Cholesterol Crystal Embolism Presenting as AKI and Pancreatitis - Case Report -

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Background:
Cholesterol crystal embolism (CCE), also known as cholesterol embolization syndrome, refers to arterio-arterial embolism of cholesterol crystals or small pieces of atheromatous material from atherosclerotic plaque, usually from the aorta but occasionally from other arteries. This can result in partial or total occlusion of small arteries, leading to tissue or organ ischaemia.

Case Report:
A 64-year-old English gentleman presented with severe back pain of few weeks duration. He had high BMI but no other medical co-morbidities and had never smoked in his life. He was found to be severely hypertensive with a systolic blood pressure above 240 mmHg. Investigations revealed a severe stage III, oligo-anuric, AKI requiring renal replacement therapy and severe thrombocytopenia with a blood film showing a few red cell fragments. He had a raised troponin consistent with a myocardial event, and his amylase and lipase were also elevated, leading to a clinical diagnosis of pancreatitis. Renal US showed un-obstructed normal sized kidneys. Subsequent CT imaging, performed to exclude aortic dissection, revealed widespread atheromatous disease of the entire aorta and iliac arteries, enlarged mediastinal and abdominal/pelvic lymphadenopathy in keeping with malignancy, and subtle sclerotic bone lesions. A PSA done in context of these findings came back at 604 ug/l leading to a diagnosis of metastatic prostate cancer, for which hormonal therapy was started. Based on his initial findings, a working diagnosis of TTP was made, and he commenced plasma exchange therapy. However, his ADAMTS13 came back normal, and so the diagnosis was revised to TTP secondary to pancreatitis or paraneoplastic as a result of prostate cancer. With this in mind, his PLEX was discontinued, and following this and with control of his blood pressure, his thrombocytopenia resolved. However, he remained dialysis-dependent. A renal biopsy was initially delayed by a significant upper gastrointestinal bleed with endoscopy showing diffuse erosive duodenitis and a bleeding ulcer. Once this had settled a subsequent renal biopsy showed cholesterol emboli with acute and subacute tubular injury, and importantly, no thrombotic microangiopathy. Unfortunately, he remains dialysis dependent with no recovery.

Discussion:
This case is in keeping with CCE. Our patient has extensive atheromatous disease, and CCE is known to present with thrombocytopenia and end-organ damage; in particular AKI and pancreatitis (which explains the back pain) are both recognised. Upper gastrointestinal bleeds are also common and are often secondary to infarction resulting from occlusion of small vessels. His undiagnosed hypertension is a risk factor for atherosclerosis and in this presentation has been exacerbated by his back pain. Obesity is another risk factor. The prognosis correlates with the degree of the underlying atherosclerosis and is overall poor. The prostate cancer here is likely to be an incidental diagnosis which has been picked up at the same time.

Conclusion:
CCE typically happens following arteriography, cardiac catheterisation, vascular surgery, trauma to the abdomen or over anti-coagulation. The diagnosis was more difficult in our patient as he did not have any of these prior to presentation. Lab testing is generally nonspecific and definitive diagnosis depends upon pathologic specimens.