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ABSTRACT

Renal artery aneurysms are rare visceral aneurysms that occur in otherwise normal kidneys often with no background of atherosclerotic disease. The vast majority of renal artery aneurysms may remain asymptomatic and be detected only upon abdominal imaging for other illnesses. Rarely, they may be associated with resistant hypertension or co-existent renal artery stenosis. The risk of rupture and potential harm to the renal parenchyma may warrant repair even in asymptomatic patients. This brief review looks at the local experience of managing two patients with this condition along with available therapies for management in the modern era where the standard of care is renal preservation.

Keywords: Renal artery aneurysm, Renal artery stenosis, Auto transplantation, Renal transplantation

INTRODUCTION

Aneurysms occurring in the renal artery are rare and have an estimated incidence of 0.09-1.3% in the general population [1,2]. While autopsy findings report a lower rate, more frequent use of diagnostic imaging in recent times has resulted in the slightly higher incidence of incidental aneurysms. A Renal Artery Aneurysm (RAA) is defined as a localised segment of dilatation in the renal artery that exceeds twice the diameter of the adjacent 'normal' artery. The vast majority of such RAAs remain asymptomatic and are detected only as an incidental finding at abdominal imaging for other illnesses. Less commonly, RAA can cause symptoms such as hypertension, loin pain and haematuria. The indications for active intervention have been historically defined as RAA which are symptomatic or >2 cm in diameter [3,4].

Furthermore, RAA occurring in women of child-bearing age have been recommended repair due to the presumed increased risk of rupture and mortality during pregnancy [1]. Nevertheless, these indications and their validity have been questioned in recent times due to lack of robust evidence regarding the actual risk of expansion and rupture. The primary aims in definitive management are to repair an aneurysm while preserving renal function. Here we discuss the clinical cases of two patients with RAA and their subsequent management with aneurysm repair and autotransplantation.

CASE 1

A 36 year old female presented to the Emergency Department with lower abdominal pain associated with her menstrual periods. She had had a history of irregular periods and was currently under investigation by the gynaecologists. She was afebrile and had normal blood pressure. Her basic blood biochemistry including the renal functions was normal. Her urine analysis showed pyuria (25-30 pus cells per high power field) and was started on treatment for possible urinary tract infection.

A trans-abdominal ultrasound scan (USS) of the kidneys showed possible aneurysmal dilatation in the hilar arteries of the right kidney. A CT renal angiogram was performed for better visualisation of renal artery anatomy. This revealed a 3.9 cm aneurysm in the distal right renal artery close to the hilum, involving the hilar branches. It appeared to involve both main segmental renal arteries (Figure 1).

Considering her age and the size of the aneurysm, it was decided to offer her open surgical reconstruction.

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J Renal Transplant Sci, 1(1): 24-28

A short segment (8 cm) of the right great saphenous vein in the thigh was harvested and preserved in heparinised saline for arterial reconstruction. A right nephrectomy was performed via a trans-costal incision under general anaesthesia. The kidney was flushed and preserved in cold Histidine-Tryptophan-Ketoglutarate (HTK) solution. The aneurysmal segment was excised with a 2 mm margin. The superior segmental artery was anastomosed end-to-end to the saphenous vein graft. The inferior segmental branch was anastomosed to the side of the venous extension in an endto-side manner. All anastomotic reconstructions were done with 7/0 polypropylene suture (**Figure 2**). The reconstructed kidney was auto-transplanted into the right iliac fossa akin to a standard allotransplantation. The cold ischaemia time was 42 min.



Figure 1. CT renal angiogram showing the 3.9 cm extraparenchymal renal artery aneurysm in the left kidney.



Figure 2. Schematic diagram depicting the excision of renal artery aneurysm and arterial reconstruction using autogenous saphenous vein.

CASE 2

A 62 year old patient was being investigated for resistant hypertension by the Department of Internal Medicine. He had no other co-morbidities and he was a non-smoker. He was currently on three anti-hypertensives at their maximal dosages. His cardiac function was normal. An USS of the

Gunawansa N

abdomen had revealed a possible RAA in the right kidney. His renal functions were marginally elevated (serum creatinine 1.6 mg/dL).

