Sudden death from rupture of cerebral abscess into subarachnoid space

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DESCRIPTION

A Mexican man in his mid-30s with a medical history of atrial septal defect presented with sudden onset of left lower extremity weakness and a 2-week history of fever, headache and purulent rhinorrhoea. On examination, he was alert, had no cranial nerve deficits and had full strength except for mild weakness in his left lower extremity. CT head showed two right frontal hypodense lesions with surrounding oedema, mass effect and subfalcine herniation (**figure 1A**). Given probable recent sinusitis and the radiological features described above, the cerebral abscess was considered as the most likely diagnosis.

He was started on vancomycin, cefepime, amphotericin B, mycobacterium RIPE therapy and dexamethasone. However, the next day, he suddenly deteriorated losing all cortical and brainstem function. He was started on hypertonic saline and measures for intracranial pressure control without improvement. Emergent MRI showed ring enhancement of

the previously identified lesions but no new herniation (**figure 1B and C**). He had no improvement in the examination, and the next day, a nuclear medicine cerebral flow study revealed the absence of all perfusion. He was ultimately pronounced brain dead.

An autopsy confirmed the presence of two frontal abscesses, with the most anterior extending to the superficial surface of the frontal lobe (**figure 1D**). Microscopy revealed bacterial abscess with gram-positive cocci in clusters and gram-negative rods. Abscess exudate was found in the subarachnoid space resulting in meningeal inflammation (**figure 1E and F**). Given no significant worsening of mass effect, herniation or evidence of rupture of abscess into intraventricular space, rupture of abscess into subarachnoid space was considered the cause of sudden deterioration and brain death.

Although rare, this complication has been described before.¹ The source of abscess, in this case, was likely local spread of a sinus infection, but the presence of

Figure 1 CT (A) and MRI brain (B and C) showing right frontal hypodense lesion with surrounding oedema, mass effect and ring enhancement with gadolinium. Gross section of brain showing right frontal abscesses (D). Histological slide with H&E stain showing left frontal (E) and left occipital (F) exudate from abscess rupture and meningeal inflammation.

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an atrial septal defect also predisposed him to paradoxical embolism and cerebral abscess.² He was HIV negative and not otherwise immunosuppressed. Little is known about what increases the risk for rupture into the subarachnoid space, but multiloculation and close proximity to the ventricles increase the risk for intraventricular rupture.³ It is important to be aware of this complication, as it underlines the importance of early medical and surgical management of cerebral abscess. Further research should be done to see if these factors influence the risk of rupture into the subarachnoid

space and to determine what patients are at risk for this catastrophic complication.

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Learning points

- ▶ Prompt recognition and treatment of cerebral abscess is crucial in preventing devastating complications.
- ▶ Rupture of abscess into subarachnoid space should be recognised as a possible mechanism for sudden deterioration in the absence of other complications such as intraventricular rupture and hydrocephalus, intracranial pressure crisis or seizures.

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