The identification and management of the profunda femoris artery as a rare source of a late type II endoleak

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ABSTRACT

Internal iliac artery aneurysms are found in 20% of abdominal aortic aneurysm cases, with a high mortality rate in the event of rupture. Type II endoleaks are a common complication after endovascular intervention. Transarterial or direct sac puncture techniques have superseded open surgical repair due to the challenging nature open surgery presents in accessing the feeding vessel(s). We describe the rare source of a late type II endoleak feeding from the profunda femoris in an 83-year-old man after fenestrated endovascular aortic aneurysm repair and concurrent embolization of the right internal iliac artery for treatment of a juxtarenal abdominal aortic aneurysm and internal iliac artery aneurysm. (J Vasc Surg Cases Innov Tech 2021;7:759-62.)

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Since their inception in the 1990s, endoleaks have become a well-described complication associated with endovascular aortic aneurysm repair (EVAR), particularly with repair of complex aneurysms. Further subclassified into five subtypes, type I and III endoleaks pose the highest risk of aneurysmal rupture due to the ongoing perfusion of the native aneurysmal sac, generally requiring prompt reintervention. Type II endoleak (T2E) remains the most commonly encountered, occurring in up to 27% of EVAR.[1] However, unlike its counterparts, the management of T2E remains debatable, with the EUROSTAR registry demonstrating no statistical difference in aneurysm rupture rates at 2 years in patients with and without T2E.[2] Therefore, most T2E are managed conservatively, with spontaneous thrombosis seen in up to 50% of cases.[1,2] We describe a unique case of the profunda femoris artery as a cause of a T2E in a previously excluded, expanding internal iliac artery aneurysm (IIAA). Consent for publication and images used was obtained from the patient.

CASE REPORT

An 83-year-old man with a background history of atrial fibrillation, tachy-brady syndrome requiring a permanent pacemaker, coronary artery bypass grafting, ischemic cardiomyopathy, chronic renal impairment, obstructive sleep apnea, hypertension, and dyslipidemia was referred to our center 5 years ago after the incidental discovery of an asymptomatic juxtarenal 3.9 cm abdominal aortic aneurysm (AAA) with an associated right common iliac artery aneurysm (CIAA) measuring 2.9 cm and right IIAA measuring 3.0 cm (Fig 1). Given the anatomy and size of the iliac aneurysms, prompt open repair was recommended; however, this was delayed by the need for cardiac intervention and patient preference.

After the initial review, he underwent coronary artery bypass grafting and subsequently required replacement of his permanent pacemaker. This was complicated by a cerebral vascular event postoperatively, requiring a period of rehabilitation, further delaying the possibility for vascular intervention. Computed tomography angiography (CTA) 18 months later demonstrated an interval increase in size of the AAA, CIAA, and IIAA, now measuring 5.5 cm, 3.7 cm, and 4.4 cm, respectively (Fig 1). The infrarenal neck was unsuitable for standard EVAR, measuring less than 9 mm from the lowest left renal artery (RA). He underwent fenestrated endovascular aortic repair with a custom-made four-vessel fenestrated Cook Alpha Endograft with stenting of the superior mesenteric artery and bilateral RA with Bentley BeGrafts via bilateral femoral artery access. Intraoperatively, good apposition was noted between the aortic wall and coeliac fenestration with decision not to proceed with stenting due to ongoing difficulty tracking a sheath into the coeliac artery. The right IIA was cannulated and occluded with Abbott Amplatzer Vascular Plugs to the anterior and posterior divisions of the IIA. The right limb of the graft was extended into the mid-right external iliac artery and the left to the distal CIA with Cook Zenith Alpha limbs (ZISL-11-93 and ZISL-20-77, respectively). Completion angiogram demonstrated a possible

