Spontaneous Resolution of Post Traumatic Acute Interhemispheric Subdural Haematoma

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ABSTRACT

The authors report a case of spontaneous resolution of large Post Traumatic Acute Interhemispheric Subdural Haematoma on conservative management. The follow up was done by serial C.T. scans of the head over a period of 4 weeks.

Keywords: Acute Interhemispheric Subdural Haematoma, C.T. Brain finding, Spontaneous resolution.

INTRODUCTION

Acute post traumatic subdural haematomas usually occur over the cerebral hemispheres. But occasionally they present in the interhemispheric fissure. Small Acute Interhemispheric Subdural Haematoma usually resolves by itself over a period of time. Large post traumatic Acute Interhemispheric Subdural Haematoma are relatively uncommon. They may present with headache, vomiting, altered sensorium, seizures, hemiparesis or hemiplegia and language dysfunction. The management options range from craniotomy and evacuation of the haematoma to conservative management.

CASE REPORT

A 57 year old female patient was admitted with history of fall following a synocopal attack with forehead and nose hitting the floor. After the fall she was found in a state of unconsciousness with incontinence of urine & motion and recurrent vomiting. She was a diabetic and hypertensive for the last 20 years. She had an attack of stroke 4 years back and developed (R) sided weakness and dysphasia, from which she recovered partially. After that she was able to walk with difficulty but used to get recurrent falls.

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On Examination
She was semiconscious, disoriented, aphasic, pupils were both equal and reacting to light, (R)sided hemiplegia (R) UMN facial weakness and(R) plantor was upgoing BP 160/90. PR 88/mts. RBS-297 mg% SPO2-97% Cardio vascular system and Respiratory system examination revealed no abnormality. Her haematological profile was normal. Bio-chemical studies showed evidence of hyponatraemia which was corrected with hypertonic saline infusion

C.T scan of the head at the time of admission showed 20 mm thick. Acute hyperdense subdural haematoma extending along the full length of Inter hemispheric fissure on the (L) side with 5mm. midline shift to the right and mild compression of (L) lateral ventricle. Since it was a large haematoma associated with signs and symptoms of increased intracranial tension and the presence of focal neurological deficit, craniotomy and evacuation was advised. But the relatives were not willing for surgery. Hence the patient was managed conservatively with anticonvulants, antioedema measures, antihypertensives, antidiabetics, physiotherapy and supportive measures. Repeat C.T. head after 1 week showed no change in the size of the haematoma. The patient showed clinical and neurological improvement over a period of 4 weeks. She became conscious, started talking, (R) hemiplegia showed slow recovery. Repeat C.T. head after 4 weeks showed evidence of significant resolution of Inter hemispheric subdural haematoma.

DISCUSSION
Inter hemispheric subdural haematoma was first described by Aring and Evans in 1940 in an article “Aberrant location of subdural haematoma” published in the journal Archives of Neurology and Psychiatry. Large Inter hemispheric subdural haematomas are relatively uncommon lesions and usually seen in patients with bleeding disorders. In majority of cases they are associated with trauma. The predisposing trauma is frequently of low velocity and rupture of parasagittal bridging veins in the interhemispheric fissure is presumed to be the cause of haematoma. The interhemispheric bleeds are usually unilateral. In the present case there was a history of fall, but there was no evidence of bleeding disorder and the haematoma was located on the (L) parafalcine region. The patient presented with altered sensorium, vomiting, (R) sided hemiplegia and aphasia.

Conservative treatment and surgical evacuation of the haematoma are equally favored in the management of these patients. Surgical removal of the solid clot from interhemispheric space proved successful, but some times this can be a dangerous procedure due to the proximity of sagittal sinus and para sagittal bridging veins (Gannow 1961). Hence surgical treatment is to be reserved for those patients who have signs of increased intracranial tension and progressive neurological deficit and conservative management is to be preferred for those patients who show clinical and neurological improvement and those who have concurrent risk factors and poor general condition. In our case even though we have advised surgery and evacuation of haematoma, the relatives were not willing for the same. Hence surgery deferred and patient was managed conservatively.
END NOTE

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REFERENCE