7. Brcic I, Brcic L, Kuzmanic D, Coric M, Coric M. Fibronectin glomerulopathy in a 34-year-old man: a case report. Ultrastruct Pathol. 2010;34:240–2.
8. Nadamuni M, Piras R, Mazbar S, Higgins JP, Kambham N. Fibronectin glomerulopathy: an unusual cause of adult-onset nephrotic syndrome. Am J Kidney Dis. 2012;60:839–42.
9. Otsuka Y, Takeda A, Horike K, Inaguma D, Goto N, Watarai Y, et al. A recurrent fibronectin glomerulopathy in a renal transplant patient: a case report. Clin Transplant. 2012;26 Suppl 24:58–63.
10. Ertoy BD, Kutlugun AA, Bresin E, Piras R. A case of familial glomerulopathy with fibronectin deposits caused by the Y973C mutation in fibronectin. Am J Kidney Dis. 2013;61:514–8.
11. Yoshino M, Miura N, Ohnishi T, Suzuki K, Kitagawa W, Nishikawa K, et al. Clinicopathological analysis of glomerulopathy with fibronectin deposits (GFND): a case of sporadic, elderly-onset GFND with codeposition of IgA, C1q, and fibrinogen. Intern Med. 2013;52:1715–20.
12. Assmann KJ, Koene RA, Wetzels JF. Familial glomerulonephritis characterized by massive deposits of fibronectin. Am J Kidney Dis. 1995;25:781–91.
13. Gemperle O, Neuweiler J, Reutter FW, Hildebrandt F, Krapf R. Familial glomerulopathy with giant fibrillar (fibronectin-positive) deposits: 15-Year follow-up in a large kindred. Am J Kidney Dis. 1996;28:668–75.
14. Ishimoto I, Sohara E, Ito E, Okado T, Rai T, Uchida S. Fibronectin glomerulopathy. Clin Kidney J. 2013;6:513–5.
15. Sato H, Matsubara M, Marumo R, Soma J, Kurosawa K, Taguma Y, et al. Familial lobular glomerulopathy: first case report in Asia. Am J Kidney Dis. 1998;31:E3.
16. Chen H, Bao H, Xu F, Zhu X, Zhu M, He Q, et al. Clinical and morphological features of fibronectin glomerulopathy: a report of ten patients from a single institution. Clin Nephrol. 2015;83:93–9.

Genyang Cheng[a,b,1], Zheng Wang[a,b,1], Wenming Yuan[a,b], Yanna Dou[a,b], Dong Liu[a,b], Jing Xiao[a,b], Zhanzheng Zhao[a,b,*]

[a] *The Nephrology Center of the First Affiliated Hospital of Zhengzhou University, Zhengzhou, China*
[b] *Zhengzhou University Institute of Nephrology, Zhengzhou, China*

* *Corresponding author.*
E-mail addresses: 13938525666@139.com, zhanzhengzhao@zzu.edu.cn (Z. Zhao).
[1] These authors contributed equally to this work.

http://dx.doi.org/10.1016/j.nefroe.2017.01.008


# True brachial artery aneurysm following vascular access for haemodialysis in renal transplant patient. Two case reports☆

## Aneurisma humeral verdadero en relación con acceso vascular en paciente trasplantado renal: a propósito de 2 casos clínicos

*Dear Editor,*

Humeral artery aneurysms (HAA) are an uncommon condition, usually secondary to trauma, infection or connective tissue disease.[1,2] They have also been described in the context of arteriovenous fistulas (AVF) for haemodialysis; the majority are anastomotic or venous pseudoaneurysms, with few true degenerative arterial aneurysms. We present two cases of true HAA:

Case one is a 44-year-old male with rapidly progressive glomerulonephritis who started haemodialysis at age 20 (1978) through a left radiocephalic AVF. During the course of his illness he received two kidney transplants. In 1996 a new radiocephalic AVF was performed on the contralateral arm due to thrombosis of the previous fistula. In 2003, an asymptomatic true HAA of 6 cm diameter was detected and confirmed by arteriography. Aneurysmal resection was performed with interposition of the inverted internal saphenous vein extracted from the left lower extremity. Two years later, a 4.5 cm aneurysmal dilatation of the venous graft was detected (Fig. 1A) and it was repaired by PTFE prosthesis interposition. During the follow-up, no other complications were detected, with exitus in 2009 from metastatic clear-cell renal cell carcinoma.

