Sudden right-sided hemiparesis in a middle-aged woman

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A 58-year-old woman presented as an emergency having been found collapsed at home. She had a 2-hour history of difficulty moving her right limbs and problems with her speech. She had been active prior to the onset of symptoms. Clinical examination of the cardiovascular and respiratory systems was unremarkable. She was fully orientated with a Glasgow Coma Scale (GCS) score of 14/15 (E4 V4 M6), with normal sized and normal reacting pupils. There was right-sided weakness of the lower facial muscles and increased tone and reduced power (3/5) of the right limbs. Both plantar responses were extensor, but there were no reflex or sensory abnormalities. Full blood count, urea and electrolytes, clotting function, electrocardiogram and chest X-ray were all normal.

Two days after admission she deteriorated to a GCS of 10/15 (E3 V2 M5). A computed tomography (CT) scan revealed a right-sided high-density collection with associated mass effect (figure).

Questions

1. What three features does the CT scan show?
2. What is the phenomenon called whereby the intracranial lesion is on the same side as the focal neurological signs?
3. How is it caused in this case?
Answers

QUESTION 1
Right-sided acute subdural haematoma; marked mass effect compressing the right lateral ventricle and displacing the midline structures; contralateral hydrocephalus.

QUESTION 2
Kernohan’s notch.

QUESTION 3
Displacement of the midbrain away from the haematoma leading to contralateral compression of the cerebral peduncle. Since this is above the medullary pyramidal decussation, it produces signs ipsilateral to the haematoma.

Discussion

The phenomenon of compression of the crus cerebri against the free edge of the tentorium cerebelli by a contralateral supratentorial mass was first demonstrated by Kernohan and Woltman in 1928 at the Mayo Clinic. The syndrome was first noted in a patient with a primary brain tumour, however, it is also seen, although rarely, after traumatic brain injuries. The compression causes grooving with underlying tissue damage in the anterolateral aspect of the cerebral peduncle. In trauma, the condition is due to the effect of significant shift secondary to oedema or haemorrhage. The resulting compressive forces cause the herniating midbrain to impinge on the contralateral edge of the tentorium, hence damaging the descending corticospinal and corticobulbar tracts.

Other possible causes of an ipsilateral hemiparesis include a primary brainstem lesion, a contralateral contusion, pressure necrosis secondary to elevated intracranial pressure, or arterial infarction. Primary brainstem lesions tend to produce severe and prolonged impairment in the level of consciousness, associated with typical damage in the dorsolateral region of the brainstem. Large haematomas can produce a direct compressive or diffuse ischaemic necrosis if the intracranial pressure or cerebral perfusion pressure reaches a sufficient level.

Magnetic resonance imaging (MRI) has been shown to be superior to CT in the detection of traumatic lesions, brainstem, or white matter injuries and, in view of the other possible diagnoses, its use has been advocated to assess the full spectrum of lesions in unusual presentations.

The phenomenon itself, in heralding a descending transtentorial herniation with brainstem displacement and cisternal compression, may lead to a poor prognosis with a mortality of greater than 50% in severe cases. However, CT scanning and the initial severity as indicated by GCS, may not be predictive of eventual function if early rehabilitation is undertaken.

Following craniotomy and evacuation of the haematoma the patient’s right-sided weakness gradually improved and 5 days later she was transferred for further rehabilitation.

Final diagnosis

Right-sided acute subdural haematoma causing ipsilateral physical signs.

Keywords: hemiparesis; Kernohan’s notch

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References