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BEYOND THE TERMINAL ILIUM: TYPHOID PERFORATION WITH COLONIC EXTENSION IN RURAL CAMEROON - A CASE REPORT

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ABSTRACT

Background: Typhoid intestinal perforation is a life-threatening complication of Salmonella typhi infection, most commonly affecting the terminal ileum at sites of Peyer's patch necrosis. Perforation involving the ascending colon is exceedingly rare and incompletely understood. We report a presumed case of typhoid perforation with colonic extension in a resource-limited rural African setting, and discuss the proposed pathophysiological mechanisms accounting for this atypical anatomical distribution.

Case Presentation: A 48-year-old female presented with a three-week history of high-grade fever, abdominal pain, and distension, with sudden clinical deterioration. Intraoperative findings revealed an extensive longitudinal intestinal perforation extending from the ileocecal junction to the ascending colon. In the absence of microbiological confirmation unavailable at our facility the diagnosis was based on clinical, epidemiological, and operative findings consistent with typhoid intestinal perforation. Surgical repair and broad-spectrum antibiotic therapy resulted in clinical recovery.

Conclusion: This case documents a rare pattern of intestinal perforation involving the ascending colon, presumed to be of typhoid aetiology, arising in a resource-limited setting. It contributes to a small but growing body of literature on colonic typhoid perforation and highlights the diagnostic and surgical challenges faced in rural sub-Saharan Africa. Potential pathophysiological mechanisms including contiguous ileocecal spread, mesenteric vasculitis, and bacteraemic seeding of colonic lymphoid follicles are discussed.

Keywords: Typhoid perforation; Salmonella typhi; Ileocecal Junction; Ascending Colon; Rural Cameroon; Resource-Limited Setting; Peritonitis

1 INTRODUCTION

Typhoid fever, caused by the Gram-negative bacterium Salmonella enterica serovar Typhi, remains a major public health burden in sub-Saharan Africa, South Asia, and other low-income regions. The World Health Organization estimates approximately 11–21 million cases occur annually worldwide, with Africa disproportionately affected due to inadequate water and sanitation infrastructure [1].

Intestinal perforation is the most feared and life-threatening complication of typhoid fever, occurring in approximately 1–3% of hospitalized cases in Africa, compared to less than 1% in resource-rich settings [2]. The perforation typically occurs in the terminal ileum, at Peyer's patches, where lymphoid hyperplasia, necrosis, and ulceration develop during the second and third weeks of illness. Single perforations of the terminal ileum are the most frequently reported pattern [3].

Perforations extending beyond the terminal ileum particularly those involving the ileocecal junction and the ascending colon are exceedingly rare. The few reported cases in the literature describe multiple discrete perforations rather than a single longitudinal tear extending across an extended anatomical segment [4,5]. To our knowledge, a longitudinal perforation extending continuously from the ileocecal junction to the ascending colon has not previously been described in the Cameroonian or Central African literature.

We present this case to contribute to the limited documentation of atypical typhoid intestinal perforation patterns in rural sub-Saharan African settings, and to highlight the diagnostic and surgical challenges encountered in resource-limited environments.

2 CASE PRESENTATION

A 48-year-old female subsistence farmer from a rural community in Northwest Region, Cameroon, presented to our health facility with a three-week history of high-grade fever, generalized abdominal pain, and progressive abdominal distension. The pain was initially periumbilical, poorly localised, and associated with anorexia, nausea, and loose stools. In the 24 hours prior to presentation, the patient reported a sudden worsening of abdominal pain, rigidity, and inability to pass flatus or stool.

There was no history of prior treatment for the current illness at the time of presentation. The patient had not received typhoid vaccination. She had no known chronic medical conditions, was not on regular medications, and denied any history of tuberculosis, HIV, or prior abdominal surgery. She had no known drug allergies. Family and social history were unremarkable. The patient had no access to piped water and relied on communal water sources.

On examination, she was acutely ill-looking, febrile (temperature 38.9°C), tachycardic (pulse 118 bpm), hypotensive (BP 88/56 mmHg), tachypnoeic (RR 26/min), with SpO_2 of 94% on room air. She was pale and appeared dehydrated. Abdominal examination revealed a grossly distended, rigid abdomen with generalized tenderness and guarding. Bowel sounds were absent. There was dullness to percussion in the flanks bilaterally, suggesting free fluid. Rectal examination revealed an empty rectum with tenderness on the right side.

Available laboratory investigations showed a white cell count of 15.3×10^9/L with a neutrophilia of 83%. Haemoglobin was 8.2 g/dL. The Widal test was reactive -TO titre 1:160 and TH titre 1:320, which in the clinical context was considered supportive of typhoid infection. Malaria rapid diagnostic test (RDT) was negative. HIV testing was Non-Reactive. Blood and stool cultures could not be done due to laboratory limitations. Renal function and electrolytes could not be assessed at our facility.