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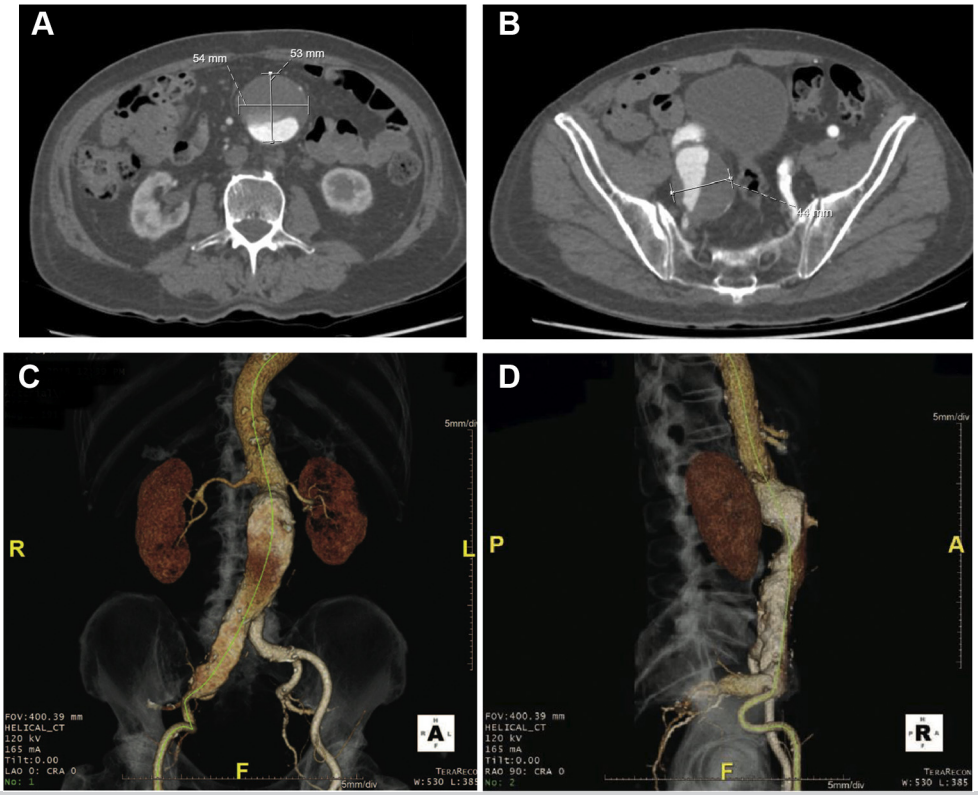

Fig 1. Computed tomography angiogram (CTA) demonstrating the initial infrarenal aneurysmal and right common iliac aneurysm **(A, B)** with interval increase in size over the following 18 months. Preoperative 3D centerline reconstructions demonstrating the angulated neck with minimal proximal seal zone inadequate and the large right common and internal iliac artery aneurysm (IIAA) **(C, D)**.

type 1A endoleak; however, because of the difficulty of the procedure, radiation exposure, and contrast load, intraoperative decision was made for progress CTA and reintervention at a later date (Fig 2). The procedure was complicated by maldeployment of the closure device on the right, requiring open repair of the femoral access site.

Day 4 postoperatively, he underwent a CTA that did not demonstrate any further endoleak on arterial and delayed phases, with a patent aortic endograft and visceral stents. He was discharged on day 11 postprocedure after inpatient rehabilitation. He was followed up at 1 and 6 months with aortoiliac duplex ultrasound (DU) that did not demonstrate any endoleaks and stable aneurysm sac size. At 12-month follow-up, he remained asymptomatic; however, DU demonstrated a possible T2E, and no growth of the residual aneurysm sac size was noted. This was managed conservatively with planned repeat imaging in 3 months. At 15-month review, DU demonstrated enlargement of the right CIAA, which was further correlated with CTA, which demonstrated an interval increase in the residual AAA sac, measuring 57 $\times$ 58 mm as well as the right CIAA measuring 42 $\times$ 39 mm likely from a T2E.

Selective digital subtraction angiogram via a left radial approach identified two culprit feeding arteries to the right IIA endoleak, namely, a branch of the left superior gluteal artery and a branch of the right profunda femoris artery (Fig 3; Supplementary Video 1, online only). The profunda femoris artery was confirmed to be contributing to the endoleak on selective angiography. After case review at a multidisciplinary meeting, he underwent embolization of the right IIA nidus with 32 mL of Onyx 34 via US-guided direct sac puncture. At 1- and 2-month follow-up, he remained well, with aortoiliac DU demonstrating no further endoleak and slight regression of sac size. He is planned for further review at 6 months after embolization with a surveillance DU.

DISCUSSION

T2E refer to the continued retrograde sac filling from branch vessels. T2E are further subclassified into types IIa and IIb, referring to a single causative or multiple causative vessels.[3] Given the high rate of spontaneous resolution, the accepted criteria for T2E intervention include persistent endoleak for greater than 6 months, sac enlargement >5 mm, large aneurysmal nidus, >3 feeding arteries, or a feeding artery diameter >4 mm.[1,3] Recent literature however suggests that delayed T2E, defined as >1 year after EVAR, are less likely to resolve than early T2E, requiring early intervention in the presence of sac growth.[4]

Treatment options for T2E include embolization of the feeding vessels and nidus via transarterial or translumbar

