

Case Report

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Bilateral Renal Artery Aneurysms: about a Case

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SUMMARY

A renal artery aneurysm is defined as a dilated segment of the renal artery that exceeds twice the diameter of a normal renal artery. Although rare, the diagnosis and incidence of this entity has steadily increased with the routine use of cross-sectional imaging. In some cases, renal artery aneurysms can be clinically telling and potentially lethal. However, knowledge about their occurrence, natural history and prognosis with or without treatment is still limited. The purpose of this article is to review the recent literature concerning renal artery aneurysms, with particular attention to pathophysiology, aspect of imaging, indications for treatment, different technical options, post-procedural complications and treatment outcomes.

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Introduction

Renal artery aneurysms (RAAs) represent a rare, clinical entity with an estimated incidence of 0.09 % on autopsy studies [1]. In spite of this fact, their frequency is increasing due to the routine use of imaging for various intra-abdominal pathologies.

Although RAAs present commonly as an incidental finding, several studies have shown that this anomaly causes hypertension and life-threatening retroperitoneal hemorrhage, secondary to spontaneous rupture [2- 4].

Despite being a well-known clinical entity since its first description by Rouppe in 1770, the current knowledge of RAAs presents some controversial issues regarding physiopathology, indications for treatment, different technical options, post-procedure complications, and treatment outcomes [5]. This article aims to review the most recent literature available concerning RAAs, to offer the treating physician a wide-scope and updated practical guideline for their adequate management.

Patient and observation

It's a case of a 37-year-old female patient who suffered from renal colic without any medical or surgical history. On abdominal ultrasound, there was a doubt about the presence of bilateral kidney stones. As there was no clinical improvement under appropriate treatment, a abdominal CT scan was performed after injection of iodinated contrast in arterial phase in parenchymal window showing bilateral saccular aneurysms of the prepyelic and retropyelic renal arteries.

Epidemiology

At the time when most of the information on RAAs came from autopsy studies, rather than angiography, it was considered to be

extremely rare. However, in angiographic and cross- sectional imaging studies, the incidence of RAA is found to be significantly higher, about 70-fold the rate obtained from post-mortem reports in retrospect [6,7]. The reason for this difference between necropsy series and imaging series may be that renal artery aneurysms often are quite small and some- times intrarenal, and therefore are not easily visualized at a routine autopsy.

The occurrence of renal artery aneurysms in reported series of patients undergoing renal angiography varies between 0.3 % and 0.7 % [7-9]. However, if microaneurysms and dissections are excluded, the incidence is approximately 0.1 %, and shows an equal distribution between men and women [1, 4, 7, 10].

Pathogenesis

The pathogenesis of RAAs is well-described. A true aneurysm contains all three layers of the vascular wall, which undergoes progressive dilation and thinning. The majority of these cases probably result from an abnormality in the medial arterial wall, since the most common histopathological finding is reduced smooth muscle and a deficiency of the arterial media with loss or fragmentation of the internal elastic lamina. This anomaly may be congenital in origin, and would be further stressed by various acquired factors that also contribute to some degree [1, 4, 11, 12]. Degeneration of elastic fibers and mediolysis lead to a weakening of vessel wall, which results in expansion of the vessel due to high intraluminal pressure. In fact, in one series by Henke et al., arterial fibromuscular dysplasia was shown to be the most prevalent vascular disease associated with RAAs [13].

Atherosclerotic lesions are usually present. Although frequently cited as a common cause, Stanley et al. proposed that the typical calcific atherosclerotic change in many of these aneurysms is more likely to be a secondary result, given the lack of similar findings in the adjacent vessels, rather than the primary causative process [4].

The increased blood flow, intraabdominal pressure, and vessel wall transformation due to the hormonal and metabolic changes associated with gestation are believed to be contributory. Although less common, congenital syndromes and collagen disorders are possible causes of multiple, idiopathic, true RAAs [14,15].

