

Pneumococcal meningitis masquerading as subarachnoid haemorrhage

Taposh Chatterjee, John R Gowardman and Tony D Goh

A 43-year-old woman taking warfarin for past venous thrombosis presented with 4 days of flu-like symptoms and deterioration in level of consciousness. Computed tomography suggested subarachnoid haemorrhage, and magnetic resonance imaging showed widespread cerebral infarcts. However, these seemed out of proportion to the amount of haemorrhage, and lumbar puncture revealed meningitis caused by Streptococcus pneumoniae. (MJA 2003; 178: 505-507)

COMPUTED TOMOGRAPHY (CT) is a vital investigation in acute medicine, but CT appearances may occasionally be misleading, as illustrated here.

Clinical record

Presentation (Day 0): A 43-year-old woman was admitted to the emergency department (ED) in June 2002 with deterioration in her level of consciousness. She had a 4-day history of non-specific flu-like symptoms. On the day of admission, her husband had noted her to be lucid at 09:00 but found her semi-comatose when he returned home at 14:30. She had a past history of venous thrombosis, thought to be secondary to the presence of anticardiolipin antibody and lupus inhibitor, for which she took warfarin. She used topical and parenteral corticosteroids to control severe eczema, was a non-smoker and did not take the oral contraceptive pill.

On examination, her score on the Glasgow Coma Scale (GCS) was 7/15. Pupils were equal and reactive, and no definite signs of meningism could be elicited. Respiratory rate was 24 breaths/min, with oxygen saturation of 99% on supplemental oxygen, 15 L/min via a Hudson mask. Tympanic temperature was 40.8°C, blood pressure 168/92 mmHg and pulse rate 128 bpm in sinus rhythm. Serum blood glucose level was 7.8 mmol/L (reference range [RR], 3.6–5.8 mmol/L), white cell count was $18.6 \times 10^9/L$ (RR, $4.2\text{--}11.0 \times 10^9/L$), with toxic changes on the blood film, and platelet count was $357 \times 10^9/L$ (RR, $150\text{--}460 \times 10^9/L$). The international normalised ratio (INR) was 1.8 (RR, 0.8–1.3), with normal values for fibrinogen and fibrin cross degradation products, and activated partial thromboplastin time. C-reactive protein level was raised at 402 mg/L (RR, 0–10 mg/L). Other laboratory results, including renal function, were within the reference ranges.

In the ED, the patient underwent tracheal intubation because of her low GCS score, and was subsequently sedated with morphine and midazolam. Blood was taken for

culture, and intravenous ceftriaxone (2 g daily) was begun. Chest radiography showed segmental right upper-lobe consolidation.

Non-contrast CT of the brain was performed during her transfer to the intensive care unit (ICU). This was reported as showing subarachnoid haemorrhage, with increased density in the subarachnoid space, particularly within the basal cisterns (Box A). A neurosurgeon was consulted, and subarachnoid haemorrhage with aspiration pneumonia was diagnosed. The differential diagnosis of meningitis was thought less likely because of the CT appearance. A nimodipine infusion was begun, and cerebral angiography was planned. Her high INR was normalised with 4 units of fresh frozen plasma.

Day 1: At 06:00 the next morning, the patient's pupils became unequal and non-reactive. This was thought secondary to either vasospasm or a re-bleed. Urgent repeat contrast CT at 07:00 showed a parietal infarct, but no further bleeding or signs of raised intracranial pressure. The hyperdensity in the basal cisterns was less apparent, but there was subtle hyperdensity within the subarachnoid space, around the cerebral sulci near the vertex.

On subsequent review, the amount of haemorrhage suggested by the cranial CT scans appeared insufficient to account for the patient's clinical condition.

Diffusion-weighted magnetic resonance imaging (MRI) at 13:30 showed multiple acute cerebral infarcts in the territories of the right middle and posterior cerebral arteries, as well as in the watershed zone between the middle and anterior cerebral arteries (Box B). There was no abnormal enhancement of the leptomeninges to suggest infectious meningitis, and, in particular, no basal leptomeningal enhancement to suggest granulomatous meningitis (Box B). In the absence of abnormal leptomeningeal enhancement, the presence of increased signal within the subarachnoid space was thought to be due to subarachnoid haemorrhage (Box B).

Magnetic resonance angiogram and venogram showed no aneurysm or dural venous sinus thrombosis. Appearances were again thought most likely to represent primary subarachnoid haemorrhage, with acute cerebral infarction secondary to vasospasm. However, meningitis with secondary cerebral infarction was raised as a differential diagnosis, and lumbar puncture was recommended.

