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Case Report

Microbiology Section

Campylobacter Infection Associated Intestinal Perforation: A Case Report

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ABSTRACT

Campylobacter is an agent of diarrhoeal illness and can potentially lead to intestinal obstruction. This condition manifests as acute abdomen and requires exploration. A 22-year-old male who had been receiving empirical treatment for pulmonary tuberculosis for four days was admitted with severe abdominal pain. Laparotomy revealed intestinal obstruction and perforation. Histopathological examination of the intestinal wall and omentum showed granulomatous lesions suggestive of tuberculosis. On the third day after laparotomy, the patient developed septicaemia. Blood and peritoneal fluid yielded *campylobacter* on culture. The present case report emphasises the importance of considering *campylobacter* infection in cases of intestinal obstructions and granuloma formation.

Keywords: Granuloma, Intestinal obstruction, Tuberculosis

CASE REPORT

A 22-year-old male was brought to the hospital with mild abdominal pain and dyspnoea. He was admitted to the Department of Pulmonary Medicine. His medical history revealed recurrent evening fevers and weight loss over the past seven months. He also experienced nocturnal cough, dyspnoea on exertion, loss of appetite, and vomiting for the past two weeks. Physical examination showed dullness on the right-side of the thorax upon percussion and crepitations upon auscultation. A chest Computerised Tomography (CT) scan revealed moderate pleural effusion on the right-side, along with multiple enlarged necrotic mediastinal and right supraclavicular lymph nodes. Based on these findings, the possibility of pulmonary tuberculosis or lymphoma was considered [Table/Fig-1].

Laboratory tests for infections including Human Immunodeficiency Virus (HIV), Hepatitis B, and Hepatitis C were negative. Liver and renal function tests were normal. The patient's haemogram showed haemoglobin level was 12.4 grams/100 mL, Erythrocytes Sedimentation Rate (ESR) was 14 mm/hour, total white blood cell count was 4710/cubic mm, with neutrophils at 74%, lymphocytes at 20%, eosinophils at 4%, and basophils at 2%. Thoracoscopy did not detect any pleural thickening. Pleural biopsy on histopathological examination could not detect tuberculosis and Acid-fast Bacilli (AFB) were absent in the sputum and pleural fluid. The Cartridge-based Nucleic Acid Amplification Test (CBNAAT) using the GeneXpert system from Cepheid failed to detect genes of *Mycobacterium tuberculosis* in the pleural fluid. Based on the radiological findings, the patient was started on Anti-tuberculous Treatment (ATT) with an intensive phase therapy of a fixed-dose combination containing four drugs: isoniazid (H) 75 mg, rifampicin (R) 150 mg, pyrazinamide (Z) 400 mg, and ethambutol (E) 275 mg (HRZE). The patient was discharged with instructions to take four tablets per day based on their weight category.

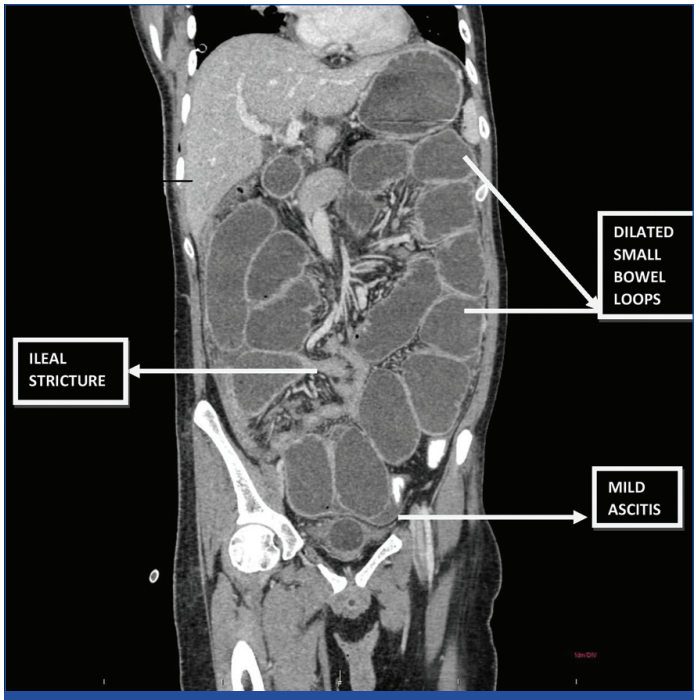
However, after four days, the patient experienced severe abdominal pain and was admitted to the General Surgery Department. An abdominal CT scan revealed features of subacute small bowel obstruction with a transition point at the mid-distal ileum, likely indicating a stricture. The scan also showed free fluid in the peritoneal cavity with peritoneal thickening and enhancement, necrotic nodes in the mediastinum, extensive mesenteric and omental fat stranding, a mild hydropneumothorax on the right-side, mild pericardial effusion, and hepatosplenomegaly [Table/Fig-2]. During laparotomy, an ileostomy was performed for acute intestinal obstruction and perforation. Following the surgery, the patient developed fever and signs of septicaemia. *Campylobacter* was isolated from the blood and peritoneal wash collected during the laparotomy [Table/Fig-3]. Subsequent biopsy of the intestinal wall revealed granuloma suggestive of tuberculosis. On the fourth day following the laparotomy, despite aggressive treatment, the patient unfortunately died from cardiopulmonary arrest. Molecular analysis identified the isolated strain of *Campylobacter* as *C. jejuni* [Table/Fig-4a,b].

