CASE REPORT

Massive lower gastrointestinal haemorrhage in a teenager caused by *Campylobacter enteritis*

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SUMMARY

A 14-year-old boy presented with a very unusual complication of massive lower gastrointestinal bleeding mimicking bleeding from Meckel's diverticulum who needed blood transfusions and required mini-exploration. He was found to have *Campylobacter enteritis* with bleeding from multiple mucosal ulcers in the ileocaecal region, which is presented with a review of the literature.

BACKGROUND

Campylobacter enteritis is a significant cause of diarrhoea and generally a mild disease. In some cases repeated stool cultures and antibiotic therapy may be indicated to diminish the morbidity. *Campylobacter* can produce minor self-limiting intestinal bleeding but massive lower gastroinstinal (GI) bleeding as a complication is extremely rare.[1] Only one case of such complication has been reported in adults and none in children.[2] Massive GI haemorrhage is a rare complication of *Campylobacter jejuni* infection. We report a 14-year-old boy who presented with massive lower GI bleeding secondary to *C enteritis*.

CASE PRESENTATION

A 14-year-old boy was admitted to a university hospital without the paediatric surgical services with a 1 day history of rectal bleeding which had increased in frequency associated with sticky loose stools. Abdominal pain was diffuse and crampy. Loose stools were six per day without any blood, which lasted for 2 days. There was no fever, chills, nausea or vomiting. There was abdominal pain and discomfort while passing stools and had lost weight since last week. He had non-traumatic nose bleeding on two occasions for the past 2 days. He had malaise and dizziness associated with significant pallor. He passed a large smelly stool of black tarry colour and fresh blood followed and splashed in the toilet. Since then he opened bowels 10 times with 20–50 mL of fresh red coloured blood. He had visited a farm 1 week before his illness.

He had been diagnosed with supraventricular tachycardia 4 years previously, which was ablated 2 weeks before and he had been prescribed 75 mg aspirin daily. On admission his pulse was 121/min and regular, his blood pressure 112/65 mm Hg and temperature 36.8°C. He was pale and listless. His chest was clear and abdomen was soft. There was mild tenderness in the epigastric region. There were no local lesions in the perineum and perianal regions. Per rectal examination was normal.

INVESTIGATIONS

The patient's initial haemoglobin was 11.7 gm%, white blood cell (WBC) 6.93×10^9/L and platelets were 313×10^9/L. His serum biochemistry, coagulation screen and ECG were all normal. A diagnosis of aspirin-induced bleeding or infective colitis was considered and he was admitted for observation. He was given intravenous ranitidine overnight. His pallor increased and he felt dizzy. His pulse increased to 132/min and haemoglobin dropped to 9.5 gm%. He was very pale and anaemic clinically with severe malaise. The malaena and fresh rectal bleeding continued.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of massive lower GI bleeding included anatomic, vascular, neoplastic and inflammatory causes (infectious and non-infectious). Infectious causes thought of were *Salmonella, Shigella* and *Escherichia coli*.

TREATMENT

Aspirin was stopped after discussion with his consulting paediatric cardiologist at the regional children's hospital. He was started on tranexamic acid 10 mg/kg three times a day. He underwent upper GI endoscopy at the referring university hospital, which showed normal oesophagus, stomach and duodenum without any evidence of fresh or old bleeding. He was then transferred to us as he continued to have persistent lower GI bleeding. His haemoglobin dropped to 9.5 gm% despite having transfused four units of bloods in order to stabilise him before transfer.

He underwent examination under anaesthesia, which did not reveal any mass, and lower GI endoscopy showed no local lesions in the anus, rectum or colon but the lumen was full of blood clots and bowel mucosa appeared congested. Biopsies were taken. A mini exploration via the right iliac fossa transverse skin crease incision and grid iron approach showed no evidence of Meckel's diverticulum or any other surgical lesions but there were few clots in the distal 5 or 6 cm of the terminal ileum with thickening of the ileocaecal region and enlarged multiple firm lymph nodes were noted. The colon was filled with blood clots. He received three doses of perioperative intravenous antibiotics. His bleeding stopped and the haemoglobin stabilised.

