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[Case Study] Crohn's Disease Presenting As Acute Abdomen: A Case Report

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Abstract

An inflammatory condition, Crohn's Disease (CD) can affect any portion of the GI system. Tuberculosis, ulcerative colitis, irritable bowel syndrome, and other gastrointestinal disorders share many signs and symptoms of CD. A third of patients have involvement in the small intestine, especially the terminal ileum; 20% have colon-only involvement, and about half have both colon and small intestine involvement. The most typical CD consequences, such as intestinal obstruction with segmental thickening and fibrosis, may occur in severe cases. Despite the extensive range of diagnostic methods available, including colonoscopy, barium x-rays, CT scans, and ultrasonography, a conclusive diagnosis of CD is still challenging, and there is no one "gold standard" sign of the disorder. Crohn's disease should be considered a differential diagnosis in those with an acute abdomen, especially if they have a long history of vague abdominal issues. We discuss a patient with an acute abdomen who was admitted to our hospital and was later found to have a small intestinal obstruction. He was managed medically. After an exploratory laparotomy, histology confirmed Crohn's disease from the excised bowel parts.

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Introduction

Crohn's Disease (CD) is an inflammatory disorder and may affect any part of the gastrointestinal tract (GIT)^{1]}. The signs and symptoms of CD are similar to those of tuberculosis, ulcerative colitis, irritable bowel syndrome, and other gastrointestinal illnesses. It may involve systems other than GIT. The anatomical regions affected and the type of inflammation present determines the clinical appearance of Crohn's disease^[2]. Crohn's disease presents with a wide range of symptoms overlapping various disease processes, necessitating a high suspicion of diagnosis^[3]. Crohn's disease is a challenging diagnosis; however, morbidity and mortality of the patients can be reduced if diagnosed appropriately and the patient remains compliant with the medical management. We present a case of acute abdomen admitted to our hospital and diagnosed as a case of intestinal obstruction. An exploratory laparotomy was done, and histology showed Crohn's disease from the excised bowel parts.

Case presentation

An 18-year-old male was admitted to the surgical inpatient department with a complaint of abdominal distention for two weeks. It was associated with abdominal pain, dull aching in nature, and non-radiating. He had a history of vomiting, which was non-bilious and non-blood mixed. He also developed fever for ten days on and off, which was associated with chills and headaches. Physical examination revealed a well-built man with a blood pressure of 110/70 mmHg, heart rate of 84 beats per minute, respiratory rate of 21 breaths per minute, and a temperature of 98 °F. The patient appeared appropriate for his age and was alert and oriented to person, place, and time. The skin was warm and dry; edema was absent. The head, eyes, ears, nose, and throat revealed no abnormalities. Pulmonary and cardiovascular systems showed no abnormalities. The abdomen was flat and soft with no masses or organomegaly; bowel sounds were present and increased. Bowel frequency was decreased, and diffuse tenderness to palpation was present. Murphy's sign was negative, and hernias were absent. Rectal examination revealed no fistulas or fissures. There was a normal anal sphincter tone and no palpable masses. There were no genitourinary, neurological or musculoskeletal deficits.

The metabolic profile was unremarkable except for hyponatremia (Na-119 millimol/liter). Complete Blood Count was normal. A fever panel was negative. Montoux test showed no induration after 72 hours of intradermal injection of tuberculin PPD. The chest radiograph was normal. Multiple lymph nodes were seen on the USG of the abdomen/pelvis, some of which were sub-centimetric in the short axis and involved the mesentery and retroperitoneal area. USG-guided FNAC of inguinal lymph node showed compatibility with reactive lymphadenitis, negative for malignant cells.

Computed tomography of the abdomen and pelvis showed mucosal hyperenhancement, bowel wall thickening,

engorgement of the vasa recta, and submucosal edema in both small and large intestines.

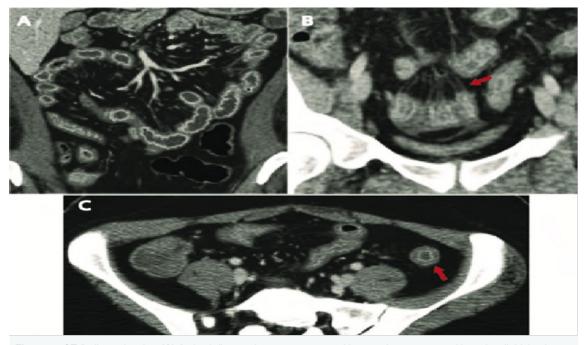


Figure 1. *CT* findings showing **(A)** Active inflammation causes mucosal hyperenhancement and bowel wall thickening greater than 3mm. **(B)** Engorgement of the vasa recta adjacent to an inflamed loop of bowel is a specific finding in active *CD* and has been coined the "comb sign." **(C)** Submucosal edema yields a characteristic "target sign."