DISCUSSION

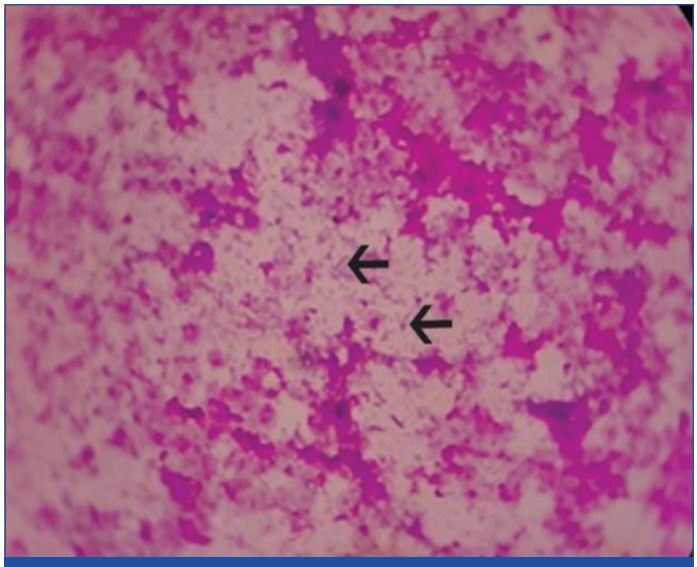
Campylobacters are spiral microaerophilic Gram Negative Bacilli (GNB) and are important agents of enteritis [1,2]. Acute diarrhoeal

Admission day	Symptoms	Physical examination findings	Investigations and findings	Clinical diagnosis	Treatment and outcome
1st admission 4th day	Dyspnoea, mild abdominal pain, history of weight loss	Dullness and crepitations on right-side of chest	CT scan of chest: mediastinal lymph nodes and fluid collection in right pleural cavity Pleural fluid AFB examination and molecular test for MTB: Negative	Pulmonary Tuberculosis (PTB)	Intensive phase therapy for PTB with HRZE Patient was discharged
IInd admission-first day	Severe abdominal pain	Signs of acute abdomen	CT abdomen: Intestinal obstruction, fluid in peritoneal cavity Histopathological examination of intestinal wall and omentum: granuloma Fluid from peritoneal washing culture: *Campylobacter* was isolated	Tuberculosis of intestine *Campylobacter* infection leading to perforation	Laparotomy and correction of obstruction
Third day	Fever	Signs of septicaemia	Blood culture: *Campylobacter* was isolated	*Campylobacter* septicaemia	Treatment with meropenem and Erythromycin
Fourth day		Signs of sepsis			Patient expired

