CROHN’S DISEASE CO-EXISTING WITH BLEEDING PEPTIC ULCER DISEASE - A CASE REPORT

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ABSTRACT
Crohn’s disease is a chronic idiopathic inflammatory condition of the gastrointestinal tract, which rarely affects the oesophagus and stomach together. The usual sites of involvement are the terminal ileum and proximal colon. It is rarer still for gastric Crohn’s disease to present for the first time with haematemesis resulting from acute upper gastrointestinal haemorrhage, especially in an African setting where the disease is thought to be rare. Oesophageal Crohn’s disease is usually asymptomatic for a long period of time, and may come to medical attention when dysphagia or complications such as stricture or fistula occur in the oesophagus, or when extra-oesophageal manifestations occur. We present a 67 year old Nigerian farmer who was brought into our emergency room at the Irrua Specialist Teaching Hospital, Irrua, from his farm with a history of sudden onset haematemesis. The clinical diagnosis was moderate to severe upper gastrointestinal haemorrhage secondary to non-steroidal anti-inflammatory drug use. He improved on medical treatment, including blood transfusion. Upper gastrointestinal endoscopic diagnosis was peptic ulcer disease of the oesophagus and stomach and a small hiatus hernia. However, histology of biopsied specimens from the oesophagus and stomach showed typical multiple non-caseating granulomas with associated features of chronic inflammation in keeping with Crohn’s disease.

Keywords: Crohn’s disease, Oesophagus, Stomach, Upper gastrointestinal tract bleeding

INTRODUCTION
Crohn’s disease is a chronic transmural inflammation that may involve any part of the alimentary tract from mouth to anus, and is associated with many extra-intestinal features.

It most frequently manifests as abdominal pain and diarrhoea, and is often complicated by intestinal fistulation or obstruction or both. Crohn’s disease typically affects the ileum, colon or perianal region. Involvement of the oesophagus and stomach is uncommon, and isolated involvement of the oesophagus and stomach is even rarer.¹

Case Report
Mr. O P is a 67-year-old Nigerian farmer who presented at the accident and emergency unit of the Irrua Specialist Teaching Hospital, Irrua in December 2010, with a 2-hour history of sudden onset haematemesis. He was working at his farm when he suddenly developed haematemesis estimated to be about 300mls in volume. There were no associated blood clots in the vomitus. He later became weak but experienced no altered level of consciousness. There was no history of melena, haematochezia, or
haematuria at presentation, however he started passing melaena after 24 hours on admission. There was no current or past history of jaundice or recurrent abdominal pain. A history of weight loss, or swelling in any part of the body was absent. He however, had a history of chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs) in combination ("Alabukun", Piroxicam, Diclofenac) on a daily basis in the last one month for low back and joint pains. He was not a known peptic ulcer disease patient. He neither consumed alcohol nor smoked cigarettes. He was not hypertensive or diabetic. He was married with children.

Clinical examination revealed an elderly man who was conscious, not pale and not dehydrated. He was not dyspnoeic at rest and was not icteric. There was no central or peripheral cyanosis. He had no oral ulcers or peripheral lymph node enlargement. There was neither digital clubbing nor other peripheral stigmata of chronic liver disease. He had a pulse rate of 102bpm; his blood pressure was 100/60mmHg in the supine and 80/60mmHg in the erect positions respectively. First and second heart sounds only were heard on auscultation. The liver which was tipped was smooth and soft with a span of 10cm. There was no clinical evidence of splenomegaly or ascites. Digital rectal examination was normal.

The initial clinical assessment was upper gastrointestinal (UGI) haemorrhage secondary to chronic NSAID abuse.

He was treated with 2 pints of whole blood, intravenous normal saline fluid, intravenous omeprazole and triple regimen for Helicobacter pylori infection eradication.

Results of laboratory investigations are as follows: PCV:35%; TWBC: 5,300/mm$^3$; RBS:145mg/dl; Urea:74mg/dl; Na$^+$:133mmol/L; K$^+$:3.4mmol/L; HBsAg and Anti-HCV were non-reactive; prothrombin time was 12 seconds; abdominal ultrasound was essentially normal.

While on admission, his clinical status improved; there was no further episode of haematemesis. He was discharged to do UGI endoscopy which was done 11 days after presentation. The result of the endoscopy revealed features of Oesophagitis with areas suspicious for Barrett’s, a small hiatus hernia and an ulcer measuring 3-4mm with a clean base at the pylorus along the lesser curvature of the stomach. There was no evidence of ongoing or recent bleeding in the oesophagus, stomach or duodenum. An endoscopic diagnosis of peptic ulcer disease was made. A Pentax EPK-700 endoscopy system was used for the procedure.

