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Octreotide LAR for severe obscure-overt gastrointestinal haemorrhage in high-risk patients on anticoagulation therapy

We read with interest the letter by Krikis *et al* (*Gut* 2005;**54**:171–2). We report on two patients with a poor clinical history (recurrent gastrointestinal haemorrhage, older age, severe comorbidity, anticoagulation, contraindication to β-blockers), successfully treated using only octreotide long-active release (LAR).

A 73-year-old man was admitted six times (32 days of hospital stay) in the previous six months because of recurrent episodes of melaena. Medical history included type 1 diabetes, three myocardial infarctions and multivessel disease treated by triple-bypass graft surgery (left ventricular ejection fraction 35%), ischaemic stroke due to total occlusion of the right carotid artery, surgically solved, and occlusion of the left carotid artery of 40%, treated with acenocoumarol. Haemoglobin fell to 7–8 g/dl in all admissions developing stable angina pectoris, without overcoagulation, requiring a transfusion of 25 units of packed red blood cells. Three upper endoscopies, two colonoscopies with ileal intubation, small bowel series radiography, computed abdominal tomography, capsule endoscopy and radionuclide scanning did not find the source of bleeding.

A 69-year-old man was admitted ten times during 14 months for recurrent episodes of melaena (122 days of hospital stay). He smoked 30 cigarettes per day. His medical history included obesity, heart failure, chronic obstructive pulmonary disease on long-term oxygen therapy, obstructive sleep apnoea and atrial fibrillation on acenocoumarol therapy. Haemoglobin fell to 6–7 g/dl in all admissions, with exacerbation of previous diseases. No overanticoagulation was detected and a transfusion of 37 units of packed red blood cells was required. Two upper endoscopies, two colonoscopies, small-bowel series radiography, computed abdominal tomography and radionuclide scanning did not achieve diagnosis. Capsule endoscopy showed diffuse angiodysplasias from the jejunum to the ileum.

Because of the medical conditions and a high rate of bleeding, rescue treatment in both patients was started with octreotide LAR 20 mg intramuscularly once a month. Neither blood transfusions nor admissions were needed after 9 months of follow-up, and no side effects or anticoagulant interaction was detected.

Obscure gastrointestinal haemorrhage represents 5% of gastrointestinal bleeding. Some patients are not suitable for endoscopic or surgical management because of severe comorbidity, lack of diagnosis of the source of bleeding or diffuse lesions non-responsive to conventional treatments, and may be at high risk of re-bleeding, especially if active bleeding or lesions are detected in the small bowel.[1 2] The effectiveness of hormonal treatment is unclear and burdened by adverse effects.[3 4] Octreotide can control bleeding from the gastrointestinal angiodysplasia and variceal bleeding,[5 6] probably by decreasing splanchnic and portal blood flow. Octreotide LAR, a depot formulation, is administered once monthly, with a similar efficacy and safety profile as octreotide, and does not require hospital admission. In addition to the report by Krikis *et al*, we found only two other reports on the usefulness of octreotide LAR for severe obscure gastrointestinal bleeding,[7 8] but not in older anticoagulated patients with a poor prognosis. Moreover, the economic and medical resources saved and the psychological benefits for patients and families in an outpatient management make octreotide LAR an attractive, effective, safe and comfortable treatment for some specific cases of obscure gastrointestinal bleeding, like ours, thanks to conservative management.

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Acute surgical abdomen—an atypical presentation of *Plasmodium vivax* malaria

A search of the existing literature did not identify any previously reported cases of acute abdomen as atypical features of *vivax* malaria. The case presented here illustrates the ubiquitous nature of this disease with its unusual clinical manifestations in the UK.

A 51-year-old Asian man presented to the acute surgical admissions unit with a 1 week history of left iliac fossa pain, malaise and non-bilious vomiting. No other bowel or urinary symptoms were present. His medical history included asthma, which was well controlled with inhalers. He had no history of any previous abdominal surgery.

