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Case Report

Kernohan's Notch Syndrome in Acute Subdural Hematoma Secondary to Ruptured Middle Cerebral Artery Aneurysm

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ABSTRACT

An 82 year old male was brought unconscious to the Emergency Department (ED). On examination, he was found to have a Glasgow Coma Scale (GCS) score of 6 (E1M4V1), with increased tone and hyperreflexia in the right arm and right leg. CT scan of the brain showed a large right sided Subdural Hematoma (SDH) and a Subarachnoid Hemorrhage (SAH) in the suprasellar cistern and Sylvian fissure. An aneurysm of the right middle cerebral artery was identified as the likely source of bleeding. The clinical and imaging correlation was consistent with the Kernohan's Notch Syndrome. The patient was managed conservatively and sadly died four days after admission.

BACKGROUND

Case report

An 82 year old Caucasian male was found to be unresponsive in bed in the morning by his wife. His wife noticed that he was becoming increasingly confused and less mobile 4-5 days prior to the day of his presentation to the ED. He had a history of ischemic heart disease, mild left ventricular systolic dysfunction and atrial fibrillation, which was being managed with warfarin. An incidental cerebral aneurysm of the right middle cerebral artery was identified 7 years previously and was being managed conservatively. There was no history suggestive of chest infection, urinary symptoms, fall, headache or any recent change in medication.

On examination, his Glasgow Coma Scale (GCS) score was 6 (Eye opening 1, Motor response 4, Verbal response 1), respiratory rate was 20/min, oxygen saturation 92% on air, heart rate 98 bpm, blood pressure 131/88 mmHg and temperature 38 0C. He had bi-basal crepitations and pitting edema up to the mid shin level due to mild Left Ventricular Failure (LVF). The pupils were equal (3 mm) and sluggish in reaction and there was increased tone and hyperreflexia in both right upper and right lower limbs with an extensor plantar response on the right side.

Investigations

His serum Sodium was 136mmol/l. International Normalized Ratio (INR) was 2.2. Inflammatory markers and lactic acid levels were normal. Chest x-ray revealed cardiomegaly, blunting of the costophrenic angles and upper lobe diversion, consistent with LVF. Arterial blood gases showed type one respiratory failure, which was corrected with supplemental oxygen. CT of the brain revealed a large subdural

hematoma causing right uncal and subfalcine herniation (Figure 1,2). In addition, subarachnoid hemorrhage was noted in the suprasellar cistern and in the right Sylvian fissure. A subtle rounded hyperdensity measuring 15mm along the M1 (sphenoidal) segment of the middle cerebral artery was suggestive of cerebral aneurysm which was the likely source of bleeding.

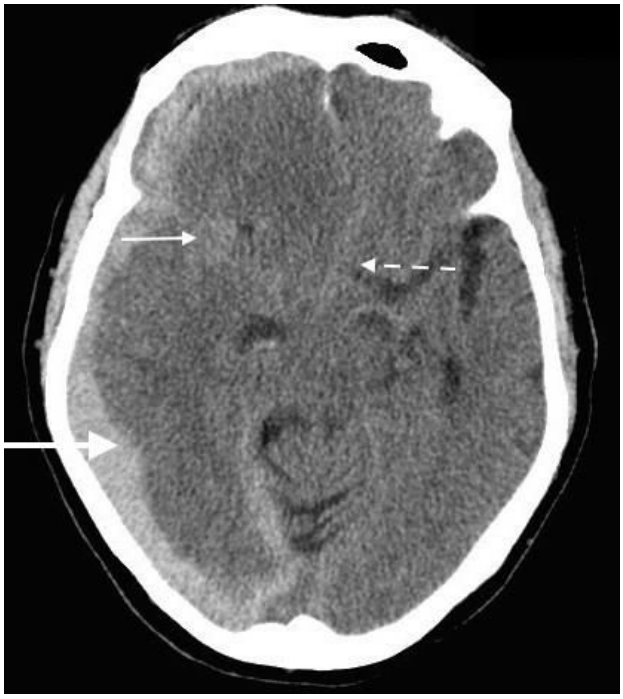

Figure 1: CT scan of the brain. The large arrow indicates subdural hematoma. The small arrow points to middle cerebral artery aneurysm. The dashed arrow is directed at subarachnoid hemorrhage in the suprasellar cistern.

