

11. National Institute for Clinical Excellence. Principles for best practice in clinical audit. Oxon: Radcliffe Medical Press; 2004. p. 1–196.
12. Jamtvedt G, Young JM, Kristoffersen DT, O'Brien MA, Oxman AD. Does telling people what they have been doing change what they do? A systematic review of the effects of audit and feedback. *Qual Saf Health Care*. 2006;15:433–6.
13. De Vos M, Graafmans W, Kooistra M, Meijboom B, Van Der Voort P, Westert G. Using quality indicators to improve hospital care: a review of the literature. *Int J Qual Health Care*. 2009;21:119–29.

I. Pomar Ladaria^a, Y. Armendáriz Cuevas^a,
J. Lopez Pisón^{a,b,*}, L. Monge Galindo^{a,b}

^a *Unidad de Neuropediatría, Hospital Universitario Miguel Servet, Zaragoza, Spain*

^b *Instituto Aragonés Ciencias de la Salud, Hospital Universitario Miguel Servet, Zaragoza, Spain*

*Corresponding author.

E-mail address: jlopezpi@salud.aragon.es (J. Lopez Pisón).

doi:10.1016/j.nrleng.2011.01.014

Extracranial internal carotid artery aneurysm: An uncommon disease of the supra-aortic arteries[☆]

Aneurisma de arteria carótida interna extracraneal. Una patología infrecuente de los troncos supraaórticos

Sir,

Carotid aneurysm is a rare pathology with an incidence of 0.3% among aneurysms in general. Many cases remain asymptomatic until ischemic neurological symptoms appear, so their early detection and treatment are important. Their most common location is at the level of the carotid bulb and in the proximal internal carotid,¹ and the treatment of choice is open surgery with exclusion of the aneurysm and restoration of circulation through a graft.

We report a patient with an aneurysm of the extracranial internal carotid artery, which was treated by resection and graft interposition.

The patient was a 67-year-old male who was referred to our outpatient service due to the incidental finding of an abdominal aortic aneurysm (AAA), 4.8 cm in size, during the course of an ultrasound study for toxic syndrome.

The patient presented the following as history: allergy to penicillin and pyrazolones, arterial hypertension, former smoker, former drinker, COPD, chronic liver disease, chronic anaemia, vertigo syndrome and non-specific memory loss disorder.

A vascular examination found increased left carotid pulse, soft painless abdomen with a pulsating mass and distal pulses present at both ends.

Vascular studies were initiated with full blood tests, abdominal computed tomography angiography (CTA), ankle-brachial indices and Doppler ultrasound of the supra-aortic trunks (SAT).

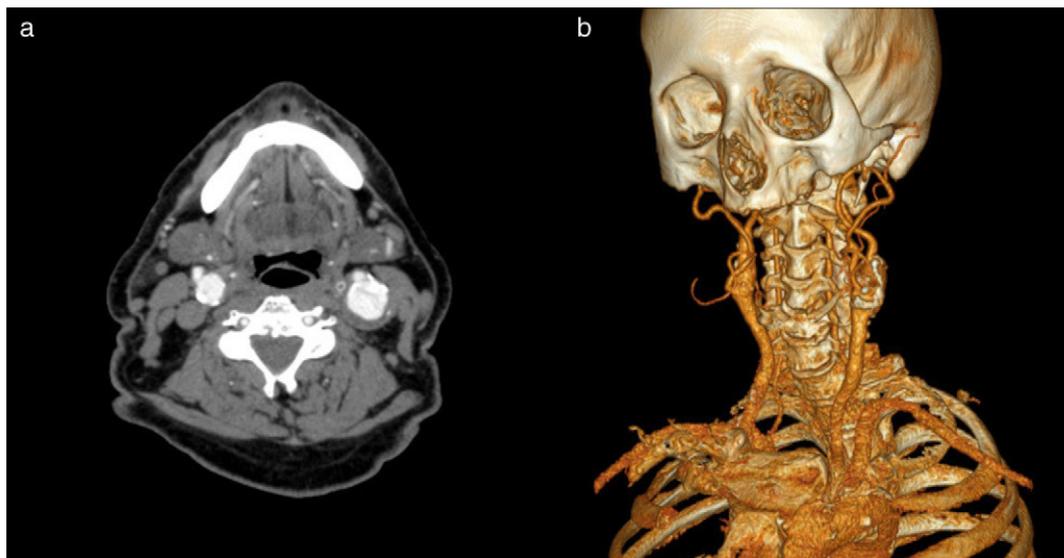


Figure 1 Cervical CTA. Axial section of the left side (a) and 3D reconstruction of the right side (b), which demonstrates the existence of left carotid aneurysm.

