A 73-year-old woman presented to the emergency department with progression of pain in the lower abdomen. The pain developed two months earlier, following pneumonia treated with antibiotics. Initially, it was accompanied by a short episode of diarrhoea and vomiting, which spontaneously resolved. She had a documented history of rheumatoid arthritis, diabetes mellitus type 2, hypertension, hypercholesterolaemia, myocardial infarction and cholecystectomy.

On admission there were no other abdominal symptoms. She was only having one bowel motion a day with lactulose intake. Her daily oral food intake was normal and there was no history of weight loss. Her rheumatoid arthritis has been stable for years. She stopped smoking more than 20 years ago. Current medication consisted of insulin, prednisone, a depot methylprednisolone every ten weeks, nitrazepam, acetylsalicylic acid, diclofenac, omeprazol, bumetanide, metroprolol, folic acid and lactulose. Physical examination revealed a moderately ill patient with a blood pressure of 140/80 mmHg, a pulse rate of 85 beats/min and a body temperature of 37°C. On auscultation active bowel sounds were heard. On palpation a localised tenderness in the lower abdomen, somewhat more pronounced in the right lower quadrant, was found. Blood tests revealed 17.2 x 10^9 leucocytes (88% neutrophils, 8% monocytes, 4% monocytes), CRP 144 mg/l, creatinine 69 µmol/l, alkaline phosphate 146 U/l, ASAT 11 U/l, ALAT 22 U/l, γGT 25 U/l, amylase 63 U/l, lactate 1.8 mmol/l and glucose 15.1 mmol/l. The urine examination was normal, except for glucosuria. The gynaecological examination was normal. A contrast-enhanced CT scan of the abdomen is presented in figures 1 and 2.

WHAT IS YOUR DIAGNOSIS?

See page 285 for the answer to this photo quiz.

Figure 1 A transverse contrast-enhanced abdominal CT scan of the patient

Figure 2 Coronal reconstruction of the abdominal CT scan
The abdominal contrast-enhanced CT scan shows mild diverticulosis of the sigmoid (solid arrows, figure 1), with infiltration of the surrounding mesenteric fat (arrowheads, figure 1) extending along the inferior mesenteric vein (open arrow, figure 1). The wall of the inferior mesenteric vein is thickened and the lumen shows filling defects (arrowheads, figure 2) consistent with thrombosis. The splenic and portal veins are open, and there are no signs of appendicitis. These findings are very consistent with a thrombophlebitis of the inferior mesenteric vein complicating a mild sigmoid diverticulitis. Thrombotic events of the mesenteric veins lack specific clinical symptoms and laboratory data. Mesenteric vein thrombosis is diagnosed in 5 to 15% of all mesenteric ischaemic events,1 usually in the superior mesenteric vein. In about 75% of the patients it occurs secondary to abdominal inflammation, cancer, coagulation disorders, recent abdominal surgery, or cirrhosis in portal hypertension.1,2 Thrombophlebitis of the inferior mesenteric vein secondary to diverticulitis occurs infrequently,3 and may be complicated by sepsis and intrahepatic abscesses. CT imaging helps to diagnose this complication at an early stage and can significantly improve the previously reported high mortality and morbidity rates associated with this condition. Conservative therapy with antibiotics, which target Gram-negative bacilli, anaerobes and enterococci, can lead to resolution of the thrombosis.4 Although the effect remains controversial, in most cases anticoagulant therapy is started. Elective surgery may be performed to eradicate the primary inflammatory process when antibiotic therapy fails. Immediately after admission, this patient was started on anticoagulant therapy. Because the abdominal pain persisted and the CRP concentration rose to 246 mg/ml, intravenous antibiotic therapy was started within 48 hours, resulting in a clinical improvement. Blood cultures obtained on the first and second day of admission were negative. After one week the antibiotics amoxicillin/clavulanic acid were continued orally and the patient was discharged. At follow-up the patient had made a full recovery, laboratory parameters normalised, and the CT scan no longer showed abnormalities.

REFERENCES