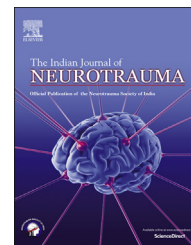


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Case Report

Pediatric post-traumatic subdural hygroma associated with a giant frontotemporal arachnoid cyst



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ARTICLE INFO

Article history:

Received 29 April 2013

Accepted 10 November 2013

Available online 2 December 2013

Keywords:

Arachnoid cyst

Subdural hygroma

Pediatric trauma

Management

Radiology

ABSTRACT

We report a rare case of a post-traumatic subdural hygroma occurring with a giant frontotemporal arachnoid cyst in a 6-year old child and discuss its pathogenesis and management implications. Awareness regarding the association of a subdural hygroma associated with an arachnoid cyst is of immense clinical importance as the former may precipitate intracranial hypertension in a previously asymptomatic arachnoid cyst and yet may remain undetectable on a CT scan.

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1. Introduction

An arachnoid cyst is a development anomaly that may remain asymptomatic or may manifest features of raised intracranial pressures or seizures. Rarely an arachnoid cyst can present

with an intracystic hemorrhage, a subdural hematoma or a subdural hygroma either spontaneously or following trauma.^{1–5} We report the rare case of a post-traumatic subdural hygroma occurring with a giant frontotemporal arachnoid cyst and discuss its clinical implications.

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<http://dx.doi.org/10.1016/j.ijnt.2013.11.003>

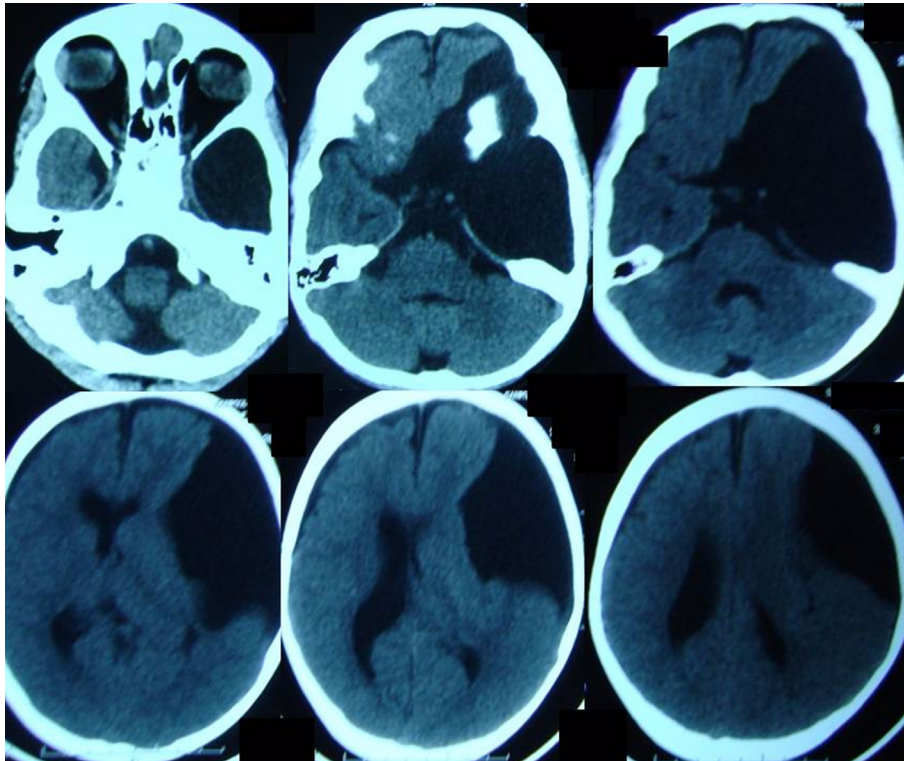


Fig. 1 – CT scan done immediately following injury showing the frontotemporal giant arachnoid cyst with midline shift and effacement of ipsilateral lateral ventricle.

2. Case report

A 6-year old girl, following a traffic accident, sustained mild bruises without loss of consciousness or seizures. The CT performed immediately after the accident showed a hypodense cystic lesion in the middle cranial fossa on the left side involving the entire sylvian fissure and displacing temporal and frontal lobes. There was mass effect in form of effacement of ipsilateral lateral ventricle without any evidence of hematoma, bleed or overlying bony injury (Fig. 1). She continued complaining of occasional mild-to-moderate holocranial headache that resolved over 10 months time. An MRI repeated 10 months after the accident showed the T1 hypointense and T2 hyperintense cystic lesion in the frontotemporal, region with a left sided frontoparietal subdural hygroma. On contrast administration there was enhancement of walls of the hygroma. There was no midline shift or hydrocephalus (Fig. 2). As the child had no evidence of raised intracranial pressure, leukocytosis or febrile episodes, no operative intervention was undertaken and she has been kept on regular follow up.

