Fig: Section from stomach showing hypertrophic nerve bundles (N) in outermost layer of muscularis propria (M). The adjacent serosa (S) is unremarkable (H & E, 23 x).

Rosai-Dorfman disease, where one could encounter multiple systemic neoplasms.

We speculate that the enterochromaffin cell hyperplasia (primary or secondary) was an initiating event in the neural hypertrophy. The hypertrophy could alternatively represent an occult non-familial type of neurofibromatosis.

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Vesical varix in cirrhosis of liver

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Vesical varices in portal hypertension are rare. We report a patient with portal hypertension who developed recurrent painless hematuria. Cystoscopy was normal. Doppler ultrasound and MR angiography showed a dilated paraumbilical vein within the falciform ligament coursing down to the urinary bladder wall and draining into the right internal iliac vein.

He underwent liver transplantation for decompensated chronic liver disease. He is in good health and has not had further episodes of hematuria. [Indian J Gastroenterol 2000;19:193-194]

Key word: Hematuria

Uncommon sites of portal-systemic collaterals in portal hypertension include the duodenum, jejunum, ileum, colon, rectum and peritoneum. Bleeding has been reported from peristomal varices and infrarenal sites such as ovary and vagina. We report a patient with cirrhosis of liver who developed hematuria from a vesical varix.

A 27-year-old man was diagnosed to have cryptogenic cirrhosis of liver with portal hypertension; he had grade 1 esophageal varix and was on drug prophylaxis (propranolol) 20 mg twice daily and isosorbide-5-mononitrate 20 mg twice daily) to prevent variceal bleeding. He presented with recurrent episodes of gross painless hematuria (5 times in 45 days). Each episode lasted 24-36 hours and resolved spontaneously. Clinical examination was unremarkable.

The hemoglobin level decreased from 11 g/dl to 9.4 g/dl. His urine showed numerous red blood cells and 2-4 leukocytes, without any casts. He had microproteinuria (186 mg/1800 ml). During the first two episodes of hematuria, no organism was found on urine culture. There was asymptomatic bacteriuria on urinary culture (insignificant colonies) during the latter three episodes. There was no leukocytosis; platelets were 50-60 x 10^9/L. He had borderline elevation of liver enzymes, normal bilirubin and alkaline phosphatase levels, with normal renal profile. The coagulation parameters were normal.

Cystoscopy did not reveal any mucosal abnormality, stone or tumor. Intravenous urogram was normal. Ultrasonography with color Doppler showed a small echogenic liver, and a patent portal vein measuring 16 mm in diameter. The paraumbilical vein was dilated and measured 20 mm (within the falciform ligament), extending to the posterior abdominal wall. The vessel extended downwards in the right paramedian region to the urinary bladder wall. The varix on the bladder wall measured 21 mm in diameter and was found to indent the bladder lumen. Low-velocity venous (non-pulsatile) pattern of flow was noted on spectral Doppler. Its further continuity could not be determined. Magnetic resonance angiography (Fig) of the lower abdomen showed a tortuous varix in the pelvic region. Anteriorly, it could be traced to the abdominal wall and posteriorly to the region of the right internal iliac vein. Red cell tagged scan done during an episode of hematuria showed a hot spot in the bladder corresponding to the area of the varix.

Following two episodes of spontaneous bacterial peritonitis and three episodes of encephalopathy, he underwent orthotopic liver transplantation. The radiological findings were confirmed. He is in good health after transplant and has not had further episodes of hematuria.

The most common causes of painless gross hematuria include malignancy and tuberculosis of the urinary tract. Hematuria has been reported in patients with cirrhosis of the liver: both these patients, however, had undergone interventions that could alter portal pressure dynamics and flow pattern. Our patient had received
VIPoma of pancreas in a child

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An eleven-year-old girl had massive watery diarrhea. She was found to have pancreatic VIPoma. It responded favorably to surgical resection of the tumor. There was no tumor recurrence at 18 months of follow-up. [Indian J Gastroenterol 2000;19:194-195]

Key words: Diarrhea, pancreas tumor, vasoactive intestinal polypeptide

Tumor secreting vasoactive intestinal polypeptide (VIP) has been identified as a cause of secretory diarrhea. VIPoma arising from the pancreas is rare in children, most reported ones being ganglioneuromas or ganglioneuroblastomas in the neck, thorax, adrenals or pelvis. We report a child with pancreatic VIPoma.

An eleven-year-old girl presented with intermittent, large-volume diarrhea of two years' duration. The stool volume was more than two liters per day and diarrhea persisted during fasting. There was no history of laxative abuse or abdominal surgery. The diarrhea was not associated with abdominal cramps, joint pain, fever, skin rash or lymphadenopathy. Physical examination revealed moderate dehydration and her weight was below the third percentile for age.

Investigations: Routine laboratory tests including liver profile were normal. Serum creatinine was 2.6 mg/dL, sodium 130 mEq/L, chloride 109 mEq/L, potassium 2.9 mEq/L (less than 2 mEq/L on three occasions during hospital stay), bicarbonate 14 mEq/L, magnesium 1.82 mEq/L, calcium 10 mg/dL, phosphorus 2.1 mg/dL; normal serum immunoglobulins, lipid profile and prothrombin time. Urine osmolality was 238 mOsm/Kg H2O. Twenty-four-hour urinary excretion of sodium was 47 mEq/L, potassium 13 mEq/L, calcium 65 mg/dL and phosphate 88 mg/dL. She tested negative for antibodies to human immunodeficiency virus (ELISA). Fasting gastric juice pH was 5.0.

Stool examination was negative for parasites and pathogens on several occasions. Seventy-two-hour fecal fat excretion was 35 g whereas urinary d-xylene excretion test was mildly impaired. Fecal potassium and sodium concentrations were 78.8 mEq/L and 30 mEq/L, respectively, with stool osmolality of 226 mOsm/Kg H2O.

Barium meal follow-through examination was normal.

References