have appeared in literature. Due to the severity of symptoms and signs, most cases present immediately after the injury; delayed presentation of colonic perforation following compressed air injury is uncommon.\(^2\)

A 17-year-old man presented with peritoneal discomfort, generalized pain in the abdomen and bleeding per rectum, three days after his co-workers in a huddling industry placed the nozzle of a compressed air pipe (7 Kg/cm\(^2\)) near his anal region. He had features of peritonitis. X-ray abdomen showed air under the domes of diaphragm. At laparotomy, there were multiple seromuscular tears with perforating necrotic mucosa involving the entire large gut, along with colic perforation and fecal peritonitis.

Large-gut irrigation with warm saline was done with a 30-F Malecot catheter placed in the colic perforation, with the effluent coming out per anum through a proctoscope. The colonic perforation was closed, colonic seromuscular tears repaired, and ileostomy performed, distal ileal stump closed and a thorough peritoneal lavage given. Ileostomy started functioning on day 2, but on day 6 the patient died due to peritonitis and abdominal pain with distension. Ultrasoundography showed interloop fluid collection suggestive of colonic leak. The patient was treated conservatively and discharged on day 10 in good health. After 2 months, barium enema showed normal colon and ileostomy closure was done.

Compressed air injury of the colon is primarily an industrial injury and occurs as a result of tragic practical jokes. In experimental studies on cadavers, it was shown that the normal intestine ruptures at 0.49 to 0.88 Kg/cm\(^2\) pressure.\(^3\) Compressed air first produces tears in the muscular and serous coats; the mucosa is the last to be damaged because of its elasticity.\(^2\) The patient usually presents with severe peritonitis and shock, but initial symptoms may be mild if the injury does not cause complete rupture of the colon. One case report mentioned continued sepsis in the postoperative period suggestive of a delayed second perforation and need for re-exploration.\(^2\) In the present case, large-gut irrigation cleared the fecal matter from the colon and end ileostomy helped in fecal diversion.

We suggest that in patients presenting late after compressed air injury of the colon, intraoperative large gut irrigation should be performed along with resection or repair of the colon and fecal diversion.

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References

Colon metastasis from squamous cell carcinoma of esophagus

Carcinoma esophagus can metastasize to unusual sites. We report a patient with squamous cell carcinoma of the esophagus with metastasis to the stomach and colonic wall.

A 60-year-old man presented with progressive dysphagia since 3 months. Physical and routine biochemical examination was within normal limits. Barium swallow and endoscopy revealed irregular narrowing in mid esophagus; biopsy revealed moderately differentiated squamous cell carcinoma. All routine metastatic work-up was normal.

Exploration of the abdomen revealed enlarged colic lymph nodes, a small nodule of 3 cm \(\times\) 3 cm size in the lesser curvature of the stomach and an annular, hard lesion on the left side of the transverse colon. In view of this, radical esophagectomy was deferred. Local excision of the stomach nodule, biopsy from colic lymph node and excision of the colonic growth with colo-colic anastomosis were done. Histology revealed moderately differentiated squamous cell carcinoma deposits at all three sites. The colonic growth involved the muscular layer; the serosa and mucosa were spared. The patient was treated with adjuvant radiotherapy and chemotherapy.

Carcinoma in the middle-third of the esophagus metastasizes to colic lymph nodes in approximately one third of cases.\(^1\) Colic lymph nodes ultimately drain into the cisterna chyli, which receives lymph from two major pathways: (i) the intestinal trunk draining the celiac and superior and inferior mesenteric lymph nodes, and (ii) right and left lumbar trunks, which drain the lateral aortic, iliac and sacral nodes.

In the present case there presumably was retrograde lymphatic permeation of tumor cells from the obstructed cisterna chyli. Non-involvement of serosa and mucosa supports this postulate. We cannot explain the absence of other mesenteric lymphadenopathy.

