0011 Adrenal gland infarction as the first manifestation of systemic lupus erythematosus

Halil Yildiz[1], Chantal Lefebvre[1]

[1]cliniques universitaires St Luc, Brussels, Belgium

A 29-year-old woman presented for severe lumbar and left flank pain since 2 days. She feel tired and noticed a weight gain (10 kg) in 6 months. She had no past medical or family history. Vital signs were normal.

Laboratory investigations were the following: CRP level at 75 mg/L; neutrophile count at 8890/µL; LDH level at 317 IU/L, creatinine level at 1.5 mg/ dL, estimated glomerular filtration rate (eGFR) 50 mL/min/1.73 m, platelet count at 116 000/mm^3 and serum albumin 15 g/L.

A contrast-enhanced computed tomography (CT) of the abdomen (Figure 1A) was performed and interpreted as normal. Unfortunately, the pain rose up and fever (38.2 °C) appeared. Therefore, five days later, a second abdominal CT scan was performed and showed unilateral left adrenal gland hemorrhage (Figure 1B). A new lecture of the first scan showed unilateral left adrenal gland infarction.

Investigations revealed significant proteinuria (6.7 g/L), a positive ANA (titre 1/1260, homogeneous pattern), low complement C3 and C4 at 84 mg/ mL and 12 mg/mL (0.84 g/L and 0.12 g/L), respectively, elevated double-stranded DNA antibodies at 81 units Thrombophilia screening including antiphospholipid antibodies was normal. A kidney biopsy was performed and was highly suggestive of lupus glomerulonephritis. A treatment with steroids and mycophenolate mofetil was began and showed a rapid improvement. After 8 months of follow up, the patient goes well and blood tests completely normalized.

Literature key-points: Adrenal gland infarction is rare. Pathogenesis is likely due to microvascular or main adrenal venous thrombosis in procoagulable states and during episodes of hypotension, and may lead to infarction. Usually this may be complicated by hemorrhage due to ischemic necrosis or reperfusion injury. On CT-scan,[1] adrenal infarction showed thickened, hypodense poorly enhancing infarcted adrenal glands. In the other hand, adrenal hemorrhage characteristically appear round or oval, with surrounding stranding of the periadrenal fat evident.

Adrenal infarction due to hypercoagulable states has been best described in the setting of antiphospholipid-antibody syndrome[2] but also in condition like heparin-induced thrombocytopenia,[3] nephrotic syndrome (like in our case) and myeloproliferative disorders.[4]

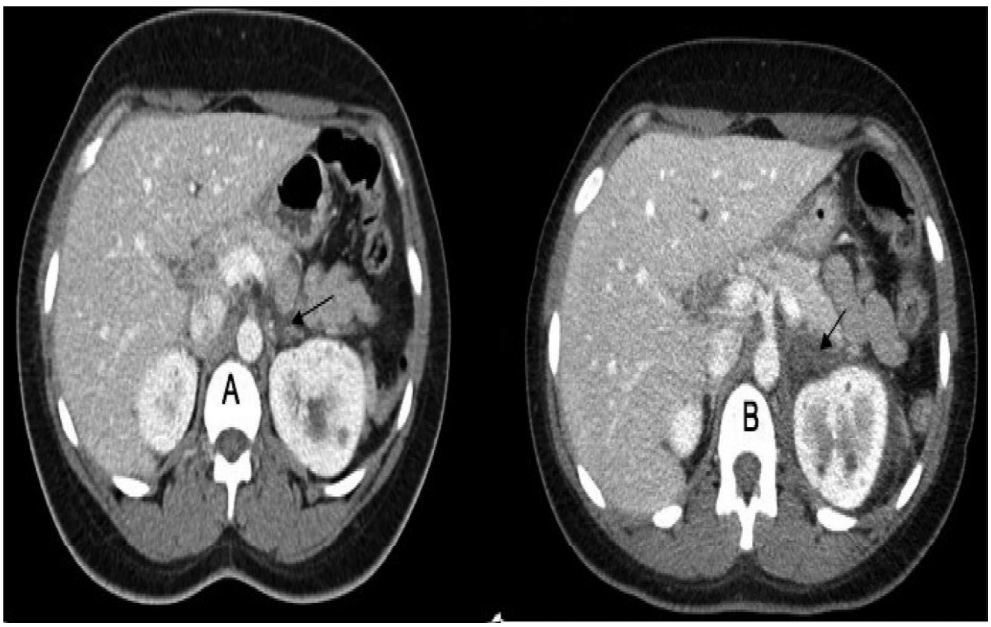

Figure 1A showing adrenal gland infarction (arrow) and Figure 1B showing adrenal gland hemorrhage (arrow) 5 days later.

References

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