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Faciobrachial Monoparesis Secondary to Borderzone Infarct : A Case Report

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Abstract :

Faciobrachial monoparesis is presumed to be secondary to lesions involving Heubner artery(a proximal perforating branch from anterior cerebral artery) or lateral lenticulostriate artery(branch of middle cerebral artery). Here we report a case of faciobrachial monoparesis secondary to borderzone infarcts.

Key Words : large vessel stroke, borderzone infarcts, faciobrachial monoparesis.

Introduction :

Small vessel strokes are secondary to occlusion of small penetrating arteries. Border zone or watershed infarcts are ischemic lesions that occur at the junction between two main arterial territories. