Figure 2: H&E stained sections (10x100 X) showing mononuclear infiltrates and erythrophagocytosis

Discussion

Gall bladder is more likely to perforate in cases of obstruction leading to raised intra luminal tension. Non obstructive cholecystitis is unlikely to result in perforation.\(^1\) Perforation complicating acute acalculus cholecystitis is often seen in association with acute infections like pneumonia, viral influenza, and in particular with typhoid where the causative organism *Salmonella typhi* multiply in bile in very high titers and are further concentrated in gall bladder.\(^2\) Perforation in such cases is likely to be caused by intense inflammation coupled with infection with more virulent organisms and existence of a immuno compromised state leading to thrombosis of the blood vessels. This in turn causes transmural necrosis and perforation. Younger patients in an immuno compromised state also have a higher incidence of perforation.\(^3\) Gall bladder perforations due to typhoid fever, though known, have been rarely reported over the past two decades\(^4\)\(^,\)\(^5\). Most of the reported cases of enteric gall bladder perforation date back to 1970s.

Peritonitis due to gall bladder perforation is associated with high mortality as quoted by Essenhigh (39.1%)\(^1\). This high mortality makes timely diagnosis and management of perforated gall bladder highly crucial. Diagnostic peritoneal lavage in gall ladder perforation yields bile with or without blood. However, the presence of bile indicates biliary tract injury and is not specific for gall bladder perforation.

USG is useful in 70% of cases in diagnosing gall bladder perforation and detecting the defect in gall bladder wall and hence it can be used as first line imaging modality for evaluating such a case\(^6\). The features of non-traumatic gall bladder perforation on USG are free fluid and fluid collections close to the gall bladder fossa, thickened gall bladder wall – 7 mm (range 3-20 mm) and localized collection in gall bladder wall. CT scan shows free fluid and localized perihedral and gall bladder fossa collections. The gall bladder can be accurately identified and the site of perforation is often demonstrated.

Perforation of the gall bladder must be treated surgically when the diagnosis is made or suspected. The preferred procedure is emergency cholecystectomy. Cholecystostomy is an acceptable alternative especially in poor risk patients.

References

ranges, except C-reactive protein which was markedly elevated (185 U; range 0-5). In addition, coagulation studies revealed a prolonged prothrombin time (PT) of 15.1 sec (range 9-13), international normalized ratio (INR) 1.27 (range 0.85-1.15) and activated partial thromboplastin time (APTT) 41.1 sec (range 26-38).

Abdominal ultrasonography revealed free intraperitoneal fluid collection whereas chest and abdominal radiographs were unremarkable. Due to acute abdominal findings and signs of sepsis, an urgent abdominal CT scan was obtained which revealed marked thickening mainly involving the right colon wall (Figure 1), along with ascites (Figure 2). Based on these findings the diagnosis of acute infectious colitis was considered and thus we decided not to proceed to an exploratory laparotomy. After blood and stool cultures were obtained, replacement therapy with fluids and electrolytes was initiated and the patient was placed on antibacterial therapy with ciprofloxacin and metronidazole. After 12 hours, his general condition began to improve steadily and he was discharged home 6 days later, after complete resolution of his symptoms. Stool cultures yielded Salmonella spp, whereas blood cultures were negative. Colonoscopy performed after the acute phase of the disease revealed findings consistent with infectious colitis (Figure 3).

Discussion

Salmonella gastroenteritis is the most common clinical syndrome caused by non-typhoidal salmonella. After an incubation period of 6-48h patients usually present with nausea, vomiting, abdominal cramps and diarrhea. Invasive disease with bacteremia has been reported in fewer than 5% of the cases, whereas unnecessary surgical interventions to treat suspected acute abdominal conditions have been performed in patients with salmonellosis, increasing morbidity and mortality rates. Clinical presentation is often indistinguishable from gastroenteritis caused by other pathogens. However, most patients have mild to moderate symptoms and the disease tends to run a short self-limited course. In our case, although the patient was non-immunocompromized he had a very severe clinical course. We speculate that this was likely due to the ingested dose of the bacteria as it has been identified as an important determinant in the incubation period, symptoms and severity of acute salmonellosis.

Colonic involvement in salmonellosis was first reported in 1969 when Boyd described necropsy findings of 6 patients with infections caused by S. typhimurium. The clinical course of salmonella colitis varies from 1 week to 2-3 months with an average duration of 3 weeks. Early diagnosis and appropriate treatment is mandatory to prevent potentially serious complications including toxic megacolon requiring aggressive therapy, sepsis and bleeding.

Despite the high incidence of salmonellosis in the general population there are only a few reports in the literature describing CT or other radiological findings of salmonella colitis. This likely happens because most infected patients do not seek medical assistance, do not undergo diagnostic evaluation and are treated empirically with supportive therapy and broad-spectrum antibiotics.