Stool studies did not detect ova, parasites, clostridium difficle, and other bacterial pathogens. The presumptive diagnosis of Crohn's disease was made based on historical information, physical examination, and diagnostic testing. As colonoscopy is contraindicated during acute inflammation, it was postponed. Therefore, attention was focused on managing the acute inflammatory stage of the presumptive diagnosis.

The initial management plan was to keep the patient nil per oral; intravenous fluid (normal saline) started. Serum sodium monitoring was done every six hours, and daily inputs and outputs were recorded. Salt capsules were added. Intravenous antibiotic meropenem and DVT prophylaxis was started. Intensive Care Unit care, neuromonitoring, and chest physiotherapy were done. Oral antibiotics like Rifaximin, azithromycin, and metronidazole were added. Inj. celemin (5%) was added to 500 ml of Normal saline and was given intravenously over five hours every 24 hours due to poor oral nutrition. The patient was managed conservatively with antibiotics, proton pump inhibitors (PPI), steroids, and other supportive medication. After the patient's symptoms subsided, intravenous fluid therapy was switched to oral fluids, and the patient was allowed to resume a regular diet. After acute inflammation subsided, a colonoscopy was performed.

Colonoscopy findings: 1. multiple patchy erythematous erosions and ulcerations 2. Loss of vascular pattern and friable mucosa 3. Circumferential cobblestone appearance. Such findings were present in both small and large intestines.

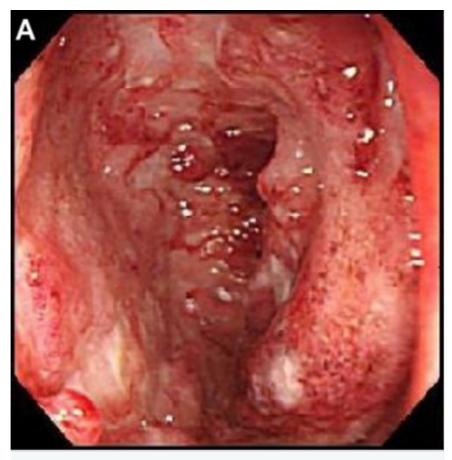


Figure 2. Colonoscopy showing deep ulcers (white areas) and circumferential cobble stone appearance between 50 cm and 55 cm from the anal verge.

The patient was started on oral mesalazine and prednisolone. An exploratory laparotomy was done for small bowel obstruction, and a part of the ileum was excised. Histology revealed trans-mural inflammation and granulomas consistent with Crohn's disease. Quantiferon test, AFB stain, and culture were negative.

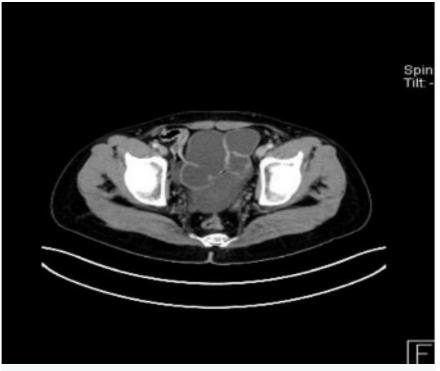


Figure 3. Small bowel obstruction secondary to Chron's disease.

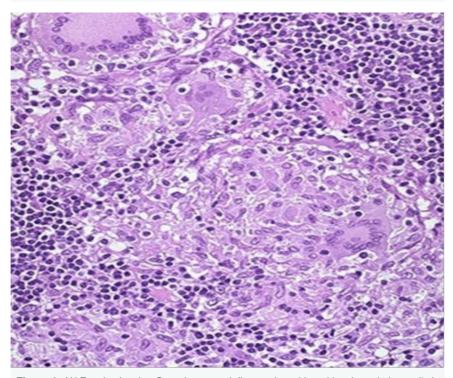


Figure 4. *H&E* stain showing Granulomatous inflammation with multinucleated giant cells in terminal lleum.