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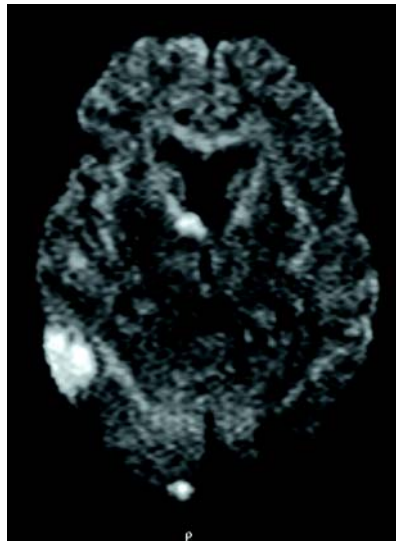
Brain imaging in a patient with pneumococcal meningitis

A: Computed tomography (CT) on Day 0



Non-contrast CT scan, showing increased density within the basal cisterns (arrow A) and along the sylvian fissures bilaterally (arrow B), suggesting subarachnoid haemorrhage.

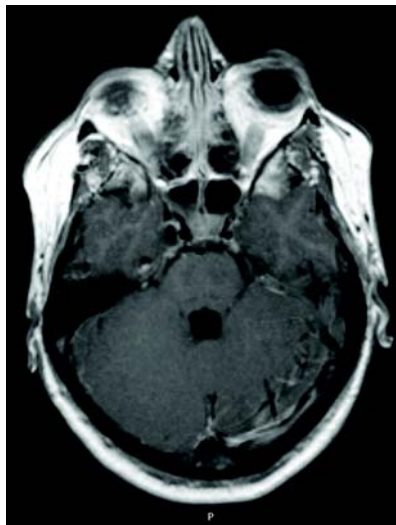
B: Magnetic resonance imaging (MRI) on Day 1



Diffusion-weighted MRI scan showing multiple foci of increased signal intensity in the anterior part of the right thalamus, posterior right temporal lobe and right occipital lobe. Increased signal intensity was also seen in the right frontal and parietal lobes (not shown).

This appearance was consistent with multiple acute infarcts in the territories of the right middle and posterior cerebral arteries.

Not shown: Multiple small acute infarcts were also seen in the watershed zone in the centrum semi-ovale between the territories of the right middle and anterior cerebral arteries.



Gadolinium-enhanced T1-weighted MRI scan showing no abnormal enhancement of the leptomeninges, including the basal leptomeninges. This suggests no evidence of a granulomatous meningitis.

Not shown: Increased signal intensity was seen within the subarachnoid space overlying the cerebral sulci of both cerebral hemispheres on the FLAIR sequence (a T2-weighted sequence that nullifies the signal from cerebrospinal fluid [CSF], improving detection of lesions within the subarachnoid space and brain parenchyma¹). This is a non-specific finding in a wide range of conditions, principally subarachnoid haemorrhage and meningitis. In our patient, the absence of leptomeningeal enhancement on the post-contrast images favoured a diagnosis of subarachnoid haemorrhage.

Lumbar puncture was subsequently performed after correction of residual coagulopathy, and revealed an opening pressure > 35 cmH₂O, a white blood cell count in cerebrospinal fluid (CSF) of $1510 \times 10^6/L$ (all polymorphs), red blood cell count of $160 \times 10^6/L$ (decreasing on subsequent tubes, with xanthochromia not detected), and protein level of 2291 mg/L (RR, 150–450 mg/L). Gram-positive diplococci were visible on Gram stain, and the CSF was positive for pneumococcal antigen. Blood taken on admission subsequently grew *Streptococcus pneumoniae*. After consultation with the infectious diseases team, we changed the antibiotic regimen to intravenous ceftriaxone (2 g) and vancomycin (1 g) twice daily. The diagnosis was revised to primary pneumococcal pneumonia with secondary pneumococcal meningitis.

Course: After 11 days' treatment in the ICU, the patient was transferred to a ward. On Day 13, she developed a large pulmonary embolus confirmed by CT angiogram and was readmitted to the ICU for respiratory support. She recovered and subsequently resumed anticoagulation therapy with heparin and warfarin. She was discharged back to the ward on Day 18.

At review a month later: She was undergoing rehabilitation, and was lucid and able to move with a frame, but remained blind in both eyes.

Discussion

Cranial CT scanning is a vital diagnostic tool in patients presenting with acute alteration in level of consciousness. Our case posed a diagnostic difficulty because of the unusual appearance on initial brain imaging. The initial CT scan showed increased density in the subarachnoid space, which is mostly caused by subarachnoid haemorrhage. Based on the imaging findings alone, the most likely diagnosis was therefore felt to be subarachnoid haemorrhage with secondary vasospasm causing cerebral infarction. However, there seemed to be a disparity between the amount of subarachnoid blood and the patient's clinical condition. This was reinforced by MRI, which showed extensive infarction in different vascular territories. This degree of infarction secondary to vasospasm would be unusual without widespread haemorrhage or thick focal clot, which were not seen. Additionally, vasospasm secondary to subarachnoid haemorrhage tends to peak 4–12 days later, while our patient showed signs of neurological deterioration less than 24 hours after admission.

