Renal Artery Aneurysm Causing Symptomatic Hydronephrosis

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Introduction

Renal artery aneurysms occur in only up to 0.3% of the population [1]. They are usually an incidental finding but have been known to cause hypertension, pain, haematuria and hydronephrosis [1-3]. Dedicated CT angiography is needed to diagnose a renal artery aneurysm hence they are not always obvious on conventional CT. There have been numerous reports of renal artery aneurysms that were originally thought to be cysts [1,4,5].

This is a case report of a patient who presented with loin pain and hydronephrosis originally thought to have a classic Pelviureteric Junction Obstruction (PUJO) but was later identified as having a PUJO secondary to a renal artery aneurysm.

Case Report

A 63-year-old female presented with frequency and dysuria, and despite oral antibiotics developed severe left flank pain and fevers requiring admission for IV antibiotics. She denied fevers or sweats. On examination she was haemodynamically stable with severe percussion tenderness on the left but there was no palpable mass or audible bruit. Her full blood count was normal other than a white cell count was 18.0×10^9/L with a CRP of 104. Renal function was normal with eGFR=90 ml/min and Cr 42 μmol/L. She had microscopic haematuria and urine MCS revealed an Escherichia coli which was resistant to amoxicillin and trimethoprim.

A CT intravenous pyelogram was ordered which revealed left sided hydronephrosis with a collapsed ureter at the PUJ. There was suspicion of a small (15×8 vmm) irregular enhancing area at the PUJ felt to represent a small transitional cell carcinoma or an artefact secondary to PUJ stenosis. There were normal appearances of right kidney and ureter.

She was investigated endoscopically for a suspected urological malignancy. A retrograde pyelogram failed to show any filling defects and ureteroscopy revealed no mucosal lesions and appearances were consistent with a left PUJO. A ureteric double-J stent was placed at the time to address the obstruction and associated infection. At this stage the leading differential was worsening PUJO in the setting of infection.

The ureteric double-J stent was removed following resolution of the infection as she had severe stent irritation with urinary frequency. A Mag 3 scan with Lasix washout was performed which showed a differential renal function of 44% on the left and 56% on the right. The Lasix half clearance time on the right was less than 4 minutes and greater than 20 minutes on the left consistent with high grade left PUJO.

Given the Mag 3 findings, coupled with her episode of pain and sepsis, it was decided that the patient should proceed to elective laparoscopic pyeloplasty. A repeat CT IVP was performed 2 months after her initial presentation which showed changes consistent with left PUJO. At operation, a ureteric double-J stent was placed into the left collecting system and the patient was then repositioned for a laparoscopic pyeloplasty. Intra-operatively the upper ureter was densely adherent to a pulsatile mass suspected to be a renal artery aneurysm. There was also a crossing vessel just cranial to the point of obstruction. The ureter was carefully freed from the obstructing aneurysm, the ureteric double-J stent was left in situ and the pyeloplasty was abandoned due to the risk of haemorrhage. It was also thought that if the ureter was freed, and the aneurysm addressed that the patient’s symptoms might resolve.

The patient was referred for a CT angiogram and vascular consult for guidance on how to manage the aneurysm. CT angiogram revealed a 12 mm aneurysm arising from the anterior branch of the left renal artery, directed posteriorly to lie at the level of the PUJ. It was remarked that the appearance of the left renal artery was consistent with fibro muscular dysplasia.

Vascular surgery was consulted in regards to management of the aneurysm. There was thought to be a miniscule risk of rupture so the aneurysm in itself did not require treatment. Another option was to try and treat the aneurysm with a view to relieving pressure on the PUJ. A covered metals tent across the mouth of the aneurysm would be technically difficult, result in devascularisation of a portion of the kidney and may not guarantee resolution of symptoms if the thrombosed aneurysm failed to shrink. An open surgical approach to the aneurysm was discounted due to the high risk of bleeding and damage to the main renal artery. It was therefore concluded that the most definitive treatment would be open pyeloplasty with a view to nephrectomy if this wasn’t possible. Conservative management was not appropriate given recurrent pain and sepsis. Endopyelotomy was contraindicated due to high risk bleeding.

