# A Case of Spontaneous Bilateral Renal Artery Dissection Following Robotic Surgery

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## CASE PRESENTATION

Our patient is an otherwise well 69-year-old man who re-presented to hospital 2 days following an uncomplicated robotic-assisted laparoscopic prostatectomy (RALP).

Our patient's only significant medical history was ventral ectopic beats for which he was regularly on aspirin and Ménière disease. A transperineal biopsy had revealed Gleason 4 + 3 = 7 prostate cancer, on which basis he had proceeded to RALP with a standard bilateral pelvic lymph node dissection. Baseline blood work prior to surgery revealed normal renal function with urea 6.3 mmol/L and creatinine 95 μmol/L (estimated Glumerular Filtration Rate (eGFR) 70). His prostate-specific antigen was 3.8 ng/mL.

The operation itself was routine and uneventful. The patient was positioned in the modified Z Trandelenburg position with shoulder support, with 25-degree head down, a position we have previously demonstrated to reduce physiological strain.[1] Records of the blood pressure during the case (from invasive and noninvasive sources) did not show any evidence of hemodynamic instability during either the operation or in recovery, with a peak blood pressure of 158/94 mmHg and a low of 85/52 mmHg. The total operative time was 2.5 hours. No abnormalities were observed by any member of the operative team. The initial postoperative recovery was also uneventful, and the patient was discharged home on the second postoperative day as planned.

On the afternoon of the day of discharge, the patient experienced sudden right flank pain lasting for several minutes followed by intense nausea and 2 episodes of vomiting. He called the hospital for advice, and was instructed to re-present for assessment. At the time of representation, he was pain free and no longer vomiting although he remained nauseated.

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## INITIAL INVESTIGATIONS AND DIFFERENTIAL DIAGNOSIS

A baseline set of bloods taken on readmission revealed a number of abnormalities. The electrolyte panel showed acute kidney injury (AKI) with urea 7.6 mmol/L and creatinine 170 μmol/L (eGFR 35). Liver function test showed an isolated elevation in lactate dehydrogenase to 820 U/L. At this time the C-Reactive Protein was 57 mg/L.

Given the nonspecific nature of the presentation, the differential diagnosis at this time was very broad. The most likely diagnosis was felt to be a concomitant gastrointestinal illness, with less likely candidates including a postoperative collection, or injury to a viscous structure. To exclude these later injuries, a contrast computed tomography (CT)-abdomen/pelvis was ordered on postoperative day 3.

As shown in Figure 1, the CT scan did not demonstrate any abdominal collections, and instead showed an infarction of the inter-polar region of the right kidney. However, as the scan had not been performed with an angiographic phase, it was not possible to thoroughly assess the renal artery. Hence, it was felt that the etiology of the infarct was embolic in nature and the patient was commenced on anticoagulation. We noted also the aberrant vascular anatomy, with an accessory artery providing uninterrupted supply to the polar regions of the right kidney.

## WORKUP AND FURTHER INVESTIGATIONS

On the basis of this working diagnosis the cardiology and nephrology teams were involved. A transthoracic echo did not reveal a cardiac thrombus or any other structural heart abnormalities. On the advice of the nephrology team, a CT angiogram was performed to exclude renal artery dissection.

This angiogram, shown in Figure 2, demonstrated a right renal artery dissection. We also noted a dissection on the left side just proximate to the trifurcation of the renal artery into its major tributaries, with some evidence of extension of the dissection into the superior branch. This was

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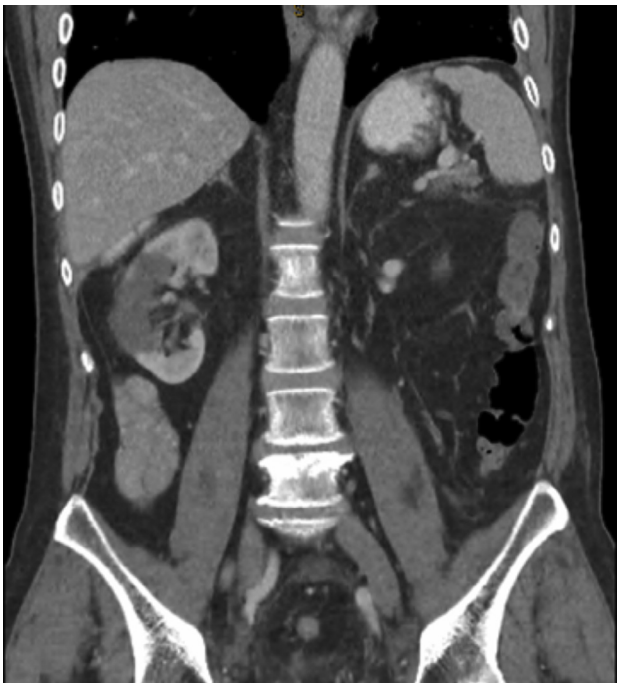

**Figure 1.** CT abdomen on postoperative day 4 showing wedge infarction of interpolar region of right kidney. CT, computed tomography. (Color version available online.)

