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Case Report

Microbiology Section

An Unusual Case of Perforation Peritonitis: A Diagnostic Dilemma-Bacterial or Parasitic?

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ABSTRACT

Large intestinal perforation due to mixed parasitic and bacterial aetiology is a rare entity. We report the case of a 63 years old man, who underwent a therapeutic paracentesis for ascites, which revealed eggs of hookworm, along with *E.coli.* The patient was initially treated conservatively, until his vitals stabilised, following which, he was operated for the perforation. He improved subsequently and was discharged. To the best of our knowledge, this is the third case of an intestinal perforation attributed to a parasitic (hookworm) cause. Hence, this report highlights the datum, to consider parasites in the evaluation of perforation peritonitis, apart from trauma and common infections like *Salmonella typhii* and *Mycobacterium tuberculosis* relevant from the Indian perspective.

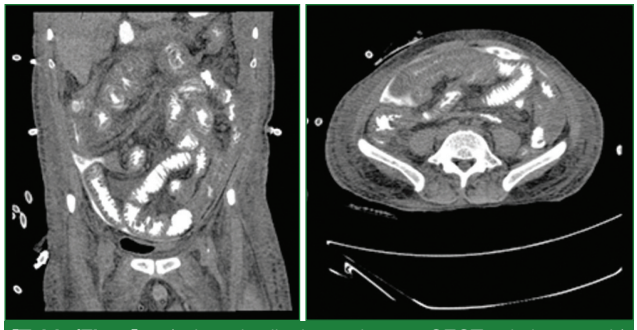
Keywords: *Escherichia coli*, Hookworm, Peritoneum

CASE REPORT

We report the case of a 63-year-old man, farmer by occupation, who presented to the Emergency Department with high grade, intermittent type of fever (103.2°F) and severe pain abdomen, localised to right upper quadrant since the last 2 days. The patient also complained of shortness of breath, although he did not have chest pain or any cough. There was general weakness, malaise and myalgia, in addition to loss of appetite. However, there was no significant loss of weight. The patient reported to have fever 15 days prior to this episode, which was not recorded and for which he was treated with antibiotics elsewhere the details of which were not available. Over a period of time he noticed yellowish discolouration of his sclera and subsequently developed haematuria. There was no comorbid history of diabetes mellitus/hypertension/tuberculosis/steroid therapy/immunosuppression or any past surgical interventions. No history of alcohol abuse, however positive smoking history of 15 years with one pack per day. There was no history of similar complaints in any other family members.

On physical examination, the patient was conscious, co-operative and oriented to time, place and person. On admission, the patient was found to be tachypnoeic with rhonchi on auscultation and an oxygen saturation of 84%, in view of which endotracheal intubation was done and patient was put on intermittent mechanical ventilation. On per abdomen examination, there was gross distension with tenderness in the right hypochondrium and the epigastric region, along with hepatomegaly and evidence of free fluid in the abdomen. Urine output was good, although bowel movements were altered to an extent that patient did not pass stool for the duration of four days since hospitalisation.

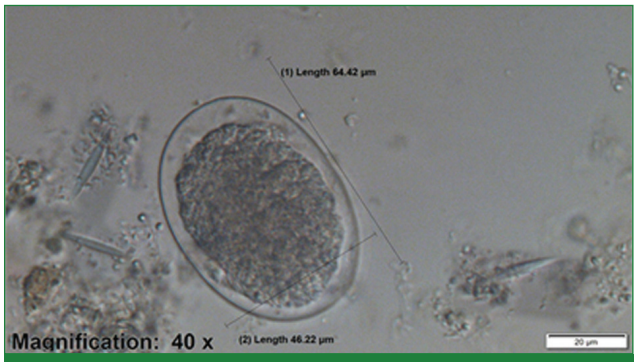
Radiological findings (non contrast CT-scan) on admission revealed gross hepatomegaly with a large hypoechoic lesion in the right lobe, moderate to severe ascites, bilateral pleural effusion along with pneumoperitoneum. Subsequent contrast enhanced CT-scan, a week later, revealed a focal defect in the anterior wall of caecum with extravasation of oral contrast in the right lumbar region, tracking down into the pelvis [Table/Fig-1].



[Table/Fig-1]: a) Longitudinal section - CECT abdomen; b) Transverse section - CECT abdomen.