OUTCOME AND FOLLOW-UP

His postoperative period was uneventful except for urinary retention due to pain, which required

catherisation. His haemoglobin was 11.5 gm%, white blood cell count 6×10^9/L and platelets 205×10^9/L. The microbiology and infection control department from the referring hospital contacted us and informed that he has grown *Campylobacter proctocolitis* from his initial stool sample. The microbiology department at our hospital has grown the same species, which was sensitive to ciprofloxacilin and no other organisms were grown. Public health authorities were informed and he was isolated and a course of ciprofloxacilin was completed.

Repeat stool cultures after 48 h of antibiotics were negative and the histology of the rectal and colonic biopsies showed active chronic inflammation possibly secondary to *C proctocolitis*. He was started again on aspirin and follow-up at 8 years showed him to be completely asymptomatic and well.

DISCUSSION

C jejuni, a Gram-negative spiral shaped bacterium, is a frequent cause of food-borne GI illness in humans throughout the world. Illness with *C jejuni* ranges from mild to severe diarrhoeal disease. *C jejuni* is a newly recognised enteropathogenic bacterium. Acute lower GI bleeding is an extremely rare complication in *C enteritis*. Massive bleeding has been reported as a rare manifestation of intestinal infections and infestations in the tropics.[3–6]

C jejuni and *C coli* infect the healthiest of persons resulting in self-limiting diarrhoea often accompanied by mucus and blood in the stool. The severity of their symptoms and signs provided difficulty in distinguishing this diagnosis from acute surgical emergencies, in particular acute appendicitis and inflammatory bowel diseases.[7 8] It may cause haemorrhage from the ulceration of the stomas, solitary caecal ulcer, toxic megacolon, perforative peritonitis, intestinal obstruction or may develop typical Fisher's syndrome with neurological features.[9–15]

The most important radiological features were thickening of ileal mucosal folds, of interhaustral indentations and of the ileocaecal valve, lymphoid hyperplasia and microulcerations.[16] Radiology, as well as endoscopy, are both non-specific in *C jejuni* enterocolitis. The importance of radiology is to exclude more typical features of other causes of inflammatory bowel diseases. Moreover, before the result of the stool culture is available, the radiological features should suggest the suspicion of an acute infectious enterocolitis by *C jejuni* as possible diagnosis. The Meckel's scan is positive only in 15% of cases and the services are often not available during weekends.

Campylobacter causes self-limiting disease but bloody diarrhoea is also known.[17] A conservative approach may be suggested as first-line therapy. Drug of choice is erythromycin and ciprofloxin is effective in 90% of cases.[17] Whenever possible, the algorithm for evaluating massive lower GI bleeding should be followed as suggested by University of Pennsylvania Health System.[18] Based on the evidence report, the availability of particular tests and clinical experience, the committee developed an algorithm for the management of patients with acute lower GI bleeding which emphasises colonoscopy, contrast-enhanced CT with interventional radiology procedures and a nuclear imaging scan using ^{99m}Tc-labelled red blood cells, in that order, to localise the site and deal with it accordingly.

Surgery is inevitable in patients suffering from massive bleeding and in patients with recurrent bleeding. The *C enteritis* is often self-limiting, but in view of the severity of complications, we recommend that symptomatic *C enteritis* should be treated with appropriate antibiotics to prevent subsequent complications, since severe bleeding can occur at the time of apparent recovery.

Learning points

- Massive gastrointestinal haemorrhage is a rare complication of *Campylobacter jejuni* infection.
- Clinicians should be alerted to the possibility of the occurrence of severe hemorrhagic enteritis as a life-threatening complication in *Campylobacter*-induced enteritis.
- Our case is the reminder of the fact that this increased morbidity can occur long after the initial abdominal pain, diarrhoea and fever have settled.
- Careful food preparation and cooking practices may prevent some *Campylobacter* infections
- Whenever possible, the algorithm for evaluating massive lower gastrointestinal bleeding developed by University of Pennsylvania Health System should be followed.

