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# P428 -A hidden cause of pulmonary oedema revealed by diagnostic persistence and teamwork.

Doctor Stephanie Urwin[1], Doctor Paul Walker[1], Doctor Ahmed Kaabneh[1], Doctor Neil Alexander Hoye[1], Doctor Jonathan Stephen Murray[1]
[1]*South Tees Hospitals NHS Foundation Trust, Middlesbrough, United Kingdom*

Presentation and Background: A 64 year old lady presented to hospital with acute breathlessness. She had a long history of end-stage kidney failure due to chronic glomerulonephritis (aged 20). She had received a third kidney transplant 6 years earlier and her serum creatinine was noted to be elevated (291 µmol/l) from baseline (150 µmol/l). She reported passing less urine and her chest x-ray demonstrated pulmonary congestion. Her blood pressure was noted to be relatively high compared to clinic readings. She did not respond to high dose diuretics and soon required dialysis to manage pulmonary oedema associated with worsening acute kidney injury (AKI, peak creatinine 526 µmol/l).

Two years earlier she had a similar presentation, though she had responded to medical therapy. Of note a doppler ultrasound scan to assess for transplant renal artery stenosis (TRAS) had been negative at that stage.

Investigations: Although TRAS was not demonstrated 2 years earlier, the current clinical presentation led to this diagnosis being reconsidered; once again imaging (this time CT angiogram, figure 1) did not demonstrate TRAS. Because the clinical picture was so compelling and the patient was seemingly dialysis-dependent, however, the renal and radiology teams further reviewed her CT angiogram. This collaborative reassessment driven by clinical context concluded that, due to surgery-related artefacts at the site of the kidney transplant (arrow, figure 1), it was difficult to definitively exclude vessel stenosis close to the origin of the transplant renal artery on this scan. A subsequent intra-arterial angiogram revealed a culprit stenosis in the external iliac artery (arrow, figure 2) near to the transplant renal artery anastomosis.

Management and outcome: Angioplasty of the iliac artery stenosis precipitated a prompt improvement of the patients symptoms, associated with an improvement in her urine output and kidney function (discharge serum creatinine 115 µmol/l, well below her baseline). When her symptoms and AKI recurred again 2 months later, imaging indicated the stenosis had recurred - further angioplasty with stenting (arrow figure 3) has provided definitive treatment for the patient to date.

Discussion and learning points: Reported prevalence of TRAS varies between 1 and 23%[1]. This range likely reflects its different clinical manifestations and various imaging modalities employed. Non-functionally significant TRAS may be an incidental finding, though it typically presents with post-transplant hypertension and can cause flash pulmonary oedema and acute transplant dysfunction - as the case presented highlights. TRAS typically occurs 3 months to 2 years post transplantation[2] when risk factors include cytomegalovirus infection[3] and surgical factors[4]. As the case presented illustrates, however, clinicians should remain vigilant for TRAS as it can present several years post-transplant, when it may reflect atherosclerotic disease of the transplant renal artery or the adjacent iliac artery[5]. The presented case also underlines the importance of collaborative working between renal and radiology teams, incorporating careful discussion of clinical context and index of suspicion; this justified more invasive imaging which "revealed" a unifying diagnosis that was effectively "hidden" on a preceding scan - and enabled effective treatment for our patient.