A CT angiogram was performed with adequate hydration and sodium bicarbonate to minimise contrast nephropathy. The angiogram revealed a 2.2 cm RAA at the hilum, involving 2 of the segmental arteries (**Figure 3**). Based on these findings, it was decided to offer him open surgical reconstruction. A right nephrectomy was done, and the kidney was cold-perfused with HTK solution. The aneurysm was excised and an ex vivo arterial reconstruction was performed similarly to the procedure done in case 1 (**Figure 4**). The reconstructed kidney was auto-transplanted into the right iliac fossa, in the same manner as the previous patient. The cold ischaemia time was 38 min.



Figure 3. CT renal angiogram reconstructed image showing 2.2 cm extra-parenchymal renal artery aneurysm in the left kidney.



Figure 4. Ex vivo repair of the renal artery aneurysm.

The post-operative recovery was uncomplicated and his renal functions remained stable. The blood pressure showed better control although not achieving complete normalcy. He was discharged on day 7 with a serum creatinine of 1.5

SciTech Central Inc. J Renal Transplant Sci (JRTS)

mg/dL and blood pressure of 140-90 mm Hg on two antihypertensives. He remains asymptomatic after discharge with excellent renal perfusion and no evidence of aneurysm recurrence.

DISCUSSION

RAA constitute approximately 20% of all visceral artery aneurysms. The anatomical classification of RAA is based on its location to the renal parenchyma. Extra-parenchymal aneurysms comprise about 85% of all RAA while the remaining 15% are intra-parenchymal. Approximately 20% of RAA may be bilateral while 30% may have multiple RAAs on the same side [5,6]. Both patients discussed above had single RAAs in the extra-parenchymal region. The vast majority (70%) of RAA are saccular in configuration while the remainder are either fusiform or associated with renal artery dissection. The reported incidence is slightly higher among females due to co-existent fibromuscular dysplasia [7]. Furthermore, females also have been reported to have a somewhat higher risk of rupture when affected during pregnancy and child-bearing age (8-10). The natural history of asymptomatic RAA is a matter of debate, with earlier reports estimating the risk of annual rupture at 14% while more recent studies with a long-term follow-up report it between 3-5% [8].

While the majority of RAA are asymptomatic as in our first patient, the most frequent symptom attributed to it is hypertension as in the second patient. The pathophysiology of hypertension in RAA is thought to be due to associated renal artery stenosis or micro-emboli from an aneurysm causing recurrent micro-infarcts in the kidney [4]. Rarely, a large RAA may also cause kinking of the main renal artery resulting in hypertension. The impact of RAA repair on hypertension is unclear with some authors reporting complete resolution after repair while others were claiming that it had minimal or no effect. Our second patient showed a definite reduction in blood pressure and better control after surgery although not achieving complete normotensive status [9,10].

The association between RAA and women of child-bearing age is quite complicated. There is no definite evidence of an increased incidence of RAA during pregnancy [1]. However, in the event of pre-existing RAA, the risk of rupture during pregnancy is raised presumably due to a combination of factors such as increased intra-abdominal pressure, increased intra-vascular volume and hormone-induced vascular changes.

Due to the existing uncertainty regarding the actual natural history of asymptomatic RAAs, there appears to be a lack of consensus in the management strategy. Although a threshold diameter of >2 cm has been taken as the indication for repair even if asymptomatic, some recent reports have suggested that this may be too aggressive and recommended serial imaging and observation. Klausner et al. [5] reported their

experience of successfully managing 88 asymptomatic RAA >2 cm with close observation and serial imaging with aneurysm expansion rate of only 0-086 cm per year [3].

Elective open repair

Once a decision is made regarding intervention, the most conventional approach is open surgical repair. The exact surgical approach depends on the location of the aneurysm in relation to the renal hilum and the hilar divisions. Repair of the aneurysm while reconstructing the hilar arterial divisions and renal functional preservation is considered the standard of care [11].

Proximal RAA in the main renal artery that is saccular and solitary can be treated by tangential excision and repair with a vein patch [6]. Patch angioplasty repair is recommended over the direct repair to prevent subsequent stenosis. RAAs that are more distal and closer to or involving the segmental arterial divisions, as seen in our two patients, require excision and reconstruction. This is also the treatment method of choice for fusiform aneurysms and RAA with associated proximal renal artery stenosis. The aneurysmal segment is excised in total, and the segmental branches are reconstructed using autogenous vein graft as discussed above [12,13].

