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Colitis after polytrauma: Case report

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Abstract—Across the medical literature, delayed diagnosis and treatment leads to more costly and worse outcomes. Rehabilitation patients, especially those with polytrauma, often have a complex mixture of medical, social, and psychological health problems that can impair effective diagnosis and treatment. The case presentation describes the procession toward the diagnosis of ulcerative colitis in a preinjury asymptomatic male, suggesting a potential mechanism for its emergence and describing the effect of delayed diagnosis on the efficiency of rehabilitative care. As such, the differential diagnosis for early posttraumatic diarrhea should remain broad, particularly if unexplained or ineffectively controlled.

Key words: Active Duty, amputation, comorbidity, Department of Veterans Affairs, gastroenterology, polytrauma, rehabilitation, traumatic brain injury, veteran, ulcerative colitis.

INTRODUCTION

Rehabilitation patients, especially those with polytrauma, often have a complex mixture of physiologic, social, and psychological comorbidities that interfere with participation in interdisciplinary rehabilitation. These comorbidities may not be identified in the acute care setting, which leads to worse clinical outcomes, increased length of stay, and increased cost [1–5]. Interdisciplinary rehabilitation improves outcomes and reduces length of stay when appropriately implemented [6]. It is therefore important to properly identify comorbidities and barriers to participation in therapy in order to maximize the effectiveness of rehabilitation.

This case report describes a servicemember whose greatest limitation to therapy participation was diarrhea. In acute settings, the typical differential diagnosis for diarrhea in a polytrauma patient with a prolonged hospital stay is broad but is typically focused on acute and subacute explanations. In this patient's case, these included diet, psychogenic, infection, medication use or withdrawal, bowel resection, ischemia, and disuse malabsorption. Here, we propose that ulcerative colitis (UC), a chronic idiopathic illness not on the typical differential for acute causes of diarrhea, may have been catalyzed by the stresses of polytrauma and therefore been responsible for diarrhea in this patient. Including chronic conditions in the differential diagnosis is important, because failure to consider these diagnoses may result in delayed treatment, worse outcomes, and limited progression in therapy.

Abbreviations: C. diff = clostridium difficile, CT = computed tomography, FIM = Functional Independence Measure, GI = gastroenterology, IBD = inflammatory bowel disease, PRC = polytrauma rehabilitation center, TPN = total parental nutrition, UC = ulcerative colitis.

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CASE: METHODS AND RESULTS

Patient is a previously healthy and motivated 41-year-old African-American male Active Duty service-member who experienced a severe traumatic brain injury with posttraumatic amnesia of 3 wk duration and multiple orthopedic injuries after massive trauma sustained when his parachute malfunctioned during a training jump. Patient experienced aortic injury and subsequent hemorrhagic shock requiring massive transfusion and intubation. Acute hospitalization was complicated by hypertension, ventilator-associated pneumonia, bilateral lower-limb amputations, small bowel resection due to ischemia, pelvic fractures, right elbow fracture, heterotopic ossification, total parental nutrition (TPN) use for 3 wk, urinary retention, abdominal midline wound, and pain. Upon admission to the polytrauma rehabilitation center (PRC), his total Functional Independence Measure (FIM) score was 56.

In the acute care setting, the patient had four loose stools per day with no documented diagnosis. Clostridium difficile (C. diff) toxin polymerase chain reaction assay was negative on multiple occasions. These symptoms continued upon admission to the PRC. The primary differential diagnosis included withdrawal from 100 μg fentanyl patch discontinued approximately 24 h prior to his rehabilitation admission, short bowel syndrome from 1 to 2 ft of small bowel resection, and disuse from prolonged TPN use. Infectious etiologies were lower on the differential based on absence of risk factors, fever, or leukocytosis. Pertinent medical history included a negative colonoscopy in 2009 and history of hemorrhoidectomy. Based on the differential diagnosis, he was initially managed with soluble fiber, colestipol, and loperamide, which achieved constipation. Patient subsequently developed hematochezia along with increasing abdominal discomfort without rebound tenderness. On hospital day 10, gastroenterology (GI) specialists were consulted for evaluation of diarrhea, abdominal pain, and bright red blood per rectum. A computed tomography (CT) scan of the abdomen showed gallstones (**Figure 1**), and the GI specialists recommended endoscopic retrograde cholangiopancreatography with sphincterotomy. Despite this procedure, the patient's symptoms continued. As evidenced by only a 3-point improvement in total FIM score from admission to hospital day 30 (FIM efficiency = 0.10), therapy progress was slow. Participation was severely limited by the diarrhea and the associated symptoms of pain, poor sleep, and fatigue. The bloody diarrhea progressed to become intractable with up to 14 stools per day, accompanied by severe malnutrition, despite augmenting the bowel regimen with iron, bismuth sulfate, and dietary modification to slow bowel propulsion. Abdominal imaging studies remained unremarkable and C. diff assay was negatives times four. Fecal leukocytes were positive and C-reactive protein was elevated to 12 (normal: <3). Flexible sigmoidoscopy performed by the GI specialists on hospital day 30 showed transmural inflammation (**Figure 2**), and biopsy returned cryptitis and crypt abscess consistent with inflammatory bowel disease (IBD) (**Figure 3**). The patient was diagnosed with severe UC; placed on a nothing per mouth diet; and started on TPN, steroids, and mesalamine. Surgical consultation was obtained as a precaution to discuss any surgical options. Over the ensuing days, the patient experienced progressive resolution as documented by clinical, colonoscopic, and pathologic evaluation. After diagnosis and treatment, he progressed well in therapy, tolerated a regular diet, was fitted with bilateral lower-limb prosthetics, and achieved a discharge FIM score of 112.