Case two is a 55-year-old male, former smoker and with uncontrolled arterial hypertension, who started haemodialysis at age 40 (2000) through a left radiocephalic AVF due to malignant nephroangiosclerosis, receiving a kidney transplant in 2001. He had a past medical history of popliteal aneurysm in the right lower limb, operated with a

DOI of original article:
http://dx.doi.org/10.1016/j.nefro.2016.09.009.
☆ Please cite this article as: Fernández Prendes C, Zanabili Al-Sibbai AA, González Gay M, Carreño Morrondo JA, Alonso Pérez M. Aneurisma humeral verdadero en relación con acceso vascular en paciente trasplantado renal: a propósito de 2 casos clínicos. Nefrologia. 2017;37:96–98.

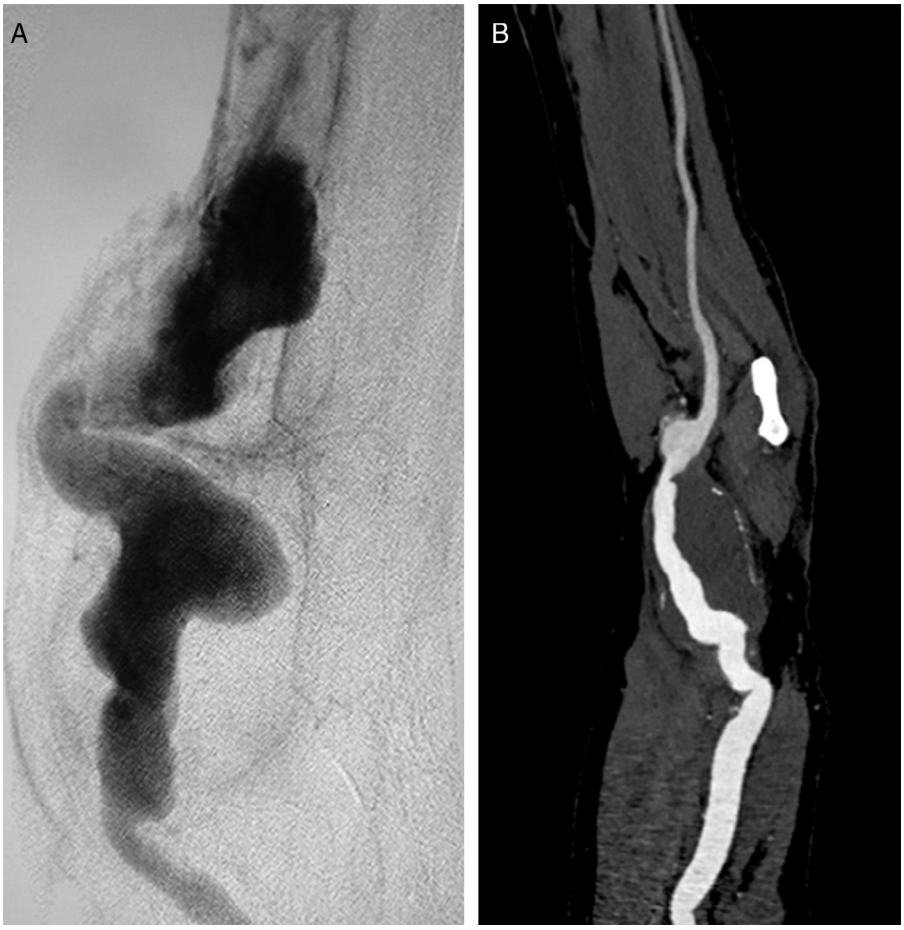


**Fig. 1 – Selective arteriography: (A) humeral artery with a maximum diameter of 6 cm. (B) Three-dimensional reconstruction of computerised angiotomography, showing aneurysmal dilatation of the humeral artery, with a maximum diameter of 4.5 cm and a single outlet through the ulnar artery.**

