3. Jenne CN, Urrutia R, Kubes P. Platelets: bridging hemostasis, inflammation, and immunity. Int J Lab Hematol. 2013;35:254–61.
4. Okyay GU, Inal S, Oneç K, Er RE, Paşaoğlu O, Paşaoğlu H, et al. Neutrophil to lymphocyte ratio in evaluation of inflammation in patients with chronic kidney disease. Ren Fail. 2013;35:29–36.
5. Martínez-Urbistondo D, Beltrán A, Beloqui O, Huerta A. The neutrophil-to-lymphocyte ratio as a marker of systemic endothelial dysfunction in asymptomatic subjects. Nefrologia. 2016;36:397–403.
6. Reddan DN, Klassen PS, Szczech LA, Coladonato JA, O'Shea S, Owen WF Jr, et al. White blood cells as a novel mortality predictor in haemodialysis patients. Nephrol Dial Transplant. 2003;18:1167–73.
7. Huarte-Loza E, Barril-Cuadrado G, Cebollada-Muro J, Cerezo-Morales S, Coronel-Díaz F, Doñate-Cubells T, et al. Grupo de Consenso de la Sociedad Española de Diálisis y Trasplante Nutrición en pacientes en diálisis. Consenso SEDYT. Dial Traspl. 2006;27:138–61.
8. Lou M, Luo P, Tang R, Peng Y, Yu S, Huang W, et al. Relationship between neutrophil-lymphocyte ratio and insulin resistance in newly diagnosed type 2 diabetes mellitus patients. BMC Endocr Disord. 2015;15:9.
9. Turkmen K, Erdur FM, Ozcicek F, Ozcicek A, Akbas EM, Ozbicer A, et al. Platelet-to-lymphocyte ratio better predicts inflammation than neutrophil-to-lymphocyte ratio in end-stage renal disease patients. Hemodial Int. 2013;17:391–6.

Venice Chávez Valencia[a,c,*], Citlalli Orizaga de la Cruz[a], Oliva Mejía Rodríguez[b], Sergio Gutiérrez Castellanos[a], Francisco Alejandro Lagunas Rangel[c], Martha Eva Viveros Sandoval[c]

[a] *Hospital General Regional No. 1, Instituto Mexicano del Seguro Social (IMSS), Morelia, Michoacán, Mexico*
[b] *Centro de Investigación Biomédica de Michoacán, Morelia, Michoacán, Mexico*
[c] *Universidad Michoacana de San Nicolás de Hidalgo, Facultad de Ciencias Médicas y Biológicas, Maestría en Ciencias de la Salud, Morelia, Michoacán, Mexico*

[*] *Corresponding author.*
E-mail address: drvenicechv@yahoo.com.mx
(V. Chávez Valencia).

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Embolism as a cause of early thrombosis of arteriovenous fistula of hemodialysis patients☆

Embolismo paradójico como causa de trombosis precoz de fístula arteriovenosa para hemodiálisis

Dear Editor,

Thrombosis of the vascular access (CA) is usually due to technical problems and/or small arterial or venous size, obesity, advanced age, diabetes mellitus, female sex and AVF in the wrist.

We present a case, not previously described, of early AVF thrombosis secondary to paradoxical embolism caused by a patent foramen ovale (PFO). He is an 80-year-old man; after 7 years in PD is switched to because of poor dose of dialysis. A left humerus-perforan vein AVF was made that quickly thrombosed at 24 h. During Re-intervention it was observed permeable vessels and a thrombus are detected in the middle third of the humeral artery suggesting an arterial embolism. Thromboembolectomy was successful and anticoagulation was prescribed. The ECG presented sinus rhythm and the echocardiography showed no abnormalities. Transesophageal echocardiography (TEE) is performed, showing PFO with early bubbles after the injection of agitated saline solution. In the descending thoracic aorta, a giant fibrocalcified atherosclerosis plaque with irregularities and thrombotic content occupying 40% of the light was observed.

Thereafter he presented transient cerebrovascular event with loss of intellectual abilities with lower limb chronic small vessel ischemia and injuries suggestive of cholesterol crystals atheroembolism which caused with severe pain, livedo reticularis, cyanotic punctate lesions and cyanosis of the first toe of the left foot with preserved pulses. Then, Sintrom® was discontinued and it was changed to subcutaneous LMWH 60 mg/24 h, aspirin and clopidogrel were added and statins dose was increased to 80 mg daily as recommended by vascular surgery. The patients lost 15 kg in 8 months, albumin 2.1 g/L and CRP was 57 mg/L with severe anemia despite increased EPO. The patient decided to voluntarily stop HD

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Paradoxical embolism as the cause of AV thrombosis has not previously been described in the literature. Arterial embolism is a rare cause of fistula thrombosis,[1] and even less frequent is paradoxical embolism,[2] which is the passage of a venous thrombus located in the right cardiac cavities to the arterial circulation through a cardiac defect. The most frequent is PFO, with a prevalence of 25% by autopsy and 35% by TEE.[3]

The definitive diagnosis is made if a thrombus going through a septum defect is observed by ultrasound, but a diagnosis also made by: (a) systemic arterial embolism, in the absence of atrial fibrillation, from source located in the left heart cavities or proximal arteries; (b) right-to-left short circuit at any level, and (c) venous thrombosis and/or pulmonary embolism or (d) if PFO is detected, as in the present case.[3]

Echocardiogram is one of the most useful tools for the diagnosis of emboligial heart disease. With the Valsalva maneuver and the introduction of intravenous contrast a PFO can be identified (sensitivity: 60% and specificity: 78%).[3] The diagnostic possibilities improve with TEE.

