A 78-year-old man was admitted to our ICU for supportive care with a diagnosis of hypovolaemic shock due to bleeding. His medical history revealed insulin dependent diabetes mellitus (IDDM) with retinopathy, polyneuropathy and nephropathy (creatinine clearance 40 ml/min), cerebrovascular accident, hypertension and upper digestive tract bleeding. Recently he had undergone a supragenual amputation following femorocrural bypass surgery of the right leg. On physical examination the patient was in shock with a blood pressure of 90/40 mm Hg, heart rate 80/min (on Beta-blockade). Heart and lung examination was normal. Abdomen: few bowel sounds, painful but not tensed, normal percussion of spleen and liver. The wounds on his right leg looked clean. Laboratory results showed a metabolic acidosis with a slightly raised lactate (3.3 mmol/L). The haemoglobin level was 3.2 mmol/L, coagulation times were prolonged: APTT 64 and INR 1.62. Creatinine level had risen from 93 to 140 micromol/L. The patient was intubated and ventilated, arterial and central venous catheters were placed. Low-dose norepinephrine and four units of red blood cells were given. No bleeding-focus was found on gastroscopy and coloscopy. Abdominal ultrasound revealed an irregular structure dorsally and caudally of the left kidney. Views were poor because of adiposity and bowel gas.

A CT-scan of the abdomen revealed an inhomogeneous collection with different intensities from the left kidney to the left psoas muscle. The left psoas muscle was swollen. A psoas bleed with a retroperitoneal haematoma was diagnosed.

The hypovolaemic shock in this patient was due to a spontaneous bleed in the psoas muscle. The patient had been treated on the surgical ward with therapeutic doses of nadroparin, a low molecular weight heparin (LMWH), to prevent progression of arterial occlusive disease. Since the patient had renal dysfunction, it is possible that the nadroparin had accumulated.

In the literature, bioaccumulation of LMWH appears to be largest in severe renal impairment (creatinine clearance less than 30 ml/min) using therapeutic doses [1]. Until more data is available, periodic monitoring of anti-Xa activity can be recommended to detect accumulation and minimize the risk of bleeding [2]. Over 200 cases of psoas haematoma following anticoagulant treatment are reported in the literature. Ultrasound may establish the diagnosis but can be misleading. CT or MRI is often required to confirm the diagnosis.

Therapeutic options include optimization of coagulation, surgery, percutaneous drainage and embolization. In femoral neuropathy secondary to psoas haematoma, surgical evacuation will provide a quicker and more complete recovery from femoral paralysis [3]. In this case, discontinuation of nadroparin therapy together with optimization of haemoglobin level stabilized the patient. No further interventions were needed and his recovery was uneventful.

References