northern India who develop acute hepatitis E a few weeks after they arrive in Mumbai for treatment of cancer at our center.

Our recent experience adds further evidence. One of us (YS) attended the annual conference of the Indian Society of Gastroenterology held in November 2001 at Lucknow. A week after returning to Mumbai, he developed prodromal symptoms. Biochemical hepatitis was documented four days later, followed by testing IgM anti-HEV positive. Serum bilirubin level peaked (at 10 mg/dL) in a further two weeks. Biochemical recovery was noted approximately five weeks after first testing; clinical normalcy took a further two weeks. The sequence of events fits into the epidemiology of HEV.2

All travelers to endemic regions must be advised to take adequate precautions. This is even more important for immunocompromised patients and pregnant women, as the case fatality of acute HEV hepatitis is high in these individuals.3

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References

Primary hypertrophic enteropathy

Primary hypertrophic enteropathy, with marked hypertrophy of the muscular coat of the small bowel without any known cause, is a primary visceral myopathy causing acute or chronic intestinal pseudo-obstruction.

A 50-year-old woman presented with pain and distension of the abdomen since two years. The pain was associated with infrequent vomiting. She had had similar attacks recurrently since two years. There was no significant family history. Systemic examination was normal. At emergency laparotomy, a firm stricture of 1 cm length in the ileum was resected, with end-to-end anastomosis. The postoperative period was uneventful, and the patient has been asymptomatic for the two years.

Histology of the resected specimen revealed normal ileal mucosa and submucosa. The muscle coat showed marked hypertrophy (Fig). The myenteric plexus was normal. There was no evidence of malignancy or any other pathology.

Myopathies of the gastrointestinal tract produce...
due to common carotid artery occlusion.

A 42-year-old man had chronic small-volume diarrhea with bleeding per rectum; a diagnosis of UC was made based on findings at colonoscopy and rectal biopsy elsewhere. Despite treatment with prednisolone enema and oral 5-aminosalicylic acid (5-ASA), he had frequent relapses of UC. He presented to us with a severe relapse, which was treated successfully with intravenous followed by oral corticosteroids; this was tapered off, and 5-ASA was continued. Colonoscopic and histologic evaluation at this stage showed pancolitis.

A year later, he developed sudden left-sided hemiparesis, which progressed to hemiplegia over a 24-hour period, while he had another relapse of bloody diarrhea. This was preceded by pain in the right periauricular area for 3 days. There was no history of headache, vomiting, convulsion, alteration of sensorium, diabetes mellitus or hypertension. He was treated with oral corticosteroids and 5-ASA elsewhere for one week. Plain CT scan of head showed an ischemic infarct abutting the right lateral ventricle. He presented to us a month after the onset of hemiparesis. Examination revealed weak right carotid and radial pulses, blood pressure of 120/60 mmHg in the left arm and 80/60 mmHg in the right arm, normal higher mental functions and cranial nerves, left hemiparesis (muscle power 2/5) and normal sensory function, no cardiac murmur or arterial bruit. Chest and abdomen were unremarkable.

Investigations: hemoglobin 10.2 g/dL, total leucocyte count 7000/mL, platelet 190,000/mL. Carotid Doppler study showed absence of blood flow in the right common carotid artery up to the base of skull. Magnetic resonance imaging showed infarct in the right internal capsule area; magnetic resonance angiography (Fig.) failed to visualize the right common carotid artery in the neck and right middle cerebral artery at the base of the brain. Protein S and C levels were normal. Azathioprine (AZA; 100 mg/day) was started and prednisolone was gradually tapered off. Oral 5-ASA and active physiotherapy for hemiparesis were continued. Angiography done 4 weeks later showed complete block of the right common carotid artery with normal aorta and renal arteries. Three months later, UC was in remission, and left hemiparesis had improved.

Vascular events have been reported in patients with UC. The frequency of thromboembolic complications in UC has varied from 1.3% to 39% in clinical series.6 In a review of stroke in patients with inflammatory bowel disease, around 70% were found to occur during an acute flare-up of the bowel disease.6 Rarely, involvement of large arteries of the brain in association with TA has been reported in UC.6 Rarity of thrombosis of large arteries and significant difference in blood pressure in the upper limbs led us to suspect TA in our patient. Although right subclavian or brachio-

Fig: Magnetic resonance angiography showing right and left vertebral arteries and left common carotid artery with its branches; the right common carotid artery is not visualized.

cephalic artery involvement was not found on angiography, this might be related to treatment with corticosteroids and AZA for one month before angiography. Considering the autoimmune pathogenesis of both UC and TA, an association is not unexpected. Given the good therapeutic response of TA to immunosuppressive therapy, particularly AZA, this drug in combination with prednisolone may be started early in such patients.

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