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Benign Transient Intracranial Hypertension in an Infant Case Report

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ABSTRACT

A four month old boy was admitted with a fever and vomiting for 2 days duration. The child had a minor fall at home two days prior to hospital admission. On physical examination, he was febrile and irritable but consolable. Vital parameters were stable, Occipitofrontal circumference at admission was 42.5 cm (75 thcentile) with bulging and tense anterior fontanelle. The ophthalmologic evaluation was negative for papilledema or venous congestion. Neurological examination did not reveal cranial nerve involvement, sunset sign ,or pyramidal signs. There was no otitis media or focal signs of infection. Chest, cardiac and abdominal examinations were normal. Our patient had no history of recent vaccination, steroid, or Vitamin A ingestion. Clinical possibilities of acute meningitis, intracranial bleeding, obstructive hydrocephalus, and intracranial hypertension were considered. The child was evaluated along these lines, and urgent Cerebral ultrasounds followed by CT head were performed (Fig. 1 and 2). Neuroimaging revealed features of increased intracranial pressure, without intracranial bleeding, intracranial space-occupying lesion, or venous thrombosis. Blood investigations with sepsis markers were performed which showed Hemoglobin, total count and differential count, serum C- reactive protein, and procalcitonin were within normal range. The child had no evidence of sepsis in investigations. A lumbar puncture was performed, opening CSF pressure was 11 cm H_2O (Normal value 5-7 cm H_2O). CSF analysis was not suggestive of infection or inflammation. A clinical diagnosis of Intracranial Hypertension was made. The child was evaluated for metabolic and endocrine causes of raised intracranial pressure, investigations revealed normal serum calcium, serum electrolytes, thyroid profile, normal serum level of vitamin D 25-OH 36 mg mL^{-1} (normal value >30 ng mL^{-1}) and serum Vitamin A level 0.40 µg mL^{-1} (normal value 0.14-0.52 µg mL^{-1}).The child was empirically started on antibiotics, he was closely monitored for new symptoms and clinical deterioration; bulging anterior fontanelle and fever resolved within 48 hrs and his head circumference reduced from 42.5 cm to 41 cm. Antibiotics were stopped once blood and CSF cultures were sterile. The child was followed up at 4 weeks and 8 weeks after discharge, he remained asymptomatic.

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Key Words
Hypertension, Infant, vomiting, ultrasounds

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DISCUSSION

Fever, vomiting, and bulging anterior fontanelle in infants is a classical triad of symptoms of acute meningitis. However, a small percentage of patientswith intracranial hypertension unrelated to CNS infection may present with similar clinical features resulting in a diagnostic dilemma.Bulging and palpable tension of the anterior fontanelle in infants is an indicator of increased intracranial pressure (ICP) and raises serious concerns for intracranial pathology such as meningitis, hydrocephalus, cerebral edema, intracranial bleeding, space-occupying lesion, or idiopathic intracranial hypertension (IIH).Increased ICP is typically accompanied by headache, vomiting, altered mental status, acute onset squint, diplopia or papilledema.

Acute meningitis is the most common clinical diagnosis in this clinical scenario, however, other causes of fever with bulging anterior fontanelle in infants should also be considered.Idiopathic intracranial hypertension (IIH) may progress rapidly henceit requires prompt recognition, therapeutic CSF drainage and medications to reduce CSF pressure. Lumbar puncture is a diagnostic as well asa therapeutic procedure in intracranial hypertension, nevertheless, due precautions must be taken and contraindications for lumbar puncture should be ruled out.The entity known as transient intracranial hypertension of infancy represents a benign cause of acutely increased ICP and is typically transient and innocuous, however, it needs to be differentiated from other serious conditions.

The pathophysiology of benign transient intracranial hypertension in infants is poorly understood. Transient intracranial hypertension has been described in several clinical settings. It has been associated with roseola infantum, coronavirus-associated disease, measles infection, and DPT vaccination administration.This raises a possible explanation of fever affecting CSF flow dynamics in susceptible individuals in the absence of meningeal inflammation. There is little data on how temperature affects cerebral hemodynamics in clinical settings,body temperature affects cerebral hemodynamics as evaluated by Transcranial color-coded duplex sonography (TCCDS) when the temperature rises cerebral blood flow- velocity (CBF- V) increases in parallel and vice versa when temperature decreases.

