

Endoleak-induced DIC presenting as massive chest wall haematoma in a patient on dual antiplatelet therapy

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DESCRIPTION

A 76-year-old man was admitted complaining of fatigue/lassitude over 1 week and the appearance of a mass on his chest. Pallor, tachycardia, scattered superficial haematomas and a large tender mass over his right lateral chest were found. The ECG and chest X-ray were unremarkable.

He had a long history of hypertension, heavy smoking with chronic obstructive pulmonary disease (COPD) and extensive atherosclerotic cardiovascular disease (ASCVD) involving the coronary arteries (myocardial infarction and stenting 20 years prior) and aorta (endovascular repair of thoraco-abdominal and abdominal aneurysms and femoro-femoral bypass for thrombosed left iliac artery 10 years prior). He was being treated with valsartan, bisoprolol, atorvastatin and dual antiplatelet therapy (DAPT): aspirin (100 mg/day) and clopidogrel (75 mg/day).

Haemoglobin (Hb) was 54 g/L (mean corpuscular volume (MCV) 79, red cell distribution width (RDW) 19.5%, reticulocytes 5%), white cell count $15 \times 10^9/L$, platelets $90 \times 10^9/L$, then $77 \times 10^9/L$, with prolonged prothrombin time (PT) (17 s) and activated partial thromboplastin time (aPTT) (52.6 s), decreased fibrinogen (113 mg/dL, then 89 mg/dL, N 200–400) and D-dimer $>20\,000$ ng/mL ($n < 500$). Blood smear showed thrombocytopenia and few schistocytes. Blood urea nitrogen (BUN) was 38 mg/dL, creatinine 1.5 mg/dL. Ferritin was increased (475 mg/dL), while urinalysis, haptoglobin, lactate dehydrogenase (LDH) and liver enzymes and function were normal. Recently, Hb was 101 g/L, creatinine 1.1 mg/dL. There was no history of trauma or melaena. Nasogastric tube fluid and rectal examination were normal.

Chest and abdominal CT revealed a massive subcutaneous chest haematoma (figure 1A), and extensive repaired thoraco-abdominal aneurysms with large thrombus and endoleaks into enlarged aneurysm sacs (figure 1B–D). DAPT was stopped. Four units of packed red blood cells and platelet transfusions were given, with cryoprecipitate and intravenous hexacapon. He readily stabilised and was referred to vascular surgery for further treatment of the aneurysms.

DAPT inhibits platelet function by different complementary mechanisms and effectively prevents thrombosis after stent insertion, at the price of up to 2%–4% major bleeding at 12–24 months.¹ Our patient's DAPT was prolonged beyond current indications (usually, 12 months), but well tolerated

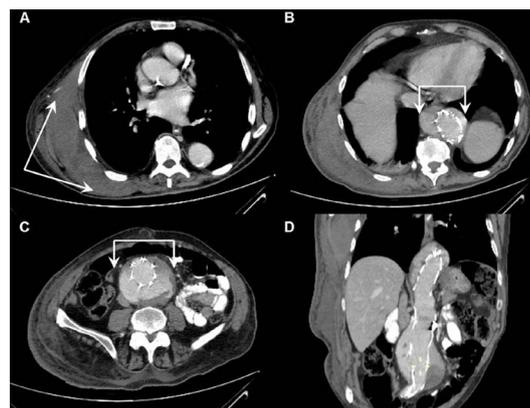


Figure 1 Chest wall haematoma and thoracoabdominal aortic aneurysm with endoleak. Contrast enhanced CT images of the chest and the abdomen show an extensive right chest wall haematoma (A, arrows) and large thoracoabdominal aortic aneurysm (B and C, arrows). The extent of the stented aneurysm is demonstrated on the oblique coronal reformat (D). Contrast is noted in the partially thrombosed enlarged aneurysmal sac, indicating the presence of an endoleak.

until his admission. While gastrointestinal, urinary tract and intracranial bleeding are well-known DAPT adverse effects, DAPT-associated bleeding can also occur at unusual sites.² Retroperitoneal, perirenal, omental, rectus sheath, spinal (extradural, epidural), lung and liver haematomas were described in isolated reports, and spontaneous soft tissue haematomas can also rarely occur.²

Here, in addition to DAPT which had been taken uneventfully long-term, a new player on the field tipped the scales causing severe bleeding. Laboratory studies indicated that significant disseminated intravascular coagulation (DIC) developed (International Society of Thrombosis and Haemostasis (ISTH) score 6), with conspicuous absence of all its usual causes.³ DIC is a rare but well-established complication of aortic aneurysms.^{4,5} DIC-associated bleeding may even be an unusual *presenting* symptom of a hitherto unrecognised aneurysm.⁶ Intriguingly, the development of leakage into the excluded aneurysm sacs,⁷ so-called endoleak, with expanding aneurysmal sac as seen in our patient (figure 1) could have triggered his DIC. Endoleak-induced DIC is an exceedingly rare complication, reported in ~10 patients, to the best of our knowledge,⁸ and not listed among the possible aetiology of DIC.³ This association needs to be better recognised since it can be the initial and *presenting*



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Learning points

- ▶ Clinically overt disseminated intravascular coagulation can be the presenting symptom of an aortic aneurysm, but also of the development of endoleak in repaired aneurysms.
- ▶ Dual antiplatelet therapy can be associated not only with gastrointestinal, urinary tract and intracranial bleeding but also with bleeding at unusual sites, such as a massive subcutaneous haematoma.
- ▶ In a patient who has bled, iatrogenic causes must be carefully evaluated and more than one such cause may occur concurrently.

manifestation of an endoleak, as our patient demonstrates. It has been proposed that turbulent flow liberates coagulated material from the aortic sac, exposing denuded endothelium and tissue factor, leading to activation of coagulation factors, excess generation of thrombin, chronic consumption of clotting factors and simultaneous excess plasmin generation and fibrinolysis of the clots.

Thus, endoleak-induced DIC combined with DAPT was associated with massive unusual subcutaneous bleeding and severe anaemia. It led to spotting DAPT treatment that was prolonged beyond current indications, as well as to the diagnosis of previously unsuspected endoleaks in repaired aortic aneurysms.

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