The acute medical abdomen: Henoch–Schönlein purpura presenting with peritonitis and nephritis

Introduction

Henoch–Schönlein purpura is an immunoglobulin A-mediated small vessel vasculitis. Although predominantly a disease of childhood (Lawee, 2008), as this case demonstrates it is an important differential for the acute abdomen in the adult.

Discussion

Although the aetiology is unknown, Henoch–Schönlein purpura typically occurs in the autumn or winter months and is often preceded by an upper respiratory tract infection (Saulsbury, 2002; Brogan, 2007). It classically presents with palpable purpura, abdominal pain and arthritis, and although this triad of symptoms is rarely seen in any other condition, they can be variably present making the diagnosis difficult. Moreover, adults tend to develop a more atypical and severe clinical syndrome (Lawee, 2008).

Figure 1. Plain radiograph of the abdomen showing no evidence of bowel obstruction and reduced bowel gas pattern.

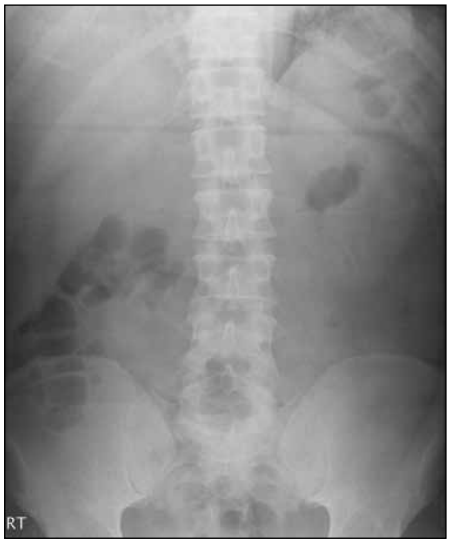


Figure 2. Computed tomogram of the abdomen demonstrating thickening and inflammation of the jejunum and congestion of the small bowel mucosa.

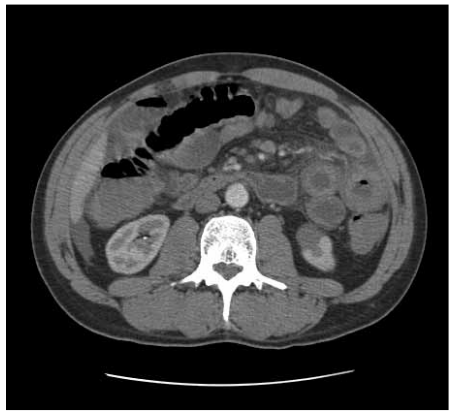
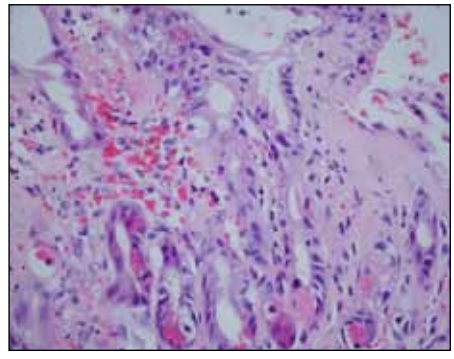


Figure 3. Jejunal biopsy under high power demonstrating ischaemic damage to the small intestinal mucosa.



Case Report

A 41-year-old man presented with a 6-day history of worsening generalized, colicky abdominal pain associated with fever, non-bloody diarrhoea, vomiting and joint swelling. This was preceded by an upper respiratory tract infection for which he was prescribed antibiotics. On examination, the patient was febrile with generalized abdominal tenderness and peritonitis localized to the right iliac fossa. Investigations revealed an elevated white cell count of 14.0×10^9/litre (neutrophils 11.0×10^9/litre) and a C-reactive protein of 120 mg/litre. Abdominal X-ray (*Figure 1*) showed a decreased bowel gas pattern. An initial diagnosis of antibiotic-related colitis was made.

The patient's symptoms did not resolve and computed tomography scanning of the abdomen was performed and revealed focal jejunal thickening with associated mesenteric inflammation and free fluid within the pelvis (*Figure 2*). This suggested either inflammatory bowel disease or focal bowel ischaemia, and since the clinical picture favoured the former, broad spectrum intravenous antibiotics and steroids were commenced.

The patient later developed generalized peritonitis and underwent laparotomy. At surgery, a large amount of serosanguinous free fluid was found, a 30 cm segment of jejunum was thickened and inflamed and the small bowel mucosa was congested. The abdominal cavity was washed out and a jejunal biopsy was performed which demonstrated vasculitis (*Figure 3*).

