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Two different intracranial haemorrhages and one cerebral infarction: what are the odds?

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DESCRIPTION

A 60-year-old epileptic woman was brought to A&E obtunded. Before 1 h, she fell onto the ground and hit her head after developing a seizure. Her Glasgow Coma

Scale (GCS) was 6/15. An urgent CT brain scan was done (figure 1) and the patient was immediately transferred to the intensive care unit. The family declined neurosurgical intervention. On day 4, she became comatose with a GCS

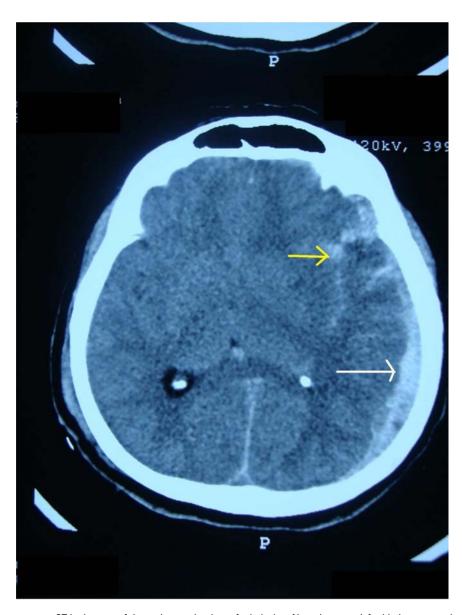


Figure 1 Urgent non-contrast CT brain scan of the patient at the time of admission. Note the acute left-sided crescent-shaped subdural haematoma (white arrow). In addition, the left Sylvian fissure contains blood, signifying the presence of subarachnoid extension (yellow arrow).

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Figure 2 Non-contrast CT brain scan of the patient on day 4. Note the hypodense area (white arrow) at the territory of the left posterior cerebral artery (PCA); this is acute ischaemic stroke caused by compression of the proximal part of the left PCA against the free edge of the tentorium by the mass effect of the left-sided subdural haematoma. The latter has become denser, pointing to a recurrent bleed. The cytotoxic oedema of the infarcted area had augmented the already high intracranial pressure.

of 3/15. Another CT brain scanning was ordered (figure 2). The patient was neither hypertensive nor diabetic, and no history of cardiac diseases was obtained. Her 12-lead ECG showed sinus tachycardia with a regular rate of 138 beat/min.

The appearance of a new hypodense area is consistent with an acute and completed ischaemic infarction in the territory of the left posterior cerebral artery.

Abe $et\ al^1$ found that most of the ischaemic brain damage noted in patients with acute subdural haematomas (SDHs) was an aftermath of arterial compression secondary to brain shift and herniation syndromes rather than the direct effect of the haematoma itself upon the underlying brain. Furthermore, they suggested that this ischaemic brain damage adversely affects the morbidity and outcome, and the difficulty in preventing ischaemic damage in cases with marked brain shift leads to poor outcome in patients with acute SDHs. According to Wani $et\ al,^2$ these infarctions are 'commonly seen' in acute SDHs. In figure 2, we can notice that the SDH has become denser, signifying the occurrence of a recurrent bleed.

Learning points

- Head trauma can result in more than one type of intracranial haemorrhage.
- ► The asymmetric increment in the intracranial pressure as well as the midline shift induced by acute subdural haematomas can result in secondary compression on major brain arteries with subsequent ischaemic infarctions. The latter vascular event challenges the management and deteriorates the prognosis.
- ► This type of arterial occlusion is common in acute subdural haematomas. The treating medical/surgical team should vigilantly search for this complication when patients develop new focal or lateralising signs or a rapid downhill course ensues.

Competing interests None

Patient consent Obtained.

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