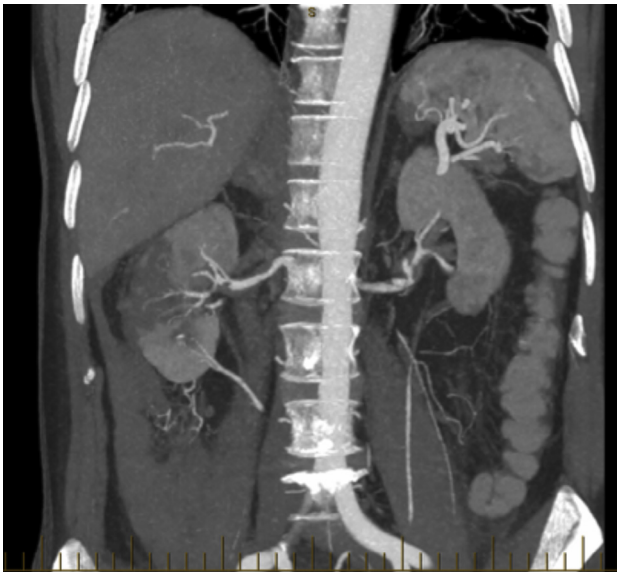

**Figure 2.** CT angiogram on postoperative day 5 showing right-sided dissection with good contrast uptake to the left kidney. By this time the infarction of the right side is well developed. Note also the accessory artery going to the inferior pole of the right kidney. CT, computed tomography. (Color version available online.)

also causing stenosis; however, at this time there was still good contrast uptake in the left kidney. On the basis of there having been no antecedent trauma (either intraoperatively or subsequently), this was determined to be a bilateral spontaneous renal artery dissection (SRAD).

## PROGRESS AND MANAGEMENT

By the time of the second CT scan the AKI had worsened, with the creatinine now 235 μmol/L (eGFR 23). The patient's nausea was also still present, and was resistant to all anti emetics.

Consideration was given to either attempting endovascular repair or open repair of the left renal artery. We initially discounted the possibility of an endovascular repair as the interventional radiologist involved believed that the chances of successfully stenting a lesion as close as this to the trifurcation was limited and associated with significant risk of further renal damage.

A number of approaches to open repair were considered, including direct repair of the dissection or an autotransplant. However, this was also felt to be too risky as the location of the dissection made either approach technically difficult, with a reasonable to high probability of failure.

We elected to treat conservatively with therapeutic anticoagulation via an intravenous heparin infusion, with serial bloods tests and Doppler ultrasounds of the renal artery to assess progress. However, by postoperative day 6, the renal function had deteriorated to a creatinine of 270 μmol/L (eGFR 20), and the Doppler ultrasound showed resistive waveforms distal to the dissection and markedly dampened flows within the renal parenchyma.

With the likelihood of rapid progression onto renal replacement therapy, the decision was made to proceed to endovascular repair, as by now the risk of further damaging the left kidney was outweighed by the possibility of salvaging at least some function. Angiography at this time, shown in Figure 3, clearly demonstrated the SRAD, and it proved possible for the vascular surgeon to place a stent in the distal part of the renal artery.

## OUTCOME

Following the angiogram and insertion of stent, the patient's renal function stabilized with a creatinine between 280 and 295 μmol/L (eGFR 18-19). Serial Doppler ultrasound assessment of the left renal artery has demonstrated a stable resistive index of 0.75. Interestingly, the patient's nausea—present since his re-presentation—settled within a day of the stent being placed.

In the week following his stenting, the patient developed moderately severe new onset hypertension with systolic blood pressures of up to 185 mmHg; however, these have been controlled effectively with metoprolol and amlodipine. Use of agents to block the renin-angiotensin system was not considered, given their potential risk of further renal deterioration in context of AKI and recent contrast exposure. The patient stayed in hospital for a week following his angiogram for observation and to control the new onset hypertension, after which he was discharged.

At the time of writing, the patient remains well at home. At a month following the RALP the creatinine is now 190 μmol/L (eGFR 30). We will be following him up with repeat blood tests and Duplex ultrasounds.