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Case Report
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Vascular Anatomical Variants of Multiple Territories in a Patient with Posterior Circulation Infarcts: A Case Report

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Abstract

Introduction: Stroke in posterior vascular territory is less frequent that anterior infarcts, in some cases the anatomical variations can generate an increase in the probability or even be the explanation of the decrease in flow to a certain territory, it is usual to find some anatomical variants in each person, however multiple malformations are not usually as frequent and less in the central nervous system where there are vital structures that require the maintenance of blood flow.

Case Report: We present an unusual clinical case based on a patient with headache and cerebral, thalamic and right occipital infarcts associated with multiple congenital and secondary anatomical variants in the cerebral vascular circulation. Our patient had multiple vascular variants including azygos anterior cerebral artery, hypoplasia of the left A1 segment and, anterior communicating artery, aortic origin and, hypoplasia of the left vertebral artery with left main terminal branch Posterior Inferior Cerebellar Artery (PICA), old occlusion of the amputated basilar artery at the top with collateral circulation by bilateral Anterior Inferior Cerebellar Artery (AICA) arteries and, *via* PICAs.

Discussion: The set of brain anatomical variants found in the anterior and posterior circulation and post-infarction changes *via* bilateral AICA and right PICA is rare in the literature, especially about the clinical presentation of the patient. Following the above, it is important to report this case due to the low prevalence of the set of anatomical variants described and the absence of similar cases described in the literature.

Keywords: Brain anatomical variants; Acigos artery; Brain infarction posterior circulation; Infarction basilar artery

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Introduction

Anatomical variants in the central nervous system are frequent, especially in the Willis Polygon, which has a classic structure only in 20% to 25% of people [1,2]. The variants can be classified as fenestrations, duplications, hypoplasia's, agenesis, infundibular origins and, persistent communicating anomalous anastomosis [2,3].

These variants are mainly found incidentally but sometimes are associated with ischemic events that tend to differ from the classically described vascular territories, the most clinically relevant variants are: 1) Bilateral infarcts by anterior Azygos and, Percheron artery 2) Infarcts of Choroidal or Heubner's arteries and, 3) Stem syndromes [3].

The objective of this article is to present multiple vascular anatomical variants documented in a patient who was admitted to our hospital with a right posterior cerebral artery infarction.

Case Presentation

A 56-year-old man with a history of migraine, without cardiovascular risk factors, presented with intense holocranial headache, stabbing type, sudden loss of left peripheral vision and, left upper limb paresthesia, subsequently decreased alertness with retrograde amnesia of the event. Neurological examination showed lower left quadrantanopsy and, hypoesthesia of the left upper limb, the initial head Computed Tomography (CT) was normal (Figure 1).

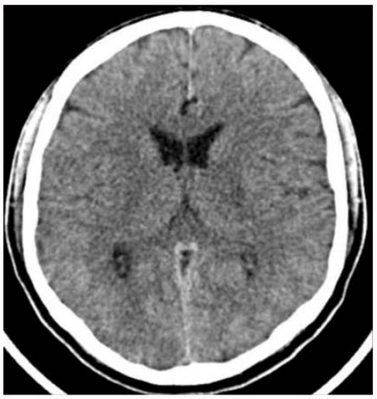

Figure 1: Simple computed tomography, axial brain slices, at the level of the thalamus and occipital lobes, without observing alterations.

Later he presented an increase of the support polygon and, lateralization in gait to the left, the Contrast-enhanced Magnetic Resonance Image (C-MRI) revealed areas of restricted diffusion in the right thalamus, posterior horns of the lateral ventricle and, right occipital lobe, compatible with acute multiple brain infarction in the right Posterior Cerebral Artery (PCA) territory (Figure 2). Angiotomography was performed revealing a filling defect of the Basilar Artery (AB), absence of flow within the PCA, hypoplasia of the pre-communicating portion (A1) of the left Anterior Cerebral Artery (ACA) and, in the intracranial portion (V4) of the left Vertebral Artery (VA).

Additionally, a subsequent pan-angiography confirmed the occlusion at the top of the basilar artery, absence of the Posterior Communicating Arteries (PCoA) without direct cortical irrigation, since this irrigation was given by an extensive network of vascular collateralization to the ischemic territory from the Anteroinferior Cerebellar Arteries (AICAs) and, the right Posterior inferior Cerebellar Artery (PICA). It also noted a hypoplastic left vertebral artery with its origin at the aortic arch, hypoplasia of the left A1 segment and, absence of anterior communicating artery, with azygos system from A2 but dependent on the right ACA (Figures 3-5). Capillary and venous phases with normal characteristics, dural sinuses with flow pattern and normal appearance. Malformations of the brain parenchyma were not observed.

