is necessary to re-emphasize the point made by Sood et al.1 that history of intake of medicines from alternative systems of medicines should form an inherent part of history-taking. It is also important to educate patients regarding the dangers of self-prescribing of even "herbal" medications and to inform their doctors regarding the intake of such drugs.

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References

Barotrauma: a cause of hemobilia

The most common cause of hemobilia in the Western world is iatrogenic, mostly as a consequence of percutaneous liver procedure; this is followed by non iatrogenic trauma, most commonly road traffic accidents. We report a patient with hemobilia caused by barotrauma who was successfully managed with transarterial embolization (TAE) of hepatic artery by cyanoacrylate.

A 10-year-old boy had an injury over the abdomen when a firecracker exploded while he was leaning over it. He had severe right upper abdominal pain, which subsided within a day. Two days later he had episodes of heematemesis. Examination revealed mild pallor; there were no external injury marks. Endoscopy revealed blood in the duodenum. Ultrasonography was normal. Contrast-enhanced CT scan showed pseudoaneurysm in relation to the hepatic artery. The patient continued to have episodes of melena and was transfused six units of blood. Transarterial catheterization was done via the femoral route, right hepatic artery was selectively catheterized (Fig) and cyanoacrylate embolization was done as close to the lesion as possible. A subsequent film showed complete embolization and presence of glue cast in the region of the pseudoaneurysm. The hemobilia stopped completely. The patient was discharged after 72 hours. One year later he is symptom-free.

Hemobilia after blunt trauma is relatively uncommon.1 To the best of our knowledge this is the first case of hemobilia after a blast by firecracker leading to barotrauma. The blast could have led to shock waves that caused shearing force in the liver area involving the branch of the hepatic artery, leading to formation of pseudoaneurysm. Associated parenchymal injury predisposes to bile stasis. Bile has been shown to inhibit fibrin formation and granulation in liver wounds.2 This expanding pool of necrotic tissue may eventually erode into hepatic blood vessels allowing fistula formation.

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References

Brunneroma causing gastric outlet obstruction

We read with interest the report by Wagholikar et al.1 of a young patient presenting with melena due to chronic bleeding from a Brunneroma. We wish to report a lady who presented with chronic gastric outlet obstruction due to circumferential Brunner gland hyperplasia of the proximal duodenum.

A 45-year-old housewife presented with 8-year history of episodes of non-bilious vomiting, each lasting 3–4 days. Clinical examination revealed visible peristalsis in the lower abdomen. Relevant biochemical and hematological investigations were normal. Upper gastrointestinal endoscopy on at least two occasions earlier were reported as normal.

In the present admission, endoscopy showed a large stomach with a narrow first part of duodenum and inability to pass the scope beyond; a few superficial erosions were seen. Barium series showed a massively dilated stomach with a smooth narrowing at the pyloro-duodenal junction (Fig).
The gross specimen showed stomach wall measuring 7-11 mm in thickness (the mucosa was 2-4 mm in thickness, and muscularis propria 6-8 mm), with a narrow pyloro-duodenal canal that just admitted a fine lacrimal probe. Histology showed diffuse Brunner gland hyperplasia with no atypia. The hypertrophied glands extended circumferentially across the entire duodenum and measured 6-8 mm in thickness. Gastric wall biopsies revealed normal to atrophied mucosa and hypertrophied subjacent muscularis propria measuring 6-8 mm.

In a follow up of nearly 2½ years, she had had no further episodes of vomiting and has gained appetite and weight.

Obstruction of the duodenum in these instances is usually due to polypoidal lesions; occasionally, the diffuse circumferential hyperplasia variety is severe enough to cause gastric outlet obstruction or duodenal stenosis. Only one other case report has been described with features of duodenal stricture/stenosis due to diffuse circumferential hyperplasia.²

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References

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Correction
In the Letter "Immune responses induced by two dose strengths of an yeast-derived recombinant hepatitis B vaccine in adolescents" (2003;22:71-2), the name of the 7th author should read as Prasad Kulkarni instead of Alka Kulkarni. We regret the error.