femoropopliteal bypass with inverted saphenous vein, which required supracondylar amputation due to acute thrombosis. The AVF was closed in 2007 due to vein aneurysmal degeneration. In 2015, an asymptomatic true HAA of 4.5 cm in diameter was documented by Doppler ultrasound and confirmed by computerised axial angiotomography (CT angiography) (Fig. 1B). Aneurysmal resection and inverted homolateral basal vein interposition were performed. At the 6-month follow-up, the bypass remained permeable and free of complications.

HAAs have been described in the context of AVF for haemodialysis, although most are anastomotic or venous pseudoaneurysms. True degenerative aneurysms are very rare, with an estimated incidence of 0.17%.[1] True HAAs are most often associated with radiocephalic AVFs (60%), followed by brachiocephalic AVFs (36%), and generally appear 7–19 years after it placement.[3] Unlike aortic, femoral, and popliteal aneurysms, HAAs do not appear to be associated with synchronous aneurysms in other locations.[2] All of this suggests a etiopathogenic origin different from other true degenerative aneurysms.

Several mechanisms have been described that cause a significant increase in arterial diameter after the creation of an AFV; an increase in intra-arterial blood flow generates fissures in the elastic fibres in the internal lamina creating a predisposition to aneurysmal degeneration; in addition, an increase in the wall tension increases the release of endothelial factors such as nitric oxide.[1,4,5] These mechanisms are neither prevented nor avoided by closure or thrombosis of the AVF.[3] Kidney transplantation has been associated with aneurysmal arterial progression proximal to the AVF, while treatment with steroids and immunosuppressants has also been associated with an increased incidence of HAA.[4,6,7] The two cases presented here were kidney transplant patients who received immunosuppressive therapy for more than 10 years and developed HAA 15 and 25 years after creation of the AVF, respectively.

Their most frequent clinical manifestation is in the form of an asymptomatic pulsatile mass, although they can cause pain and paresthesias due to local compression. Distal embolisation has been observed in 28–30% of cases, while the other ischaemic presentations are unusual and rupture is extremely rare.[3,8,9] The initial diagnostic test is colour Doppler ultrasonography, although CT angiography is the most commonly used technique for surgical planning.[10]

Due to their low frequency, therapeutic indications are usually based on those accepted for popliteal aneurysms. Surgery is generally indicated for asymptomatic HAAs measuring 3 cm or more. For those measuring 2–3 cm, surgery is

recommended if compressive symptoms or distal embolisation are present.[8] First-line treatment is usually aneurysmal resection, maintaining arterial continuity by direct suture if possible. If revascularisation is required, the use of autologous grafts is preferred; if these are unavailable, prosthetic grafts or allografts would be used.[3,8–10] In a systematic review published in 2015, with 12 selectable articles and 23 cases described in total, the mean permeability was 12 months (1–38 months) after autografting and 6 months (1–48 months) after PTFE interposition.[10]

Although systematic monitoring by ultrasound in patients with an AVF is not recommended, probably because it is not cost-effective; however it would be reasonable to perform a physical examination of the AVF at follow-up visits or at haemodialysis sessions.[9] If aneurysmal degeneration is suspected it would be advisable to request a ultrasound to make early diagnosis and avoid possible thromboembolic and/or compressive complications.