Haematological findings revealed leucocytosis. Liver and renal biochemistry profiles were deranged. Thus, a working diagnosis of acute viral hepatitis with a differential of leptospirosis was made in view of ongoing hepatorenal syndrome.

Empirical treatment with piperacillin-tazobactum (4.5 gm, intravenous, eight hourly) and metronidazole (loading dose: 1 gm, intravenous; maintenance dose: 1 gm, intravenous, twelve hourly) was started and given for 4 days intravenously. Blood cultures and endotracheal aspirate cultures were sterile after 6 days and 72 hours of incubation, respectively. A surgical opinion was sought, which inferred a provisional diagnosis of



[Table/Fig-2]: Hookworm egg Charcot-Leyden crystals (x40).

a ruptured amoebic liver abscess with amoebic typhlitis. But due to the weak general condition, surgical intervention was not warranted and conservative management was planned.

In view of gross ascites, two peritoneal drains and a pigtail catheter were inserted, with an average daily drain output of 2000 mL. On microscopy of the drain fluid, there were many pus cells while on culture, there was heavy growth of *E.coli* (MALDI-TOF, Biomerieux, France). The antibiotic susceptibility testing was performed using Vitek 2 system (Biomerieux, France). The isolate was resistant to the aminoglycosides (amikacin, gentamicin), penicillin and cephalosporins (ampicillin, ceftriaxone, cefotaxime, cefepime), fluoroquinolone (ciprofloxacin), β-lactam-β-lactamase inhibitors (amoxicillin-clavulanic acid, cefoperazone-sulbactum, piperacillin-tazobactum), cotrimoxazole and carbapenems (imipenem) sensitive only to colistin and tigecycline.

The drain fluid and aspirated pus sent for parasitological workup, revealed numerous eggs of hookworm in addition to Charcot Leyden crystals and polymorphonuclear leucocytes [Table/Fig-2]. A repeat sample was again asked to confirm the findings which showed similar picture. The presence of inflammatory cells, Charcot-Leyden crystals and the repeat sample with similar findings ruled out sample contamination. Agar culture of the stool sample was done to identify the rhabditiform larvae which morphologically resembled that of *Ancylostoma duodenale*. Serology for *Entamoeba histolytica* was positive (ELISA kit, SCIMEDX Corporation), although stool examination and liver abscess aspirate did not reveal any morphological forms of *E.histolytica*.

The patient was treated in the intensive care with positive pressure ventilation, with nasogastric tube feeding and had been put on Meropenem (1 gm intravenous, 8 hourly) and Colistin (loading dose: 9 million IU, intravenous; maintenance dose 4.5 million IU, intravenous, twelve hourly) in view of the multi drug resistant *E.coli* isolate. Specific treatment for the intestinal perforation (exploratory laparotomy with resection and anastomosis of the devitalised bowel loops) couldn't be initiated as the patient's vitals were not stable. However, the patient improved over a period of time with anti parasitic therapy (Metronidazole 1 gm intravenous, 12 hourly and diloxanide furoate 500 mg via nasogastric tube, 8 hourly) for 5 days and albendazole 400 mg, via nasogastric route, single dose following which he underwent resection and anastomosis procedure. He tolerated the surgical procedure well and was eventually discharged.The patient's consent was not taken as all the samples were procuredas part of routine diagnostic workup. Further, the patient did not return for subsequent follow-up.