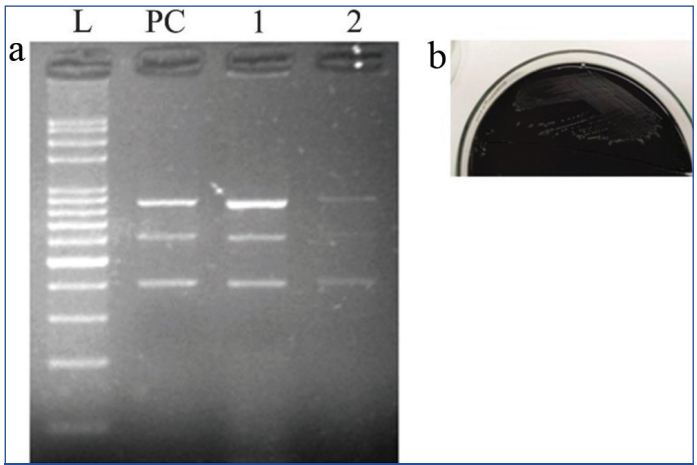
[Table/Fig-1]: Clinical events.



[Table/Fig-2]: Computerised Tomography (CT) scan of abdomen.



[Table/Fig-3]: *Campylobacter* in blood culture (Arrow heads; Gram Stain, 100x magnification).



[Table/Fig-4]: a) Multiplex PCR profile of *Campylobacter jejuni* using 16S rRNA (816 bp), map A (586 bp) and cad F genes (416 bp). L: Molecular ladder; PC: Positive control (*C. jejuni*); Lane 1 and 2: Samples from blood and peritoneal fluid; b) Growth of *campylobacter* on campy medium.

illness caused by *Campylobacter* species is usually seen in infants and is a significant cause of food poisoning [3]. The disease typically presents as loose stool with mucus and sometimes blood. Intestinal symptoms are usually self-limiting or can be relieved with antibiotics. Inflammatory changes can be observed through endoscopic examination and histopathology of the intestine. Since most infections are self-limiting, patients may not seek medical assistance. In the case presented, there was a history of loss of appetite and vomiting, but not diarrhoea. Infections can lead to intestinal obstruction and perforation, and timely identification of the cause can save the patient [4]. In the present case, the patient required emergency laparotomy due to intestinal obstruction. Life-threatening complications such as septicaemia have also been reported [5]. Invasive infections can progress to peritonitis from enterocolitis, preceded by intestinal obstructions [4]. In the presented case, intestinal perforation was only discovered during exploratory laparotomy and managed accordingly. Patients with complications usually have a history of previous gastroenteritis. In the presented case, the patient was initially investigated for respiratory illness and not for gastrointestinal disease during the first admission. The radiological findings suggested pulmonary tuberculosis, and ATT was initiated. After four days of ATT, the patient was admitted on an emergency basis due to abdominal symptoms. Histopathological examination revealed granuloma in the intestinal wall and omentum. Mesenteric lymph nodes were also involved. Granulomatous enteritis and reactive mesenteric lymph nodes can be observed in *Campylobacter* infections [6]. Although chronic granulomatous infection with weight loss is not commonly reported in humans, such clinical presentations have been documented in animals in the literature [7]. There are no reports of granulomatous infection with weight loss caused by *Campylobacter* infections in humans, but in veterinary practice, there are reports of granulomatous enteritis with weight loss and septicaemia [7]. In the present patient, evidence of tuberculosis could not be confirmed through pleural biopsy and pleural fluid analysis. However, granulomas were found in the intestinal wall and omentum.

Other reported complications associated with *Campylobacter* infections include irritable bowel syndrome, Guillain-Barré syndrome, and reactive arthritis [8]. However, in the presented case, there were no symptoms suggestive of these conditions, except for a history of loss of appetite and vomiting.

Having epidemiological knowledge is crucial for timely treatment. In the population of the presented case, *Campylobacter* infections have been reported, albeit infrequently [9]. These infections are considered zoonotic and are often associated with food poisoning. The bacteria can spread through contaminated water and food, particularly poultry [10]. Due to their requirement for microaerophilic conditions and selective media for isolation, the reporting of these bacteria from clinical samples is limited, potentially representing only a small portion of the actual cases.

Campylobacter infections typically respond to erythromycin in uncomplicated enteritis. However, drug resistance has been reported [11]. Therefore, the possibility of drug resistance should also be considered when treating such infections.

CONCLUSION(S)

The present case is presented to raise awareness that in cases of intestinal obstruction and perforations, infectious causes, particularly *Campylobacter* infection, should be considered and promptly treated to save the patient.

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