Conversely, the wall of a pseudoaneurysm is partially made up of the tissues surrounding the vessel, because the occurrence of this event is usually due to a tear of the vessel wall and a periarterial hematoma [16,17]. The focal disruption in one or every layer of the artery causes a saccular outpouching at the weakened area. Pseudoaneurysms can develop as the result of blunt or penetrating trauma, inflammation either in the arterial walls or the adjacent tissues (which erode into the artery), infection, and iatrogenic trauma secondary to surgical, endoscopic, and radiologic procedures [15, 18].

Intrarenal aneurysms are either true or false aneurysms within the renal parenchyma. They deserve special classification because their management is usually with nephrectomy or endovascular treatment. Intraparenchymal aneurysms are believed to arise primarily from inflammatory changes of the vessel wall, and commonly develop into microaneurysms.

Classification

RAAs may also be classified based on their morphology and anatomic location. According to these features, Rundback et al. categorized RAAs into three types. This classification is of outmost importance, as it provides guidelines for an adequate management approach [19]. Type 1 includes saccular aneurysms arising from the main renal artery or a large segmental branch. Type 2 includes fusiform aneurysms, and type 3 includes intralobar artery aneurysms (Figure 1).

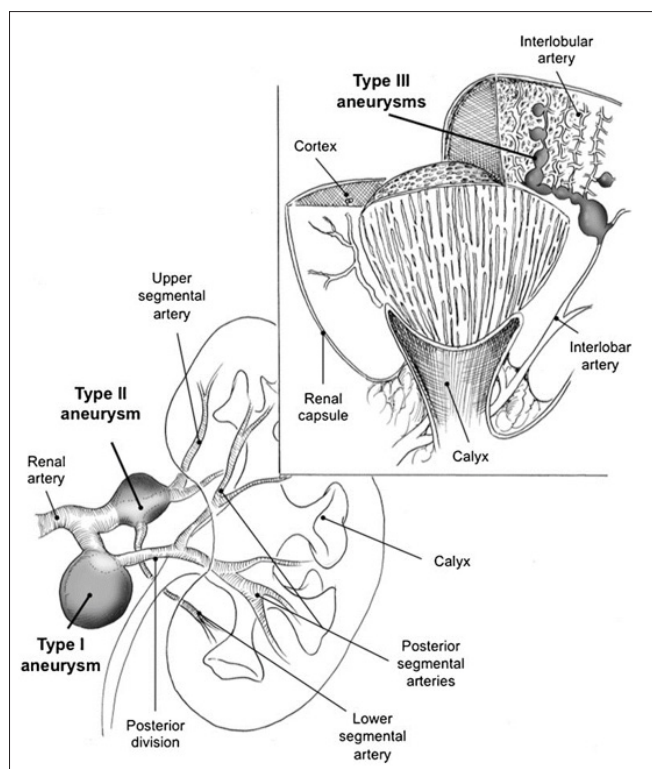


Figure 1: Rundback et al.'s classification of RAAs. Type 1 includes saccular aneurysms arising from the main renal artery or a large segmental branch. Type 2 includes fusiform aneurysms, and type 3 includes intralobar artery aneurysms

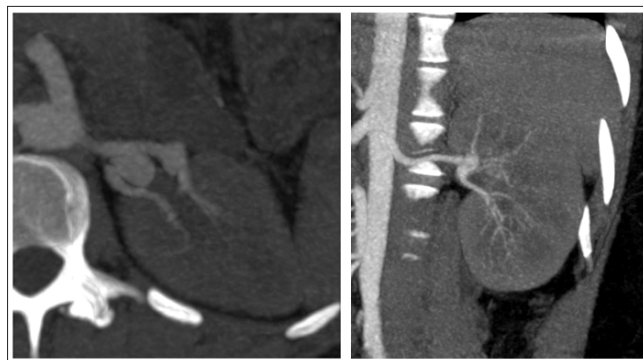


Figure 2: Abdominal CT scan in axial section after injection of iodinated contrast in arterial phase in parenchymal window