In the event of extensive fibromuscular dysplasia or aortic atherosclerosis, other bypass reconstruction is possible with inflow from the splenic artery in the left (splenorenal), the hepatic artery in the right (hepato-renal) or the common iliac arteries (ilio-renal). Autogenous vein grafts are recommended in these reconstructions owing to the better long-term patency rates compared to prosthetic grafts.

Ex vivo reconstruction and auto-transplantation

Where the location and size of the RAA preclude safe exposure and reconstruction, the recommended approach is ipsilateral nephrectomy and ex-vivo reconstruction as was performed in our patients. Although this may add to the overall ischaemia time of the kidney, the advantages are the technical ease of reconstruction in a bloodless environment at a safe and comfortable depth to the surgeon [14]. This technique involves ex vivo renal flushing and cold preservation similar to a renal allotransplantation [15,16]. This may be done by the division of the renal artery and vein only while keeping the ureter in place or by complete division of the ureter as in a donor nephrectomy. If the ureter is not divided, the flushing and cooling of the kidney can be done on the abdominal wall, and the kidney can be autotransplanted in an orthotropic manner in the native renal bed. Alternatively, the kidney can be removed by the division of the ureter as in our patients and be auto-transplanted into the iliac fossa where the vascular reconstruction and anastomosis becomes easier albeit the need for a second surgical incision.

J Renal Transplant Sci, 1(1): 24-28

Meticulous pre-operative imaging and planning are imperative in minimising the cold and warm ischaemia times during the reconstruction, thereby maximising the eventual functional preservation. Appropriate patient positioning, the preliminary harvest of the saphenous vein graft and having the cold preservative solutions ready and in-place all contribute to minimising of the ischaemia time.

Total or partial nephrectomy

In the event of an intraparenchymal RAA, partial or rarely complete nephrectomy may be required, unless they can be treated by endovascular interventions. Total nephrectomy may also be indicated as a salvage procedure in patients with ruptured RAA with haemodynamic instability. RAA occurring in conjunction with renal malignancy or a nonfunctioning kidney may also be treated by total nephrectomy.

Endovascular treatment

Saccular aneurysms with a narrow neck can be treated by coil embolisation and occlusion. RAA with wider neck has also been successfully treated by embolisation using concomitant bare-metal stents to cover the aneurysm opening, thereby preventing distal embolisation to the renal parenchyma. Other embolisation techniques have employed ethylene alcohol polymers or glue as liquid embolisation agents instead of coils [11]. The apparent advantages of embolisation are the avoidance of major complex surgery and the ability to offer salvage treatment in cases of intraparenchymal and ruptured RAA [17,18]. Tsilimparis et al. [19] compared the overall outcomes following surgical versus endovascular repair of RAAs and reported comparable peri-operative results in terms of 30 day morbidity, mortality and long-term renal function. However, the use of endovascular therapy is limited by favourable anatomy with incomplete sealing and possible aneurysm reexpansion. Furthermore, the long-term success rate of such procedures is not clearly documented and needs further evaluation.

RAA located proximally in the main renal artery with adequate proximal and distal landing zones can also be treated with covered stents [20]. The obvious limitation is the scarcity of such aneurysms with sufficient proximal and distal artery length and the risk of stent migration and thrombosis. However, one advantage of this treatment modality is its use in co-existing RAA and renal artery stenosis.

CONCLUSION

RAAs are rare visceral aneurysms that may be detected incidentally at abdominal imaging or investigation for resistant hypertension. The relative scarcity of long-term data has contributed to a lack of consensus in optimal management guidelines. The actual risk of rupture may be significantly less than initially estimated resulting in a growing argument for conservative management with close surveillance. In the event of symptomatic or large RAAs, the optimal treatment modality depends on a combination of factors including patient's general health status, anatomical location of the aneurysm, renal function and available expertise. Regardless of the treatment modality, the standard of care should be complete aneurysm excision and reconstruction with renal functional preservation.

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J Renal Transplant Sci, 1(1): 24-28

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