The treatment of PFO is anticoagulation and, in selected cases, surgical closure or percutaneous closure, with a morbidity of 10%, may be considered.

One important finding in this patient was the presence of a large fibrocalcified atherosclerotic plaque in the descending aorta containing a thrombus. This type of plaques is associated with a high risk of stroke and peripheral embolization.[4] In renal patients abdominal aorta calcification is observed in 81% of patients and it is severe in 51% of cases.[5] Vascular calcification is an independent risk factor for cardiovascular mortality. These plaques are the cause of 2 types of emboli: thromboembolism and atheroemboli (emboli by cholesterol crystals). The latter are characterized by the release of small emboli either spontaneously (25%) or provoked by interventions such as cardiac catheterization, arteriography, peripheral interventions, or cardiac surgery.[6] In this case, the fistula intervention may have been a precipitating factor.

Mortality in patients with extensive atherosclerotic plaques in the thoracic aorta is 20% at 3 years, cerebrovascular events are observed in 20% of patients, other types of embolic events are seen in 7% and 1% may have cholesterol crystals embolism.[6]

In patients with atherosclerotic aortic plaques, a high incidence of atheroembolism has been attributed to anticoagulants treatment; however recent randomized clinical trials do not seem to confirm this.[7]

The treatment includes antithrombotic therapy and statins. Presently is recommended either with aspirin 50–325 mg daily or clopidogrel 75 mg daily versus the combined treatment[8] or warfarin administration.[9] There are no data on the use of non-vitamin K dependent oral anticoagulants and therefore they are not recommended.

This case illustrates an exceptional complication of AVF, but given the high frequency of atherosclerosis in HD patients it should be considered.

REFERENCES

1. Bordils A, García R, Fonseca JA, Mcclean R, Campos C, Abad N, et al. Embolismo arterial de fístula arteriovenosa en paciente en hemodiálisis. NefroPlus. 2009;2:40–2.
2. Loscalzo J. Paradoxical embolism: clinical presentation, diagnostic strategies, and therapeutic options. Am Heart J. 1996;112:141–5.
3. Hara H, Virmani R, Ladich E, Mackey-Bojack S, Titus J, Reisman M, et al. Patent foramen ovale: current pathology, pathophysiology, and clinical status. J Am Coll Cardiol. 2005;46:1768–76.
4. Tunick PA, Rosenzweig BP, Katz ES, Freedberg RS, Perez JL, Kronzon I. High risk for vascular events in patients with protruding aortic atheromas: a prospective study. J Am Coll Cardiol. 1994;23:1085–90.
5. Honkanen E, Kauppila L, Wikström B, Rensma PL, Krzesinski JM, Aasarod K, et al., CORD Study Group. Abdominal aortic calcification in dialysis patients: results of the CORD study. Nephrol Dial Transplant. 2008;23:4009–15.
6. Scolari F, Ravani P, Gaggi R, Santostefano M, Rollino C, Stabellini N, et al. The challenge of diagnosing atheroembolic renal disease: clinical features and prognostic factors. Circulation. 2007;116:298–304.
7. Blackshear JL, Zabalgoitia M, Pennock G, Fenster P, Strauss R, Halperin J, et al. Warfarin safety and efficacy in patients with thoracic aortic plaque and atrial fibrillation. SPAF TEE Investigators. Stroke Prevention and Atrial Fibrillation. Transesophageal echocardiography. Am J Cardiol. 1999;83:453–5.A9.
8. Amarenco P, Davis S, Jones EF, Cohen AA, Heiss WD, Kaste M, et al., Aortic Arch Related Cerebral Hazard Trial Investigators. Clopidogrel plus aspirin versus warfarin in patients with stroke and aortic arch plaques. Stroke. 2014;45:1248–57.
9. Bhatt DL, Fox KA, Hacke W, Berger PB, Black HR, Boden WE, et al., CHARISMA Investigators. Clopidogrel and aspirin versus aspirin alone for the prevention of atherothrombotic events. N Engl J Med. 2006;354:1706–17.

María Dolores Arenas[a,*], Rebeca Pérez[b], Jose Angel Rodriguez[c]

[a] *Servicio de Nefrología, Vithas Hospital Internacional Perpetuo, Alicante, Spain*
[b] *Servicio de Cirugía Vascular, Vithas Hospital Internacional Perpetuo, Alicante, Spain*
[c] *Servicio de Cardiología, Vithas Hospital Internacional Perpetuo, Alicante, Spain*

[*] *Corresponding author.*
E-mail address: lola@olemiswebs.net (M.D. Arenas).

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