Acute inhalational tobacco poisoning in children

Sir,

We report here an 11-year-old girl admitted with sudden alteration of sensorium with nausea, vomiting, headache, and muscle weakness. On examination, she was afebrile, pulse rate was 90/min, respiratory rate was 20/min, blood pressure was 130/80 mm Hg, and capillary blood glucose was 141 mg/dL. She had no skin ulcer. Neurological examination revealed normal pupil, Glasgow Coma Score (GCS) of eight without meningeal irritation, cranial nerve involvement, or neuro deficit. She had diffused pain abdomen with passage of stool with altered blood. Detailed history revealed that the child used to work in a tobacco industry for packing chewing tobacco or Khaini. She belonged to low socioeconomic strata. She was compelled to work on part-time basis to support her family. She worked in small premises lacking proper ventilation and sanitation, wearing gloves without face mask. Her father had a monthly income of INR 3400. For every 100 packing, she was given INR 10.

Investigation revealed hemoglobin of 11.8 g/dL, total leukocyte count of 14,400/mm³, polymorphs 80%, lymphocytes 18%, platelet counts 2.4 lakhs/mm³, and erythrocyte sedimentation rate (ESR) 23 at the end of first hour. Biochemistry showed serum sodium 136 meq/L, potassium 5.0 meq/L, calcium 8.6 mg/dL, and blood glucose 96 g/dL. Renal and liver function tests were normal. Chest X-ray and sonography of abdomen were normal. Provisional diagnosis of unknown poisoning was made at emergency. Surgical consultation ruled out surgical etiology. GI endoscopy was deferred as gastrointestinal manifestations became passive soon. Serum nicotine level was elevated (92 µg/L). Urine nicotine level was not measured due to financial constraint.

Gastric lavage was given and supportive management initiated with gastro-protective agents, anti-spasmodics, and empirical antibiotics. GCS improved after 2 h, but sensorium returned to normalcy the next day. She was discharged later with an advice to attend school and abstain from working in tobacco industries.

The child from a tobacco industry had sudden reversible altered sensorium with diffuse abdominal pain and elevated blood nicotine level, which confirmed it to be a case of acute tobacco inhalational poisoning in the absence of evidence of dermal or oral exposure. Acute nicotine poisoning is characterized by nausea, vomiting, headache, muscle weakness, and dizziness. It occurs mainly due to green tobacco sickness (GTS), where nicotine absorbs through skin of the workers who cultivate and harvest tobacco.[1] It occurs rarely in children due to exposure through other routes like enema, inhalation, etc., Some cases of acute tobacco poisoning in children have been reported due to GTS,[1] tobacco enema,[2] or topical application for eczema.[3]

Nicotine is the active ingredient of tobacco. It can be absorbed through lungs, skin, gastrointestinal tract, buccal and nasal mucosa. It acts on noncholinergic presynaptic and postsynaptic receptors. Nicotine and its metabolic product can be detected in urine, serum, and saliva.[4] Acute nicotine poisoning is diagnosed by elevated blood nicotine level. Nicotine inhalation toxicity data are scarce.[5] To the best of our knowledge, acute tobacco inhalational poisoning has not been reported in children earlier. The child labor involved in such a hazardous job may be responsible for serious poisoning as well as tobacco addiction in the long run.

Rakesh Mondal, Abhishek Roy, Goutam Mukherjee¹, Asok K Mandal
Departments of Pediatrics and Obstetrics, North Bengal Medical College and Hospital, Darjeeling, West Bengal, India

Address for correspondence:
Dr. Rakesh Mondal,
Balarampur, Mahestala, Kolkata, West Bengal, India.
E-mail: ivanrakesh2001@gmail.com

References

Sir,

Subgaleal hematoma is a potentially life-threatening extracranial bleed that occurs most commonly in neonates after difficult instrumental deliveries. Its occurrence beyond the neonatal period is rare and is often associated with head trauma involving tangential or radial forces applied to the scalp causing emissary veins traversing the subgaleal space to be ruptured.

Large subgaleal hematoma due to trivial head trauma in a parahemophilic patient with no reported literature is interesting to describe. Parahemophilia, a relatively rare hemorrhagic disorder affecting both sexes, is due to a congenital and frequently familial deficiency of FV. This autosomal recessive disorder varies greatly in severity. The most commonly reported symptoms are bleeding from mucosal surfaces and postoperative hemorrhage. However, hemarthroses, intramuscular, and intracranial hemorrhages can also occur. Subgaleal hematoma associated with parahemophilia, as seen in our case, is yet to be reported in the literature.

A 15-year-old boy presented with progressive head enlargement in a period of 3 days after sustaining trivial head trauma due to assault. He had history of soft tissue and joint bleeding since childhood. There was no history of recent drug intake or fever. On clinical examination, he was fully conscious and alert with no neurological abnormality, but appeared ill with severe pallor. There were no lymph node enlargements or other manifestations of bleeding tendency such as purpura or ecchymosis. The abdominal examination was normal. Head examination revealed large soft tissue swelling of the entire scalp demonstrated by pressure indentation with normal overlying skin. The head circumference was 67 cm [Figure 1]. Computed tomography (CT) scan of head showed large subgaleal hematoma involving both side with no intracranial hemorrhage, no midline shift, and no skull fractures [Figure 2].

Suspicion of associated coagulopathy arose in our mind. The screening coagulation tests revealed a prolonged activated partial thromboplastin time (aPTT) and marked prolongation of prothrombin time (PT) [Table 1]. Determination of coagulation factor activities yielded normal results, while FV activity was 17% (normal value > 60%). The liver function test was within normal limit. The plasma concentrations of d-dimers, fibrinogen, antithrombin III, protein C, protein S, and plasminogen were found to be normal. There were no antiphospholipid antibodies and no lupus anticoagulant.

The patient received five units of fresh frozen plasma (FFP) transfusions, without complications. A pressure bandage was applied to the scalp and forehead. When aPTT and PT returned to normal range, the hematoma resolved and the head size became normal with improvement of the general condition [Figure 3]. We investigated available members of the family and found the patient's brother with the same deficiency (FV activity, 35%). Other family members were free from same disorder.

Most reported cases of subgaleal hematoma are neonates and the reported incidence of subgaleal hematoma ranges from 1.6 to 3/1000 live births.

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<td>Test &gt;60.0 seconds, within 5-7 seconds of control</td>
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Table 1: Laboratory data demonstrating the patient's coagulation profile

Large subgaleal hematoma as a presentation of parahemophilia.