Discussion

Crohn's disease is one of the two major types of idiopathic, chronic intestinal inflammation considered to be a type of

inflammatory bowel disease (IBD). The disease is named after Burrill B. Crohn, MD, who, with colleagues, published a landmark paper describing the disease entity of regional ileitis based on 14 cases in 1932^[4]. It can affect any area of the gastrointestinal tract from mouth to anus; however, it most commonly affects the ileum^[5]. CD is almost always found in the small intestine and colon. CD may rarely affect the stomach and duodenum, and the frequency of gastroduodenal CD is reported to range between 0.5% and 4.0% in all patients with CD. The incidence and prevalence of CD in East Asia are lower than in western countries but are rising^[6]. There have been no extensive epidemiologic studies conducted on a nationwide scale to examine CD in Nepal.

CD affects men and women equally in all age groups, with predilection in the second and third decades with familial preponderance in a few cases^[7]. The etiology of CD is currently unknown, although several theories have been issued, such as the involvement of genetic factors, environmental factors (including diet), and infective agents. CD is directly correlated with a triad of predisposing factors, including genetic problems, immune system malfunctions, and environmental factors^[5]. CD usually presents with abdominal pain, mainly due to the involvement of the ileum, blood-stained diarrhea, and anemia. Some may have a low-grade fever, nausea, and vomiting. Sometimes the initial presentation of regional enteritis can closely imitate acute appendicitis, with focal right lower quadrant pain, fever, and leukocytosis. The diverse presenting symptoms and imitation of other disease entities make it imperative that clinicians maintain a high degree of suspicion to make the diagnosis of Crohn's disease^[4]. Normal healthy bowel can be found between sections of the diseased bowel. Transmural inflammation of the intestine is the hallmark of CD, with granulomatous features seen on biopsy^{[8][9]}.

The diagnosis of Crohn's disease is based on the combination of clinical history, physical examination, and endoscopic, radiologic, histologic, and laboratory findings. Despite the wide range of diagnostic techniques available, such as ultrasonography, barium x-rays, CT scans, and colonoscopy, a definitive diagnosis of CD remains elusive, and there is no single "gold standard" sign of the condition^[10]. The long-term management of Crohn's disease usually requires a multifaceted approach, including specific therapy for the inflammatory process along with adjunctive care to minimize symptoms and comorbidities. The most common medications used in Crohn's disease therapy include 5-aminosalicylic acid (5-ASA), corticosteroids, antibiotics, immunosuppressive agents, and biologic agents that are chimeric monoclonal antibodies. 5-ASA is commonly a first-line agent in Crohn's disease. Medical therapy for Crohn's disease is individualized based on the patient's disease location, extent, and response to treatment. Nutrition is vital in treating Crohn's disease because the symptoms and pathophysiology can lead to dehydration, malnutrition, and vitamin deficiencies. In our case, nutrition was addressed through fluid resuscitation and providing a well-balanced diet as tolerated. Different malabsorption syndromes can occur based on the location and extent of intestinal involvement. Nutritional support should be patient-specific, based on the nature of the disease, and directed by the patient's physician, with possible dietician consultation. Patients often require vitamin and mineral supplementation, including vitamin B12, folic acid, fat-soluble vitamins, and calcium. Periodic checks of nutritional status may also be needed^[4].

Though surgery is necessary to alleviate obstruction, repair a perforation, treat an abscess, or close a fistula, a careful approach to the patient is essential regarding intervening or continuing conservative management to avoid life-threatening

consequences. Several complications may occur with Crohn's disease; stenosis or obstruction of the small intestine or colon, extensive ileal mucosal damage, malabsorption syndromes, urinary calcium oxalate stones, gallstones, gastrointestinal bleeding and colorectal cancer, especially in long-standing disease^{[11][4]}. Fissures or cracks may appear, and anal involvement can lead to fistulas and abscesses^[12]. It may also present with extra-intestinal manifestations like skin or mouth lesions, joint pain, and eye irritation. Affected children may have delayed milestones^[13]. The outcome of Crohn's disease has improved with appropriate medical management. It's a serious condition, yet it's not fatal. The risk of surgery or accompanying diseases causes death in these patients. Although patients are asymptomatic, they should have a follow-up annually, and any new symptoms should be taken seriously^[14].

In our case, the patient's most recent postoperative abdominal CT scan and colonoscopy revealed disease-free status. He is under maintenance dose of oral mesalazine and prednisolone. He has been followed every three months or as needed in the surgical outpatient clinic with a satisfactory clinical outcome.

