however, available information suggests that patients with biliary carcinoid have an overall favorable prognosis after aggressive surgical management.[1,6] The mean disease-free follow up after resection in one study was 32 months (range, 3 months-20 years).[6] Hence surgery with curative intention should be considered wherever possible.

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Thyrotoxicosis co-existing with ulcerative colitis

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A 32-year-old lady was admitted with complaints of recurrent episodes of vomiting and loose stools associated with tenesmus and blood and mucus. She also had a neck swelling since 11 years of age, and had features of thyrotoxicosis for five to six months. She responded to balsalazide only after her thyroid status was controlled with carbimazole. [*Indian J Gastroenterol* 2005;24:263-264]

Autoimmune thyroid diseases are well known to complicate ulcerative colitis, but the association of thyrotoxicosis with ulcerative colitis is not common.[1,2] Control of active colitis is possible only after treatment of hyperthyroidism.

A 32-year-old lady presented with complaints of recurrent vomiting and bloody diarrhea with tenesmus. There was no significant past medical illness. Examination showed an anxious patient with tachycardia and exophthalmos. There was a multinodular goiter with no bruit. Abdominal examination was normal.

Investigations showed leukocytosis with elevated ESR. Colonoscopy showed active ulcerative colitis involving the left side of the colon; this was confirmed on histology. Thyroid profile showed undetectable serum TSH with elevated free T3 and T4 levels. Technetium-99m thyroid scintigraphy showed increased uptake in both the lobes suggestive of a large multinodular goiter. Anti-thyroid antibodies were negative. She was treated with balsalazide and topical steroids, with no improvement for the initial 7 days. She was then started on carbimazole and low-dose propranolol. A week later she showed improvement in her thyrotoxic and bowel symptoms and was in complete remission of ulcerative colitis when reviewed one month later. She is in remission on maintenance dose of balsalazide and carbimazole for the last 6 months.

Though autoimmune thyroid disorders are well known to complicate chronic ulcerative colitis, the coexistence of hyperthyroidism and ulcerative colitis is very rare.[1,2] The incidence of thyrotoxicosis in patients with ulcerative colitis is 0.8% to 3.7% and the prevalence of ulcerative colitis in patients with hyperthyroidism is 1.3%.[2,3] Some investigators have suggested only an incidental association of these two diseases,[1] while others reported a relationship between the two.[3] Psychological stress may exacerbate either disease.[2,3]

Hyperthyroidism intensifies the systemic manifestations of ulcerative colitis and renders its management difficult.[1,4] The rapid metabolism of the drugs for treating ulcerative colitis or their rapid transit through the gut may prevent them from attaining effective concentrations.[4] Hence before the colitis can be effectively controlled, treatment of the thyrotoxicosis is essential.

Silent hyperthyroidism in ulcerative colitis has also been reported.[2] Also some of the features of active colitis may obscure an early diagnosis of coexisting thyrotoxicosis. Hence laboratory tests for thyrotoxicosis are indicated in patients with refractory ulcerative colitis with or without thyroid enlargement.[4]

Either one of the diseases in active stage can induce the other.[2] Since our patient had a toxic multinodular goiter, the chance of other nodules turning autonomous in the future is high. So the treatment of choice is total thyroidectomy with thyroid hormone replacement.

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Splenic rupture as a complication of colonoscopy

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Splenic rupture is a rare but potentially lethal complication of colonoscopy. We report a 66-year-old man who developed abdominal pain 6 hours after diagnostic colonoscopy. His clinical status deteriorated rapidly. Splenic rupture was identified at laparotomy. He recovered after surgery. [*Indian J Gastroenterol* 2005;24:264-265]

Perforation and hemorrhage of the colon are the most common complications of colonoscopy.[1] Less commonly reported complications include pneumothorax, pneumomediastinum, volvulus, hernia incarceration, retroperitoneal abscess and emphysema, bacteremia, endocarditis, vasovagal reaction, and bronchospasm.[2] Splenic rupture is a rare but potentially lethal complication.[3]

A 66-year-old man presented to the emergency department with history of left upper quadrant abdominal pain, worse with deep inspiration, 4 hours prior to admission. The patient had undergone outpatient colonoscopy for evaluating cause of anemia, 10 hours prior to admission. The procedure could not be completed due to colon redundancy. It had shown multiple telangiectasias throughout the colon. Six hours after colonoscopy he developed left upper quadrant abdominal pain associated with vomiting.

Past medical history was significant for hypertension, ischemic heart disease and left-sided stroke. He was a cigarette smoker, and had received atenolol, diltiazem, ASA, nitrocantin, and warfarin. Upper GI endoscopy, performed for chronic anemia, showed gastritis and gastroesophageal reflux disease.

Examination revealed an acutely ill man with blood pressure 80/60 mmHg, pulse rate 100 beats/min, respiratory rate 26 per min, and normal temperature. The abdomen was diffusely tender with hypoactive bowel sounds. The rectum was empty. *Investigations*: WBC 6800/mm^3; hemoglobin 7.4 g/dL (value was 10.1 g/dL 1 month ago), and platelet count 79,000/mm^3. Coagulation studies were marginally deranged; blood chemistry was normal.

After resuscitation, laparotomy was done. There was no evidence of colonic perforation, but the spleen showed multiple lacerations. Splenectomy was performed. The 80-gram spleen had multiple lacerations on its medial aspect. Histology showed small and medium-sized vessels with hyaline arteriosclerosis, compatible with long-standing hypertension.

Six days after surgery he developed retrosternal pain with sweating. ECG revealed ST-segment elevation in the inferior and posterior leads associated with elevations of troponin I and CK-MB. The patient was transferred to the CCU and received appropriate care. He recovered uneventfully and was discharged 2 weeks after admission.

Wherry and Zehner[3] reported the first case of splenic rupture after colonoscopy. Splenic rupture is an extremely uncommon complication of colonoscopy; the overall incidence is 0.004%. Although several mechanisms have been suggested,[4] splenic rupture has been reported in cases in which no problems were encountered, the entire colon was visualized, and no biopsy or polypectomy was performed. The complication rate of colonoscopy was not related with either the level of experience or the number of prior or annual colonoscopies.[2]

Most patients with splenic rupture exhibit the usual features of intra-abdominal hemorrhage. However, asymptomatic rupture of the spleen after colonoscopy has been reported.[5] In the vast majority of cases, pain begins within 24 hours after the procedure, but rarely can occur several days after the procedure.[6] A standard chest radiograph should be done first to rule out perforation. Abdominal CT is sensitive and specific and may help decide which patient needs surgery or can be managed nonoperatively as in closed subcapsular hematoma.[6] A bedside ultrasonography can be useful for quickly identifying free fluid in the abdomen in the unstable patient, although gas in the bowels after colonoscopy may limit its usefulness.

After resuscitation, the hemodynamic status of the patient, along with the findings on abdominal CT, will determine the best course of management for the individual patient.

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