[☆] Please cite this article as: Argilés Mattes N, et al. Aneurisma de arteria carótida interna extracraneal. Una patología infrecuente de los troncos supraaórticos. *Neurología*. 2011;27:53–55.



Figure 2 Selective left carotid arteriography.

The abdominal CTA confirmed the presence of an infrarenal AAA, of 4.5 cm maximum size, with elongation of common iliac arteries. It also revealed the presence of hepatosplenomegaly with ascites and mediastinal and abdominal lymph node formations. The patient presented ankle-brachial indices of 1 on the right side and 0.86 on the left. Finally, the SAT ultrasound found an ectasia in the right distal internal carotid and a true aneurysm of 4 cm maximum diameter at the level of the carotid bulb, extending into the left proximal internal carotid, with a mural thrombus in its interior.

To confirm these findings and plan adequate treatment, we performed CTA and SAT arteriography. The CTA confirmed the existence of a left carotid aneurysm of 3 cm maximum diameter, with a thrombus in its interior and right internal carotid artery ectasia (Fig. 1). The arteriography demonstrated the existence of this aneurysm, without showing significant stenosis in the distal exit (Fig. 2). The patient was rapidly intervened. We carried out resection of the carotid aneurysm with replacement of the flow through interposition of a 6-mm Dacron prosthetic graft. The clamping time was 25 min without shunt placement. The patient presented a good postoperative evolution without any complications, so he was discharged after 3 days.

Aneurysms of the extracranial internal carotid artery are rare, accounting for only 0.1%–2% of carotid interventions.² Their aetiology is varied; some of the most common are atheromatous degeneration (in 40% of cases) and trauma.³ Most cases are asymptomatic and are diagnosed incidentally.

Up to 40% of carotid aneurysms appear with neurological deficits, most of them as episodes secondary to cerebral transient ischemic attacks (TIA), established cerebrovascular accidents (CVA) or ischemic retinopathy by embolisms.⁴ Other symptoms may be due to compression of the aneurysm on surrounding structures, such as cranial nerves.^{5,6} The diagnosis is confirmed by CTA or MRI angiography; the angiography can delineate extension, detect stenosis and help during surgical planning.

With respect to treatment, surgery is indicated in most cases because the risk of vascular events in patients managed conservatively is over 50%.⁷ Conservative treatment is considered only in cases of very small or asymptomatic aneurysms located in the most distal section, near the base of the skull, due to the high surgical risk involved.

The current technique varies according to the type of aneurysm. In saccular and fusiform aneurysms with local extension and in an anatomically accessible neck, resection of the aneurysm and replacement by vein or prosthetic graft is the preferred technique and that with a better prognosis.

The endovascular option, using a covered stent for the exclusion of the aneurysm, has represented an improvement in the repair of these aneurysms. It is less aggressive and should be considered in cases of extensive aneurysms with a high location and a difficult surgical approach, or in patients with high co-morbidity for surgery, since they present a higher rate of neurological complications than conventional treatment, with a morbidity–mortality of about 7%.^{8,9}

In our case we opted for surgical treatment of the left carotid aneurysm due to its large size and consequent high risk of embolisms. The decision to use open surgery was based on the low surgical risk and the accessible anatomy.