Figure 3: Abdominal CT scan in axial section after injection of iodinated contrast in arterial phase in parenchymal window



Figure 4: 3D reconstruction of an abdominal CT scan in arterial window

Natural History and Clinical Presentation

The natural course of an RAA is still uncertain and considerable controversy exists about its clinical importance [1, 4,7,11, 20]. At present, most RAAs are asymptomatic and do not pose any significant clinical problems, being discovered incidentally on examinations performed for unrelated conditions [4,7,21]. However, about 30 % of patients develop symptoms, including uncontrolled intractable hypertension, shock, or less frequently, other symptoms such as flank pain, thrombosis and hematuria [1].

Hypertension The most common symptom found in renal artery aneurysms is hypertension, with a reported incidence as high as 90 % [7,11,12, 22]. However, it still remains uncertain whether RAAs are a cause or an effect of hypertension. It is important to separate patients with a combination of a significant renal artery stenosis,

often caused by fibrous dysplasia and an aneurysm, from those with aneurysm alone. Most patients with fibromuscular dysplasia are healthy, young, hypertensive women, and on angiography the renal artery appears as a string of beads, with the aneurysm at the renal artery bifurcation. In these cases, the hypertension can be attributed to the renal artery stenosis and activation of the renin-angiotensin system, with increased angiotensin II levels resulting in fluid retention and vasoconstriction [22].

Conversely, hypertension in renal artery aneurysms in the absence of a hemodynamically significant stenosis is not as well understood. Possible explanations contributing to high blood pressure in these cases are mechanical kinking of the renal artery, segmental renal parenchymal ischemia, and flow turbulence inside the aneurysm changing the hemodynamics [23, 24].

Cummings et al. demonstrated that those patients who have renal artery aneurysm combined with a hemodynamically significant stenosis, as verified by renal venous renin studies, could be cured with surgery [26]. Those without evidence of stenosis and with negative renin studies remained, however, hypertensive—even after a technically successful reconstruction in this study. Therefore, hypertension alone in a patient with a renal artery aneurysm may not be a formal indication to operate, and probably cannot improve the blood pressure situation unless it is combined with a stenosis, which has been demonstrated to be hemodynamically significant. In that case, the primary indication to operate is not the aneurysm but the stenosis. In contrast, the blood pressure response and the reduction in antihypertensive medications cited in other reports appear similar in patients treated for a renal artery aneurysm with associated renal artery stenosis, and in those without coexisting stenosis [2].

Aneurysm Rupture Renal artery aneurysms may present as a life-threatening intraabdominal hemorrhage secondary to acute rupture [26]. Earlier reports give an extremely high risk for rupture. Ippolito reported that 24 out of 169 patients with a non-calcified aneurysm ruptured, and Cerny claims that 30 % of intrarenal aneurysms ultimately rupture [3,27]. In recent reports, five small series of patients with renal artery aneurysms have been followed for a period of 3–10 years without operative treatment [1, 4, 7,11]. No ruptures have been reported in these studies, thus suggesting that renal artery aneurysms usually show a slightly different natural history than other visceral counterparts with a significantly lower risk of rupture [1,21]. Mortality derived from rupture is also considerably low (i.e., around 10 %), but loss of the kidney under these circumstances is almost inevitable [4].

It has been suggested that asymptomatic aneurysms smaller than 1.5 cm with circumferential calcification are unlikely to rupture [3, 4, 11]. Conversely, the risk for rupture is reported to be high in non-calcified saccular aneurysms when accompanied by hypertension (i.e., >20 % risk of rupture) and in pregnant women, especially during the third trimester, being a life-threatening condition associated with maternal mortality of around 50 % and fetal mortality of 80 % [3,11,28-32].

