Chronic Subdural Hematoma

Chronic subdural hematoma has a protean manifestation in infancy and childhood. It may, therefore, often go undiagnosed unless its possibility is actively considered and relevant investigations are asked for. Further the confirmation of the diagnosis has become a simple affair with the availability of CT Scan. These points are clearly brought out in the case under report.

A 7-month-old female, born after a gestational period of 28-30 weeks and weighing a 'little over 1 kg' at birth, was admitted to the Pediatrics services of J.N. Medical College Hospital with a history of irregular fever, dry cough and intermittent episodes of rapid breathing for a month. Increasing irritability and head banging appeared a day prior to hospitalization. She was seen by her local doctor who asked for a lumbar tap which was reported to be normal. She developed generalized tonic spasm lasting for 15-20 minutes twelve hours after lumbar tap. It was controlled with parenteral diazepam but the convulsions recurred within a few hours. The baby stopped opening her left eye after the attack of seizure and became increasingly stuporous and finally lapsed into deep coma. She was referred to us at this stage. Physical examination revealed an afebrile child weighing 3.25 kg. Her pulse rate, respiratory rate and BP were 115/min, 70/min 132 mm of Hg (systolic), respectively. All the peripheral pulses were well felt and were regular but of high volume.

A working diagnosis of meningitis (partially treated or tuberculous) with raised intracranial tension and seizures was made. Investigations for sepsis screening were advised. Meanwhile, she was given antibiotics (crystalline penicillin + chloramphenical) in antimeningitic doses along with mannitol, phenytoin and blood transfusion. Her head size however, increased by 0.3 cm in the next 36 hours. On review of the case at this stage, a diagnosis of meningitis seemed unlikely in view of the clinical profile and laboratory findings. Possibility of a space occupying lesion in the form of subdural effusion or chronic subdural hematoma was considered. Transillumination test was negative. On repeated questioning the parents came out with the history of fall from a 3 feet high cot. Symptoms suggestive of head injury, following the fall were absent. CT Scan head revealed the diagnosis of chronic subdural hematoma (Fig.). It was drained through a frontal burrhole. The baby regained consciousness in the next 24 hours. She was left with no neurologic deficit except 3rd left cranial nerve palsy. Normal reaction to light and full range of movements in the left eye reappeared by 4th post-operative day. The ptosis however was persisting on the 6th day after surgery when the patient left against medical advice.

Seizures, tense anterior fontanel, enlarging head size, impaired consciousness,
Fig. A hypodense lesion between the inner table of the skull and the brain parenchyma in the left frontoparietal region causing collapse of the ventricles on the same side and shift of the brain to the right. B&C. Areas of decreased attenuation in the left cerebral hemisphere suggestive of cerebral edema.

Summing up, the possibility of chronic subdural hematoma should be considered in an infant with this clinical profile, specially if the head size is rapidly increasing despite decongestive therapy. Ultrasound or CT Scan of the head should be done to rule out the possibility.

REFERENCES


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irritability, failure to thrive, anemia and unexplained fever as documented by others (1,2) were all present in the case under review. However, vomiting which is almost invariably present (1) was conspicuous by its absence in our patient. Retinal or subhyaloid bleed that are pathognomonic of chronic subdural hematoma was also not seen in this infant. Ipsilateral 3rd cranial nerve palsy as noted in the patient under review has not been reported earlier. Though head injury is the usual initiating factor it may be so insignificant as to have been forgotten or disregarded. The parents may come out with the history only on repeated questioning as seen in this case.

Visceral Leishmaniasis Treated with Rifampicin and Cotrimoxazole

Visceral leishmaniasis is a parasitic disease treated conventionally by stibogluconate or, in stibogluconate resistant cases, pentamidine. The more toxic amphotericin B is recommended in cases not responsive to these drugs. Other drugs including rifampicin, INH and cotrimoxazole have been used in therapy of visceral leishmaniasis with inconsistent results (1). We