At the time of her open procedure for definitive management of her symptoms it was again noted that the renal artery was densely adherent to the ureter. The ureter and renal pelvis where both densely fibrotic, possibly exacerbated by the ureteric stent. The pyeloplasty was abandoned in favour of the nephrectomy. Pathological examination of the specimen revealed chronic inflammatory changes in the renal pelvis and proximal ureter consistent with an indwelling ureteric double-J stent and recent infection. The renal artery was divided proximal to the aneurysm to exclude any possible risk of rupture.

Discussion

Fibro muscular dysplasia is by far the most common cause of renal artery aneurysm however trauma, dissection and inflammatory and congenital causes also exist [6]. The detection of asymptomatic Renal Artery Aneurysms (RAA) is increasing due to the widespread use of imaging. However, they remain a rare entity and as such they are not
always evident, or perhaps not considered as a differential diagnosis in the setting of a mass involving the PUJ. Whilst they are often visualised as a ‘mass’ on ordinary CT, angiography is required to accurately diagnose and evaluate a renal artery aneurysm [3].

RAAs are usually asymptomatic but can be associated with hypertension, haematuria, pain or obstruction [6]. Several reviews including one of 252 aneurysms in 168 patients did not comment on any of them being associated with hydronephrosis and only 6 cases have been discussed in the literature to our knowledge [1-4,6-10]. It is thought that hydronephrosis is a rare complication as the larger aneurysms are more likely to occur in the main artery or its primary branches which are unlikely to be in a position to obstruct the urinary tract [2]. All of the reported cases were treated, 2 by nephrectomy and 2 angiographically, 1 by in-situ revascularisation and the last by “ex situ” reconstructive arterial surgery and auto transplantation. Hydronephrosis secondary to a renal artery aneurysm, although a rare phenomenon needs to be treated before permanent renal injury occurs [2]. Even when aneurysm size remains stable hydronephrosis can be progressive [3].

Yang et al. discussed a case where a 10 cm aneurysm caused hydronephrosis and cortical thickening [1]. The renal arterial branch supplying the artery was able to be identified and ligated. As the aneurysm was defunctionalised and parenchymal damage had already occurred it was not removed. The kidney was salvaged in this case with resolution of the hydronephrosis as ligation of the feeding vessel was possible but Bernhardt described a similar case where a nephrectomy was performed [4]. In cases where ligation is not possible and embolization is not likely to cause shrinking of the aneurysm nephrectomy may be necessary to relieve obstruction [1].

Miyaokawa et al. went on to analyse 412 cases of RAA in Japan over a 40 year period [2]. Less than 3% (12) of these cases presented with hydronephrosis. Five were treated by nephrectomy, 2 by embolization of the renal artery and 4 were able to be treated with the kidney salvaged. No operation was documented for the last case.

Treatment options include primary in vivo repair or saphenous vein grafting, percutaneous stent grafting or embolization and ex situ repair with auto transplantation [8,11]. Nephrectomy while not the preferred option is sometimes unavoidable particularly where repair of the aneurysm is not likely to resolve the secondary obstruction as in this case [4]. Failure to address the obstruction also could lead to further episodes of pyelonephritis.

The risk of rupture is said to be low but is clearly the most catastrophic complication [12]. Several series have shown that rupture is more likely to occur in non-calcified or incompletely calcified renal artery aneurysms at a rate of up to 30% [13,14]. Hypertension is a significant risk factor for rupture, as is size (>15 mm) and the risk is also increased in pregnancy [7,15]. Other complications include arteriovenous fistula formation, hypertension, thrombosis and infarction secondary to embolization. Indications for surgery include hydronephrosis, pregnancy, aneurysm greater than 2 cm or enlargement on serial imaging. Surgical correction is also favoured in younger patients, particularly women of childbearing age [7,8].

Conclusion

While renal artery aneurysms associated with hydronephrosis are rare their treatment can be problematic and outcomes uncertain. Multidisciplinary treatment may be needed. Treatment should not be delayed due to the risk of parenchymal damage.

References