Hemorrhagic Stroke in the Context of Anatomical Variations in the Circle of Willis - A Perspective on Neurological Rehabilitation

Ana-Maria Dumitrescu[1], Anca Sava[1], Lucia Corina Dima-Cozma[2], Liliana Mihailov[3], Cristina-Maria Gavrilescu[4], Claudia Florida Costea[5], Irina Luciana Gurzu[6], Bodan Gurzu[7], Roxana Gabriela Cobzaru[8], Dana Teodora Anton Paduraru[9], Roxana Florentina Șufaru[1]*, Liviu Ciprian Gavril[1], Ana-Maria Haliciu[1], Ana-Maria Rădulescu[1] and Carmen Valerica Rîpă[8]

[1]Department of Morphofunctional Sciences I, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[2]IST Medical Department, Grigore T. Popa University of Medicine and Pharmacy, Iaкi, Romania
[3]Department of Palliative Care, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[4]Department of Biomedical Sciences, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[5]Department of Clinical Ophthalmology, ñProf. Dr. N. Obluò Emergency Clinical Hospital, Iaьi, Romania, Grigore T. Popa University of Medicine and Pharmacy, Iaкi, Romania
[6]Department of Preventive Medicine and Interdisciplinarity, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[7]Department of Morphofunctional Sciences II, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[8]Department of Microbiology and Parasitology, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania
[9]Department of Mother and Child Medicine, "Saint Mary" Emergency Clinical Hospital for Children, Iasi, Romania, Grigore T. Popa University of Medicine and Pharmacy, Iasi, Romania

Abstract

we considered to be most relevant for the topic, thus laying the basis for a descriptive literature review.

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e CoW has been divided in two di erent parts: an anterior one, composed of ACoA and A1 precommunicating segments of the two ACAs and a posterior one composed of the P1 precommunicating segments of the two PCAs [3]. ere are other Indian authors that consider a "classical" circle of Willis as symmetric bilaterally, in the shape of a "ring" of vessels [4]. Judging a er its general external aspect, the circle of Willis can stand out as typical or atypical. e typical aspect of the circle of Willis has been described in several studies as having all its component arteries as well as its communicating segments present and of normal dimensions [5]. In terms of anatomical variations, the most common ones that are recurrently described in the actual literature are as follows: hipoplasia, duplication or triplication, fetal type artery, absence, fenestration.

Hipoplasia refers to a diameter of an artery that is less than 1mm, with the exception of PCoAs and ACoA [4]. Fenestration is the type of anatomical variant described by the presence of two arterial canals for a singular vascular route [6]. Duplication or triplication of an artery refer to the presence of suplimentary arteries. e fetal type artery can be either partial, de ned by a PCoA diameter that is less than 1 mm and the P1 segment of the PCA less than 1 mm, or total, when the PCoA diameter is greater than 1 mm and the P1 segment of PCA is missing.

e absence is an anatomical variation referring to some arteries that are practically missing from the con guration of the vascular circle[4,6].

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e main cause of stroke development at the level of the CoW is represented by the thrombotic occlusion, secondary to atherosclerosis**.**

e most frequent areas of its development is at the bifurcation of PCAs. Principal risk factors for the development of stroke, either ischemic or hemorrhagic, are high blood pressure and atherosclerosis, under the condition of Internal Carotid Artery (ICA) occlusion and bilateral PCoA hipoplasia/aplasia (absence of the vessel lumen) [7].

e hipoplasia of the ipsilateral Vertebral Artery (VA) is an additional risk factor to the presence of PCoA hipoplasia, because it increases the arterial pressure within the controlateral vertebral artery in the case of hypertensive patients. Frontal haematoma can appear in the case of patients presenting unilateral PCoA hipoplasia or aplasia, in the context of an elevation of blood pressure within the carotidian system, due to the fact that blood cannot communicate with the vertebral-basilary system for depressurizing the CoW. e unilateral partial fetal type PCA that has anastomosis with the basilary artery through a very thin arterial branch can determine the formation of a right hemispheric paradoxal infarction, if the patient presents systemic atherosclerosis, complicated with ulcerations and calci cations and high tensional values. A er thrombolysis, the blood penetrates the infarction zone and this way the hemorrhagic stroke is produced. Acute pancreatitis can determine a metabolic encephalopathy, but also a fatal intracerebral hematoma [8]. In a study published in 2018, Mukherjee et al. suggests that the presence of anatomical variants in the CoW can lead to a change in the microembolic circulation at the level of the constituent arteries in the circle and can have as e ect the development of infarction in some distal areas of the brain, less common [9]. e hipoplasic arteries present a patent lumen, therefor, in the absence of stenosis at the level of big proximal arteries, the communicating arteries do not a ect the blood ow volume to the brain, unless there are several risk factors involved such as: hypertension, atherosclerosis, insulin-requiring Type-2 diabetes mellitus, hemorrhagic acute pancreatitis, hepatic cirrhosis or disseminated intravascular coagulation [10]. It is documented in literature that chronically elevated tensional values that lead to a higher risk of cardiovascular pathology have most commonly stress as main trigger [11].

