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LETTERS TO THE EDITOR

Common origin of carotid and subclavian arteries as an exceptional anatomical variant of the aortic arch

Origen común de arterias carótidas y subclavias como variante anatómica excepcional del arco aórtico

Dear Editor:

The origin of the supra-aortic trunks at the level of the aortic arch can present several different anatomical dispositions, with the morphology of a right brachiocepahlic trunk and an independent origin of the internal carotid and left subclavian arteries the most common anatomical form, present in approximately 80%of cases.[1,2]

The longest case series in the literature by Natsi et al[3] describes up to 8 different types of anatomical variations in the origin of supra-aortic trunks in 633 brain arteriograms. Both carotid arteries stemming from a common trunk and both subclavian arteries coming from another, separate trunk is characteristic of birds and represents an exceptional finding in humans with a prevalence rate of 0.16-1%[3,4] This variant does not necessarily represent a pathophysiological mechanism, due to haemodynamic compromise or cerebral vascular disease,[5] although it must be considered when facing an eventual intervention in the aortic arch.

We report the case of a left-handed, 30-year old male with no known drug allergies or cardiovascular risk factors in whom a diagnostic arteriogram is performed of the brain and supra-aortic trunks to complete a study due to stroke-like manifestations with recurrent, temporary focal deficits, consistent with altered speech and motor-sensory deficit on the right-hand side of his body. After carrying out the radiographic study, a common origin in two, separate trunks for both common carotid arteries and both subclavian arteries was seen (figs. 1 and 2). This finding was incidental

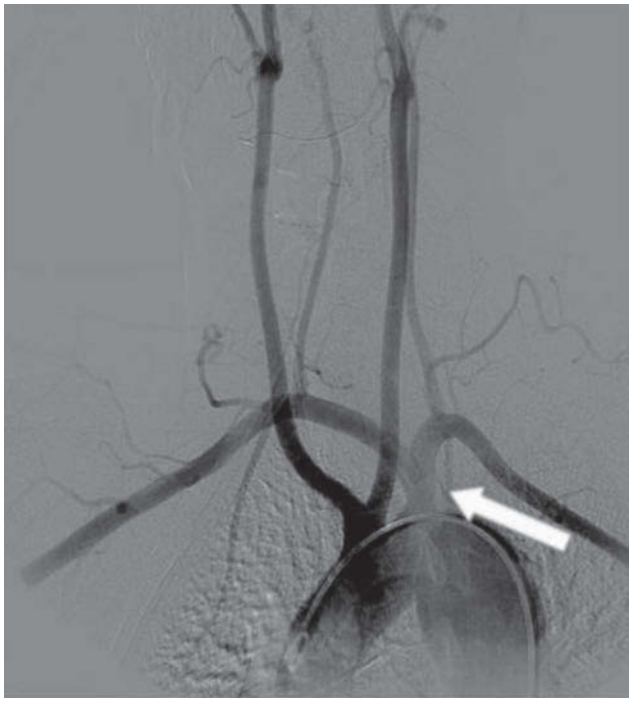

Figure 1 Arteriogram of the supra-aortic trunks. White arrows points to the common trunk of both subclavian arteries.

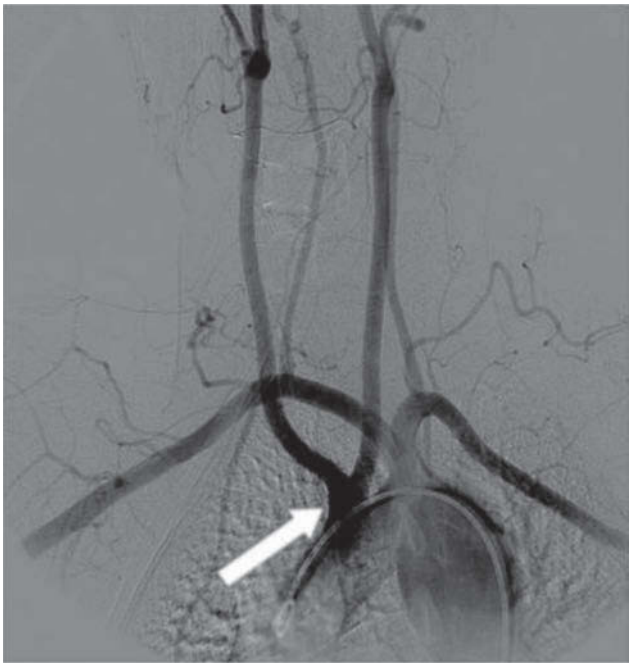

Figure 2 Arteriogram of the supra-aortic trunks. White arrows points to the common trunk of both common carotid arteries.

and apparently unrelated aetiologically with the patient's clinical symptoms.

