Staged Endovascular Repair Resulting in Renal Salvage

Endovascular management of a renal artery aneurysm with large arteriovenous fistula and giant venous aneurysm.

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enal artery aneurysms (RAAs) associated with arteriovenous fistulas (AVFs) that involve the native kidney are very rare lesions. Large AVFs may be secondarily complicated with the formation of giant venous aneurysms. Management of these rare clinical entities will usually necessitate a nephrectomy. However, using currently available advanced endovascular devices and techniques, these large and complex fistulas may be obliterated to allow renal salvage. In this article, we describe a case of an RAA associated with a renal AVF and a giant tortuous venous aneurysm, which was managed with staged endovascular repair.

In the first stage of the procedure, an Amplatzer vascular plug (AVP, St. Jude Medical, Inc., St. Paul, MN) was used to obliterate the fistulous tract, and a covered stent was used to partially cover the orifice of the RAA. In the second stage, endovascular coiling of the RAA was performed using retractable Interlock coils (Boston Scientific Corporation, Natick, MA). Flow to the renal parenchyma was preserved, resulting in renal salvage. On 4-year follow-up, the renal artery is patent, and the arterial and venous aneurysms remain obliterated; the renal functions are normal.

CASE REPORT

A 57-year-old man presented to the emergency department with a 3-week history of right-sided abdominal pain described as shooting and burning in quality with no radiation. It appeared to worsen with eating. Initial work-up was conducted under the assumption of acute cholecystitis, and an ultrasound was performed. Ultrasound demonstrated cholelithiasis without evidence for cholecystitis. Incidentally, it showed a dilated tortuous vascular structure in the region of the hilum of the right kidney. Subsequently, contrast-enhanced computed tomography

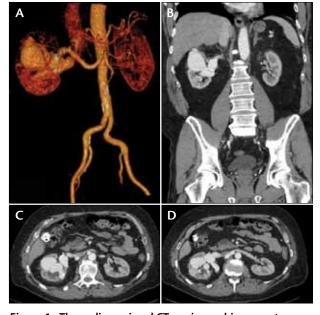


Figure 1. Three-dimensional CT angiographic reconstruction showing right RAA, AVF, and large venous aneurysm (A). Axial and coronal images demonstrating a 2.5-cm-diameter right RAA with peripheral calcifications and a fistulous tract leading to a giant 4.1-cm-diameter renal venous aneurysm (B through D).

(CT) showed two aneurysms and an AVF (Figure 1). There was a 2.5-cm RAA with peripheral calcifications, arising from the distal aspect of the right renal artery, close to the bifurcation. The apex of this aneurysm was drained through a short 2-cm fistulous tract directly to a large tortuous venous structure, which communicated with the renal vein and drained into the inferior vena cava.

The venous aneurysm measured 4.1 cm at its largest dimension. Given the large size of the venous aneurysm

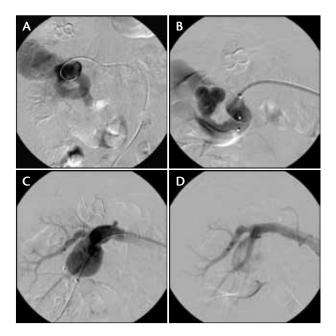


Figure 2. Renal arteriography with the catheter in the RAA showing the RAA, fistulous tract, and venous aneurysm (A). Arteriography demonstrating deployment of a 16-mm AVP in the fistulous tract (B). After deployment of the AVP, the venous aneurysm is no longer visualized, but the RAA is seen filling and is in close proximity to the bifurcation of the renal vessel (C). After a covered stent was deployed, there is partial coverage of the neck of the aneurysm; however, persistent filling of the RAA is seen (D).

and the fact that patient had pain, further treatment was pursued due to the risk of rupture. The urology department proposed nephrectomy because the vascular structures could not be reconstructed. We were then consulted for angiography with possible endovascular embolization to preserve renal function. At this time, a more thorough medical history was performed, which revealed that the patient had a remote history of trauma. The patient was kicked in the abdomen by a horse when he was 18 years old. This may or may not have been the etiology of this renal AVF. It is more likely that this patient had a congenital RAA, which gradually enlarged and ruptured into a branch of the renal vein, resulting in an AVF and secondary formation of a large venous aneurysm.