Ultrasound of the abdomen revealed free intraperitoneal fluid and increased peritoneal echogenicity. Plain abdominal X-ray could not be done in our facility. A diagnosis of typhoid intestinal perforation with generalized peritonitis was made on clinical and radiological grounds.

The patient was resuscitated with intravenous crystalloids (1 litre normal saline over 30 minutes), commenced on intravenous ceftriaxone 2g daily, metronidazole 500mg three times daily, analgesia and transfused 1 unit of whole compatible blood. Following haemodynamic stabilization, she was taken to the operating theatre for emergency exploratory laparotomy.

Intraoperative findings were remarkable. Upon entering the peritoneal cavity, approximately 1.5 liters of feculent fluid with fecal contamination were encountered. Exploration of the small and large bowel revealed an extensive longitudinal tear of the intestinal wall beginning at the ileocecal junction and extending continuously along the antimesenteric border of the ascending colon, measuring approximately 8cm in total length. The edges of the perforation were

oedematous, friable, and necrotic, consistent with the ischaemic sequelae of typhoid-associated vasculitis of the Peyer's patches. The terminal ileum proximal to the perforation appeared congested but intact. The rest of the bowel was viable. No other discrete perforations were identified.

Surgical management consisted of thorough peritoneal lavage with warm saline, primary repair of the perforation in two layers (inner continuous absorbable suture, outer interrupted Lembert sutures), and placement of an abdominal drain. The abdominal wall was closed in layers.

Postoperatively, the patient was managed in the ward with continuation of intravenous ceftriaxone for 10 days, metronidazole for 7 days, and nasogastric decompression. Oral feeding was resumed on day 3. The postoperative course was uncomplicated. The patient was discharged on day 7 postoperatively in stable condition. She was seen at follow-up 2 weeks later and was well.

3 DISCUSSION

Typhoid intestinal perforation carries mortality rates of 10–32% in sub-Saharan Africa, with outcomes strongly dependent on the interval between symptom onset and surgical intervention [2,3]. The present case illustrates both the clinical severity of this complication and the significant diagnostic constraints that characterize its management in rural African health facilities.

3.1 Early Gastrointestinal Manifestations and Prevention of Perforation

Gastrointestinal involvement in typhoid fever follows a predictable temporal pattern corresponding to the phases of bacteraemia. During the first week, systemic features predominately fever, headache, and malaise. Intestinal symptoms emerge in the second week as bacteraemic seeding of intestinal lymphoid tissue intensifies. Early markers of gastrointestinal involvement warranting heightened clinical vigilance include abdominal distension, change in stool character (constipation transitioning to peasoup diarrhoea), localised right iliac fossa tenderness, and hepatosplenomegaly[7]. Rose spots, though rarely visible in dark-skinned individuals, may provide an additional diagnostic clue [8].

Perforation can be prevented or its severity mitigated through early and appropriate antibiotherapy. Third-generation cephalosporins (ceftriaxone) and fluoroquinolones (where sensitivity allows) initiated during the first week of illness significantly reduce the risk of intestinal necrosis and perforation [9]. Vaccination with the Vi-conjugate typhoid vaccine represents the most effective upstream prevention strategy, particularly in endemic communities with limited access to safe water and sanitation [1]. In this case, the patient had neither received vaccination nor accessed early medical care, likely contributing to the advanced disease state at presentation.

3.2 Mechanisms of Colonic Perforation in Typhoid Fever

The pathophysiology of classical typhoid perforation is well established: Salmonella typhi colonizes Peyer's patches aggregated lymphoid follicles present exclusively in the small intestine, predominantly in the distal ileum triggering a cascade of lymphoid hyperplasia, mucosal ulceration, transmural necrosis, and eventual perforation [10]. As rightly noted in the peer review of this manuscript, the colon does not contain Peyer's patches, and therefore classical Peyer's patch-mediated perforation cannot account for colonic involvement.

However, several alternative mechanisms have been proposed in the literature to explain colonic typhoid perforation, and are relevant to the anatomical findings in this case:

• Solitary colonic lymphoid follicles: The colonic mucosa contains scattered solitary lymphoid nodules that, while less prominent than Peyer's patches, may become hyperplastic and ulcerated during the bacteraemic phase of typhoid. This mechanism has been proposed to account for isolated colonic perforations documented in published case series [4,5,6].

• Contiguous ileocecal spread: The ileocecal junction represents an anatomical zone of transition between small and large bowel. A primary ileocecal perforation may extend contiguously into the adjacent ascending colon via direct transmural propagation, particularly when disease is advanced and the tissue planes are compromised by oedema and necrosis. This mechanism plausibly accounts for the longitudinal distribution of the perforation observed intraoperatively in this case.

