A 58-year-old woman with a history of type-2 diabetes, hypertension, and myocardial infarction, presented with acute onset of severe continuous pain of her right lower and upper abdomen, and vomiting. The pain started at 3 o’clock in the night, during sleep. Physical examination revealed a moderately obese woman with a normal pulse, blood pressure and body temperature. There was tenderness of the right upper abdomen and costovertebral angle. Blood and urinalysis revealed no abnormalities except an elevated C-reactive protein (CRP) of 12 mg/l (normal <10 mg/l). An echography of the abdomen and a nonenhanced CT scan (figure 1) were normal. The following day the pain increased and blood analysis showed a white blood cell count of 13.3 x 10⁹/l and a lactate dehydrogenase (LDH) of 494 U/l (normal 110 to 200 U/l). CRP 17.3 mg/l. Urinalysis demonstrated microscopic haematuria. The surgical consultant, unaware of the first CT scan, ordered a contrast-enhanced CT scan (figure 2).

**WHAT IS YOUR DIAGNOSIS?**

See page 83 for the answer to this photo quiz.
The contrast-enhanced CT scan revealed a wedge-shaped perfusion defect in the lower pole of the right kidney (arrow, figure 2), consistent with a renal infarction. The patient was treated with heparin and acenocoumarol. The pain resolved after one week.

Acute renal infarction is often an overlooked or delayed diagnosis.1-4 Because its presentation is nonspecific, it is frequently mistaken for more commonly encountered diseases such as ureterolithiasis, pyelonephritis, appendicitis, diverticulitis, biliary obstruction and torsion of pelvic masses. Patients with acute renal infarction typically present with nonspecific symptoms of acute onset of low back pain, abdominal pain or flank pain with nausea, vomiting, hypertension and sometimes fever. Physical examination reveals costovertebral angle tenderness, which is a characteristic sign of acute renal infarction. About 90% of patients have increased LDH levels. Other common laboratory findings include leukocytosis, haematuria and proteinuria. Aspartate aminotransferase, alkaline phosphatase and CRP are also useful markers, but less specific.4 These laboratory abnormalities may be absent in case of very early presentation after onset of the pain.4 The major causes of an acute renal infarction are atrial fibrillation, and valvular or ischaemic heart disease. Additionally, other causes of acute renal infarction include trauma, hereditary or acquired clotting disorders, both intravenous and nasal cocaine abuse,4 vessel anomalies, medical interventions such as surgery for valve replacements, kidney transplantation, endovascular catheterisation and application of intraluminal stents, sickle cell disease or sickle cell trait and malignant disease.4 None of these diseases were observed in this patient and a magnetic resonance angiogram did not show arterial vessel disease.

Spiral (helical) computed tomography without contrast is usually the preferred initial test for flank pain, being the imaging technique of choice for the diagnosis of kidney and ureteral stones, which are more common than renal infarction. However, contrast enhancement is required when unenhanced CT reveals no abnormalities because the diagnosis renal infarction will be missed as happened in this case. The triad of acute flank pain, high serum LDH and microscopic haematuria should make the physician aware of a possible renal infarction.

REFERENCES