Cerebral infarction has been well reported in bacterial meningitis in both adults^{2,3} and children,^{4,5} and is thought to result from an intense inflammatory response in the cerebral vascula-

ture. It is particularly noted in pneumococcal meningitis. Although pneumococcal meningitis was not diagnosed initially in our patient, broad spectrum antimicrobials, to which the organism was fully sensitive, were fortuitously begun on admission. The delay in ascertaining the correct diagnosis created a dilemma about subsequent anticoagulation, which probably could have been recommenced earlier.

Increased density in the subarachnoid space on CT has been described in tuberculous meningitis.⁶⁻⁸ However, this was unlikely in our patient — the condition is still uncommon in the Western world, and contrast enhancement of the basal leptomeninges (a sign of granulomatous meningitis) was absent. Increased density in the subarachnoid space has also been reported in anoxic encephalopathy,⁹ but our patient had no history of hypoxia or prolonged ischaemia. In addition, although the acute infarcts were within regions of the brain susceptible to acute hypoxic injury, they were all within the right cerebral hemisphere. With a global insult such as hypoxia, bilateral lesions would be expected.

To our knowledge, there has previously been only one English-language article describing a patient with acute purulent meningitis mimicking subarachnoid haemorrhage on CT scan.¹⁰ Presumably this appearance is caused by the high protein concentration of the purulent exudate in pyogenic meningitis, as noted in our case. Although this is uncommon, we present this case to alert both radiologists and clinicians to the presence of this atypical appearance on imaging and to highlight the need for careful evaluation of such patients. The case also illustrates that results of

investigations should not be interpreted in isolation from the clinical picture. We suggest that lumbar puncture should be performed if the clinical presentation is atypical or not in keeping with the radiological findings.

Acknowledgements

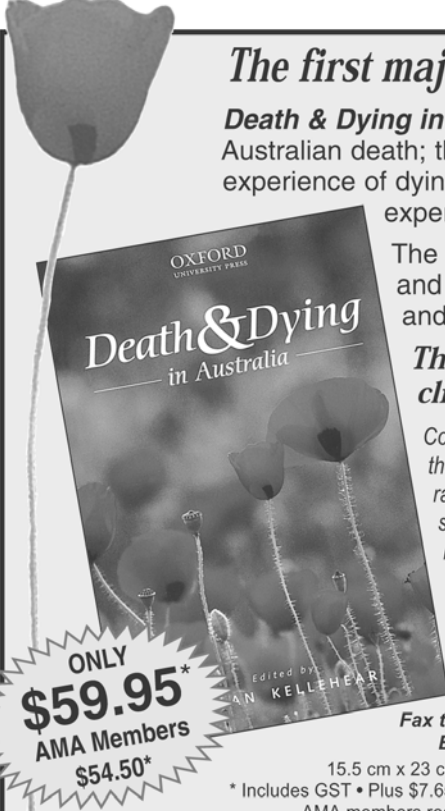
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References

- Hajnal JV, Bryant DJ, Kasuboski I, et al. Use of fluid-attenuated inversion recovery (FLAIR) pulse sequences in MRI of the brain. *J Comput Assist Tomogr* 1992; 16: 841-844.
- Pfister HW, Borasio D, Dirnagl U, et al. Cerebrovascular complications of bacterial meningitis in adults. *Neurology* 1992; 42: 1497-1504.
- Westrate W, Hijdra A, de Gans J. Brain infarcts in adults with bacterial meningitis. *Lancet* 1996; 347: 399.
- DiNubile MJ, Boom WH, Southwick FS. Septic cortical thrombophlebitis. *J Infect Dis* 1990; 161: 1216-1220.
- Stovring J, Snyder RD. Computed tomography in childhood bacterial meningitis. *J Pediatr* 1980; 96: 820-823.
- Upadhyaya P, Bhargava S, Sundaram KR, et al. Hydrocephalus caused by tuberculous meningitis: clinical picture, CT findings and results of shunt surgery. *Z Kinderchir* 1983; 38 Suppl 2: 76-79.
- Bhargava S, Gupta AK, Tandon PN. Tuberculous meningitis — a CT study. *Br J Radiol* 1982; 55: 189-196.
- Rovira M, Romero F, Torrent O, et al. Study of tuberculous meningitis by CT. *Neuroradiology* 1980; 19: 137-141.
- al-Yamany M, Deck J, Bernstein M. Pseudo-subarachnoid hemorrhage: a rare neuroimaging pitfall. *Can J Neurol Sci* 1999; 26: 57-59.
- Mendelsohn DB, Moss ML, Chason DP, et al. Acute purulent leptomeningitis mimicking subarachnoid haemorrhage on CT. *J Comput Assist Tomogr* 1994; 18: 126-128.

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