3. Discussion

The simultaneous presence of a subdural hygroma with a frontotemporal arachnoid cyst in our patient may be related to the post-traumatic or post-Valsalva maneuver rapid rise of intracystic pressure of the pre-existing arachnoid cyst that caused its rupture into the subdural space. A one-way valve

mechanism between the subarachnoid space and the arachnoid cyst may have also led to a rapid increase in its pressure with consequent rupture of its cerebrospinal fluid into the subdural space.^{1–5} The subdural hygroma may grow by increase in its oncotic pressure due to fluid loculation precipitating further ingress of fluid from the subarachnoid space along the osmotic gradient. The enhancement of the subdural membrane in our patient may have been related to the development of inflammation or a subdural empyema. However, the lack of fever or leukocytosis and, the resolution in the clinical symptomatology over time without antibiotics eliminated this possibility. The other likely explanations included the presence of hemorrhage along its walls or the development of a thick membrane of a chronic subdural hygroma that may also show enhancement. Spontaneous resolution without intervention is usually the most likely course in these lesions; however, craniotomy and fenestration, subduro-peritoneal shunt, subduro-cisternal shunt or subdural drainage have been tried for resolving a symptomatic subdural hygroma associated with an arachnoid cyst.^{1–3,6–9}

Awareness regarding the association of a subdural hygroma with an arachnoid cyst is of paramount clinical importance in children. In our patient, the CT scan done immediately after the child sustained trauma did not reveal the subdural hygroma coexisting with the arachnoid cyst but showed its indirect presence due to the presence of the midline shift and ipsilateral lateral ventricular effacement. A subsequent MRI done after 10 months when the child had become asymptomatic revealed the subdural hygroma as well as the arachnoid cyst but the midline shift had resolved. A CT image may, therefore, not be able to

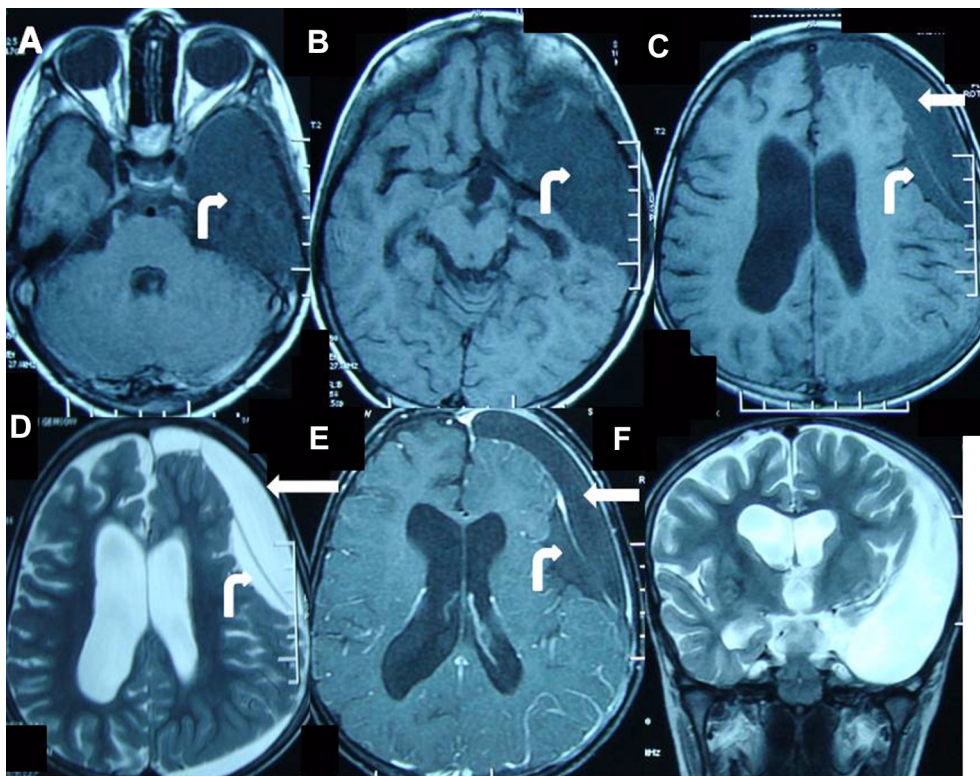


Fig. 2 – a–c: T1 axial; d: T2 axial; e: contrast T1 axial; and e: coronal T2 showing the frontotemporal arachnoid cyst (curved arrow) with the coexisting subdural hygroma (straight arrow). There is enhancement of membrane of the subdural hygroma (Figure 2e). The midline shift and ventricular effacement have resolved (Figure 2f).

detect the simultaneous presence of a subdural hygroma causing a sudden increase in symptoms of headache or raised intracranial pressure in a pre-existing arachnoid cyst. The treating physician may be unable to comprehend the reason for change in clinical status of the child as the CT image may appear unchanged from previous scans. This may induce a false sense of security precipitating the risk of a catastrophic herniation due to rapid rise of raised intracranial pressure. This situation may be avoided by detecting the simultaneous presence of the subdural hygroma on MR imaging and by carrying out serial imaging to detect a temporal sequence of progressive brain shift over time.

Conflicts of interest

All authors have none to declare.

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