Two days post-laparotomy the patient developed a polyarthritis and a purpuric rash over the limbs. Blood tests revealed an erythrocyte sedimentation rate of 65 mm/hour; rheumatoid factor, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, complement C3 and C4 levels were unremarkable. The rash and arthritis improved, but 2 weeks later the patient developed macroscopic haematuria. Urinalysis demonstrated proteinuria; serum albumin was 20 g/litre and serum urea and creatinine were 8.4 mmol/litre and 135 μmol/litre respectively. A 24-hour urine collection showed a protein loss of 0.5 g/24 hours and a creatinine clearance of 53 ml/min. Core biopsy of the renal cortex and medulla revealed focal, segmental proliferative glomerulonephritis, consistent with an immunoglobulin A nephropathy. The patient was managed with an angiotensin-converting enzyme inhibitor, aspirin, furosemide, prednisolone and mycophenolate and made a full recovery.

Ms Kaji Sritharan *is Specialist Registrar in Vascular Surgery, St Thomas' Hospital, London SE1 7EH,* **Dr Ahmed Karim** *is FY2 in the Department of Accident and Emergency, Royal Free Hospital, London,* **Ms Samia Ijaz** *is Specialist Registrar in Colorectal Surgery on the Northwest Thames Surgical Rotation, London and* **Mr Sebastian Raymond** *is Consultant General Surgeon, Hemel Hempstead NHS Trust, Hemel Hempstead*

Correspondence to: Ms K Sritharan

While rash is the presenting symptom in most (83%) adult cases, abdominal pain and arthritis can precede its development (Pillebout et al, 2002). Importantly, the former may mimic an acute abdomen, leading to unnecessary explorative laparotomy, and approximately 6% of patients will develop gastrointestinal complications, such as intussusception, gastrointestinal bleeding, bowel ischaemia and perforation, necessitating surgery (Cull et al, 1990; Katz et al, 1991). Overall 1% of adults with Henoch–Schönlein purpura will go on to develop chronic kidney disease and since this can develop up to 4 months after the initial presentation it is essential to follow up all patients (Pillebout et al, 2002).

Conclusions

Henoch–Schönlein purpura-related peritonitis and nephritis in the adult is rare, but the former may lead to unnecessary laparotomy and the latter is associated with considerable morbidity. Henoch–Schönlein purpura should therefore be actively considered as a diagnosis in all patients where the presentation of an acute abdomen is atypical. BJHM

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Tuberculous psoas abscess mimicking soft tissue tumour

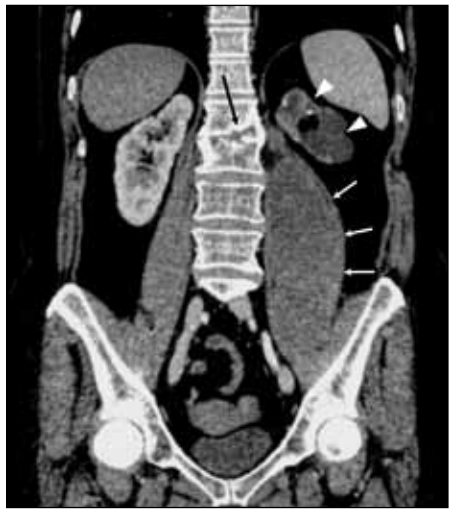
A 62-year-old Caucasian school dinner lady presented to her family doctor with a 2-year history of recurrent urinary tract infections. She was otherwise well, with no significant past medical history. Full blood count, urea and electrolytes, and bone biochemistry were normal. Urine culture was sterile. An abdominal ultrasound scan detected a left-sided mass in the iliolumbar region and scarring of the left kidney. The patient was referred to the authors' sarcoma unit with a suspected retroperitoneal soft tissue tumour causing obstructive renal damage.

A computed tomography scan of the trunk demonstrated a large cystic mass within the left psoas muscle. The left kidney was atrophied, with parenchymal calcification and cystic change, although it was not hydronephrotic. Additional computed tomography findings included mediastinal lymph node calcification and destruction of the L1/2 intervertebral disc with partial collapse of adjacent vertebral bodies (*Figures 1* and *2*). These radiological signs suggested a unifying diagnosis of multi-focal tuberculosis, with a psoas abscess and involvement of the left kidney and spine. The diagnosis was confirmed upon aspiration of pus from the psoas mass, which yielded a growth of acid-fast bacilli. The patient was commenced on anti-tuberculous medication and is currently well on dual agent maintenance therapy.

Dr Nevin T Wijesekera *is Specialist Registrar in Radiology,* **Dr Angela M Riddell** *is Consultant Radiologist and* **Dr Eleanor Moskovic** *is Consultant Radiologist in the Department of Radiology, The Royal Marsden Hospital, London SW3 6JJ*

Correspondence to: Dr NT Wijesekera

Figure 1. Computed tomography scan showing left psoas abscess (white arrows), atrophic left kidney (arrowheads) and destroyed L1/2 intervertebral disc (black arrow).



Discussion

Chronic tuberculosis can be difficult to diagnose as presenting lesions may mimic other pathologies leading to delays in treatment. This case highlights the importance of considering tuberculosis even in patients not traditionally thought to be at high risk. BJHM

Figure 2. Computed tomography scan showing calcified mediastinal lymph node (arrow).