Right renal arteriography via a right common femoral arterial approach was then performed under conscious sedation. The right renal artery was selectively catheterized, and right renal arteriography was performed. This showed a large renal AVF with a high-flow state. Imaging in multiple projections was performed to delineate the complex anatomy. A 2.5-cm RAA was identified just proximal to the bifurcation of the right renal artery. The neck

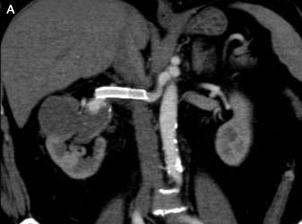




Figure 3. CT 3 weeks after the first stage of repair shows complete obliteration of the venous aneurysm. The covered stent is patent with filling of the RAA (A). The AVP is seen obliterating the fistulous tract (B).

of the aneurysm measured 13 mm in size. The neck of the aneurysm was in very close proximity (2–3 mm proximal) to the renal artery bifurcation. The tip of the RAA communicated with the renal vein through a fistulous communication, which was 2 cm long.

A giant venous aneurysm was identified, measuring approximately 4.1 cm in diameter (Figure 2A). This was a very high-flow–state AVF, and using coils alone carried a high risk of inadvertent migration of the coils into the pulmonary artery, so we decided to use an AVP to obliterate the fistulous tract. Using a 4-F Glidecath Cobra (Terumo Interventional Systems, Inc., Somerset, NJ), a 6-F sheath was advanced into the RAA, leading to the narrowest part of the fistula, which measured 12 mm. A 16-mm AVP was then used to occlude the outflow of the fistulous tract (Figure 2B). This resulted in occlusion of the fistulous tract and complete obliteration of flow into the larger venous aneurysm.

There was persistent filling of the RAA (Figure 2C). We decided to use a covered stent in an attempt to cover the neck of the aneurysm. Overlapping 7- X 38-mm and 7- X 22-mm iCast balloon-expandable covered stents (Atrium Medical Corporation, Hudson, NH) were deployed within the mid-to-distal renal artery and dilated to 10 mm. The opening of the RAA could not be completely covered because it was in close proximity to the bifurcation and carried the risk of covering a renal branch and infarcting the renal parenchyma. After placement of the covered stents, there was decreased but persistent residual flow in the RAA (Figure 2D).

Given the contrast load and complexity of the anatomy and the procedure, a decision was made to stage the procedure and bring the patient back after 3 weeks for reevaluation and further intervention. A follow-up CT examination was performed 3 weeks later, which showed patent stents with an area of persistent enhancement within the RAA, with complete thrombosis of the venous aneurysm (Figure 3A). There was mural thrombus in the RAA, and the enhancing part of the aneurysm measured 16 mm. The AVP is seen in the fistulous tract (Figure 3B).

The patient was brought back for embolization of the RAA 8 weeks after the first procedure. Renal arteriography redemonstrated that the neck of the aneurysm was only partially covered with the covered stent and was in juxtaposition to the bifurcation (Figure 4A). The RAA was selectively catheterized with a 3-F microcatheter and embolized using 14- and 16-mm, 0.018-inch retractable Interlock coils (Figure 4B). Complete thrombosis of the aneurysm was achieved (Figure 4C). Follow-up contrast CT at 2 years showed complete obliteration of the fistula (Figure 5). The entire right kidney enhances with contrast and is functional.

DISCUSSION

RAAs are rare lesions that are discovered in only 1% of renal arteriographic procedures and 0.3% to 0.7% of autopsies.² Most RAAs are asymptomatic. The clinical manifestations may include hypertension, flank pain, hematuria, and rupture. The risk of rupture is low but is associated with mortality rates as high as 80%.² Most authors recommend surgical or endovascular treatment of all aneurysms larger than 2 cm in diameter.³

Renal AVFs are exceedingly rare, with a prevalence of < 0.04%. The etiology could be congenital (25%), acquired (70%), or idiopathic (3%–5%).⁴ The majority (70%) are acquired and are iatrogenic or posttraumatic in origin and occur as a result of percutaneous renal biopsy, renal surgery (partial or total nephrectomy), or blunt/penetrating trauma. The exact cause of congenital fistulas (25%) is unknown; however, they are thought to be present at birth or to result from a congenital aneurysm that erodes into the adjacent







Figure 4. Arteriography 8 weeks after the initial repair shows the patent covered stent with obliteration of the venous aneurysm. Filling of the RAA is seen (A). A 14- and 16-mm, 0.018-inch retractable Interlock coil has been deployed within the RAA via a 3-F microcatheter (B). Complete obliteration of the RAA is seen (C).

vein. In general, congenital lesions typically have a cirsoid appearance, and acquired or idiopathic lesions are usually aneurysmal.