On examination, he was found to be clammy and to have dry mucosal surfaces. Initial observations recorded a temperature of 40.1℃ and a regular pulse of 133 beats/min, with a blood pressure of 108/51 mm Hg and a respiratory rate of 20 breaths/min. Pulse oximetry showed an oxygen saturation of 97% in room air. Cardiorespiratory examination was normal. Abdominal examination showed marked left iliac fossa tenderness with signs of peritonism. No evidence of hepatosplenomegaly was seen. Per rectal examination was normal. Chest and abdominal *x* rays were unremarkable, with no evidence of perforation. Urine analysis was negative. Blood tests gave 13.1 g/dl haemoglobin, 2.97×10/l leucocytes, 115×10/l platelets, 9.8 mmol/l urea, and 128 mmol/l creatinine. All other blood tests including serum amylase were normal. The initial clinical impression was sepsis secondary to diverticulitis with localised diverticulum perforation.

The patient was resuscitated with crystalloid, with a lack of response to resuscitation or deterioration in the clinical condition indicating an emergency laparotomy. However, over a period of observation with aggressive fluid resuscitation, he stabilised haemodynamically (pulse 80 beats/min and blood pressure 92/57 mm Hg). An abdominal/pelvic computed tomogram showed no evidence of diverticulitis or perforation. No focal small or large bowel abnormality was detected. A cardiac echo excluded infective endocarditis. Interestingly, blood films taken for haematology were reported to show *Plasmodium vivax* infestation. Further questioning revealed that the patient had returned from Pakistan 8 months before. He also admitted to not taking prophylaxis. Treatment for malaria was initiated with a 600 mg chloroquine stat dose, 300 mg once daily for 2 days. This was followed with primaquine, 30 mg once daily for 14 days. The patient was discharged 4 days later, having remained pain-free and apyrexial.

This case report shows that increased global travel results in infections in non-endemic areas.[1] Therefore, doctors in non-endemic areas need to be familiar with the clinical features of this disease (fever, chills,[2] headache, myalgia, nausea, vomitting, cough,[3] malarial paroxysm, migraine, urticarial rashes, bradycardia, postural hypotension,[4] jaundice, cerebral involvement, anaemia, thrombocytopenia, pancytopenia,[4] splenic rupture due to splenomegaly[5]), as diagnosis may not be straightforward and malaria presents in many atypical ways. Firstly, it is important to take a travel history. Secondly, symptoms and illness from *vivax* malaria may present many months after travel and exposure, possibly as a relapse from an earlier mild infection.[6] Thirdly, malaria may cause abdominal pain.[7] This patient presented with all the clinical features of localised peritonitis with concomitant dehydration and hypotension, and was being considered for an exploratory laparotomy. The diagnosis was made on the smear obtained for full blood

count. The patient recovered rapidly with malaria treatment, and all symptoms and signs resolved within days. The hypothesis behind this case report is that prolonged incubation period, relapse after months or years after the primary infection, coexistence of *P vivax* and *P falciparum* with *P vivax* suppressing *P falciparum,* and sequestration in gut and visceral ischaemia may cause intense abdominal pain.

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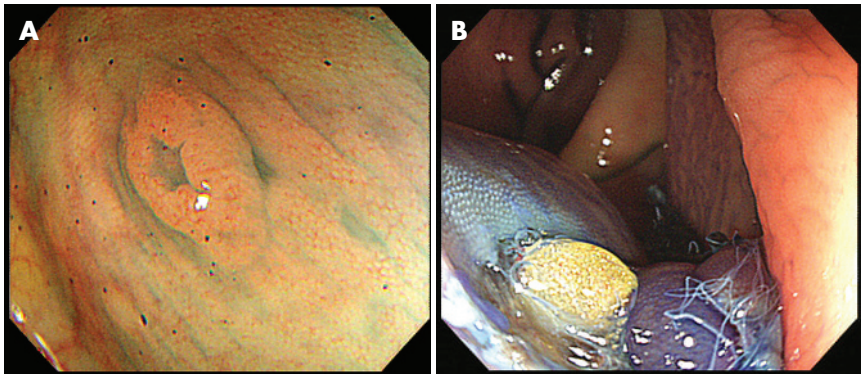

Figure 1 (A) Colonoscopy after application of 0.2% indigo carmine dye showed a diminutive Paris type 0-IIc neoplastic lesion, 1 mm diameter, in the ascending colon. (B) Colonoscopy showed the yellowish substance, which looked like a sponge and was thinner, and less yellowish than adipose tissue in the serosa, floating in the ulcer created by endoscopic mucosal resection.