References

1. Rittenhouse EA, Radke HM, Summer DS. Carotid artery aneurysm: review of the literature and report of a case with rupture into the oropharynx. *Arch Surg.* 1972;105:786–9.
2. Corteguera Torres D, Eliseo Mussenden O, Peguero Bringuez Y, García Lisame M, Díaz Hernández O. Aneurisma de la carótida extracraneal, Reporte de 2 casos. *Rev Cubana Ang.* 2007;8: 127–30.
3. Zhou W, Lin PH, Bush RL, Peden E, Guerrero MA, Terramani T, et al. Carotid artery aneurysm: evolution of management over two decades. *J Vasc Surg.* 2006;43:493–6.
4. Esteban-Jiménez O, Piñol-Ripoll G, González-Rubio F, Mauri-Llerda A, Navarro-Peman C, Mostacero-Miguel E. Ictus isquémico en un paciente con aneurismas gigantes de carótida interna. *Rev Neurol.* 2008;46:344–6.
5. Estevan Solano JM. Tratado de aneurismas. In: *Aneurismas de carótida extracraneal.* Uriach ed. Barcelona: Cayfosa; 1997.
6. Lee CC, Hsieh TC, Wang YC, Lo YL, Lee ST, Yang TC. Ruptured symptomatic internal carotid artery dorsal wall aneurysm with rapid configuration alchange. Clinical experience and management outcome: an original article. *Eur J Neurol.* 2010;17:1277–84.
7. Rossi P, Mirallie E, Pittaluga P, Chaillou P, Patra P. Bilateral extracranial aneurysm of the internal carotid artery. A case report. *J Cardiovasc Surg.* 1997;38:27–31.
8. May J, White HG, Richard W, Brennan J. Endoluminal repair of internal carotid aneurysm: a feasible but hazardous procedure. *J Vasc Surg.* 1997;26:1055–66.
9. Nishinari K, Wolosker N, Yazbek G, Vinicius C, Zotte A. Covered Stent Treatment for an aneurysm of a saphenous vein graft to the common carotid artery. *Ann Vasc Surg.* 2010;24:9–12.

N. Argilés Mattes, E. Hernández-Osma*, C. Berga Fauria, V. Sanchez Salvador

Servicio de Angiología, Cirugía Vasculay Endovascular, Hospital Universitario Joan XXIII, Tarragona, Spain

*Corresponding author.

E-mail address: eho51@hotmail.com

(E. Hernández-Osma).

doi:10.1016/j.nrleng.2011.02.003

REM sleep behaviour disorder and “pure” autonomic failure: Presentation of two cases[☆]

Trastorno de conducta del sueño REM y fallo autonómico ¿puro? A propósito de 2 casos

Sir,

The need for a diagnosis in the early stages of Parkinson's disease (PD) has led to increasingly more studies about early-stage signs and symptoms being published. A stereotyped and orderly progression of Lewy bodies in the brains of patients with PD¹ has been reported, but this α -synuclein deposit is not necessarily correlated with neuronal dysfunction or death and, therefore, with the symptoms. Thus, the first symptoms of PD can be very varied.² Longitudinal studies have shown that REM sleep behaviour disorder (RBD) can precede the diagnosis of a neurodegenerative disease (α -synucleinopathies), preferentially PD,³ in up to 45% of cases. It is known that autonomic failure is a late complication of PD and dementia with Lewy bodies (DLB), although it can sometimes constitute the presentation symptom.^{4,5} We recently described how a carrier of a mutation in the gene for α -synuclein (SNCA) presented dysautonomia as a sign of onset of her disease.⁶

We present the cases of 2 patients with idiopathic RBD and peripheral autonomic failure without signs or symptoms of neurodegenerative disease. We discuss the risk of developing synucleinopathies and the implications of an early diagnosis.

Case 1. This patient was a 66-year-old male with no family history of neurodegenerative diseases. In 2004, he attended consultation for a sleep disorder consisting of nightmares, restless sleep and falling from the bed, which the patient referred having suffered for years. A polysomnography recorded during REM sleep found increased phasic phenomena and a loss of the physiological atonia of this phase. We gave a diagnosis of RBD, which improved with clonazepam (0.5 mg). Six months later he suffered cough syncope. A vegetative nervous system study showed a fixed heart rate unchanged by the Valsalva manoeuvre, administration of ephedrine or standing. There was also orthostatic hypotension (supine position 148/80 mmHg, HR 53; standing 120/65 mmHg, HR 53). He presented noradrenaline values while lying of 26 pg/ml (VN: 100–750), which rose to 124 pg/ml (VN: 200–1700) when standing. Olfactory deficit was not detected (11/12 items identified correctly in the Brief Smell Identification Test, BSIT). There was a marked uptake decrease in myocardial-MIBG SPECT I (Fig. 1). A brain CT (MRI could not be performed due to the presence of metal particles) was normal. A 123-I-FP-CIT SPECT found no striatal uptake defects (Fig. 1). This exploration was repeated 2 years later and showed normal values, although the striated/occipital lobe uptake ratios had decreased discretely.

Case 2. This patient was a 60-year-old woman who attended consultation due to repeated syncope and orthostatic intolerance, of 10 years' evolution. She described

[☆] Please cite this article as: Tijero B, et al. Trastorno de conducta del sueño REM y fallo autonómico ¿puro? A propósito de 2 casos. *Neurología.* 2011;27:55–7.