The report of the histology of biopsied tissues from the oesophagus and gastric mucosa read: “Oesophagus mucosa – sections showed tiny fragments of tissue with some areas lined by stratified non-keratinizing squamous epithelium. The surrounding mucosa has dense lymphocytic cell infiltrates and non-caseating granuloma. Stomach mucosa section show tiny fragments of gastric tissue within which lie a chronic inflammatory lesion composed of chronic inflammatory cell infiltrates and multiple non-caseating granuloma. The gastric glands are lined by tall columnar epithelium and are few in number. Diagnosis: Oesophageal mucosa and gastric mucosa features in keeping with Crohn’s disease”.

Colonoscopy was requested for, but could not be done due to financial constraints. The patient has remained clinically stable, and on follow up at the gastroenterology clinic.

**DISCUSSION**

Crohn’s disease was first described in 1932. It is thought to be rare in Africans. There are a few reported cases of Crohn’s disease in Nigeria and these cases involved the colon. Involvement of the oesophagus and stomach is uncommon, either in association with intestinal disease or in isolation.

The symptoms commonly seen in oesophageal Crohn’s disease include dysphagia, odynophagia, heartburn, chest pain and abdominal pain. In the case of gastroduodenal Crohn’s disease, common symptoms include epigastric pain, nausea and vomiting, weight loss, early satiety, anorexia, bloating and belching while rare symptoms include malaise, pyrosis, diarrhoea, haematemesis, melaena and faeculent vomiting (in the presence of fistula). Our patient presented with haematemesis without associated dysphagia, abdominal pain or weight loss, clinical features commonly attributed to a bleeding peptic ulcer disease in our environment.

The upper gastrointestinal bleeding in this case may have been provoked by the use of multiple NSAIDs in the setting of Crohn’s disease of the oesophagus and stomach.

Endoscopy findings in oesophageal and gastric Crohn’s disease are similar to the findings in Crohn’s disease involving the intestines: these include mucosal...
oedema, focal and diffuse erythema, nodular lesions, erosions and ulcers. In our patient the endoscopic findings were features of oesophagitis and Barrett’s in the distal oesophagus and a pyloric ulcer in the stomach. Since Crohn’s disease is said to be very rare in African Nigerians, Crohn’s disease was neither suspected clinically nor endoscopically. Therefore it was not surprising that an endoscopic diagnosis of peptic ulcer disease as the cause of the UGI bleeding was made. Our case suggests that endoscopic findings may not always be relied upon to make the diagnosis of Crohn’s disease, especially in cases of UGI bleeding involving African Nigerians where Crohn’s disease is said to be rare.

Histology of biopsied tissues from the oesophagus and stomach of our patient revealed non-caseating granuloma with chronic inflammatory cells surrounding it. This was the basis for the diagnosis of Crohn’s disease in this case. It is however important to note that granuloma is not seen in all cases of Crohn’s disease. The unexpected histologic finding of Crohn’s disease in our patient emphasizes the importance of taking biopsies of lesions in the oesophagus and stomach during UGI endoscopy for histological diagnosis, as endoscopic examination alone may miss the diagnosis in some cases as exemplified by our report. Furthermore, our report would suggest the inclusion of Crohn’s disease in the differential diagnoses of UGI bleeding in the African Nigerian.

Our patient responded well to the triple regimen consisting of omeprazole, amoxicillin and clarithromycin. He was also advised to discontinue the use of NSAID s. The role of anti-secretory medication use in Crohn’s disease is not yet defined, however symptomatic relief and healing of oesophageal ulcers has been achieved with the use of proton pump inhibitors.

The use of corticosteroids and 5-aminosalicylates may be required to forestall the complications associated with Crohn’s disease such as fistula and stricture formation.

A possible limitation to this report was our inability to endoscopically or histologically exclude lower GI Crohn’s disease in our patient. However, our patient did not have lower gastrointestinal tract clinical features suggestive of inflammatory bowel disease.

In conclusion, Crohn’s disease even though thought to be rare in Africans should be considered in the differential diagnoses of upper gastrointestinal haemorrhage in Nigerians. Biopsies of upper gastrointestinal tract ulcers should be sent for histology to exclude uncommon causes of ulcers such as Crohn’s disease, as highlighted in this case report.

REFERENCES