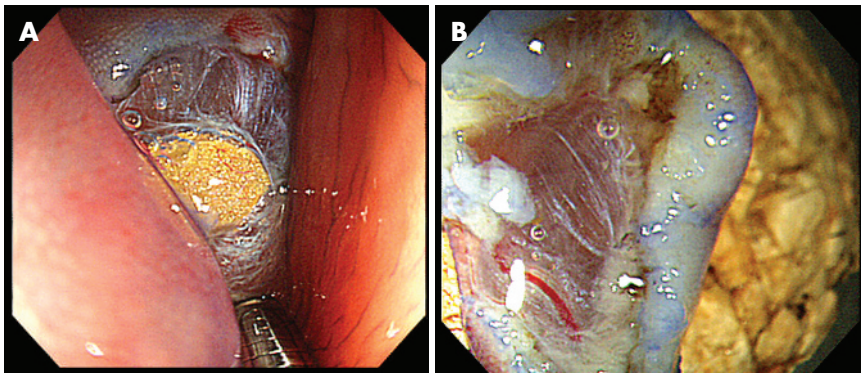

Figure 2 (A) Colonoscopy showed the yellowish substance, which looked like a sponge and was thinner, and less yellowish than adipose tissue in the serosa, floating in the ulcer created by endoscopic mucosal resection. (B) Macroscopically, the endoscopic picture showed that the muscle layer was not included in the vertical margin of the resected specimen.

Submucosal xanthachromia after endoscopic mucosal resection: laparotomy or conservative therapy?

A screening colonoscopy was performed on an asymptomatic 68-year-old woman. A diminutive 1 mm diameter Paris-type 0-IIc neoplastic lesion was diagnosed in the ascending colon (fig 1A). Further characterisation using high-magnification chromoscopic colonoscopy and 0.05% crystal violet intravital staining revealed a Kudo-type IIIs crypt architecture in the depressed component, which suggested that this lesion was limited to the mucosal layer. Endoscopic mucosal resection (EMR) was considered to be the most appropriate firstline endoluminal treatment in this case to confirm histologically the absence of neoplastic disease beyond 1000 μm in the vertical margin, where data from both Japan and Europe have shown that despite diminutive endoluminal appearances, such type 0-IIc lesions have an increased potential for local nodal disease (8–10%) in the right colon.[1 2] Further management—that is, continued endoscopic surveillance or progression to surgical resection—is therefore dependent on the "gold standard" histopathological assessment. Hot biopsy, which is a primarily ablative technique, is unable to provide this mandatory information.

The lesion was raised using a submucosal injection of normal saline solution with no evidence of non-lifting or asymmetry, and resected en bloc endoscopically. The patient reported no abdominal pain during the resection. However, a xanthachromic substance mimicking adipose tissue was observed at the vertical resection margin (figs 1B and 2A). This tissue appeared endoscopically distinct from serosal adipose tissue, diagnostic of post-EMR transmural perforation. Furthermore, the muscularis propria was not represented in the resected lesion that histologically revealed a tubular adenoma with moderate atypia only (fig 2B). No free air was present on a chest and abdominal CT. The patient was subsequenty managed conservatively without any late adverse outcome.

During EMR, transmural perforation can occur when the muscularis propria becomes entrapped below the vertical cut margin of the snare. Clinically, patients often report transient abdominal pain during resection, and subsequently a definite perforation can be identified endoscopically.[3] Furthermore, serosal adipose tissue may be directly visualised after EMR, diagnostic of transmural perforation. However, xanthachromic submucosal adiposity can also be observed after EMR, and although difficult to distinguish endoscopically from serosal fat represents a "normal" submucosal variant, particularly prominent in the proximal ascending colon and ileocaecal valve (colonic lipohyperplasia).[4] Given the increased use of EMR for the treatment of Paris type 0-II neoplastic lesions, this case serves to emphasise the importance of normal anatomical variance after resection. Submucosal xanthachromia after EMR should not be confused with the direct visualisation endoscopic appearance of serosal adipose tissue. Differentiation of these two distinct post-EMR appearances is essential to avoid misdiagnosis of perforation leading to unnecessary laparotomy.[5]

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