Other Symptoms In case series, 8–25 % of patients presented with abdominal pain [10, 11, 33]. Flank pain is the typical presenting symptom of dissection, although most spontaneous dissections are commonly asymptomatic. New onset or worsening pain may be indicative of a rapidly expanding aneurysm or impending rupture.

Renal artery aneurysms may thrombose or serve as a source of emboli that may occlude the peripheral arteries in the kidney,

causing multiple renal infarctions that may be visualized on CT-scan images [34].

Multiple peripheral intraparenchymal aneurysms, which rupture into the collecting system, may also manifest as hematuria [3]. Collecting system obstruction is a rare presentation, but it has been documented in patients with larger aneurysms [8].

Diagnosis

Improvements in imaging techniques in the past decade have led to the increased recognition of true aneurysms of the renal arteries [1, 4,7,11]. RAAs are mostly discovered incidentally on imaging obtained for unrelated reasons, and are also found during workups of uncontrolled hypertension. In a recent series, 35 % were detected during investigation for renal hypertension, whereas 26 % were discovered incidentally by angiography undertaken for some other reason [35].

An RAA can be evaluated by duplex ultrasound, spiral computerized tomography (CT), three-dimensional, contrast-enhanced, magnetic resonance angiography (MRA), or digital subtraction arteriography (DSA). Although duplex ultrasound is an inexpensive, non-invasive technique in the demonstration of an RAA, this imaging modality is highly operator- and patient body habitus-dependable, which may produce interpretation pitfalls or limitations of visibility during the examination process. Typically, duplex ultrasound images show sonolucent lesions with turbulent flow. However, RAAs may be misinterpreted as cysts or dilated renal veins [36].

Focal dilatation of the renal artery is the key feature in multidetector spiral CT imaging. This technique is widely available and has been established as a useful imaging modality for the diagnosis of RAAs, but carries the risk of exposure to ionizing radiation and nephrotoxicity from iodine injection. The conventional mode is comparable in resolution to DSA [37]. However, renal artery lesions, are better demonstrated in multiplanar reformations and volume-rendered images. Nevertheless, oblique and rotated views may be needed in the evaluation of the neck aneurysm morphology or its location in relation to branch vessel origins. Hence, in spite of the fact that DSA is a relatively invasive procedure compared with other imaging modalities, it has remained the gold standard in planning for endovascular intervention.

Contrast-enhanced MRA has shown to be promising in the demonstration of RAA, in terms of resolution and diagnostic accuracy, and bears the advantage of using non-ionizing radiation as compared to CT scanning. However, it is costly and has relatively limited availability [36,37].

Renal artery pseudoaneurysms may be distinguished from true aneurysms by imaging criteria. Imaging demonstrates focal arterial disruption in the setting of an otherwise normal artery, or perivascular inflammation with an irregular aneurysmal wall.

Indications and Contraindications

The indications for treatment of RAAs are still controversial. However, most surgeons universally accept that the indications for treatment are presentation with rupture or high risk of rupture (i.e., rapidly expanding aneurysm and females who are pregnant or contemplating pregnancy), presence of symptoms (i.e., uncontrolled hypertension from renal artery stenosis and/ or refractory to medical management, flank pain, hematuria, or renal ischemia/infarction secondary to embolization from the aneurysm sac), or large diameter (i.e., commonly greater than 2 cm).

One of the main indications to operate on an aneurysm is the risk for rupture [3]. However, the risk is smaller than generally thought, and therefore, a recommendation for surgery in a patient with an incidental RAA cannot be justified only because of the risk for rupture. The same statement can probably be made regarding the potential risk for thrombosis or embolization.

Confirmed renovascular hypertension coexistent with a combination of aneurysm and stenosis in the renal artery is, obviously, an indication to operate. In these cases, the stenosis has to be surgically treated and the aneurysm excised.

