amination has revealed evidence for an inflammatory genesis, with destruction of reticular fibers and local deposits of IgG and activated complement. Peliosis must be treated surgically, because rupture of these lesions has been reported to be fatal.

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Ulcerative colitis with sagittal sinus thrombosis with normal coagulation profile

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Thromboembolic events are serious complications in patients with inflammatory bowel diseases. We describe a 30-year-old man with ulcerative colitis complicated by sagittal sinus thrombosis with normal coagulation profile; he achieved clinical remission with subcutaneous heparin. [Indian J Gastroenterol 2000;19:88-89]

Key words: Cerebral venous thrombosis

Thromboembolic events are a serious complication in patients with inflammatory bowel diseases (IBD). Resistance of factor V to degradation by activated protein C (APC) is found in approximately 30% of patients with thrombosis. We report a patient with ulcerative colitis complicated by sagittal sinus thrombosis, with normal coagulation profile.

A 30-year-old man with ulcerative colitis in relapse was started on corticosteroids (40 mg prednisolone/day) along with 5-aminosalicylic acid, orally and as enemas. In spite of these, the frequency of stools was 3 to 4 per day with moderate bleeding. Two months later, the patient developed severe occipital headache which gradually became global; it was followed by facial swelling, especially around the eyes, and vomiting. On admission, fundus examination revealed bilateral papilledema (L>R).

CSF examination and CT scan head were normal. Anticerebral edema measures were started, but his clinical condition did not improve. MR angiography brain (Fig) revealed superior sagittal sinus thrombosis with extension into the straight sinuses and cerebral edema. Hemogram, blood sugar and renal and liver profiles were normal. Blood, stool and throat swab cultures were sterile. His coagulation profile (prothrombin time, partial thromboplastin time, lupus anticoagulant, antinuclear factor, protein C, activated protein C and protein S levels, and antithrombin III activity) was normal.

Broad-spectrum antibiotics, steroids and mannitol were started. Heparin and thrombolytic therapy were withheld because of continuing GI bleeding. Twelve hours later, left upper limb weakness and difficulty in speech appeared. On examination there was only 2/5 power in the upper left limb, left VII upper-motor-neuron type cranial nerve involvement and left-sided VI nerve palsy. Low-molecular-weight heparin was started along with intensive anti-edema measures, while monitoring for increase in bleeding from the GI tract or elsewhere.

The weakness in the upper limb and VII nerve palsy recovered in 36 hours but VI nerve palsy remained and diplopia appeared. Heparin was given for 12 days. No bleeding manifestation occurred; GI bleeding also stopped after 48 hours. Aspirin 150 mg/day was started and steroids and acetazolamide were continued. The disease has been in remission for six months. Diplopia resolved in 1 month but papilledema persists in the left eye, though it is gradually resolving.

Ulcerative colitis is associated with a procoagulant state and can induce coagulation disorders. Resistance of factor V to degradation by APC is a major cause for venous thrombosis. Various other factors, e.g., coagulation abnormalities (proteins C and S deficiency, antithrombin III deficiency), hyperhomocysteinemia, antiphospholipid syndrome and some hereditary and genetic factors have been found in a few cases. There was no acquired risk factor in this case and a search for

Fig: Magnetic resonance angiography illustrating sagittal sinus thrombosis with extension into straight sinuses bilaterally

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hereditary and genetic factors was also negative.

Derdyn and Powers reported a woman with ulcerative colitis with spontaneous cerebral hemorrhage. Another case has been described with cerebral venous thrombosis and deep limb vein thrombosis in association with exacerbation of ulcerative colitis. Laboratory investigations revealed transient functional APC resistance and mild hyperhomocysteinemia. After colectomy, the APC ratio was normal. These findings may explain the thrombophilia seen in some patients with ulcerative colitis. In another study, small vessel thrombosis was identified in the bowel of patients with Crohn’s disease. One of 17 patients with Crohn’s disease had APC resistance and 2 patients (one with Crohn’s and one of 6 with ulcerative colitis) had borderline results.

Recently, heparin has been tried successfully in the management of corticosteroid-resistant ulcerative colitis. No serious complications were observed apart from bruising at the subcutaneous injection sites. This beneficial effect of heparin, a group of sulfated glycosaminoglycans, is possibly related to its potentially anti-inflammatory effects. These include inhibition of neutrophil elastase and inactivation of chemokines.

In conclusion, this patient with ulcerative colitis developed sagittal sinus thrombosis and neurological deficit despite a normal coagulogram; he recovered smoothly on treatment with heparin.

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Retroperitoneal teratoma presenting as acute abdomen in an elderly person

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A 56-year-old man presented with acute abdomen. Clinically, he was diagnosed as having perigastric abscess. On exploration, a retroperitoneal cystic teratom was encountered. Postoperatively, he recovered uneventfully and has no residual disease two years later. [Indian J Gastroenterol 2000;19:89-90]

Key words: Cystic tumor

Primary retroperitoneal teratoma is a rare tumor in patients above the age of 50 years. Rarely, it presents as acute abdomen. When the teratoma is predominantly cystic, it can be confused with perigastric, perinephric or cold abscess. Finding multiple tissue components during exploration clinches the diagnosis.

A 56-year-old man presented with vomiting for two days and acute upper abdominal pain and distension for 24 hours. On examination, his temperature was 38°C, and he had tachycardia, abdominal tenderness and rigidity. White cell count was 22,500/mm. Biochemical parameters were normal. Plain X-ray of the abdomen showed a few dilated loops with no air-fluid levels or free gas. Ultrasonography revealed a cystic collection, 10 cm x 10 cm, irregular, with thick fluid, behind the stomach. The diagnosis was perigastric abscess.

At exploration, on opening the lesser sac, a large, irregular, avascular retroperitoneal collection was noted extending from the diaphragm above behind the transverse mesocolon. It did not appear to cross the midline. Aspiration revealed thick brownish fluid. The contents were evacuated, and resembled those of a cold abscess. However, when the opening was widened, small bits of cartilaginous material and hair were found; there was no evidence of teeth or bone. We evacuated approximately 500 mL of contents. Part of the wall was excised with the contents and sent for histological and microbiological examination. The cavity was washed with saline. We placed a 32F tube drain in the cavity; it drained 300-400 mL of serosanguinous fluid daily for six days. Postoperatively, ultrasonography showed a residual cyst, 6 cm x 6 cm, with no solid component. The drainage gradually decreased and the drain was removed on the tenth day. At the end of two years, the patient has no clinical or radiological evidence of the disease.

On histology (Fig), the cyst wall revealed fibro-collagenous tissue with inflammatory cells and a stratified squamous lining epithelium with sebaceous glands. The content of the cyst contained adipose tissue, cholesterol crystals, smooth muscle bundles and sebaceous glands. The final histological diagno-

Fig: Photomicrograph of teratoma