Contributors All the authors were involved in the care of the patient, collection, analysis and preparation of data and manuscript, in literature search and in writing, critically evaluating and finalising the manuscript.

Competing interests None.

Patient consent Obtained.

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REFERENCES

1 Reina J, Borrell N, Fiol M. Rectal bleeding caused by *Campylobacter jejuni* in a neonate. *Pediatr Infect Dis J* 1992;11:500–1.
2 Michalak DM, Perrault J, Gilchrist MJ, *et al*. Campylobacter fetus ss. jejuni: a cause of massive lower gastrointestinal haemorrhage. *Gastroenterology* 1980;79:742–5.
3 Sharma BC, Bhasin DK, Bhatti HS, *et al*. Gastrointestinal bleeding due to worm infestation, with negative upper gastrointestinal endoscopy findings impact of enteroscopy. *Endoscopy* 2000;32:314–16.
4 Watanabe T, Kudo M, Kayaba M, *et al*. Massive rectal bleeding due to ileal tuberculosis. *J Gastroenterol* 1999;34:525–9.
5 Sangkhathat S, Patrapinyokul S, Wudhisuthimethawee P, *et al*. Massive gastrointestinal bleeding in infants with ascariasis. *J Pediatr Surg* 2003;38:1696–8.
6 Kostka R, Lukas M. Massive, life-threatening bleeding in Crohn's disease. *Acta Chir Belg* 2005;105:168–74.
7 Moore MH, Davidson JR. *Campylobacter enteritis* in the acute surgical ward. *N Z Med J* 1984;97:219–20.
8 Campbell LK, Havens JM, Scott MA, *et al*. Molecular detection of *Campylobacter jejuni* in archival cases of acute appendicitis. *Mod Pathol* 2006;19:1042–6.
9 Meuwissen SG, Bakker PJ, Rietra PJ. Acute ulceration of ileal stoma due to Campylobacter fetus subspecies jejuni. *Br Med J (Clin Res Ed)* 1981;282:1362.
10 Alloy AM, Santoro JJ, Lazarus BG, *et al*. Campylobacter fetus ss. jejuni: a cause of solitary cecal ulcer. *Clin Gastroenterol* 1986;605.
11 Schneider A, Runzi M, Peitgen K, *et al*. *Campylobacter jejuni*-induced severe colitis—a rare cause of toxic megacolon. *Z Gastroenterol* 2000;38:307–9.
12 Larvol L, Zeitoun E, Barge J, *et al*. Toxic megacolon with colonic perforation complicating *Campylobacter jejuni* ileo-colitis. *Gastroenterol Clin Biol* 1994;18:281–4.
13 Perkins DJ, Newstead GL. *Campylobacter jejuni* enterocolitis causing peritonitis, ileitis and intestinal obstruction. *Aust N Z J Surg* 1994;64:55–8.
14 Kohler A, de Torrente A, Inderwildi B. Fisher's syndrome associated with *Campylobacter jejuni* infection. *Eur Neurol* 1988;28:150–1.
15 Ichikawa H, Sugita K, Fukui T, *et al*. Fisher's syndrome following *Campylobacter jejuni enteritis*—a case report and review of the literature. *Rinsho Shinkeigaku* 1995;35:391–5.
16 Demaerel P, Ponette E, Lacquet F, *et al*. The role of radiology in *Campylobacter enterocolitis*. *Rofo* 1989;150:551–5.
17 Havelaar AH, van Pelt W, Ang CW, *et al*. Immunity to *Campylobacter*: its role in risk assessment and epidemiology. *Crit Rev Microbiol* 2009;35:1–22.
18 University of Pennsylvania Health System (UPHS). *Management of acute lower GI bleeding*. Philadelphia, PA: University of Pennsylvania Health System (UPHS), 2009:6.