e current literature presents just few studies on the variations of the CoW in relation to aneurysmal development and the rupture. Changes in the haemodinamics of a vessel in the CoW, due to anatomical variations, can in uence the formation and rupture of such an aneurysm [12], in addition to capillary frailty. A study from 2012, in which there were investigated over 100 patients presenting ACoA and PCoA aneurysms, revealed the fact that the presence of anatomical variants was highly associated with the rupture of intracranial aneurysms [13].

A recent Romanian study, in which there were analized the causes of death in patients with bilateral hipoplasia of the PCoA, concludes that extended stroke could be correlated with this association of variants, especially when it associates a systemic pathology or the occlusion of an important artery assuring the irrigation of the brain [14].

In a Chinese radiology study published in 2019, in which there were analyzed over 200 patients presenting aneurysms at the level of CoW, both univariate and multivariate analysis highlighted the fact that the presence of fetal-type PCoA increased twice the risc of rupture of the aneurysm formed at the ICA-PCoA junction [15].

When fetal-type PCoA is present, it is associated with the presence of a P1 segment of the ACP that is smaller in diameter than the contralateral homologous artery. A hipoplasic VA is associated with a higher risk of atherosclerosis in the posterior circulation and, therefor, there would be a higher incidence of stroke in this region in the case of this type of anatomical variant [16]. In Germany, there has been investigated by Harati et al. the co-occurence of hipoplasia/aplasia/atresia of the VA, the aplasia of the Postero-Inferior Cerebellar Artery (PICA) and the aneurysms formed at the junction between the VA and PICA. e study concluded that VA hipoplasia on one side alone represents a risk factor for aneurysm formation on the opposite side to hipoplasia [17]. In a recent Turkish study there has been mentioned that when co-existing, the fetal-type variant of PCoA and hipoplasia of the A1 segment of ACA imply a higher rate of ACoA aneurysm rupture [18]. erefor, the concomitant presence of anatomical variants, such as hipoplasia and fetal-type, in both anterior and posterior parts of the CoW imply more o en the formation and rupture of aneurysms in the CoW.

In the case of fetal PCA, the controlateral hemispheric cerebral infarction is formatted, the "paradoxal" infarction as it is called in the specialty literature, having as risk factors hypertension and atherosclerosis. From a clinical point of view hipoplasia and aplasia of PCoA are very signi cant. e hipoplasic or aplasic PCoA can compromite the capacity of the CoW to assure collateral circulation [19]. e clinical signi cance of anatomical variants is shown especially in the case of an atypical model of stroke. In the context of big arteries occlusion or severe stenosis, the communicating arteries become crucial. In this context, the caliber of communicating arteries is inCoA

It seems that AcoA fenestration represents a predictive risk factor of death for hemorrhagic stroke, because aneurysms can develop at this level. At the same time, the anatomical variants of AcoA, in the form of hypoplasia, fenestration and absence of the vessel, represent a predictive factor of mortality risk for multiple, infra- and supratentorial ischemic strokes.

ere are also reports of associations between the presence of anatomical variants of AcoA and the development of aneurysms at the level of the anterior AcoA-ACA complex and their rupture, which o en leads to the death of those patients [21].

It appears that hypoplasia of the A1 segment of the ACA in the pediatric population is predominantly associated with headaches and dizziness, which may play a role in the incidence of neurological diseases in later life. e mechanism of these symptoms may be represented by progressive cerebral ischemia [22]. It is documented in literature that neurological symtoms in children usually appear in pathologies that a ect the brain, primarily, but, in some cases, it is important to exclude other gastrointestinal disorders such as the caeliac diasease [23].

Citation:

so that they continue to monitor their beloved ones' evolution a er coming back from medical centres or hospitals. e study also provides an insight into the anatomical underlyimg aspects of this pathology, becoming useful for neurologists, radiologists, internal medicine and family medicine practitioners, as well as for anatomists. It is noticeable that professional neurological rehabilitation can have a major impact on the outcomes of recovery in post-hemorrhagic stroke patients, if there is applied the right dose and time of speci c recovery exercices and therapy. e nal outcome of the neurological rehabilitation therapies has a focus on neuroplasticity, by brain stimulation and functional recovering, a er a hemorrhagic stroke event.

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None

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None

References

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