Early Clinical Signs, Lesion Localization, and Prognostic Factors in Unilateral Symptomatic Internal Carotid Artery Occlusion

Sibel Güler, MD, Ufuk Utku, MD, and Özer Aynaci, MD

Background: The aim of this study was to assess infarct localization, clinical signs, and prognostic factors in cases with unilateral symptomatic total internal carotid occlusion. *Methods:* In total, 101 patients who had a diagnosis of symptomatic unilateral carotid occlusion in the Department of Neurology, Trakya University Faculty of Medicine, between January 2008 and May 2012, were included in this study. The relationship between infarct localizations and prognosis of patients was evaluated by cranial magnetic resonance imaging (MRI) and diffusion-weighted MRI. The condition of ipsilateral middle cerebral artery (MCA) and posterior communicating arteries (PCoAs) was assessed by cranial and cervical magnetic resonance angiography besides opposite carotid. Patients were evaluated by modified Rankin Scale in terms of prognosis at discharge and after 3 months. Furthermore, they were evaluated in terms of risk factors, such as cigarette and alcohol use, presence of temporary ischemic attack and stroke history, hypertension, diabetes mellitus, coronary artery disease, previous myocardial infarction, hyperlipidemia, and peripheral vascular disease. *Results:* Territorial infarct was commonly seen as acute ischemic stroke pattern especially in cases with a poor MCA circulation and insufficient collateral circulation. Development of territorial stroke, occlusion of MCA, and nonvisualization of PCoA were found to be associated with poor prognosis. *Conclusions:* In unilateral symptomatic intracranial carotid artery occlusion, poor prognosis and high mortality–associated territorial stroke pattern is frequently observed. Besides, presence of severe stenosis or occlusion and absence of collateral circulation in MCA are associated with poor prognosis. **Key Words:** Symptomatic unilateral carotid occlusion—infarct pattern—Willis polygon—treatment—prognosis.

Introduction

There are 2 main mechanisms occurring in intracranial carotid artery (ICA) occlusive disease, including ischemic stroke, embolism, or severe stenosis, and hypoperfusion because of low flow that is associated with hemodynamically insufficient collateral circulation after occlusion. Therefore, acute ischemic stroke is developed after the disruption of cerebral perfusion after embolic event or hypoperfusion.[1]

In studies based on computed tomography, it was shown that significant hemodynamic stenosis or obstructions of extracranial ICA may result in hemodynamic alterations in providing hemispheric blood in distal regions. Diffusion-weighted imaging (DWI) is superior over conventional MRI and computed tomography in the detection of acute cellular damage in cerebral ischemia and especially small and multiple new ischemic lesions within first several hours.[2] In addition to border-zone infarcts that develop as a result of territorial infarct and hemodynamic insufficiency, infarct patterns, such as sectional territorial infarct and subcortical infarct, were also identified by DWI. Clinical presentation of ICA occlusion can also vary from temporary ischemic stroke, minor or major ischemic stroke to

From the Department of Neurology, Trakya University Faculty of Medicine, Edirne, Turkey.

Received December 23, 2013; revision received January 28, 2014; accepted February 5, 2014.

Disclosure: None.

Address correspondence to Sibel Güler, Department of Neurology, Trakya University Faculty of Medicine, Edirne, Turkey. E-mail: drsibelguler@yahoo.com.

1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2014.02.009

death. Here, collateral blood flow that is provided by Willis polygon is important for continuation of cerebral perfusion that is required for metabolic need in the determination of severity of ischemic lesion.[3,4] Collateral routes are used to compensate decreasing blood flow in symptomatic internal carotid disease.[5] Especially, presence of collateral flow in middle cerebral artery (MCA) is thought to be the main collateral in the progression of cerebral ischemia. In addition, anterior communicating artery and ipsilateral posterior communicating artery (PCoA) provide redistribution of blood to the brain region that is deprived of blood via collaterals of Willis.

In this study, hemodynamic roles of MCA and Willis polygon and PCoA collateral ability were compared in patients with unilateral ICA occlusion and acute ischemic stroke, and the patients were evaluated by DWI and cranial magnetic resonance angiography findings in terms of their effects on infarct localization, early clinical signs, and prognosis.

Materials and Methods

Participants

In all, 101 patients including men and women with unilateral symptomatic ICA occlusion were included. Patients were selected among individuals who were hospitalized in Trakya University Faculty of Medicine Research and Practice Hospital between January 2009 and May 2012. The ethical approval for the study was given by the Trakya University Faculty of Medicine Ethics Committee on December 07, 2013, no. 179.

Patients with a posterior circulation infarct with vertebrobasilar symptoms, whose nonatherosclerotic etiology was clearly documented (eg, arterial dissection, vasculitis, prothrombic condition, or cortical sinus thrombosis), who do not have vascular imaging, patients with AF who had cardioembolic etiology, and who were evaluated as temporary ischemic attack were excluded. In this study, differences in stroke pattern were analyzed in patients who showed acute ischemic stroke that was developed within 12 hours and who showed symptomatic unilateral ICA occlusion by carotid Doppler and cervical magnetic resonance angiography performed afterward; DWI and cranial magnetic resonance angiography were used to collect more information about the localization, distribution, and pathomechanism of stroke.

Clinical Analysis

Disability symptoms or significant neurologic deficit related to ICA occlusion site were present in all patients before MRI assessment. During discharge and assessment of 3-month prognosis, patients less than Rankin score 3 were classified as the ones with minor disability and patients who were equal and more than Rankin score 3 were classified as the ones with moderate or significant disability.

The parameters used and recorded during statistical analysis were evaluated as age, sex, stroke history, family history, cigarette use, alcohol use, hypertension, hyperlipidemia, presence of AF, presence of diabetes mellitus, presence of coronary artery disease, history of myocardial infarction, presence of peripheral vascular disease, occluded ICA side, condition of the opposite ICA, complaint, infarct pattern, presence of recurrent stroke during hospitalization, PCoA condition, MCA condition, modified Rankin Scale (mRS) score during discharge, treatment taken, and last clinical condition of the patient, respectively.

Magnetic Resonance Angiography

MCA and Willis polygon were assessed by 1.5 T magnetic resonance angiography (MRA). MCA was recorded based on the presence of flow as (1) occluded, (2) severe stenosis, or (3) open. PCoA that was included in Willis polygon was assessed. The presence of collateral flow was determined through PCoA.

Identification of Stroke Pattern

Four ischemic lesion patterns were identified in DW images based on the size, distribution, and localization of lesion.[6,7]

Territorial Infarct

Large ischemic lesion in which 1 or more major cerebral artery sites, cerebral cortex, and subcortical structures were affected is called as territorial infarct. For example, if distal MCA branches are occluded, it is evaluated as partial MCA infarct. Bifurcation without effective collateral system or proximal occlusion at the level of trifurcation is classified as large MCA infarct; total anterior cerebral artery and MCA site that are developed from distal ICA because of embolism are also classified in this way[8] (Fig 1).

Subcortical Infarct

Infarcts of deep perforated branches that are originated from distal ICA or MCA body are named as subcortical infarct. This pattern is explained as occlusion of MCA, which has open collaterals. The cause of occlusion is the embolization within MCA and it ends with large striatocapsular lesion, or it is because of the occlusion of deep perforated arteries of the carotid system and causes subcortical lesion[9,10] (Fig 2).

Internal Border-zone Infarct and Cortical Border-zone Infarct

This is the infarct pattern that is developed in the regions, which are considered to show one of the hemodynamic risk zones between major cerebrovascular areas completely or generally.[11,12] They are localized between anterior cerebral artery and MCA or between MCA and