DHF was suspected in our patient in view of an ongoing epidemic in our area, along with typical history, associated thrombocytopenia, mild elevation of transaminases, small right pleural effusion, and ascites. We believe that the colitis was part of the DHF syndrome; it regressed with recovery from dengue.

References


Correspondence to: Dr Rama Krishna, Assistant Professor. E-mail: ramakappas9@yahoo.co.in
Received August 30, 2005. Accepted October 10, 2005

Aluminium phosphide-induced esophageal stricture

Rupjyoti Talukdar, Dinesh Kumar Singal, Rakesh Kumar Tandon

Department of Gastroenterology, Pushpawati Singhania Research Institute for Liver, Renal and Digestive Diseases, Sheikh Sarai, Phase II, Press Enclave Marg, New Delhi 110 017

We report a 24-year-old woman and a 58-year-old man who developed short-segment esophageal strictures in the upper and mid esophagus two weeks after ingestion of aluminium phosphide tablets. They responded well to endoscopic dilatation. [Indian J Gastroenterol 2006;25:98-99]

Aluminium phosphide (AIP) is a lethal protoplasmic toxin and is used as a solid fumigant pesticide. It is currently the most common cause of suicidal poisoning in northern India. Its toxicity is attributed to phosphine gas (PH₃), which is liberated when AIP tablet comes in contact with water, moisture or gastric acid. The lethal dose for a 70-Kg man is 150-500 mg.

Mild intoxication is marked by retrosternal burning, nausea, vomiting, abdominal pain or headache. Moderate to severe toxicity is characterized by fatal arrhythmias and refractory cardiogenic shock with or without ARDS, culminating in a mortality of nearly 70%. In survivors, there are usually no long-term complications. We report for the first time two patients with AIP-induced esophageal stricture.

Case 1: A 24-year-old woman presented with recurrent vomiting, odynophagia and progressive dysphagia over one month. She gave history of suicidal ingestion of a fresh aluminium phosphide tablet one month back. At that time, she developed nausea, vomiting and hypotension. She was hospitalized for 15 days and was managed conservatively. She denied ingestion of any corrosive substance.

Physical examination was normal. Hematological parameters, liver function tests, serum creatinine and electrolytes were also normal. Upper GI endoscopy (UGIE) showed an esophageal stricture at 30 cm from the incisors with a circumferential ulcer. Biopsy showed thickened mucosa with congested papilla and chronic inflammation. The stroma was edematous with diffuse polymorphonuclear cell inflammation. A short-segment tight narrowing of about 3 cm was seen at the corresponding area on barium swallow examination. She was subjected to serial esophageal dilatation using Savary-Gilliard dilators, and the stricture was dilated up to 15 mm.

Case 2: A 58-year-old man presented with progressive dysphagia for about a month. He consumed a fresh aluminium phosphide tablet one and a half months back with suicidal intent. After ingestion, he had a feeling of the tablet getting stuck in the upper esophagus, following which he had vomiting. The vomitus however did not contain the tablet. He did not develop any other significant complication during that episode and was managed conservatively for 3 days in a local hospital. After discharge, he developed odynophagia for about 20 days that gradually progressed to increasing dysphagia. He denied corrosive ingestion.

Barium swallow examination revealed a short-segment narrowing in the upper esophagus. UGIE revealed a tight concentric stricture at 18 cm that was found to extend down to 20 cm after esophageal dilatation. The stomach and the duodenum were normal. Biopsy from the strictured area showed features of chronic esophagitis.

Toxic effects of AIP are usually acute. There are only a few reports of delayed toxicity, one of which was of neuromuscular weakness and the other of hemorrhagic stroke that probably resulted from vasculitis due to direct toxic effect of PH₃ on the vessel wall. In both our patients stricture developed about two weeks after ingestion and involved a short segment of the upper and mid esophagus. These developed probably due to prolonged contact of the AIP tablets with the esophageal mucosa, resulting in local mucosal ulceration. Both our patients showed good response to bougie dilatation.
Singh D, Jit I, Tyagi S. Changing trends in acute poisoning: hemoglobin 9.2 g/dL, total WBC count. He developed symptoms immediately after application of soap and water enema. The symptoms resolved gradually over four weeks. Follow up at six months and one year showed no abnormality.


References

Correspondence to: Dr Singal, B-14, Law Apartments, Karkar Dooma, Delhi 110 092. E-mail: gastro_singal@rediffmail.com

Received September 9, 2005. Accepted November 15, 2005