Renal infarction is an uncommon condition of which the true incidence remains unclear as studies in live populations are limited. It is clinically very difficult to identify and not detected by unenhanced CT imaging favoured for acute flank pain. Four principle aetiological groups have been previously identified; cardiogenic, renovascular injury/disease, hypercoagulable disorders and idiopathic infarction. The diagnosis should prompt investigation for causative pathology as swift and appropriate treatment is associated with good outcomes and a low rate of progression to renal replacement therapy.

We present a series of 5 patients diagnosed with a renal infarction in a UK district general hospital between 2013 and 2017.

* A 55-year-old female who presented with acute pancreatitis and found to have renal infarctions. Further investigations revealed a patent foramen ovale (PFO) and suggested sarcoidosis. This patient was anticoagulated and remains under follow-up.
* A 52-year-old male with breathlessness, acute kidney injury (AKI) and aphasia admitted to intensive care for haemofiltration. As well as a deep vein thrombosis, pulmonary embolism and a left middle cerebral artery territory infarction, his CT abdomen showed left renal infarction. Bubble echocardiography detected a PFO which was closed surgically. This patient has been discharged to GP follow-up with CKD 3.
* A 23-year-old male admitted with right iliac fossa pain and an AKI. A CT scan of the abdomen revealed multiple right renal infarctions. Echocardiography revealed an enlarged, failing left ventricle with a large thrombus. This patient was anticoagulated and remains under renal and cardiology follow-up.
* A 48-year-old female who presented with right-sided upper abdominal and back pain. A CT scan revealed multiple infarctions in the right kidney. This patients’ creatinine peaked at just 74. Bubble echocardiography demonstrated a PFO and lifelong anticoagulation was commenced.
* A 51-year-old female who was admitted with acute pancreatitis whose CT scan incidentally showed bilateral renal infarcts despite a creatinine of just 69. 24-hour ECG and a transthoracic echocardiogram were both normal and it was felt the infarcts were secondary to infected emboli secondary to pancreatitis. This patient was not anticoagulated and remains under follow-up.

The mean creatinine prior to diagnosis was 85 and the mean peak creatinine was 354. All five patients were followed up by the renal team; two have no CKD, two have CKD stage 3 and one has CKD stage 4. 4/5 of our population remain under renal follow-up. None have required long-term renal replacement therapy.

**Conclusion**

Renal infarct has a non-specific clinical picture and thus can present to a variety of specialties. A broader awareness of its presentation, causes and treatment is therefore needed. From our case series we have shown that outcomes are favourable if the condition is recognised and suitably managed. It is notable that three of our patients were found to have PFOs. We propose the development of a common investigation pathway that should be undertaken following the diagnosis of renal infarct. In light of the association with PFO in these patients, we suggest that be considered as part of the pathway.