Conversely, there is no clear indication to operate on a patient with an RAA and coexistent hypertension, unless it can be proven that this symptom is renovascular in origin. In the presence of flank pain or hematuria, there may be an indication to operate, but commonly the symptoms may be coincidental rather than caused by the aneurysm [3,27].

In contrast, small (<2 cm diameter) RAAs do not usually require surgical treatment and can be treated expectantly. Moreover, spontaneous cure by thrombosis has been described in small aneurysms. Regular follow-up with ultrasound or CT-scan is highly recommended if an expectant attitude is chosen.

Management

Emergency Surgical Repair In the case of acute rupture, immediate surgery is required to limit blood loss and prevent related complications, such as exsanguination and distal organ ischemia. Early arterial control through a midline approach is mandatory when the renal hilum is hampered by a large perinephric hematoma. Nephrectomy may be necessary in cases of hemodynamic instability. However, in a hemodynamically stable patient, renal artery reconstruction may be considered for renal salvage [38,39].

The management of an RAA acute rupture in a pregnant patient follows the same principles observed for a retroperitoneal hemorrhage caused by blunt abdominal trauma. Cesarean delivery should be avoided if possible, since it increases operative time and may result in additional blood loss. Specific indications for cesarean delivery include interference of the gravid uterus with exposure, fetal distress and impending or recent maternal death [40].

Elective Management Elective management of an RAA is generally undertaken to obviate the risk of rupture or to treat the symptoms from RAA. Surgical and endovascular treatment share the common goal of preventing expansion and rupture by excluding the aneurysm from high intraarterial pressure. Aneurysm isolation can be achieved by various means, and each method is selected depending on the presence of symptoms, the relative risk of rupture, the mode of presentation and the anatomical location and size of each particular lesion.

Type 1 Aneurysms They can be treated by both surgical repair and endoluminal techniques. The intervention of choice for a solitary saccular aneurysm at a proximal bifurcation is tangential excision with primary repair or patch angioplasty [41]. This procedure should be performed whenever feasible. Aneurysms with small necks may be repaired primarily; otherwise, a patch angioplasty using autologous (i.e., saphenous or gonadal vein) or prosthetic material may be needed.

Approximately 30 % of RAAs are amenable to such treatment, obtaining good anatomical and clinical results. These procedures include splenorenal bypass, hepatorenal bypass, and iliac-to-renal bypass. Partial nephrectomy may be combined with RAA repair in cases of associated renal lesions (i.e., malignancy), whereas total nephrectomy is indicated in patients with RAA in a nonfunctional kidney (i.e., severe ischemic renal atrophy or end-stage renal disease) [41, 42].

Aneurysmal morphologies that are favorable for endovascular therapy include saccular RAAs with a narrow neck and with adequate collateral flow. Therefore, patients are considered candidates for endovascular treatment if inflow and outflow vessels to and from the aneurysm can be accessed and occluded by a catheter-based system, and if end organ perfusion can be preserved by collateral flow [43-49]. An extra parenchymal saccular RAA can be isolated from the circulation by deployment of coils into the aneurysm, therefore promoting thrombosis. Endovascular coil occlusion provides an excellent option if the aneurysm presents with a narrow neck. Coils in these cases are constrained within the aneurysm sac by the narrow neck. To avoid coil migration in wider-neck RAAs, a stent is first placed across the neck of the aneurysm, constraining the coils placed through the interstices of the stent within the sac. Occasionally, glue or thrombin is injected into the coil-filled sac to promote thrombosis [49]. Saccular extra parenchymal RAAs may be excluded also using stent grafts [47]. This approach requires a normal-sized arterial segment, both in the proximal and distal aspects of the aneurysm, and cannot usually be placed at bifurcations unless a Y-shaped stent graft is utilized. However, a procedure involving the combination of a stent graft extending into one of the branches and a coiling occlusion of the other branch has been successfully performed in select cases [43-47].

