Multiple lymphomatous polyposis presenting as inflammatory bowel disease

АНШУ СРИВАСТАВА, ПУНЕТ МЕХРОТРА, РАКЕШ АГГАРИВАЛ, РАКЕШ ПАНДЕЙ, САНЯЙ КХАННА, СУБНЯШ Р НАЙК

Departments of Gastroenterology and *Pathology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow 226 014

Multiple lymphomatous polyposis is a rare manifestation of primary gastrointestinal lymphoma characterized by polypoidal masses involving several segments of the gastrointestinal tract. We report a case who initially presented with features resembling inflammatory bowel disease. [Indian J Gastroenterol1998; 17: 151-152]

Key words: Nasal polyp

Multiple lymphomatous polyposis (MLP) is a rare primary gastrointestinal (GI) lymphoma thought to arise from the mantle zone of lymphoid follicles. It is characterized by polypoid accumulation of lymphoid tissue and is classified as a small cleaved cell or centrocytic malignant lymphoma. Nearly 120 cases, including two from India, have been reported. We report a young man whose presentation simulated that of inflammatory bowel disease (IBD).

An 18-year-old man presented with bloody diarrhea and abdominal discomfort for two years. Two months later, he developed nasal obstruction and enlarged cervical lymph nodes. He also had asthenia and low-grade intermittent fever. Two months prior to presentation, he underwent surgical drainage of an ischiorectal abscess which was followed by a persistent perianal sinus.

Examination revealed anemia, pedal edema, clubbing and generalized lymphadenopathy. The liver was palpable 2 cm and was soft and smooth; there was minimal ascites. There was a persistent sinus in the perirectal area. On rectal examination, multiple polypoidal masses were felt. The right nasal cavity had a polyp in the posterior part. A diagnosis of IBD with pseudopolyp formation or multiple polypsis coli was considered.

Hemoglobin was 7.8 g/dL; ESR 60 mm in the first hour. Platelet count was normal. Renal and liver biochemistry were normal, except for raised alkaline phosphatase (399 U/L, normal <125). Serum protein was 4.7 g/dL (albumin 1.3). Colonoscopy showed multiple polyps in the rectum, and friability, edema and ulceration in the rectum, sigmoid and descending colon. Rectal biopsy was suggestive of chronic IBD (distortion and loss of crypts, cryptitis and lymphoplasmacytic infiltration of the lamina propria) with pseudopolyps. Colonic biopsies showed similar histology; however, the lamina propria contained a few atypical lymphoid cells, raising the possibility of a lymphoproliferative disorder.

Upper GI endoscopy revealed multiple small polypoid lesions in the stomach. Double-contrast barium enema showed coarse granularity and marginal scrapings in the entire colon and a fistulous tract from the rectosigmoid to the ischiorectal region. Barium meal follow-through was normal. Ultrasonography revealed retroperitoneal lymphadenopathy, hepatomegaly and minimal ascites. Histology of the stomach polyp (Fig), nasal polyp and axillary lymph node showed diffuse centrocytic malignant lymphoma. Bone marrow biopsy was normal. A diagnosis of MLP was made.

The patient was treated with three cycles of CHOP regimen, with partial response (liver and spleen regressed but lymphadenopathy persisted). He was then treated with cyclophosphamide, vincristine, adriamycin, methotrexate and intrathecal Ara-C; lymphadenopathy and hepatomegaly regressed completely. The patient was lost to follow-up.

Most patients with MLP are male and elderly; our patient was only 18 years old. The disease may involve several segments of the GI tract and intraperitoneal lymph nodes. By the time of diagnosis, involvement of extraintestinal sites is frequent. Multiple mucosal polyps in the GI tract are characteristic; occasionally, a solitary polyp is present. Nasal polyp has not previously been described; its occurrence may be explained on the basis of common origin of GI and respiratory mucosal immune system. In our patient, endoscopic features initially suggested IBD, but further investigations led to the correct diagnosis.

At histology, MLP lesions classically show non-Hodgkin's lymphoma of small cleaved cell (Working formulation) or centrocytic (Kiel classification) type. Malignant cells in MLP possess a B-cell phenotype. Patients have a JH/bcl-1 locus translocation, characteristic of lymphomas of nodal mantle cell origin, and an increased expression of receptors responsible for homing of lymphocytes to the mucosa-associated lymphoid tissue.

The prognosis is poor. Chemotherapy is the treatment of choice but leads to only partial or short-lived remission. High-dose radiochemotherapy followed by autologous stem-cell transplantation may be more effective.

References
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**Fistulization of hepatic hydatid cyst into duodenum**

TUFAIL PATANKAR, SRINIVASA PRASAD, O S ROHONDIA, J D MOHITE, PERRUMILLICHIRA JAMES, RAVIKUMAR RADHAKRISHNAN

Departments of Radiology and *Surgery, King Edward VII Memorial Hospital, Mumbai 400 012*

Though rupture of hepatic hydatid cyst is not uncommon, fistulization into the gut following rupture is rare. We report a case in whom a hepatic hydatid cyst ruptured into the first part of the duodenum. *[Indian J Gastroenterol* 1998; 17: 152]

**Key words:** Liver cyst

The liver is the commonest organ affected in hydatidosis. Communicating rupture is the commonest complication. The diagnosis of hydatid cyst before surgery is essential for optimal management of these cases.

A 35-year-old man presented with dull ache in the right hypochondrium for two months. On clinical examination, there was nontender hepatomegaly.

Plain radiograph of the chest revealed crescentic gas shadow under the right dome of the diaphragm. Dynamic contrast-enhanced CT scan showed two large, well-defined, multiseptate, nonenhancing cystic lesions in the right lobe of the liver. Air was noted in one of the cysts, tracking to the region of the first part of the duodenum (Fig). Another multiseptate cyst was seen in the pelvis between the urinary bladder and the rectum.

Exploratory laparatomy confirmed the CT findings. Pelvic and hepatic cysts were enucleated and the duodenal perforation was closed. The patient recovered uneventfully.

Lewall and McCorkell classified rupture of hydatid cysts into three types—contained, communicating, and direct. In contained rupture, the endocyst ruptures and the contents are confined within the host-derived pericyst. In communicating type, cyst contents escape via biliary or bronchial radicles that are included in the pericyst. Direct rupture occurs when both the endocyst and the pericyst tear, spilling the cyst contents into the peritoneal or pleural cavities or occasionally into neighboring structures.

Hepatic hydatid cysts rupture in 35%-58% of cases. Fistulization to the gut accounts for 0.5% of cases. Although there are reports of hepatic cysts rupturing into the stomach, fistulization into the duodenum has not been reported earlier. In our case, CT scan established the site of fistulization, thus facilitating surgical repair.

**References**