The cirsoid type of renal AVF frequently causes hematuria, as it is commonly found beneath the mucosa of the renal collecting system. The idiopathic or acquired types cause abdominal bruit, hypertension, headache, and pal-



Figure 5. Contrast-enhanced CT 2 years after the second intervention shows complete obliteration and a decrease in size of the venous aneurysm. The RAA is thrombosed with coils in place. The covered stents are patent (A). The entire renal parenchyma enhances with preserved function (B).

pitation, resulting from a large amount of blood flowing through the AVF. Idiopathic lesions are usually aneurysmalacquired AVF, which our case appears to have been. The mechanism is related to close proximity of the RAA to a renal vein branch, which becomes compressed and then stretched by the adjacent aneurysm. Eventually, erosion into the vein with formation of AVF occurs.^{5,6}

The aim of renal AVF treatment is to preserve renal parenchymal function and eradicate symptoms and long-term hemodynamic effects associated with the abnormality. Indications for treatment are a progressive increase in the size of the fistula, recurrent or persistent hematuria, and hemodynamic effects associated with the abnormality, especially decompensation, hypertension, and high-output heart failure.

From a treatment standpoint, all renal AVFs need to be treated because they seldom resolve on their own and will continue to enlarge. A small AVF can be treated with endovascular coil embolization, but owing to the concern for pulmonary embolism, large high-flow AVFs have traditionally been managed with open resectioning or ligation. Conventional RAA repair has relied on open surgical techniques, including aneurysmectomy with patch angioplasty or interposition grafting, aortorenal bypass, and extra-

anatomic bypass.¹ Ex vivo and in situ reconstruction with cold perfusion has also been performed.^{7,8} Nephrectomy is needed for instances of rupture and nonreconstructible anatomy, such as in our case.

In the last few years, with the development of advanced endovascular technology, RAAs and AVFs are increasingly being treated with various endovascular techniques and devices. RAAs have been treated with stent grafts and coils.⁹ For the management of wide-necked RAAs at the bifurcation, stent-assisted coil embolization has been used.¹⁰ Small AVFs may be treated with coils; however, treating high-flow AVFs carries the risk of pulmonary embolism. There are case reports describing the use of AVP for endovascular management of high-flow AVFs.^{11,12}

There are very few case reports of endovascular management of RAAs associated with renal AVFs. Endovascular coils have been used to exclude both the RAA and AVF. In one of the case reports, a large 4-cm RAA was excluded using large framing coils followed by several detachable coils.¹³ In another report, there was a single segmental feeder to the aneurysm, which was treated with 10-mm coils.¹⁴

In our case, we used a combination of the previously described techniques. In the first stage, we used an AVP to occlude the high-flow AVF and covered stents to exclude

the RAA. The AVP completely occluded the AVF, but the covered stents only partially excluded the RAA due to the proximity of the RAA to the branch vessels. In the second stage, we successfully used retractable Interlock coils to completely thrombose the RAA.

CONCLUSION

Our patient had an RAA with a complex highflow AVF associated with a giant venous aneurysm. Nephron-sparing reconstructive surgery was not possible, and short of endovascular intervention, nephrectomy was considered the only option. The currently available armamentarium of endovascular techniques and devices makes it possible to manage some of these high-flow and complex lesions percutaneously as an alternative to surgery. However, management of such difficult lesions may not be accomplished in one sitting or by utilizing a single endovascular device. A staged endovascular approach using various devices may be necessary, as demonstrated in this case. This patient benefited from a staged procedure, which spared the renal artery branches and preserved flow to the entire renal parenchyma, resulting in a successful and durable clinical outcome.

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