Conclusion

In individuals presenting with acute abdomen, Crohn's disease should be considered a differential diagnosis, especially if they have a long history of vague abdominal symptoms.

Consent

Written informed consent was obtained from the patient and his parents to publish this case report and any accompanying images.

References

- [^]McDowell C, Farooq U, Haseeb M. Inflammatory Bowel Disease. 2022 May 1. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan–. PMID: 29262182.
- [^]Gupta M, Goyal S, Goyal R. Crohn's disease presenting as acute abdomen: Report of two cases.N Am J MedSci. 2011 Apr;3(4):209-11. DOI: 10.4297/najms.2011.3209. PMID: 22540094; PMCID: PMC3336915.
- 3. [^]Austin L Jone and Kenneth E. Jones. Crohn's disease: a case presentation. Osteopathic Family Physician. Elsevier.
- ^{a, b, c, d}Jones AL, Jones KE. Crohn's disease: a case presentation. Osteopathic Family Physician. 2009;1(3):70-75. doi:10.1016/j.osfp.2009.06.007.
- ^{a, b}Alegbeleye BJ. Crohn's disease in a developing African mission hospital: a case report. J Med Case Reports. 2019;13(1):80. doi:10.1186/s13256-019-1971-5.
- Song DJ, Whang IS, Choi HW, Jeong CY, Jung SH. Crohn's disease confined to the duodenum: A case report. WJCC. 2016;4(6):146. doi:10.12998/wjcc.v4.i6.146
- 7. ^Alegbeleye BJ. Crohn's disease in a developing African mission hospital: a case report.J Med Case Rep. 2019 Mar

7;13(1):80. doi: 10.1186/s13256-019-1971-5. PMID: 30846003; PMCID: PMC6407268.

- [^]Lakhan SE, Kirchgessner A. Neuroinflammation in inflammatory bowel disease. J Neuroinflammation. 2010 Jul 8;7:37. doi: 10.1186/1742-2094-7-37. PMID: 20615234; PMCID: PMC2909178.
- [^]Cheema AY, Munir M, Zainab K, Ogedegbe OJ. An Atypical Presentation of Crohn's Disease: A Case Report. Cureus. Published online September 21, 2022. doi:10.7759/cureus.29431
- 10. ^Panes J, Bouhnik Y, Reinisch W, Stoker J, Taylor SA, Baumgart DC, Danese S, Halligan S, Marincek B, Matos C, Peyrin-Biroulet L, Rimola J, Rogler G, van Assche G, Ardizzone S, Ba-Ssalamah A, Bali MA, Bellini D, Biancone L, Castiglione F, Ehehalt R, Grassi R, Kucharzik T, Maccioni F, Maconi G, Magro F, Martín-Comín J, Morana G, Pendsé D, Sebastian S, Signore A, Tolan D, Tielbeek JA, Weishaupt D, Wiarda B, Laghi A. Imaging techniques for assessment of inflammatory bowel disease: joint ECCO and ESGAR evidence-based consensus guidelines. J Crohn Colitis. 2013 Aug;7(7):556-85. doi: 10.1016/j.crohns.2013.02.020. Epub 2013 Apr 11. PMID: 23583097.
- 11. [^]Lewis RT, Maron DJ. Efficacy and complications of surgery for Crohn's disease. Gastroenterol Hepatol (N Y). 2010 Sep;6(9):587-96. PMID: 21088749; PMCID: PMC2976865.
- 12. [^]Hendrickson BA, Gokhale R, Cho JH. (Clinical aspects and pathophysiology of inflammatory bowel disease. Clin Microbiol Rev. 2002 Jan;15(1):79-94. doi: 10.1128/CMR.15.1.79-94.2002. PMID: 11781268; PMCID: PMC118061.
- [^]Ephgrave K. Extra-intestinal manifestations of Crohn's disease. Surg Clin North Am. 2007 Jun;87(3):673-80.doi: 10.1016/j.suc.2007.03.003. PMID: 17560419.
- 14. [^]Campbell JP, Vaughn BP. Optimal delivery of follow-up care after surgery for Crohn's disease: current perspectives. Clin Exp Gastroenterol. 2016 Aug 8;9:237-48. doi: 10.2147/CEG.S96078. PMID: 27540307; PMCID: PMC4982489.