• Typhoid mesenteric vasculitis: Salmonella typhi bacteraemia is associated with endothelial injury and mesenteric vasculitis, resulting in ischaemic necrosis of the bowel wall independent of Peyer's patch involvement. This vascular mechanism may produce transmural injury to any segment of bowel supplied by affected mesenteric vessels, including the right colon [11].

• Bacteraemic seeding of the colonic wall: Direct haematogenous seeding of the colonic submucosa during sustained bacteraemia may produce focal inflammatory lesions that progress to ulceration and perforation in the absence of primary lymphoid tissue involvement [6].

Collectively, these mechanisms suggest that colonic typhoid perforation, while rare, is a biologically plausible complication of severe typhoid illness. The longitudinal distribution from the ileocecal junction to the ascending colon, as documented in this case, most likely reflects a combination of contiguous spread from an ileocecal focus and superimposed mesenteric vasculitis.

3.3 The Typhoid Ulcer: Pathological Basis

The typhoid ulcer develops at the site of Peyer's patch hyperplasia during the second to third week of infection. Histologically, the lesion is characterised by mucosal infiltration with macrophages, lymphocytes, and plasma cells, followed by central necrosis and sloughing of the mucosa along the long axis of the bowel producing the characteristic ovoid, longitudinally-oriented ulcer with undermined edges [10,12]. This longitudinal orientation, reflecting the anatomical alignment of Peyer's patches, explains the antimesenteric location and longitudinal axis of the tear observed in this case consistent with typhoid ulcer morphology even in its colonic extension.

Global Context: Colonic Typhoid Perforation Beyond Africa

Colonic typhoid perforation has been described in case reports and surgical series from diverse geographic regions. Bhansali (1978) described colonic perforation as a rare but recognised complication in a large Indian series, occurring in the context of advanced typhoid illness with multiple bowel segment involvement [13]. A case of sigmoid colon perforation due to typhoid in Nepal, was reportedly managed with Hartmann's procedure, in the absence of small bowel pathology [14]. Similarly, reports from Pakistan and Bangladesh have described isolated colonic typhoid perforation in patients presenting late in the course of illness [15].

Outside endemic regions, typhoid colonic perforation has been documented as an imported infection in travellers returning to Europe and North America, where it presented diagnostic challenges due to low clinical suspicion [16]. These cases collectively support the existence of colonic typhoid perforation as a distinct, if infrequent, clinical entity across diverse epidemiological settings.

3.4 Diagnostic Limitations and Transparency

The authors acknowledge that, in the absence of blood culture confirmation and histopathological analysis of the resected tissue, the aetiological attribution of this perforation to Salmonella typhi cannot be established with microbiological certainty. The Widal agglutination test, while widely used in sub-Saharan Africa, carries well-documented limitations in endemic settings due to background seropositivity and cross-reactivity with non-typhoidal Salmonella and other Gram-negative organisms [17]. A reactive Widal result in this context is supportive, but not confirmatory, of typhoid aetiology.

The diagnosis in this case rests on the convergence of: a compatible clinical syndrome (three-week febrile illness with progressive abdominal symptoms), an appropriate epidemiological context (rural Cameroon, unvaccinated, untreated communal water use), a reactive Widal test, a negative malaria RDT excluding the most common differential, operative findings morphologically consistent with typhoid ulceration (antimesenteric, longitudinally-oriented perforation), and the clinical response to anti-typhoid therapy. This pragmatic clinico-operative approach to diagnosis in resource-constrained settings is consistent with published management guidelines for suspected typhoid perforation in sub-Saharan Africa [3,9].

Future documentation of similar cases should prioritise blood culture collection prior to antibiotic initiation, and submission of perforation margin tissue for histopathological analysis, where laboratory capacity permits. These measures would substantially strengthen the evidence base for colonic typhoid perforation in African settings.

Limitations

This case has important limitations. Microbiological confirmation by blood culture was unavailable. Histopathological analysis of tissue from the perforation site was not assessed. Advanced imaging was not accessible. Long-term follow-up data are limited. Despite these constraints, the overall clinical, operative, and epidemiological picture is consistent with typhoid intestinal perforation, and the case is presented with transparency regarding the degree of diagnostic certainty.

4 CONCLUSION

We report an exceptionally rare case of extensive longitudinal typhoid intestinal perforation extending from the ileocecal junction to the ascending colon in a 48-year-old female in rural Cameroon. This case extends the known spectrum of typhoid intestinal perforation patterns and highlights the importance of early clinical recognition, prompt surgical intervention, and pragmatic management in resource-limited settings. Clinicians in sub-Saharan Africa should be aware that typhoid perforation may present with anatomically atypical patterns, and that timely exploratory laparotomy remains the cornerstone of management even in the absence of complete diagnostic workup. Greater documentation of such cases from rural African clinical settings is needed to inform surgical guidelines and training.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The author declares no competing interests.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report, including any accompanying clinical details. A copy of the written consent is available for review by the Editor of this journal.

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