In the controls by clinical neurology at 3, 10, 13 months post-infarction, he did not present subsequent neurological deficit, his return to work (mechanical technician of industrial machinery) and social was without difficulties. The control paraclinical tests at 11 months were positive antinuclear antibodies 1:320 fine granular pattern, positive antinuclear antibodies 1:320 centrosome pattern. Control images (pan-angiography, Doppler of renal arteries) and rheumatology evaluation were requested; however, as of the date of this case report, the patient has not had them performed.

In resume, the most relevant findings were right azygos ACA, absence of left A1 and, A2 segments, hypoplastic left VA with an aortic origin, absent ACoPs and, finally AB with distal flow occlusion and, extensive collateralization by AICAs and, PICAs allowing the necessary irrigation to the posterior cerebral circulation. The combination of these anatomical variants is infrequent and, even more so with the clinical findings of the patient, we started dual antiplatelet therapy with a diagnosis of minor cerebrovascular attack. Post-discharge follow-up was performed, without neurological deterioration, left lower quadrantanopsia persisted. Other studies are summarized in Table 1.

Discussion

It is important to know the cerebral vascular anatomical variants; the presences of associated cerebral vascular anatomical infarcts or arterial variants do not usually follow the classically predetermined vascular territories [3]. We will deepen those variants presented in our case.

Anterior circulation

ACA asymmetry is present in 80% of people [4]. Azygos ACA is a rare vascular variant (0.3% to 4%) [3,4], involves segment A2

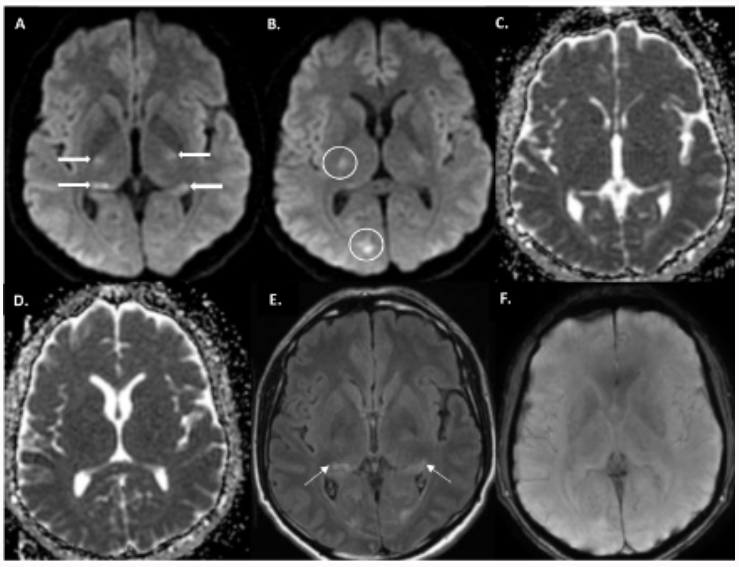

Figure 2: Axial diffusion images B-1000 and, ADC (right, middle and left, and lower right), areas of restriction to diffusion are observed on the posterior and, the upper face of the right thalamus and, right occipital subcortical. Axial FLAIR (inner row, center) hyperintense areas that correlate with diffusion. Axial SWI (lower left) without areas suggestive of hemorrhage. Findings are consistent with non-hemorrhagic acute infarcts in the territory of the right posterior cerebral artery. ADC: Apparent Diffusion Coefficient. FLAIR: Fluid Attenuation Inversion Recovery. SWI: Susceptibility-Weighted Imaging

