CASE REPORT

An unusual case of abdominal sepsis following traumatic injury

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SUMMARY

A 19-year-old woman presented to our emergency department with crampy abdominal pain and per rectal bleeding 2 weeks after falling from a horse. She had been taking regular non-steroidal anti-inflammatory drugs (NSAIDs) for analgesia. On arrival, she was tachycardic and tachypnoeic, with a lactate of 7.3 mmol/L. 'FAST' ultrasonography was unremarkable and CT scan showed thickened wall of the transverse colon. She underwent flexible sigmoidoscopy, which demonstrated "patchy inflammation and an isolated area of severe deep ulceration with nodularity and oedema". A presumptive diagnosis of "Inflammatory Bowel Disease—likely Crohn's", was made, and treated accordingly with steroids and Pentasa. Two months following discharge, the patient underwent a colonoscopy, showing a normal colon, however, "a few ulcers in the terminal ileum" were seen. She was seen by a gastroenterologist who deemed the most likely diagnosis to be NSAID-induced terminal ileitis and colitis. Having stopped the offending NSAID (and steroids), she has now made a full recovery.

BACKGROUND

Left upper quadrant (LUQ) pain in a clinically shocked patient with a history of trauma can provoke concern regarding splenic injury and a hypovolaemic cause of shock. In actual fact, this patient was systemic inflammatory response syndrome positive (tachycardic, tachypnoeic, raised white cells) and the cause of her shock was septic. The gastrointestinal (GI) side effects of non-steroidal anti-inflammatory drugs (NSAID) are well documented in the upper GI tract, however, they are seldom seen affecting the lower part of the tract. NSAID-induced colitis is a "rare but serious, sometimes fatal, complication of NSAID therapy."¹ Fewer than 100 cases of NSAID-induced colitis have been reported in previous literature.¹ The majority of affected patients seem to be elderly patients on long-term NSAID therapy. Our patient was unusual in that she was young and only on NSAIDs for a short duration.

CASE PRESENTATION

A 19-year-old woman presented to our emergency department with crampy LUQ abdominal pain and PR bleeding 2 weeks after falling from a horse. Prior to the fall, she was fit and well, and on no regular medication. There was no family history of inflammatory bowel disease. She had been taking regular NSAIDs for analgesia since the fall, mainly for musculoskeletal lower back pain. On arrival, she was tachycardic, tachypnoeic, and had a lactate

of 7.3. She was afebrile but she said she had felt feverish in the last 24 h. On examination, her abdomen was soft, but very tender in the LUQ and over the lower left ribs. 'FAST' ultrasonography was unremarkable and subsequent CT scan showed thickened wall in the transverse colon. Admission bloods included raised inflammatory markers (white cell count $40.5 \times 10^9/L$, C reactive protein 263 mg/L), haemoglobin of 167 g/dL and normal renal function. She was admitted under the surgical team as an intra-abdominal sepsis. She underwent flexible sigmoidoscopy, which showed patchy inflammation of the transverse colon, and an isolated area of severe deep ulceration with nodularity and oedema (figures 1–3). A diagnosis of colitis probably secondary to inflammatory bowel disease was made, and treated accordingly with steroids and Pentasa. She was also treated with intravenous antibiotics throughout (Tazocin and metronidazole). Following resolution of symptoms and discharge from hospital, 2 months later she underwent an outpatient colonoscopy. This showed a normal colon, however, "a few ulcers in the terminal ileum" were noticed. The patient was seen by a consultant gastroenterologist who deemed the most likely diagnosis to be NSAID-induced terminal ileitis and colitis. Having stopped the offending NSAID, steroids and Pentasa, she has now made a full recovery.

INVESTIGATIONS

CT scan on admission showed thickened wall in the transverse colon and splenic flexure with poorly enhancing mucosa, with no significant associated stranding in the adjacent fat.

Our patient underwent an inpatient flexible sigmoidoscopy including colonic biopsies. This showed patchy minimal inflammation of the mid to distal transverse colon, but severe ulceration in one part of

Figure 1 Colonic ulceration and inflammation.

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Figure 2 Inflammation and oedema in the colon.

the colon with nodularity and severe oedema. The histology from these biopsies was reported as “Biopsies of large intestinal mucosa show mild oedema of the lamina propria and mild architectural distortion in the form of crypt branching and shortening. A single crypt is distended with neutrophils and debris suggestive of a crypt abscess. There is no cryptitis and there is significant chronic inflammation. No granulomata are seen.”

Following discharge, the patient later underwent a colonoscopy, reaching the terminal ileum. This showed “a normal colon but a couple of ulcers in the terminal ileum”. Biopsies of the ileal ulcers demonstrated “Small intestinal mucosa showing focal superficial acute inflammation. No specific features. No granulomata seen.”

Urinalysis, stool culture and blood culture were all unremarkable.

DIFFERENTIAL DIAGNOSIS

Given the initial presentation, my colleagues and I were concerned over the possibility of a splenic injury causing this patients’ intra-abdominal sepsis. Fast ultrasonography did not reveal any free fluid. The CT scan was suggestive of colitis. The patient was subsequently admitted under the surgical team and

Figure 3 Inflammation, ulceration and oedema in the colon.

treated with intravenous antibiotics. Given the abnormal CT findings of the bowel, endoscopy was performed. At the time, it was thought the macroscopic and microscopic features of her presentation were most in keeping with Crohn’s disease. It was only after the patient had recovered and was seen in the outpatients department by our gastroenterology team that the diagnosis of NSAID-induced colitis and terminal ileitis was made.

TREATMENT

The patient was treated as per national guidelines on the management of Crohn’s disease.

OUTCOME AND FOLLOW-UP

The patient has made a full recovery and is symptom free at the time of writing. She has stopped taking all NSAIDs and steroids. She remains on Pentasa daily and will be seen in gastroenterology outpatients 1 year after the first appointment, with a view to stopping all medications at this point.

DISCUSSION

The gastrointestinal side effects of NSAIDs are well known in the upper GI tract, however, they seldom seem to affect the lower part of the tract. NSAID-induced colitis is a “rare but serious, sometimes fatal, complication of NSAID therapy.”¹ Fewer than 100 cases of NSAID-induced colitis have been reported in previous literature.¹ The histology of NSAID-induced colitis typically shows non-specific colitis. In making a diagnosis of Crohn’s disease, one should exclude other causes of bowel inflammation, such as NSAID-induced enteropathy.

The majority of affected patients seem to be elderly patients on long-term NSAID therapy. Our patient was unusual in that she was young and only on NSAIDs for a duration of 2 weeks.

Learning points

- ▶ Non-steroidal anti-inflammatory drug (NSAID) side effects can occur anywhere in the gastrointestinal tract.
- ▶ Colitic patients can present profoundly septic.
- ▶ Not all colitic patients have inflammatory bowel disease (IBD).
- ▶ NSAID-induced colitis can mimic IBD—particularly Crohn’s disease.

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REFERENCE

- 1 Gibson GR, Whitacre EB, Ricotti CA. Colitis induced by nonsteroidal anti-inflammatory drugs. Report of four cases and review of the literature. *Arch Intern Med* 1992;152:625–32.

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