stealth sequence, would have resulted in the avoidance of surgery in this patient, as the finding of haemosiderosis in the walls of the lesions was more consistent with a diagnosis of haemorrhagic infarction than with abscess. Conversely it could be argued that in the presence of a recent history of a regional infective process and ring-enhancing lesions on CT, aspiration was mandatory.

Conclusions

A high index of clinical suspicion for cerebral abscess is required in any patient with signs and symptoms of raised intracranial pressure and a recent history of an infective process. Cerebral abscess is a treatable condition which can progress rapidly if misdiagnosed and if there is any diagnostic doubt aspiration or surgical exploration is mandatory. The variability in the clinical and radiological manifestations, insidious onset of symptoms, and relative rarity of cerebral venous sinus thrombosis makes this condition a diagnostic challenge. One possible consequence of thrombosis is haemorrhagic infarction, which is recognised as a poor prognostic indicator. A delay in recognition can result in iso- or hypo-dense and ring-enhancing appearances on CT scanning which can be misdiagnosed as infective or malignant processes. In order to maximise the clinician's ability to correctly diagnose cerebral venous thrombosis and its sequelae the authors advocate MRI and MR venography.

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Comment

This is an interesting case. I would have probably given the same diagnosis based on the available information. One possible way to have avoided misdiagnosing this rare entity would have been to perform an MR with diffusion. It appears that the patient went straight from CT to a STEALTH MR for drainage. A routine brain MR with diffusion images may have demonstrated restricted diffusion in the brain parencyma thereby suggesting the findings of infarct as opposed to abscess. It is also possible that more than one area of restricted diffusion would have been seen, thereby suggestive of a larger parenchymal abormality.

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