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Reference

Plasmodium vivax and hepatitis E co-infection - a rare cause of malarial jaundice

Ghoshal and colleagues\(^1\) report a rare case of *Plasmodium falciparum* and hepatitis E co-infection causing hepatic failure. We report a similar patient who had co-infection of *P. vivax* and hepatitis E.

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A 38-year-old man presented with fever with chills for 10 days, high-colored urine for 8 days, melena, jaundice, decreased urine output and dyspnea for 3 days, and drowsiness for 1 day. There was no past history of jaundice, blood transfusion, high-risk behavior or alcohol abuse. On examination, the patient was drowsy and had marked pallor and icterus. Abdominal examination revealed 5 cm tender hepatomegaly without splenomegaly. A provisional diagnosis of complicated malaria with a possibility of sepsis syndrome and bleeding diathesis was kept and the patient was started on intravenous quinine, ceftriaxone and supportive treatment.

Investigations: hemoglobin 5.5 g/dL with total leukocyte count 9,000/mm<sup>3</sup> and platelets 76,000/mm<sup>3</sup>. Peripheral smear showed normocytic, hypochromic red cells with trophozoites of <i>P. vivax</i>. Blood urea was 113 mg/dL and creatinine 2.2 mg/dL. Serum bilirubin was 20.1 mg/dL (conjugated 5), serum alkaline phosphatase 338 IU/L, AST 244 IU/L and ALT 167 IU/L. Prothrombin time was 23 seconds (control 12). CSF examination was unremarkable.

The patient was resuscitated with fresh frozen plasma and packed red cells. Urine output was only 400 ml over 24 hours. He was hemodialized on the 2nd day of admission. Repeat blood smear revealed trophozoites of <i>P. vivax</i> with counts >5000/mm<sup>3</sup>. Blood cultures were sterile. Plasma hemoglobin was raised. Trophozoite immunochromatography test (ICT) was positive for <i>P. vivax</i> and negative for <i>P. falciparum</i>. On the 4th day, the peripheral blood smear was negative for malaria parasite. The serology for leptospiral infection was negative.

Over the next 10 days, the fever improved, serum creatinine fell to 1.6 mg/dL, cell counts and coagulation parameters normalized, but liver functions were still deranged (bilirubin 2.5 mg/dL, AST/ALT 79/162 U/LL). Ultrasonography revealed mild hepatomegaly. At this stage, blood was sent for viral markers for hepatitis; it revealed high titers of IgM anti-hepatitis E virus (HEV) antibodies (ELAgen HEV IgM kit; Biochem ImmunoSystems Italia S.P.A., Italy; specificity 98.6%). A final diagnosis of <i>P. vivax</i> malaria, HEV co-infection with disseminated intravascular coagulation, hepatic and renal dysfunction, and metabolite encephalopathy was made.

Jaundice in malaria may occur due to a wide variety of causes. Malarial hepatitis is usually described in patients with <i>P. falciparum</i> infection; the patients present with nausea, vomiting, fever and jaundice, with abnormal liver biochemistry. However, histology in such cases generally does not show hepatic inflammatory changes and thus the new terminology of 'malarial hepatopathy' is suggested. These patients have increased incidence of complications such as renal failure, adult respiratory distress syndrome, septicemia and high mortality.

Ours is the first reported case of <i>P. vivax</i> and HEV co-infection. The report by Ghoshal et al<sup>1</sup> was the only earlier report where HEV co-infection was reported in a patient with <i>P. falciparum</i> malaria. In our opinion, as both malaria and viral hepatitis are frequent illnesses in our country, patients with malaria and jaundice who have a clinical picture of hepatitis should be investigated for hepatitis viral markers before labeling them as malarial hepatitis.

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INDEX TO ADVERTISERS
Dr Reddy's Laboratories Front inside cover v
Glenmark Pharmaceuticals Ltd Back inside cover x

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Shree Ganesh Pharmaceuticals
USV Ltd iv