Septic Renal Infarct



Ref.no.: MP2098

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MEDICAL

Septic Renal Infarct

Clinical History

A 54 year old male patient presents with flank pain. He is an active intravenous drug user. Further questioning reveals a history of intermittent haematuria, fevers, malaise and vomiting. On examination he is hypertensive and pyrexic. Inspection of his limbs reveals Janeway lesions on his extremities and track marks from recent IV drug use. A systolic murmur is found on auscultation of his chest. Blood tests reveal elevated inflammatory markers, impaired renal function, elevated LDH and multiple bacteraemic blood cultures. Echocardiogram shows a large mobile tricuspid vegetation. He was commenced on treatment for infective endocarditis but later died from a sudden cardiac arrest.



Pathology

The specimen is the patient's kidney from post mortem examination. The kidney has been bisected with a cut half surface on display. There are multiple well demarcated wedge shaped pale yellow-white areas evident within the cortex. The base of these pyramids lies against the cortical surface and extend along the renal columns with the apex pointing toward the medulla. The largest is evident lateral upper pole of the kidney. Theses pale areas are infarcted renal tissue. There are dark irregular shaped areas which represent areas of hemorrhage.



Further Information

Renal infarction results from an interruption in the blood flow to the kidney. The kidneys receive almost a quarter of the cardiac output but have limited collateral circulation. The cortex is the most susceptible area to infarction given blood supply is from proximal to distal. The main causes of interruption of this circulation are cardioembolic disease, renal artery damage, hypercoagulable states or idiopathic.

Cardioembolic causes are the most common. These include post-myocardial infarction mural thrombi, septic emboli from infective endocarditis and emboli from mechanical valves. Idiopathic renal infarction is the second most common cause. Damage to the renal artery is the third most frequent cause and includes renal artery dissection, acute vasculitis of polyarteritis nodosa, trauma or post endovascular intervention. Hypercoagulable states are the rarest cause of renal infarcts such as hereditary thrombophilia and antiphospholipid syndrome. Infarction is bilateral in ~15% of cases.

Presentation of renal infarction depends on the underlying etiology. It can be clinically silent. Common manifestations include costo-vertebral angle pain, haematuria, hypertension due to increased renin release, nausea, vomiting and sometimes fever.

Laboratory test used to aid diagnosis include urinalysis for hematuria and serum creatinine levels which may be elevated, especially in bilateral disease. CT abdomen with contrast is the first choice radiological investigation. A wedge-shaped perfusion defect is the classic finding. Treatment varies depending on the cause of the infarction but generally involves supportive therapy and treatment of the underlying pathology.



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