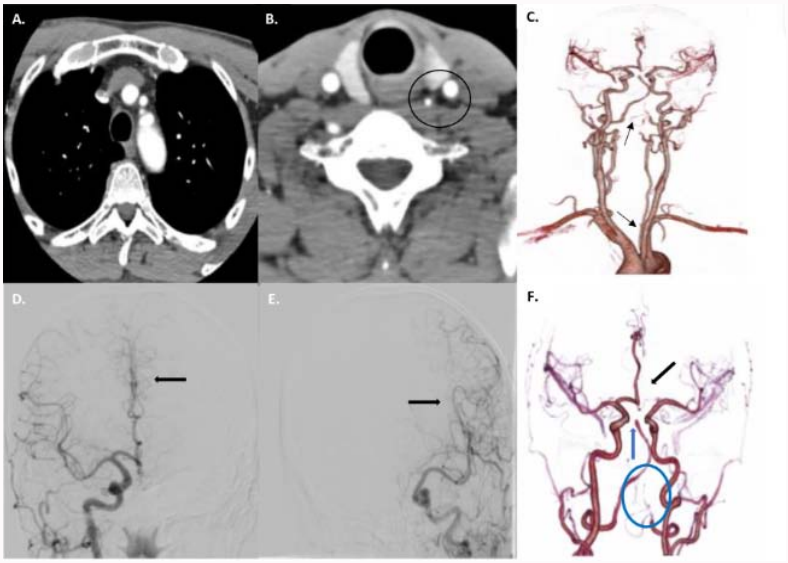

Figure 3: Line upper: Images right and, center row, axial contrasted at the level of the aortic arch and, in the neck and, image left (V1), three-dimensional tomographic reconstruction of the neck and, intracranial vessels, observing the origin of the left hypoplastic vertebral artery.
Line lower: Central and left Digital Subtraction Images (DSA), three-dimensional reconstruction of intracranial vessels. Findings: severe hypoplasia of the left A1 segment and absence of an anterior communicating artery. Azygos A2 system is dependent on the right anterior cerebral artery.

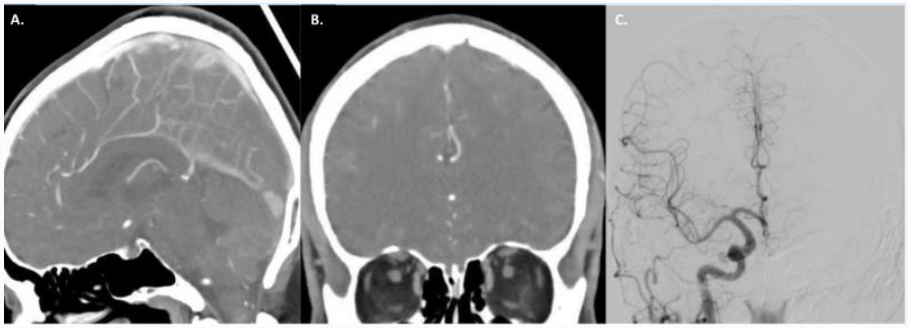

Figure 4: Sagittal and coronal arterial phase contrast-enhanced tomography images of the skull (right and center), DSA image of the right internal carotid artery on the left. Findings: The presence of azygos system in the right anterior cerebral artery from A2 at the level of the knee of the corpus callosum.

forming a single trunk in the midline, the result of an embryological persistence of the median artery of the corpus callosum, radiologically is classified in 4 types [1,5], is occasionally associated with aneurysms, holoprosencephaly, neuronal migration abnormalities and, its obstruction generates a bilateral frontal syndrome [1,4,5].

Hypoplasia of segment A1 is reported in 10% of autopsies and complete absence in 2% of angiograms [1,4]. The posterior communicating arteries may be of fetal origin, an infundibular residue, hypoplastic or unilateral absence, other types of anomalies are infrequent or have not been described [1,2].

Other anomalies of this circulation, absent in our case, include fenestration of ACA (4% of people) [2]. Duplication of the Middle Cerebral Artery (MCA) (0.2% to 2.9%), and its fenestration in less than 1% in autopsies and 0.17% in angiography [1,4].

Posterior circulation

Of all ischemic strokes, the posterior circulation represents 25% and stems 10% of cases [3,9]. Vascular variants of the posterior circulation can involve the brainstem, occipital lobes, and cerebellum, the infarcts secondary to variants are rare [1,6]. Additionally, 6% to 10% of all strokes are initially Diffusion-Weighted Imaging (DWI) negative, with a false-negative rate for posterior circulation strokes almost 10 times higher than that of the anterior circulation [9].

The most relevant abnormalities of this circulation are those that affect VA with asymmetry (70%), hypoplasia (26%) and fenestration (0.3% to 2.0%) [2,6]. PICA is absent unilaterally in 10% and bilateral in 2%. In 10% of people, this artery arises from the BA [6]. The basilar variants more frequent are fenestration (0.3% to 0.6% of angiograms), anastomosis with Carotids (0.1%), hypoplasia, and agenesis which are rare and almost non-existent [1,6].

