Renal Artery Embolism

Černý J., Petřík A., Uhlířová I.*, Hes K.**

Department of Urology, Nemocnice České Budějovice, a.s.
* Department of General Surgery, Nemocnice Strakonice, a.s.
** Department of Radiology, Nemocnice České Budějovice, a.s.

Case report

Ethiology of renal artery thrombosis and emboli

- 25% cardiac - atrial fibrillation, aortic atheroma, endocarditis
- 16% trauma - fibromuscular dysplasia, vasculitis, arteriosclerosis, aneurysm
- 32% coagulopathy - hereditary thrombophilia, hyperhomocysteinemia, antiphospholipid syndrome
- 27% idiopathic causes

Patient demographics: Woman, 48 years, BMI 25.9, truck driver
Risk factors: heavy smoker
History: no medical event
Medication: no
Lab: WBC 24.9 10³/l, fibrinogen 7.3g/l, LDH 27.3 mkat/l, CRP 160 mmol/l, microscopic haematuria
Symptoms: left flank pain, vomiting, subfebrile
Imaging: US, KUB, IVU, CT, angiography

Follow up:
Angio-CT was performed six weeks after revascularisation, the defect of upper half of left kidney is shown
DMSA renogram showed impaired function of the upper part of left kidney with 25% function three months later, it was due to inferior polar artery supply
Secondary hypertension of Goldblatt’s type or significant renal function deterioration hasn’t been developed during follow up

Conclusions
• Renal ischemia is an under-diagnosed condition
• It is possible to treat and to cure
• Minimally invasive radiological approach is the modality of the first choice
• Interval between the first symptom and revascularisation is not clearly defined
• Delay of 1 to 3 hours could predict better treatment results
• Renal ischemia is considered especially in case of a solitary kidney
• Elevation of lactate dehydrogenase, haematuria, persisting flank pain and high risk of thromboembolic event should indicate to perform enhanced CT

Angiography before intervention
Angiography 24 hours after intervention
Angio-CT 6 weeks after intervention

Further investigations:
Were performed to exclude the most frequent reasons of arterial clots.
- Cardiologic examination including ECG and heart ultrasoundography excluded atrial fibrillation, endocarditis, valvular or ischemic disease
- Haematological examination revealed hypercoagulopathy due to impaired prothrombin function
- Genetics
  - Homozygous mutation of the gene encoding prothrombin (G20210A).
  - Leiden’s mutation was not confirmed

Literature