Renal arteriovenous fistula (AVF) is often an acquired renal vascular abnormality, usually caused by biopsy, percutaneous nephrostomy, or trauma. Here, we report the endovascular management of a giant renal AVF using Amplatz vascular plug (AVP).

A 32-year-old male patient initially presented with a 6-month history of left flank pain. The pain was dull, intermittent, and non-radiating. He had a history of bullet injury 7 years back in the left flank. On physical examination, a continuous bruit was audible over the left flank. Doppler evaluation revealed a large cystic lesion at the left renal hilum with increased flow velocity and decreased arterial resistance, and mixing of arterial and venous waveform was also observed. A computed tomography angiography (CTA) was performed which revealed contrast opacification of the renal vein during the arterial phase suggesting renal AVF. A giant pseudoaneurysm (PSA) was also seen occupying the mid and lower pole of the left kidney [Figure 1A] measuring 7 cm × 6.5 cm × 6 cm. The main renal artery (MRA) was the possible feeder artery to the AVF which was seen directly opening into the PSA, suggesting an ultrashort segment of fistulous communication. Main renal vein appeared to be directly communicating with the pseudoaneurysm and was also dilated grossly with aneurismal morphology. The morphology and extension of the vascular lesion was well-demonstrated on the volume rendering technique image [Figure 1B].

The patient was referred to our department for endovascular management. Vascular access was obtained through the right common femoral artery. The right renal artery was then catheterized using 5F renal double curve catheter. Digital subtraction angiography (DSA) revealed similar findings as the CTA consistent with giant AVF fed by MRA. Subsequently, the 6F arterial sheath was upsized to a 9F guiding sheath to gain access into the renal artery ostium. Because there was hypertrophy of the MRA, a 20-mm AMPLATZER Plug II (St Jude Medical, Inc, St Paul, Minnesota, USA) was placed at the junction of the feeding artery and the PSA [Figure 1C]. Position was then confirmed by angiogram and the device was deployed. Follow-up angiogram [Figure 1D] showed non-filling of the sac. A repeat Doppler study after 12 hours showed complete non-filling of the AVF. After unremarkable recovery, patient was discharged in a stable condition and is on follow-up for the last 2 years.

Renal AVF is generally secondary to processes invasive to renal parenchyma or renal vascular system (approximately 70%) such as biopsy, percutaneous nephrostomy, and trauma or may be congenital.[1] The proper management is controversial. Patients with large AVFs and symptomatic patients are referred for treatment. Eradication of the AVF and consequent symptoms of renal AVF along with preservation of renal parenchyma are the main aims of the treatment. Indications for treatment are progressive increase in the size of AVF; non-resolving hematuria; and hemodynamic features, especially decompensation, hypertension, and high-output heart failure.[2]

Surgery in the form of arterial feeder ligation and total or partial nephrectomy is considered as the last resort...
of treatment. Coil embolization is now the standard endovascular approach to the management of symptomatic AVF. However, transcatheter embolization of large, high-flow AVF always carries a significant risk for migration of embolic material into the pulmonary arteries. This risk can be minimized with the use of an amplatzer vascular plug (AVP). AVP has many advantages over other embolic materials. Its position after the release is checked with contrast injection and can be retracted, if required, and be repositioned. Its migration risk is less than those of coils. Limitations of AVP include need of a 5–9 F sheath and unsuitability of AVP for vessels that are too small or too large. This case illustrates the feasibility of the use of AVP in the treatment of renal AVF.

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Conflicts of interest
There are no conflicts of interest.

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References

Embryogenesis of vagina and embryopathogenesis of Herlyn–Werner–Wunderlich syndrome
Sir,
I read with great interest the article titled, “Herlyn–Werner–Wunderlich syndrome presenting with infertility: Role of MRI in diagnosis” by Ahmad et al. published in the Indian Journal of Radiology and Imaging. The manuscript is excellent and informative. However, I would like to make the following contribution.

Herlyn–Werner–Wunderlich syndrome comprises of uterus didelphys, obstructed hemivagina and ipsilateral renal agenesis/anomaly. Hence, the acronym OHVIRA syndrome. The embryopathogenesis of OHVIRA syndrome is debatable. While the classical theory puts forth Mullerian (paramesonephric) roots of upper vagina, Acien’s hypothesis postulates mesonephric (Wolfian) origin of vagina in entirety except its lining epithelium from the Mullerian tubercle.

Common to both, the traditional and Acien’s view is formation of the kidney and the uterus with cervix. The former develops as a result of inductive effect of metanephric blastema (derived from the Wolfian duct at 5 weeks of gestation) into metanephros; for the latter, the mesonephros allows for proper positioning and subsequent caudal fusion of paramesonephric ducts. Wolfian birth of vagina, put forth by Acien (and supported by experiments of Sanchez on female rats), explains OHVIRA as a faulty development of mesonephros that results in three-fold effects; (a) failure of formation of metanephric blastema from the Wolfian duct and hence subsequent renal agenesis/anomaly, (b) absence of paramesonephric ducts, and (c) failure of fusion of paramesonephric ducts due to mesonephric ducts, resulting in obstructed hemivagina and ipsilateral renal anomalies.

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