Type 2 Aneurysms Fusiform aneurysms in the main renal artery or large segmental artery are best treated by surgery at specialized centers [38, 39, 41,42]. In these cases, the excision of the aneurysm paired with patch angioplasty is rarely possible, and thus, after segmental excision of the RAA, the preferred arterial reconstruction is with an autologous saphenous or gonadal vein, aortorenal bypass graft. However, if the aorta is heavily diseased by atherosclerosis, alternative bypass donor arteries may be used. The aortorenal bypass is typically constructed with an end-to-side configuration for the proximal anastomosis, and an end-to-end configuration for the distal anastomosis. Prosthetic material is also acceptable. However, vein grafts are preferred given their superior patency rates [38,39].

For aneurysms involving large arteries, the aneurysm can be “trapped” between coils placed in the parent artery distal and then proximal to the aneurysm (i.e., isolation procedure). Fusiform aneurysms involving bifurcations require endovascular exclusion with the placement of coils in the efferent and afferent arteries, to obtain complete occlusion. In these cases, perfusion of the end organ can be at least partly maintained by collateral flow. Covered stent grafts have also been described in treating these aneurysms [43-48]. Covered stents provide another means of excluding RAAs from the circulation. These stents are reserved for major branches (i.e., 6 mm or larger in diameter) of the renal artery, for which preservation of arterial perfusion is required. Limitations of covered stents include delivery systems, the size and rigidity of which preclude stent placement in distal tortuous branches. However, more flexible and lower profiled stents are now commercially available. An additional benefit of stent grafts is the ability to treat both renal artery stenosis and RAA [43].

Type 3 Aneurysms For interlobar and/or intraparenchymal aneurysms, embolization with large particles followed by coil placement in the larger proximal parent artery is probably the best option [45, 46]. However, this procedure is usually accompanied by the thrombosis of end arteries and resultant infarction. Super-selective embolization limits the infarction of renal parenchyma. This can be achieved with the use of microcatheters, which can be navigated coaxially to the site of the aneurysm, thereby limiting the number of vessels at risk. For poor-risk patients, or for aneurysms in which arterial interruption would not compromise the more distal circulation, endovascular techniques represent the most reasonable alternative [43,48].

With complex hilar or intrarenal aneurysms involving multiple arterial segments, in situ exposure of the renal hilum is difficult, and a considerable exposure to warm ischemia may accumulate while in situ repair is being attempted. Ex vivo surgery allows for adequate exposure, while maintaining the kidney with a cold preservative solution through simple continuous perfusion via the renal artery for an extended time [50, 51]. Additional surface hypothermia is also achieved with a constant drip of chilled solution onto the kidney, wrapped in an iced laparotomy pad. Once ex vivo reconstruction is complete, the kidney may be auto transplanted into the iliac fossa, or placed into the original renal fossa and revascularized by attaching the arterial graft to the aorta, and the renal vein to the vena cava or renal vein remnant. The advantages of extracorporeal reconstruction include extended time, a superficial, blood-free operating field and the possibility of needing an operating microscope.

At times, partial nephrectomy may be needed, but with improved surgical technique, renal preservation is now the standard of care. Partial nephrectomy may be combined with RAA repair in multiple RAAs, in both intraparenchymal and extra parenchymal location. Total nephrectomy is indicated in patients with multiple, large intrarenal aneurysms, not amend- able to partial nephrectomy [41, 42,52-55].

Conclusion

At present, most RAAs do not pose any significant clinical problems and usually show a slightly different natural history than other visceral counterparts, with a significantly lower risk of rupture and considerably lower mortality rates. Since RAAs are found relatively often at imaging, the question of the natural course, the treatment of choice in each clinical scenario, and the risk for potential complications before and after treatment are important in this type of aneurysm. The present article has reviewed the current literature to provide a clear depiction of the pathophysiology, natural history, and classification of RAAs, thus establishing a clinical situation-based management schedule for the treating physician.

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