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Bilateral striopallidodentate calcinosis. A presentation in the form of facial dystonia and frontotemporal dementia

Calcificación estrío-palido-dentada bilateral. Presentación en la forma de distonia facial y demencia frontotemporal

Dear Editor:

In 1860, Delacour was the first to report vascular calcifications in the basal ganglia in a male who presented rigidity, weakness in his lower limbs, and tremor.[1] Although known as Fahr's disease, this is a misnomer, since this latter author reported a case of calcinosis in the basal ganglia that does not correspond to what we currently understand this term to mean. Since the calcifications exhibit a preference for the basal ganglia and dentate nucleus, it would seem to be more appropriate to refer to it as bilateral striopallidodentate calcinosis (BSPDC).[2]

Calcification of the basal ganglia has been associated with more than 30 diseases, including infections, metabolic disorders, and genetic syndromes.[3,4] The incidence of calcifications in the basal ganglia in neuroimaging studies is 0.6%,[5] although most of them are very small and generally confined to the globus pallidus. They present clinically as extrapyramidal, cerebellar, and cognitive manifestations, with an inherited autosomal dominant variant in most cases and another, sporadic variant.

Sixty-six year old female, hypertensive, with bradycardia-tachycardia syndrome for which she had a definitive pacemaker. She had not undergone thyroid surgery nor had she received treatment with dopaminergic antagonists. No family history of dementia or extrapyramidal disorders.

The patient presents a bilateral hemifacial spasm for the last 5 years associated with jaw-closing dystonia that was treated with botulinic toxin with clear improvement of symptoms for 4 months. Over the course of the last 3 years, the patient has developed cognitive impairment with apathy, withdrawal, executive dysfunction, and emotional lability with loss of personal care.

Neurological examination: cortical functions: conscious and oriented. No aphasia, apraxia or agnosia. Cranial nerves: normal. Motor: normal tone; symmetrical, conserved strength. OTR: ++/++++. Plantar reflex: bilateral flexor. Frontal release reflexes: positive grasping and palmomental reflexes. Conserved sensitivity and cerebellum. Normal gait. Jaw-closing dystonia and bilateral asynchronous hemifacial spasm.

Neuropsychological evaluation: mild attention disorder and temporospatial disorientation, mild impairment of reading comprehension, mild deficit of episodic memory and long-term information recall. Frontal dysfunction with perseveration in graphic sequences, highly concrete thinking, limited working memory and sequencing errors in written expression.

Analyses: no alterations of interest in the blood test, coagulation, vitamin B12, folic acid, glucose, electrolytes, kidney function, liver enzymes, thyroid hormones, antinuclear antibodies, and blood proteins. Total and free parathyroid hormone, total calcium, calcium ions, and phosphorus were all normal. Genetic study for Huntington's disease, negative. X-rays of hands and feet: absence of subchondral bone cysts. Cranial CT (Figure 1): cortico-subcortical retraction with slight frontal predominance. Bilateral calcinosis at the level of the basal ganglia, thalamus, and dentate nuclei of the cerebellum.

The brain is especially well-protected against different toxins thanks to the existence of the blood-brain barrier. However, subcortical nuclei are vulnerable to several different minerals; thus, the accumulation of copper causes Wilson's disease; the accumulation of iron produces Hallevorden-Spatz's disease; the accumulation of organic mercury is the cause of Minamata's disease, and the accumulation of manganese, Parkinsonism. Different disorders produce calcification of the basal ganglia; however, the reason as to why these systemic processes bring about focal deposit in the basal ganglia is unknown. The finding of calcification in the basal ganglia in