The PCA has an absence of P1 in 10% of people, had identified four variants depending on its origin and, 15% identified fetal PCA from the Carotid Artery [6]. Percheron artery infarcts can generate bi-thalamic compromise [3]. This anatomical variant was absent in our presented clinical case.

Finally, It is also important to evaluate possible differential diagnoses with a genetic component such as Fabry, MELAS (Mitochondrial Encephalomyopathy, Lactic Acidosis, and, Stroke-like episodes), CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and, Leukoencephalopathy) and, similar [7,8]. Evidence of these pathologies doesn't exist in our patient.

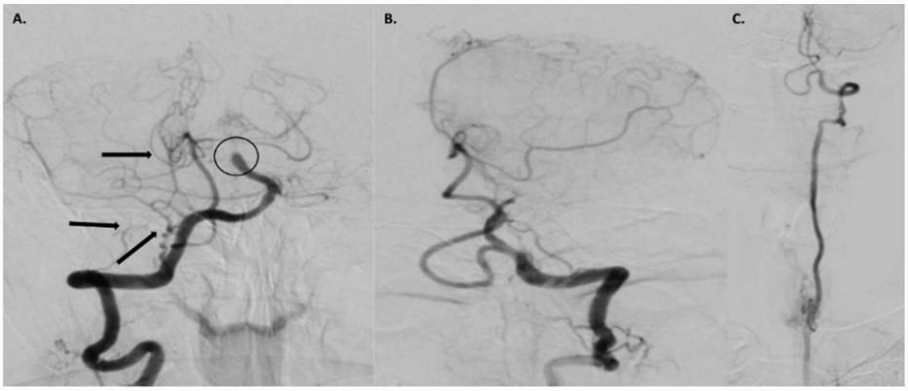

Figure 5: DSA images of the posterior cerebral circulation. Image of the right vertebral artery, center and, left vertebral artery, findings: Occlusion of the top of the basilar artery, with extensive collaterals by territories of the anterosuperior cerebellar arteries (bilateral AICA) *via* the right Posteroinferior Cerebellar Artery (PICA), in addition to severe hypoplasia of the left Posterior Communicating artery (PCo) without direct cortical irrigation, since this irrigation is caused by extensive counter flow collateralization (images on the right and center). (Image on the left) Origin of the left vertebral artery from the aortic arch (left) with little contribution to the basilar artery branch, ends and, as the terminal main branch originates the left PICA artery, besides, to the absence of flow in the posterior cerebral arteries.

Table 1: Additional paraclinical taken during hospitalization and controls at 3rd, 10th and 13th months.

Admission paraclinics.	
Hemogram: Leukocytes: 5510, Neutrophils: 3310, Lymphocytes: 1350, Hemoglobin: 15.3, Hematocrit: 44.6%, Platelets: 262000.	
BUN: 18.8, creatinine: 0.95	TP: 28, TPT: 11.06, INR: 1.01
Blood glucose: 92.7, Sodium: 139	Normal Electrocardiogram and Echocardiogram
Paraclinics at 6 months	
CRP: 0.5, Total Bilirubin: 0.78, Direct 0.23, indirect 0.55; CPR 80 (29-168), blood glucose 76, urea nitrogen: 20, creatinine 0.94; potassium 4.13, anticardiolipin IgM: negative, IgA: negative, IgG: negative; homocysteine 8.92 (normal)	
Paraclinics at 11 months	
Lupus anticoagulant: Negative, extractable Nuclear Antigen (ANE) antibody profile: negative, antinuclear antibodies: Positive 1:320 fine granular pattern, antinuclear antibodies: positive 1:320 centrosome pattern.	

Conclusion

We present a case with multiple brain anatomical variants and, radiological findings of infarction in the posterior territory, whose infrequency makes it a unique case, with clinical-radiological dissociation given the collateral circulation of the posterior territory with structural and, hemodynamic changes described. We did not find in the literature cases where a set of anatomical variants of the anterior and posterior circulation such as those presented in our case.

Ethics Approval

This article was approved by the Ethics Committee of the Hospital Universitario Nacional de Colombia.

Authors' Contribution

All authors are declared to have participated in the conceptualization, methodology, drafting, writing, and revision of this article.

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