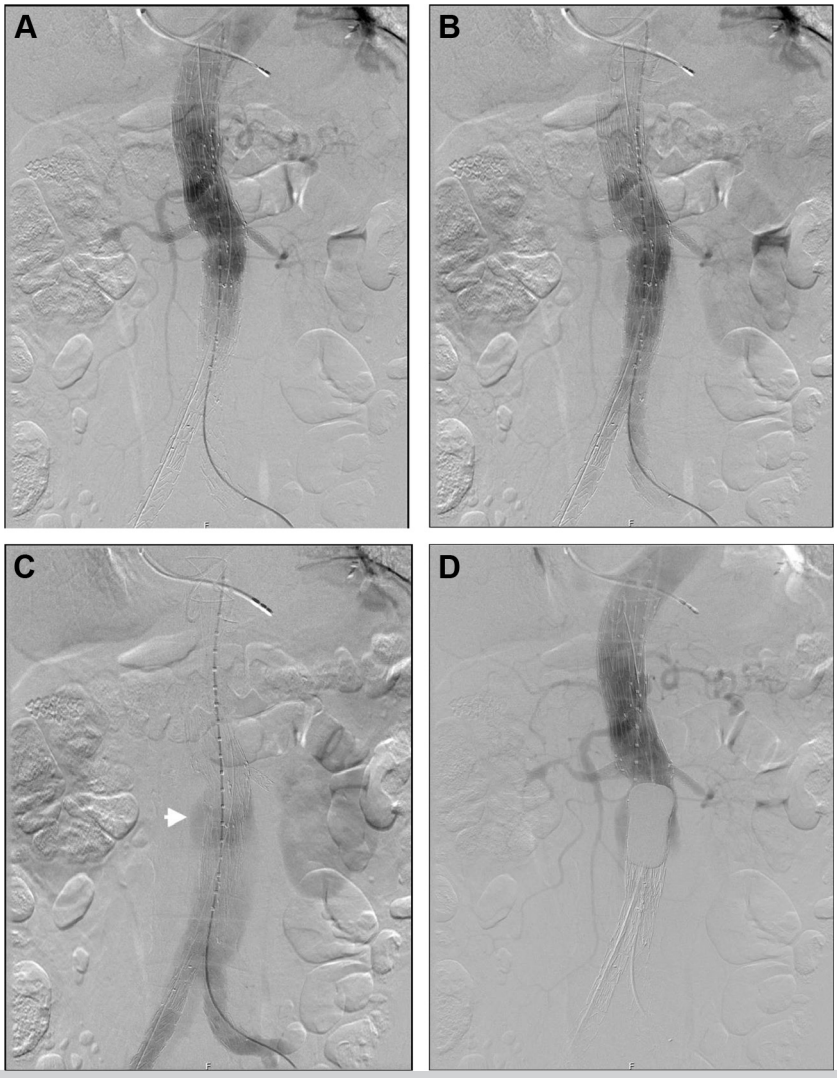

Fig 2. Completion digital subtraction angiography intraoperatively demonstrating the patent fenestrated endograft with patency of the visceral branches **(A, B)**; however, also noted is a type 1A endoleak (**C**; *arrow*) likely from the unstented coeliac fenestrated. This was further confirmed with balloon-occlusion angiography **(D)**.

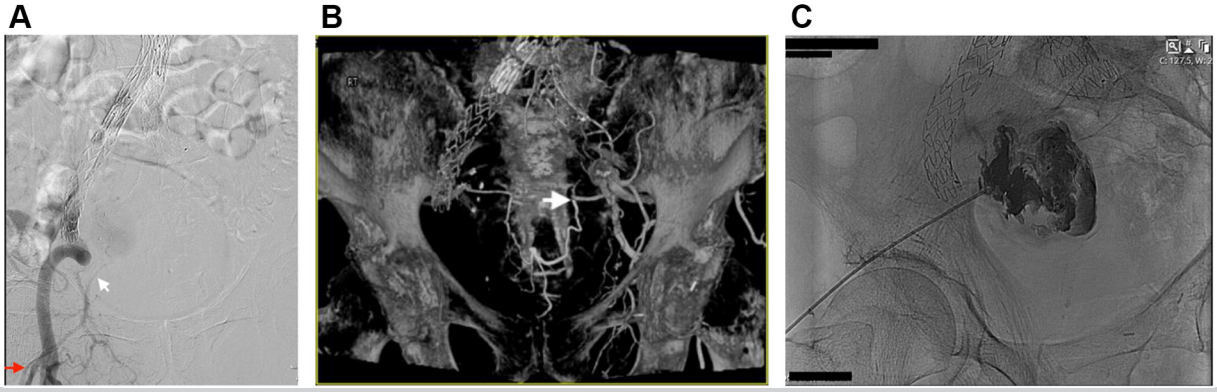

Fig 3. Selective angiography demonstrating the culprit vessels feeding the internal iliac artery type 2 endoleak from the profunda femoris artery (**A**: *red* and *white arrows*) and left superior gluteal artery (**B**; *white arrow*) and embolization with Onyx via direct sac puncture **(C)**.

access. The route of embolization is limited by the anatomy of the feeding vessels; thus, selective angiography to delineate the degree of collateralization and access vessel tortuosity is often required.[5]

The inferior mesenteric artery and lumbar arteries are the most frequently encountered vessels responsible for T2E. Less frequently encountered sources of retrograde flow include an accessory RA, gonadal artery, median sacral artery, and the IIA if not embolized before deployment of a limb into the ipsilateral external iliac artery.[6] However, the profunda femoris artery has yet to be published in the literature as a source for a late type II endoleak.[7] We describe this case to highlight the possibility of the profunda femoris artery as a cause for type IIa endoleaks, which should be considered in the event of a delayed T2E.

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