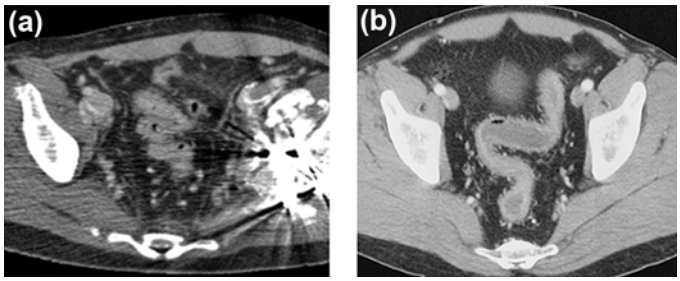

Figure 1.
Computed tomography scans of abdomen and pelvis. **(a)** Normal finding sigmoid colon seen in patient. Artifact limited view. **(b)** Typical acute ulcerative colitis finding of thickened mucosa of sigmoid colon. **Figure 1(b)** is reprinted with permission from Horton KM, Corl FM, Fishman EK. CT evaluation of the colon: Inflammatory disease. Radiographics. 2000;20(2):399–418.

DISCUSSION

UC, one of the two major disorders in IBD, is an immune-mediated chronic inflammatory condition characterized by relapsing and remitting episodes of inflammation limited to the mucosal layer of the colon. It involves the rectum and may extend proximally to the colon [7]. Typical

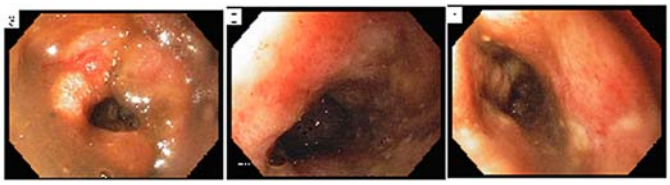

Figure 2.
Sigmoidoscopy findings. Inflammatory pattern suggestive of ulcerative colitis.

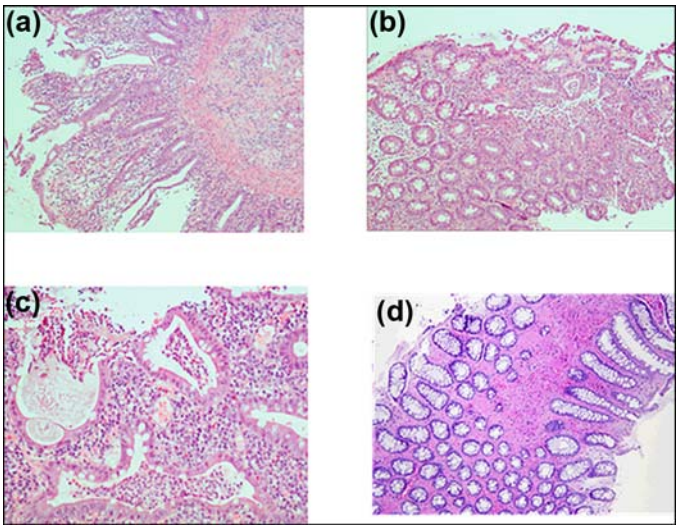

Figure 3.
Microscopic evaluation of colonic tissue. **(a)** Disease with relative sparing of submucosal. **(b)** Crypt abscess rectosigmoid consistent with ulcerative colitis (UC). **(c)** Crypt abscess and gland destruction consistent with UC. **(d)** Normal colonic mucosa (not from patient). **Figure 3(d)** is reprinted under the Creative Commons License from Oranratanaphan S, Amatyakul P, Somran J, Thumumnuaysuk S. Colonic endometriosis mimicking sigmoid cancer: A case report. Gynecol Obstetric. 2011;1:101.

presentation includes rectal bleeding, frequent stools, and tenesmus with a mean onset of symptoms to diagnosis of 10 mo [8].

In UC, the bowel wall is thin or of normal thickness, but edema, the accumulation of fat, and hypertrophy of the muscle layer may give the impression of a thickened bowel wall, especially seen in the terminal bowel region (**Figure 1**). CT scan will often show bowel wall thickness of mean 7.8 mm [9–10]. Early disease manifests as hemorrhagic inflammation with loss of the normal vascular pattern, petechial hemorrhages, and bleeding. Edema is present, and large areas become denuded of mucosa. Undermining of the mucosa leads to the formation of crypt abscesses, which is a hallmark of the disease (**Figure 3**). While laboratory studies are useful for excluding other diagnoses and assessing disease severity, the diagnosis of UC is best made with endoscopy with biopsy.

