Interhemispheric Subdural Hematoma

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ABSTRACT: Although relatively uncommon, interhemispheric subdural hematoma (ISDH) occurs more frequently than was suspected before the advent of computerized tomographic (CT) scanning. When its mass is sufficiently large to compress the medial cerebral hemisphere, specific focal neurological abnormalities may occur. These include weakness of the contralateral leg, or contralateral hemiparesis with the leg being weaker than the arm. On the unenhanced CT scan ISDH is seen as a crescent shaped, midline hyperdensity. Treatment is dictated by the clinical course. Evacuation of the hematoma by parasagittal craniotomy is recommended if the symptoms are pronounced.

RÉSUMÉ: Hématome sous-dural interhémisphérique Même si cette entité est relativement rare, l’hématome sous-dural interhémisphérique (HSDI) survient plus fréquemment qu’on ne le soupçonnait avant l’avènement de la tomographie axiale assistée par ordinateur. Quand la masse de l’hématome est suffisamment importante pour comprimer la face interne de l’hémisphère, des anomalies neurologiques focales spécifiques apparaissent. Parmi celles-ci, notons la faiblesse de la jambe contralatérale ou l’hé miparésie contralatérale, la jambe étant plus faible que le bras. A la tomographie axiale sans perfusion de produit de contraste, l’HSDI apparaît comme une zone d’hyperdensité médiane en forme de croissant. Le traitement est déterminé par l’évolution clinique. Si la symptomatologie est importante, il est recommandé de procéder à l’évacuation de l’hématome par craniotomie parasagittale.


Interhemispheric subdural hematoma (ISDH) was first described at autopsy by Airing and Evans' in 1940 and first recognized during life by Jacobsen2 in 1955. Prior to the advent of computerized tomographic (CT) scanning this lesion was very difficult to detect, a fact which accounts for the paucity of cases reported before 1974,14 and for the persistent belief that it is very rare8,11. In this paper we report two additional cases of ISDH and discuss the unique features of this lesion.

CASE REPORTS

Patient 1

An 80-year-old man fractured the neck of his right femur and struck his head in a fall. He did not lose consciousness. His past history included chronic obstructive lung disease and a myocardial infarction. Because of the latter he had been receiving oral anti-coagulant therapy. Upon admission to hospital he was alert and without neurologic deficit. Three days later he was noted to be disoriented and at examination was found to have a right hemiplegia with right facial weakness. Shortly afterwards he developed focal right sided seizures, which involved his leg more than his arm. Prothrombin time was 28 seconds (normal range 10-12), and partial thromboplastin time was 48 seconds (normal range 25-30). A nonenhanced CT scan showed a large left ISDH (Figure 1). Craniotomy was performed via a left sided parasagittal bone flap. The brain swelling. A large hematoma was encountered in the subdural space deep between the hemispheres, extending onto the tentorium. Approximately 70 cc’s of semi-liquid blood were removed with irrigation and suction. He awakened immediately after surgery, but remained confused and hemiplegic, with the leg being weaker than the arm. Prothrombin time was 54 seconds (normal range 25-30). A nonenhanced CT scan, showed a large right ISDH which was virtually identical to that in Case I (Figure 1). The blood had also tracked posteriorly to outline the tentorium. Craniotomy via a large parasagittal bone flap gave easy access to a subdural hematoma situated deep between the cerebral hemispheres and extending onto the tentorium. Approximately 55 cc’s of semi liquid blood were removed using irrigation and suction. The bridging veins were not disturbed. In the immediate postoperative period he remained stuporous with focal seizures involving the left leg. These were controlled by anti-convulsant therapy and she gradually recovered to be

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subdural space. Therefore, ISDH is usually unilateral. Associated convexity subdural hematomas were present in a number of the cases reported. Some authors consider these to represent an extension of the hematoma from the interhemispheric fissure, while others believe they originate from separate bleeding sources.

Major trauma was implicated in only a few of the cases occurring after blunt head injury. These patients were unconscious from the time of injury or shortly thereafter. More frequently the associated injury was minor, with or without a brief period of unconsciousness. Both of our patients had minor head injuries, but they were also receiving anti-coagulant therapy. Usually the onset of symptoms and signs is delayed, with the delay varying from days, to weeks, to months.

Focal neurological abnormalities have been reported in association with ISDH, and were observed in both of our patients. These include weakness of the contralateral leg or arm, paraparesis has also been described. These signs indicate a medial cerebral hemisphere lesion, have also been described in patients with interhemispheric subdural empyema and with occlusion of the anterior cerebral artery. This clinical picture has been referred to as the falx syndrome.

Prior to CT scanning, carotid angiography was the investigation of choice for ISDH. In the anterior-posterior view it shows a midline avascular space, which is produced by displacement of the callosomarginal arteries without a similar displacement of the pericallosals. Angiography has been replaced by CT scanning, and need only be considered if an aneurysm is suspected.

CT scanning gives an immediate and precise diagnosis, defining the exact location, extent and pattern of the ISDH. On the nonenhanced CT scan, its hallmark is a midline, parafalcine, crescent shaped hyperdense mass. Its flat medial border is formed by the falx and its convex lateral border by the hematoma mass bulging into the ipsilateral hemisphere. It is differentiated from subarachnoid hemorrhage and intracerebral hematoma by its size and shape, and from interhemispheric empyema by its density.

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