Colonic wall thickness is the main feature on CT indicative of acute colitis. When the lumen of the bowel is distended, the normal thickness is 1-2mm, but when it is collapsed the normal thickness is 3-4mm. Philpotts et al reported a colon wall thickness of 8.0±2.47mm in 11 patients with infectious colitis, whereas in 36% of the patients the findings were exclusively involving the right colon. In another study Horiki et al reported that the thickness of the ascending colon wall

Figure 1: Contrast-enhanced CT scan of the abdomen shows diffuse circumferential marked wall thickening (d: 2.58cm) of the ascending colon (arrow).

Figure 2: Contrast-enhanced CT scan of the abdomen shows perihepatic fluid collection (arrow).

Figure 3: Colonoscopy shows focal mucosal erythema with mucopurulent exudates.
was greater of 10mm in 45% of the patients with salmonella colitis. Finally, Balthazar et al reported a slight (3-5mm) circumferential wall thickening of cecum and descending colon in 3 patients with Salmonella infection. Compared to the above reported data, the thickness of the ascending colon wall in our patient was excessive.

The presence of ascites is another finding on CT suggestive of acute colitis and has been reported in 23-46% of the cases\textsuperscript{5,10}. CT is mainly used to evaluate patients with more severe clinical presentation and to exclude other abdominal inflammatory processes\textsuperscript{5,10}.

In most cases the definitive diagnosis of the type of colitis is based on clinical and laboratory data and colonoscopic and biopsy findings\textsuperscript{5,13}. CT findings of colitis are non-specific, but some features can be helpful in suggesting a specific diagnosis\textsuperscript{5,10}, as in our patient. These features may include the amount of wall thickness, extent of the disease, pericolic reaction, ascites, fistulae, and presence of complications\textsuperscript{13}.

We present a rare case of salmonella pancolitis in a non-immunocompromized young male patient who presented with sepsis and typical manifestations of acute abdomen. Although definitive diagnosis is made with the isolation of Salmonella spp from stool cultures, however, non-specific findings on CT scan should alert the emergency physician to consider the diagnosis of acute colitis, in patients presenting with a severe form of the disease, avoiding therefore an unnecessary laparotomy and subsequent morbidity.

References


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Extra-intestinal Hodgkin’s lymphoma in a Crohn’s disease patient on long-term azathioprine and infliximab therapy

Lymphoproliferative disorders, especially non-Hodgkin’s lymphoma, occur at an increased rate in immunodeficient or immunosuppressed patients.\textsuperscript{1} An increased risk of lymphoma has also been reported in patients with Crohn’s disease (CD).\textsuperscript{2} However, most cases of lymphoma associated with infliximab; a chimeric IgG1 monoclonal antibody that binds specifically and directly to human tumor necrosis factor-α (TNF-α); were of the hepatosplenic T cell or the B-cell non-Hodgkin’s types.\textsuperscript{3,6}

We present a case of nodular sclerosing Hodgkin’s lymphoma (HL), in a patient receiving long-term azathioprine and infliximab therapy for CD. The possible relationship between this lymphoproliferative neoplasm and concurrent therapy with TNF antagonists is explored.

Case Summary

In May 2004, a 35 year-old Saudi patient was referred to King Faisal Specialist Hospital and Research Center with active colonic CD refractory to oral steroid and sulfasalazine therapy. After clinical, biochemical, radiological and pathological assessments, the diagnosis was confirmed and the patient was started on oral daily doses of prednisone 50 mg, azathioprine 100 mg, mesalamine 3 gram, and his sulfasalazine was stopped. Prednisone treatment was tapered over 5 weeks to 10 mg daily. Three months later, he showed no significant improvement, and started on infliximab (Remicade; Schering-Plough, Holland) induction therapy of three doses of 5 mg/Kg at 0, 2 and 6 weeks after excluding hepatitis B virus, cytomegalovirus (CMV), and mycobacterial tuberculosis infections.

Patient received 18 IV infliximab infusions (each was 300 mg, with IV diphenhydramine 25 mg and hydrocortisone 100 mg pre-medication) from September 2004 till June 2007 with remarkable clinical, biochemical, and endoscopic responses. He had a smooth course, only interrupted by an episode of CMV colitis that was treated effectively with ganciclovir and follow up CMV antigenemia was negative.

In June 2007, he presented with right-sided neck swellings associated with 3 months history of intermittent fever, night sweats, and weight loss of 6 kilograms, without any diarrhoea or abdominal pains. Clinically, he was afebrile, hemodynamically stable with non-tender enlarged rubbery right cervical lymph nodes, the largest was 2x2 cm. Chest, cardiac, and abdominal examinations were unremarkable, and the clinical impression was inactive CD with new lymphadenopathy.

Laboratory investigations were as follows. WBC: 7,800/mL, hemoglobin: 110 g/L, platelets: 256,000/mL, albumin: 38 g/L, alkaline phosphatase: 239, ESR: 98 mm/hour, CRP: 174 mg/L. Excision lymph node biopsy showed classic nodular sclerosis HL with many Reed-Sternberg cell variants (lacunar cells) in a mixed inflammatory background, strongly positive for CD30 in membranous and Golgi pattern (Figure 1). Bone marrow examination and biopsy confirmed...