The diagnosis of UC in this patient was surprising because he exhibited neither symptoms prior to injury nor clear identifiable risk factors at the time of diagnosis. He had a negative colonoscopy several years earlier for hematochezia, with the colonscopy demonstrating internal hemorrhoids and polyps but no inflammation or other UC signs. Risk factors known to contribute to UC include genetic factors (e.g., first-degree relatives, Jewish descent) [11–12], autoimmune system reactions, environmental factors, nonsteroidal anti-inflammatory drug use [13], age (bimodal 15–25 and 55–65 yr) [14–15], low levels of antioxidants, psychological stress factors [16], and consumption of milk products. Although the patient received multiple antibiotics, this UC induction mechanism is only noted in case studies of children [17]. In regards to subacute de novo UC presentations, increased incidence has been noted after transplantation (a possible immunosuppression mechanism) [18], but no other such case presentations were noted in a literature search (Medline searches: "ulcerative colitis" AND polytrauma, "ulcerative colitis" AND "abdominal surgery"). This is despite the four proposed pathogenic causes of UC: dysbiosis, inflammation, impaired immunoregulation, and impaired mucosal functioning [19], all occurring in many settings of critical illness. There is no question that all four occur in the setting of a polytrauma involving bowel resection, infections, and abdominal wounds.

In polytrauma, the initial insult, critical illness, subsequent surgeries, infections, and emotional burden all alter cytokines, potentiating medical pathology. Mechanistically, polytrauma produces a proinflammatory state followed by immunosuppressive states, mediated by systemic interleukins, tumor necrosis factor, and neuroendocrine changes, along with local tissue injury factors [20–21]. A large component of this immunodysregulation is mediated by T cells [22]. Using the well-studied stress ulcer model, critical illness can produce ulcers through increased catecholamines, hypovolemia, and proinflammatory cytokines, leading to hypoperfusion and mucosal injury [23]. It is proposed that stress not only causes immediate pathology but can also produce more chronic

pathologic processes through induction of autoimmune diseases [24]. Moreover, although not clearly connected with the initial presentation of UC, these stress pathways are implicated to induce UC [25] and are known to exacerbate UC flares [26].

In this case, diagnosis of UC was delayed until day 30 of the rehabilitation admission despite loose stools being identified as pathologic during acute care stay. Sequellae included decreased therapy due to medical workup, malaise, weakness, and bowel accidents. The abdominal pain combined with impaired absorption decreased nutrient intake, impairing wound and bone healing and diminishing strength. Furthermore, pain and frequent stools impaired sleep, resulted in polypharmacy, overburdened the nursing staff, and adversely affected the patient's psychological well-being. As noted, there was limited change in FIM score during the month prior to diagnosis and treatment despite relatively rapid improvement thereafter in less than 2 mo (FIM efficiency of 0.10 vs 0.88). At time of consultation, if colonscopy had been performed, mild UC would have been the diagnosis; at time of diagnosis, the patient met 4 of 8 criteria for severe UC despite missing data for other criteria (erythrocyte sedimentation rate was not checked, he was malnutritioned, and he was on a beta blocker potentially masking other criteria) (**Table**). Confounding factors impeding consideration of the diagnosis were numerous, from the past medical history of hemorrhoids, the multiple pathologies from the polytrauma, negative imaging, and confounding infections to those created idiopathically with medications and procedures. Only after symptoms worsened to a level beyond explanation was colonoscopy reconsidered. Despite the delayed diagnosis, this patient fortunately had a good outcome as evidenced by his discharge FIM score.

CONCLUSIONS

Although in the hospital setting, new onset symptoms are likely due to acute changes; in this case, late consideration in the differential diagnosis of an idiopathic chronic condition led to delayed diagnosis and prolonged rehabilitation stay. UC currently has unclear pathogenesis with typically prolonged symptomatology prior to diagnosis. The presentation after polytrauma in a previously asymptomatic male may support physiologic stress as an underlying mechanism for development of UC. As such, UC should be considered in the differential diagnosis of early posttrauma diarrhea, particularly in the presence of associated pain.

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Table.
Criteria for evaluating severity of ulcerative colitis (Truelove and Witts criteria) [27].

Variable	Mild Disease	Severe Disease	Fulminant Disease
Stools (number per day)	<4	>6	>10
Blood in Stool	Intermittent	Frequent	Continuous
Temperature (°C)	Normal	>37.5	>37.5
Pulse (bpm)	Normal	>90	>90
Hemoglobin	Normal	<75% of normal	Transfusion required
Erythrocyte Sedimentation Rate (mm/h)	≤30	>30	>30
Colonic Features on Radiography	—	Air, edematous wall, thumb printing	Dilatation
Clinical Signs	—	Abdominal tenderness	Abdominal distention and tenderness

bpm = beats per minute.

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