Benign Intracranial Hypotension

Sir,

The syndrome of Benign Intracranial Hypotension is characterized by headache, vomiting, dizziness, diplopia, facial numbness all of which are hypostatic in nature. The atypical features include parkinsonism, fronto-temporal dementia, hypopituitarism, seizures, coma and death. The cause of this hypotension can be due to either CSF leak or can be spontaneous without any apparent cause. We report here a young woman with severe postural headache, convulsions and systemic hypotension with MRI brain findings suggestive of benign intracranial hypotension.

A 24 years female presented with low grade fever, generalized headache, mainly postural with vomiting since two months. She developed 4-5 episodes of generalized tonic-clonic convulsions on the day of admission associated with uprolling of eyeballs and sphincter incontinence. On examination, her blood pressure was 70 mm Hg systolic in supine position with severe headache on sitting. Patient was conscious, oriented with normal higher functions, cranial nerves and motor examination. Coordination was normal. She had marked neck stiffness. She had bronchial breath sounds in the right infraclavicular area. Laboratory investigations revealed Hb of 10.2 gm%, BUN 11 mg%, creatinine 0.7 mg%, serum sodium 135 mEq/l, serum potassium 3.8 mEq/l and random blood sugar was 96 mg%. X-ray chest showed the presence of right upper zone infiltration. Sputum AFB and HIV was negative. MRI brain (Fig. 1) revealed concavoconvex CSF collections along bilateral frontoparietal and temporal convexities, tentorium and interhemispheric regions with moderate degree of pachymeningeal enhancement suggestive of benign intracranial hypotension. Lumbar puncture with manometry showed low opening pressure of 40 mm H₂O with normal CSF examination. Her screening serum cortisol (8 am) was normal (15 mg/dL). A final diagnosis of Benign Intracranial Hypotension with sputum AFB negative pulmonary tuberculosis was made. A normal serum cortisol in our case ruled out coexistent Addison’s disease. She was treated with intravenous fluids, increased salt intake, oral deriphylline, phenytoin and antituberculous therapy and nursed in supine position. Her headache resolved with BP improving to 98/70 mm Hg at discharge three weeks later.

Normally CSF supports the brain such that its weight of 1500 gm amounts to only 48 gm within the cranium. Depletion of CSF volume with downward displacement of the brain causes traction on the pain sensitive structures. The Monro-Kelly thesis proposes dilatation of intra-cranial pain sensitive vascular structures as a cause of headache, which could be worsened in upright position and by Valsalva maneuver.

Diagnosis of BIH is confirmed by demonstrating decreased CSF opening pressure (< 60 mm H₂O) with characteristic MRI findings of diffuse thickened pachymeninges with gadolinium enhancement, venous sinuses engorgement, subdural collection and brain herniation. The treatment described includes conservative management with bed rest, caffeine, theophylline, hydration, increased salt intake, carbon dioxide inhalation and corticosteroid therapy. CSF leak, if detected, requires epidural blood patches, or surgical repair of meningeal tears.

Thus, BIH is an uncommon cause of very common symptom (headache) with typical neuroimaging findings and postural nature of headache could be a vital clue for suspecting the diagnosis.

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Fig. 1: T2 weighted image showing CSF (white arrows) along the fronto-parieto-temporal convexities.


Tracheoesophageal Fistula after Acid Ingestion

Sir,

A 15 years girl presented with the complaints of bouts of cough during meals and difficulty in deglutition mainly for solid foods since one month. Her old record showed that she had ingested sulphuric acid accidentally about 2 months back and was managed conservatively. On examination she was apparently normal. Pallor was present. Haemogram, renal function test, liver function test chest X-ray and ECG were within normal limits.

Barium swallow (Fig. 1) showed narrowing of lower part of esophagus with spillage of contrast material into tracheobronchial tree, through a tracheoesophageal fistula. This was confirmed by direct visualization through bronchoscope.

On endoscopy a stricture was visualized in lowered 2/3rd of esophagus along with fistula and the diagnosis of esophageal stricture with tracheoesophageal fistula was made.

Endoscopic dilatation was carried out and Ryle’s tube was passed and subsequently feeding jejunostomy carried out.

Chemicals causing esophageal injuries are alkalis and acids. Alkalis cause injury by liquefaction necrosis where as acids cause injury by coagulation necrosis. Ingestion of acids may be suicidal or accidental. The later being common in children. The amount, type, concentration and time of contact of agent all determine the extent of injury. Initial contact causes immediate changes in mucosa which progress during next 3 days. After acute phase, latent phase begins in which stricture formation may occur. The process may proceed rapidly within a month or may progress slowly. The goal of treatment is to prevent permanent injury of stricture formation in esophagus. The tracheoesophageal fistula is a communication between the esophagus and tracheobronchial tree within the neck or thorax, where two organs are contiguous, it may be congenital or acquired.

Acquired tracheoesophageal fistula following chemical injury is well reported. Such patients present with cough after swallowing, which is usually worse with liquids. The diagnosis of fistula is made by barium esophagogram, which demonstrates the tracheobronchial tree. Bronchoscopy is also essential for knowing the extent of involvement of the trachea or bronchus.

Treatment of tracheoesophageal fistula is aimed at the prevention of the passage of ingested material and saliva from the esophagus into the trachea. In this case a stricture was visualized in the lower part of esophagus with tracheoesophageal fistula, it was dilated through an endoscope and Ryle’s tube was passed and kept in situ as a stent along with a feeding jejunostomy. Patient was put on intravenous antibiotics, proton pump inhibitors and domperidone and was discharged after 1 week. Rest of the recovery was uneventful. On follow up patient progressed well, the tracheoesophageal fistula healed, patient is on regular followup now and is doing fine.

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