## REFERENCES


1. Schunn CD, Sullivan TM. Brachial arteriomegaly and true aneurysmal degeneration: case report and literature review. Vasc Med. 2002;7:25–7.
2. Gray RJ, Stone WM, Fowl RJ, Cherry KJ, Bower TC. Management of true aneurysms distal to the axillary artery. J Vasc Surg. 1998;28:606–10.
3. Chemla E, Nortley M, Morsy M. Brachial artery aneurysms associated with arteriovenous access for hemodialysis. Semin Dial. 2010;23:440–4.
4. Eugster T, Wigger P, Bölter S, Bock A, Hodel K, Stierli P. Brachial artery dilatation after arteriovenous fistulae in patients after renal transplantation. A 10-year follow-up ultrasound scan. J Vasc Surg. 2003;37:564–7.
5. Dammers R, Tordoir JH, Welten RJ, Kitslaar PJ, Hoeks AG. The effect of chronic flow changes on brachial artery diameter and shear stress in arteriovenous fistulas for hemodialysis. Int J Artif Organs. 2002;25:124–8.
6. Reilly JM, Savage EB, Brophy CM, Tilson MD. Hydrocortisone rapidly induces aortic rupture in a genetically susceptible mouse. Arch Surg. 1990;125:707–9.
7. Sato O, Takagi A, Miyata T, Takayama Y. Aortic aneurysms in patients with autoimmune disorders treated with corticosteroids. Eur J Vasc Endovasc Surg. 1995;10:366–9.
8. Marzelle J, Gashi V, Nguyen HD, Mouton A, Becquemin JP, Bourquelot P. Aneurysmal degeneration of the donor artery after vascular access. J Vasc Surg. 2012;55:1052–7.
9. Mestres G, Fontsere N, Yugueros X, Tarazona M, Ortiz I, Riambau V. Aneurysmal degeneration of the inflow artery after arteriovenous access for hemodialysis. Eur J Vasc Endovasc Surg. 2014;48:592–6.
10. Kordzadeh A, Espiney Barbara R, Ahmad A, Hanif M, Panayiotopoulos Y. Donor artery aneurysm formation following the ligation of haemodialysis arteriovenous fistula: a systematic review and case reports. J Vasc Access. 2015;16:5–12.



Carlota Fernández Prendes*, Ahmad Amer Zanabili Al-Sibbai, Mario González Gay, Jose Antonio Carreño Morrondo, Manuel Alonso Pérez

*Servicio de Angiología y Cirugía Vascular, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain*

* *Corresponding author.*
E-mail address: carlota.f.prendes@gmail.com (C. Fernández Prendes).


http://dx.doi.org/10.1016/j.nefroe.2017.01.024


# New microorganism in catheter-related bacteremia?☆

## ¿Nuevo microorganismo en la bacteriemia asociada a catéter?

*Dear Editor,*

In recent years, there is an increased prevalence of patients with advanced chronic kidney disease (ACKD) carrying tunneled central venous catheters (CVC) for haemodialysis.[1,2]

Catheter-related bacteraemia (CRB) is one of the major causes of morbidity and mortality in these patients.[2]

The microorganisms responsible for two-thirds of these bacteraemias are gram-positive bacteria,[2] not forgetting the other microorganisms.

*Ochrobactrum anthropi* is a gram-negative, non-fermenting, aerobic, mobile, oxidase- and urease-positive bacillus.[3] It is ubiquitous in nature and can be found in hospital environments. Several cases of bacteraemia associated with this microorganism have been described in immunocompromised patients, although some cases have also occurred in immunocompetent patients.[4,5] Its isolation as the cause of CRB is rare.

Two cases of bacteraemia due to this microorganism in recent months are described below in patients with a CVC for haemodialysis.

☆ Please cite this article as: Torres Aguilera E, Verde Moreno E, Muñoz P, Valerio M, Luño J. ¿Nuevo microorganismo en la bacteriemia asociada a catéter? Nefrologia. 2017;37:98–100.