Differential diagnosis

Reduced level of consciousness with lateralizing neurological signs were indicative of focal intracranial pathology such as intracerebral hemorrhage, stroke, abscess or brain tumor. As our patient was pyrexial, the possibility of meningitis, encephalitis, lower respiratory tract infection or urinary tract infection were also considered. Other common causes of reduced level of consciousness including drug overdose, head trauma, metabolic abnormalities and hypothermia were excluded based on the history and examination findings.

Treatment

The initial treatment consisted of high flow oxygen, furosemide and Tazocin (piperacillin with tazobactam) to treat the LVF and potential sepsis. Warfarin was stopped and vitamin K 10mg administered intravenously. After discussion with the regional neurosurgical unit, the decision was made that any active surgical treatment was not in the best interest of the patient, as this would lead to a poor outcome. This was in agreement with the family and advance directives expressed by the patient.

Outcome

The patient was transferred to the medical ward and sadly died 4 days later.

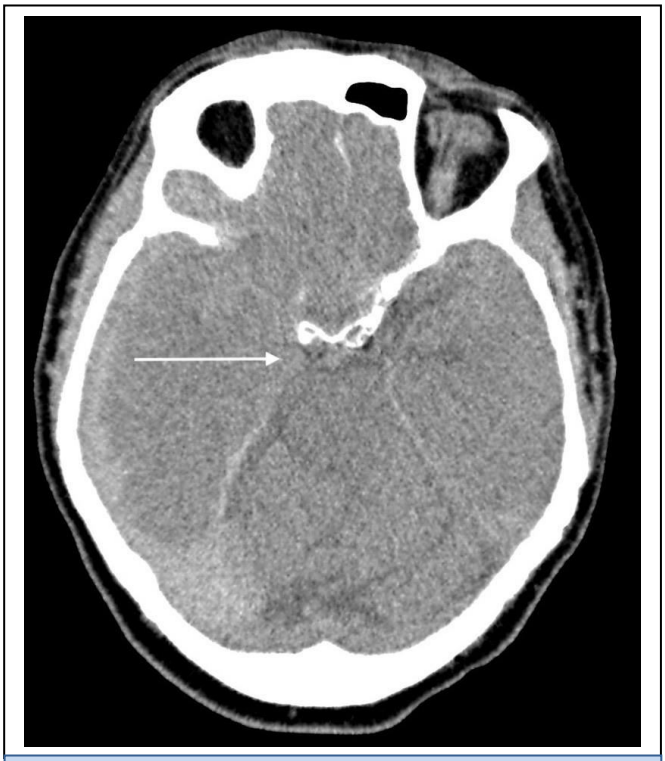

Figure 2: Axial CT images of the brain showing right uncal herniation.

DISCUSSION

This case is firstly a clinical reminder of an important and uncommon false localizing sign (Kernohan's Notch Syndrome) and secondly an example of a rare association between ruptured cerebral aneurysm and Subdural Hemorrhage (SDH). The Kernohan's Notch Syndrome (KNS) was first described by James Watson Kernohan, American pathologist in 1929 after an autopsy study revealed a notched cerebral peduncle from a contralateral herniation syndrome [1]. This is contrary to most intracerebral lesions, which cause contralateral hemiparesis. In KNS, pressure in one cerebral hemisphere causes uncal herniation, which compresses the contralateral cerebral peduncle against the rigid tentorium cerebelli [2] (Figure 3). Since the corticospinal tracts decussate at the level of lower

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medulla, the neurological deficit occurs on the same side as the hemispheric lesion [3]. In addition, patients with KNS often have mydriasis as the uncal herniation compresses the superficially running parasympathetic fibers of the oculomotor nerve. In our case pupils were equal but slow to react, which indicates that a degree of transtentorial herniation must have already occurred.

