Traumatic bidirectional renal artery thrombosis.

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Introduction

We present a case of acute blunt traumatic bilateral renal artery thrombosis in the setting of multisystem trauma. Catheter-directed thrombolysis with tissue plasminogen activator afforded kidney salvage with return of glomerular filtration rate to normal values at the time of patient discharge.

With the advent of advances in endovascular techniques and interventional radiology, the treating physician has added very useful and relatively less invasive methods of managing otherwise complex traumatic injuries compared to open surgical techniques. One such application is in the treatment of acute traumatic solid organ injuries, and some of the earliest applications related to the management of blunt spleen and liver injuries. Acute traumatic renal artery occlusion is a rare pattern of kidney injury. Methods of treatment for kidney salvage described in the past included observation, open surgical revascularization, and endovascular techniques of revascularization. Catheter-directed thrombolysis for acute renal artery thrombosis has also been described including endovascular renal artery stent placement combined with catheter-directed thrombolysis. Success rates and kidney salvage rates vary considerably and may be related to the sparse data available with such a rare injury pattern.

This case illustrates the usefulness of interventional radiology with endovascular methods in the treatment of acute traumatic renal artery thrombosis due to blunt trauma.

A 20-year-old male arrived as a level 1 trauma code after being struck by a pick-up truck traveling about 50 mph. Intubation in the emergency department occurred for airway protection and hemodynamic instability. A left chest tube yielded moderate blood egress and bilateral lower extremity deformities with open fractures were present. Findings on computed tomography (CT) imaging included a small left subdural hematoma, a left hem thorax and lung contusion. Bilateral renal infarctions with traumatic renal vascular thrombosis, and a right renal pelvis tear with minimal contrast extravasation was demonstrated. The remaining abdominal viscera were without acute injury. The right renal infarct was extensive compared to the left and involved the majority of the right kidney. A visceral angiogram confirmed bilateral renal infarctions, greater in the right kidney. The thrombus on the right renal artery was large and extending to several interlobar arteries.

A catheter was placed in the right main renal artery for infusion of tissue plasminogen activator (TPA) at 2 mg/hr for four hours after an initial bolus of 8 mg for a total dose of 16 mg. The time from injury to the treatment with TPA was approximately 5 hours. The patient was also placed on systemic heparin drip due to the bilateral nature of the renal artery thrombosis. This decision was made following consultation with a neurosurgeon as the patient also had an acute subdural bleed. A collaborative decision was reached to proceed with the systemic heparin as the risk of bilateral renal ischemia without intervention seemed to outweigh the benefits of withholding the heparin given the non-operative pattern of the subdural bleed. The systemic heparin was also applied at approximately five hours post-injury. During the time of heparin infusion, the patient remained on mechanical ventilation and was monitored closely with one hour neurologic exams for signs of deterioration. A second head CT scan was performed 12 hours post-injury which showed a stable subdural bleed with no progression.

The TPA was stopped at 9 hours post-injury for a total dose of 16 mg and the heparin was to continue with the plan for 24 hours of treatment at which time a follow-up angiogram was to be performed to evaluate renal perfusion. The heparin was stopped prematurely at 18 hours because the patient developed marked hematuria. The follow-up angiogram at that time showed resolution of the clot in the right main renal artery but there was an area of contrast extravasation in the inferior pole of the right kidney. This area of active hemorrhage was then successfully controlled by using microcoil embolization into two segmental arteries. Additionally, the kidney appeared to have restored perfusion. The patient was then managed with prophylactic heparin.

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