It is interesting to note that fever could be secondary to acute intracranial hypertension due to autonomic disturbance. Talman et al. and other experts have proposed the possible mechanism of fever secondary to raised intracranial pressureas an abnormal response of the hypothalamic nuclei responsible for thermoregulationas well as a disruption of the communicating periventricular neuronal networks.

Fever has also been well described in patients with obstructive hydrocephalus and malfunction of non-infected ventriculoperitoneal shunts emphasizing that fever is a secondary response toraised intracranial pressure. The index child had no features of infection or sepsis on blood investigations, hence transient

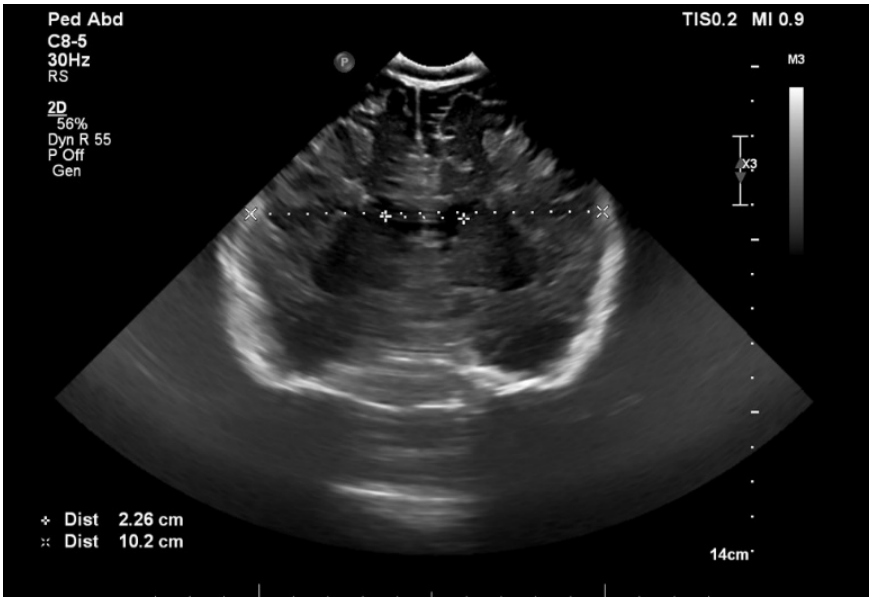

Fig. 1: Transcranial ultrasound through the anterior fontanelle in the coronal plane revealed no abnormality. The ventricular index (measured) was normal for the patient's age and gender

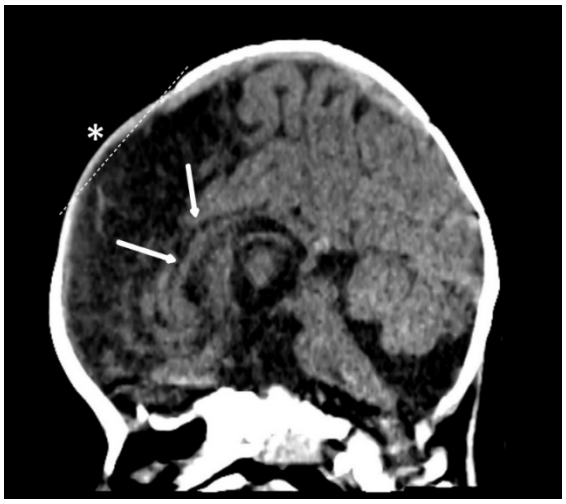

Fig. 2: CT scan of the head without contrast, taken in a supine position while the child is calm (not crying), sagittal reconstruction at the level of interhemispheric fissure revealed: bulging anterior fontanelle (asterisks) above the level of calvarium (dotted line) and bowing of corpus callosum (white arrows), suggestive of increased intracranial pressure. No other abnormality detected

raised intracranial pressure may have triggered fever in the index child. In the absence of obstructive hydrocephalus, CSF flow dysregulation may be one explanation of such clinical presentation and warrants more research in this field. It is worth following up with children for recurrence and long-termoutcomes with the headache associated with CSF dysregulation at a later age.

If a child does not improved clinically, it is important to repeat CSF analysis as early meningitis may fail to cause to an initial pleocytosis and meningitis can be missed. An infant who presents with fever and intracranial pressure of unknown origin which rapidly resolves should be reassessed at regular intervals to monitor for recurrence of raised intracranial pressure.

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