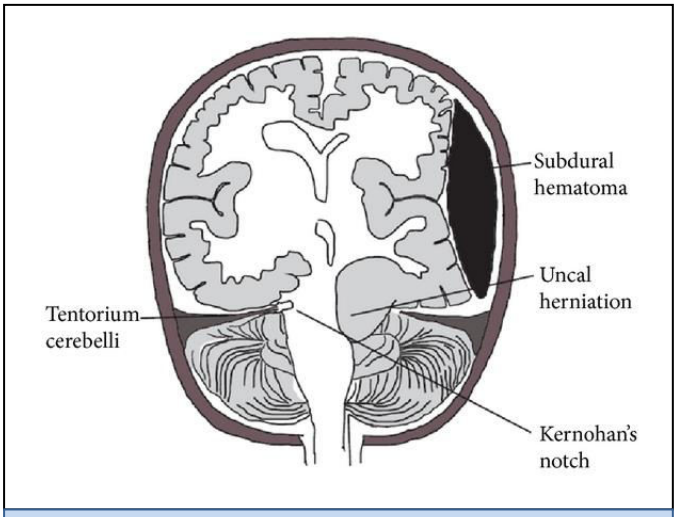

Figure 3: Diagrammatic illustration of Kernohan's Notch syndrome [3]. Subdural hematoma causes uncal herniation with subsequent compression of contralateral cerebral peduncle against the tentorium cerebelli. Since the corticospinal tracts decussate at the level of the medulla, weakness occurs ipsilateral to the lesion. (Produced with the permission from Hindawi Publishing Corporation).

The combination of ruptured cerebral aneurysm and SDH is rare. The incidence of SDH associated with aneurysmal bleed ranges from 0.5% to 4%, however a large SDH, such as in this case, with no or minimal SAH is very unusual [4-7]. Several mechanisms have been proposed to explain the occurrence of SDH in an aneurysmal rupture. Marbacher et al suggested that SDH occurs as a result of bleeding from aneurysms adherent to the subdural space after observing that most aneurysmal SDHs occur as a result of bleeding from aneurysms located in Internal Carotid Artery (ICA) and posterior communicating artery aneurysms (Pcomm) [8]. Several other studies found that ICA-Pcomm aneurysm location was an independent risk factor for the concomitant development of SDH [6,9].

Middle cerebral aneurysms, such as in our case, are only seen in approximately one fifth of cases [9,10]. Occurrence of SDH in these is likely as a result of increased pressure in the subarachnoid space, which causes a rupture of delicate arachnoid mater allowing the blood to track into subdural space [8]. Clarke and Watson proposed a different mechanism to explain aneurysmal bleeds that are associated with SDH alone without SAH [11]. They suggested that preceding small aneurysmal leaks create local adhesions of pia and arachnoid matter and when the aneurysm ruptures it does so directly into the subdural space. This is consistent with the findings of the study by Biesbroek et al., in which sentinel headache due to preceding small aneurysmal leaks, was found to be strongly associated with higher risk of subsequently developing subdural hemorrhage as a result of aneurysm rupture [6].

Historically, the prognosis of patients with aneurysmal SDH is poor. Patients often present with deepening coma and pupillary abnormalities. Unless rapid craniotomy and evacuation can be performed, patients are not likely to survive. Currently, there are no guidelines to support clinical decisions regarding the management of aneurysmal SDH due to the rarity of this condition and limited descriptive literature. Based on the experiences of the authors of several case series, it is advised that neurologically stable patients with small subdural hematomas should be managed as per guidelines for SAH management, which should include imaging of the aneurysm using Digital Subtraction Angiography (DSA) or CT Angiogram (CTA) and appropriate intervention (endovascular coiling or clipping) [5,7,8]. In patients with deteriorating neurological status, osmotherapy should be administered to lower raised intracranial pressure and SDH should be evacuated urgently [8]. Once the patient's condition is stable, definitive imaging with DSA should be performed to locate the aneurysm [8]. Unfortunately, in patients with significant comorbidities and extremely poor initial neurological and hemodynamic status, such as our patient, active treatment may not be considered to be appropriate. This should be sensitively communicated and explained to the patient's family.

CONCLUSION

Our case serves as a good reminder to consider the possibility of KNS, which results from the compression of the contralateral cerebral peduncle causing ipsilateral hemiparesis. The possibility of a subdural hematoma should also be considered in patients with lateralizing signs and reduced consciousness.

Uniquely, this patient presented with a rare combination of SDH arising from ruptured middle cerebral artery aneurysm and KNS. Literature suggests that aneurysmal SDH carries a poor prognosis and requires rapid surgical evacuation with subsequent treatment of the aneurysm. However, for patients with poor functional status active treatment may not always be appropriate.

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