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Diagnostic value of color-coded duplex sonography in patients with ischemic stroke and congenital changes in the circle of Willis

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ABSTRACT

The circle of Willis (CoW) forms the main circulatory system in the human brain. A large number of variations of the CoW is known, and also their association with ischemic stroke.

Three cases of young patients with combination of ischemic stroke and anomalies in the CoW are presented, and the value of the color-coded duplex sonography (CCDS) is compared to other imaging diagnostics such as magnetic resonance angiography (MRA) and digital subtraction angiography (DSA).

In these patients we found multiple risk factors such as stenosis or thrombosis of intracranial brain vessels, mechanical compression of vessels, a genetic mutation associated with an increased risk of thrombosis, and intake of oral contraceptives. For clinical evaluation several methods were used: detailed medical history, neurological status, laboratory examinations (complete blood count, biochemistry, lipid profile, HIV1/2, Syphilis RPR test), screening for markers associated with an increased risk of thrombosis, chest X-ray, spinal fluid study, CCDS, DSA, MRA. A full conformity in the data from CCDS and other imaging methods was found.

The authors discuss the pathogenetic role of congenital anomalies of CoW, incidence of ischemic stroke and the high diagnostic value of CCDS for finding such anomalies.

Introduction

The circle of Willis (CoW) is the anastomosed arterial ring in the brain, which integrates the internal carotid and vertebral-basilar systems. Sir Thomas Willis described the anatomy of the basal intracranial vessels for the first time in 1664.

There is considerable variability in the anatomy of the CoW, often with asymmetry, and to such extent that configuration

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can only be found in a small number of cases. A full CoW with a balanced pair of arteries was found only in 18–20% of the general population [1]. The cerebral arteries may be absent or can be found with some anomalies – hypoplastic, doubled or even tripled. Anatomical studies indicate a lack of anterior communicating artery (ACoA) in 1% of patients, the absence or hypoplasia of the proximal part of the anterior cerebral artery (ACA) in 10%, the absence or hypoplasia of posterior communicating artery (PCoA) in 30%, anterior trifurcation in 3–22% [2]. According to different authors, frequency of a hypoplastic vertebral artery (VA) varies between 1.9 and 6%; hypoplasia of both vertebral arteries is rarely found [3,4]. Fetal type cerebral blood flow can be found in about 10% of the cases [5].

Cerebral blood flow is maintained relatively stable thanks to the autoregulation of the cerebral arteries. It is defined by several factors: vascular resistance of the cerebral arteries, perfusion pressure and autoregulatory mechanisms. The cerebral perfusion pressure depends on the difference between mean arterial pressure and intracranial pressure. The autonomous control is capable of maintaining the blood flow in a wide range of variations of the mean arterial pressure. In healthy subjects, brain blood flow is relatively constant during fluctuations in mean arterial pressure of about 50–150 mmHg [6,7]. The protective role of collateral circulation is dependent on several factors, including anatomic variations, systemic arterial pressure, patient age and stage of development of occlusive disease [8,9]. The CoW is the main ''physiological anastomosis'' in the brain circulation which is able to sustain brain perfusion in cases of acute occlusion of large arteries [10–12]. Following an occlusion of a major blood vessel, the fall in perfusion pressure distally generates a gradient between adjacent arterial beds. This results in changes of blood flows' direction and speed [9,10]. It occurs almost immediately (within 1–4 s) indicating that metabolic factors are less likely related to this change [13–15]. The CoW forms the main collateral circulatory system.

Other collateral vessels are considered to be of secondary significance, such as the leptomeningeal and dural arterioles, cortical vessels, extracranial and intracranial collaterals, as well as some rarely occurring collateral vessels such as the thecal plexus, middle meningeal artery, maxillary and ethmoid artery. Anastomoses between the distal segments of the main brain arteries also contribute to collateral blood flow. The number and size of these vascular anastomoses are high in ACA and middle cerebral artery (MCA), low between MCA and PCA and with the lowest frequency between ACA and posterior cerebral artery (PCA). Various collateral connections, between vertebral and basilar segments of the posterior circulation, are provided by distal branches of major arteries in the brain.

The collateral vessels are formed during the prenatal period. Some pathophysiological conditions may lead to secondary emergence. For example, focal cerebral ischemia can lead to a release of angiogenic peptides with a known potential for collateral vessel formation. Even though these vessels may have been ''designed'' to attenuate necrotic areas, rather than increase cerebral blood flow [12]. However, angiogenesis can promote collateral growth in the periphery of the ischemic zone. The state of collateral circulation must be taken into account during the color-coded duplex sonography in the evaluation of cerebrovascular ''hemodynamically significant stenosis'' [15]. The collateral potential of the vessels is determined by the caliber of their lumen. Collateral circulation to the brain is summarized in Table 1. During carotid endarterectomy a solid example for the rapid compensatory ability of the CoW can be observed. After clamping the ipsilateral internal carotid artery, transcranial monitoring shows an increase in blood flow in the contralateral anterior cerebral artery within only two to three heartbeats.

Importance of collateral blood flow is high. It is known that acute cerebral infarction, due to occlusion of the carotid artery, has worse outcome in cases of underdeveloped collateral network. The annual stroke incidences in patients with occlusion of the carotid artery range from 0 to 5% in asymptomatic patients, up to 27% in symptomatic patients [16,6]. There is a surprisingly lower rate of stroke (4% in 10 years) in the ipsilateral side of the occluded artery, in patients with bilateral occlusive disease of the carotid artery

Table 1 – Cerebral collateral circulation.

Intracranial	Arteries connected	Connecting artery
Circle of Willis	Internal carotid artery and basilar/posterior cerebral arteries	Posterior communicating artery
	Anterior cerebral arteries	Anterior Communicating ARTERY
Vertebrobasilar and circle of Willis	Internal carotid artery and vertebral/basilar arteries	Trigeminal, otic and hypoglossal arteries
Tectal plexus	Posterior cerebral artery and superior cerebellar artery	Tectal rami, connecting supra and infratentorial arteries
Cerebral artery branches	Branches of the middle, anterior and posterior cerebral arteries	Anastomoses of terminal branches within and between arterial territories
Leptomeningeal		
Pial plexus	Neighboring branches of major cerebral arteries	Arterioles from branches of same or adjacent arteries
Meningeal	Cerebral and meningeal arteries	

Extracranial	Arteries connected	Connecting artery
Orbital plexus	Ophthalmic and middle meningeal, maxillary, ethmoidal arteries	Terminal branches
Rete mirabile caroticum	Internal and external carotid	

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