DISCUSSION

Intestinal perforation, attributed to an infectious etiology is infrequent, but can be life threatening, if not detected promptly. Among the bacterial aetiologies, enteric fever and tuberculosis constitute the commonly encountered infections in developing countries [1,2]. In rare instances, certain parasites are implicated in invading the intestinal mucosa, leading to wide spread inflammation, which subsequently evolves into a sterile abscess, or a granuloma enclosing live worms [3]. This is referred to as a helminthoma, which was initially described for the nematode, *Oesophagostomum* sp [3]. The disease is mostly observed in the tropics or subtropics, with most cases being reported primarily from Africa [4]. The larval stages of some unusual nematodes like, *Anisakis* sp. and *Pseudoterranova decipiens*, causing infection in humans,are mostly encountered in Japan, rare sites of isolation being the peritoneal cavity. It is found in association with eating of raw fish, sushi and sashimi [4-6]. The larval forms (spargana) of a few cestodes, *Spirometra mansonoides* and *Diphyllobothrium* sp, penetrate the intestinal wall, migrate through the peritoneum to reach the subcutaneous tissue, as a part of their life cycle [4]. Further, parasitic involvement of the abdominal viscera may affect the peritoneum, secondary to rupture of the underlying lesions. Peritoneal involvement in hydatid disease is an infrequent finding, being more often secondary to hepatic involvement [7]. In 1996, Karavias et al., observed among 14% of their cases with hydatid disease, to have peritoneal involvement, with or without coexisting liver disease [7]. Moreover, the trematodes implicated in peritoneal parasitosis are *Fasciola hepatica* and *Paragonimus westermani*. The metacercaria (larvae) of *Fasciola hepatica* excysts in the duodenum, migrates through the intestinal wall into the peritoneal cavity, penetrates the liver capsule to finally reach the bile ducts. The larvae of *Paragonimus westermani* excyst in the duodenum, migrates through the intestinal wall into the abdominal cavity, penetrates the diaphragm to reach the lungs. On their transit to the liver and lungs, as a part of their life cycle, the metacercaria may get entrapped in the abdominal cavity and eventually mature into adult flukes, leading to inflammatory granuloma or an abscess [3]. It is well established in literature, that hookworm is readily isolated from skin and faeces of infected persons. Although, its isolation from peritoneal effusions has not been widely reported.

Hookworms are intestinal nematodes, which cause gastrointestinal infestation, with a higher predilection for the young population and women of child bearing age. People who walk bare footed are, especially farmers are mostly affected. The bare skin of the foot is the site for hookworm penetration. In its lifecycle, the larvae undergoes a final moult

in the small intestine to form the adult worm, where they mate and thousands of eggs are released from the female worm. The various cytokines and proteins released help in intestinal attachment and multiplication.

Hookworm infections have been known to humans for quite some time. Harper in 1932 pointed out two conditions, resulting likely from hookworm infection: 1) polyarthritis associated with fever (acute rheumatism) and 2) a tendency to bleed from small vessels [8]. Koh KH et al., reported a case of a 24 years old man with irritable bowel syndrome, who on further evaluation for aggravating symptoms, was detected to have *Ancylostoma duodenale* which had invaded the jejunum and was eventually detected on capsule endoscopy [9]. The patient improved and the symptoms subsided after successful treatment with albendazole similar to our case as well [9]. Eid HO et al., reported a case of a 30 years old man presenting with an intestinal perforation, possibly caused by hookworm, which on histopathology was the size of a pinhole with accompanying non specific inflammation [1]. On the contrary, in our patient, the perforation was detected in the caecum.

The sensitivity of stool microscopy in detection of *E.histolytica* is as low as 60% with many confounding factors that render false positive results, for example, identification of macrophages as trophozoites [10]. Hence, serological evaluation, in the light of appropriate clinico-radiological context becomes imperative to render a correct diagnosis. Furthermore, in industrialised countries, serological tests may aid in identifying *E.histolytica* infection. However, its role is controversial in developing countries; *E.histolytica* is endemic and serological assays cannot differentiate between current and past infection. Further, our patient had radiological features suggestive of amoebic liver abscess in the presence of a positive serology. An amoebic typhlitis may have contributed to the large intestinal perforation in addition to hookworm infestation [10].

It may be a case of coinfection with both hookworm and *E.histolytica*, but we were unable to detect *E.histolytica* in the abscess aspirate and peritoneal fluid. A possible mechanism of isolation of hookworm in the peritoneal fluid is probably the arrested development (hypobiotic state) and longer survival in the large intestine, which may have led to intestinal obstruction and subsequently the perforation, leading to spill of the hookworm into the peritoneal cavity along with migration of the intestinal microbiota [4]. Further, *E.coli* which was isolated from the peritoneal fluid was multidrug resistant.

CONCLUSION

Here we present a unique case of coinfection of parasites, *E.histolytica* and hookworm with a bacteria, *E.coli.* The patient's clinical condition improved after the administration of anti parasitic medication and further, showed improvement after surgical procedure. Physicians should be aware of the non traumatic, infectious aetiologies of intestinal perforation while working up of a suspected case of perforation peritonitis. In fact, there is paucity of data about spillage of hookworm into the peritoneal cavity after perforation and this report is unusual due to the mixed parasitic and bacterial etiology of a complicated case of intestinal perforation in an elderly farmer.

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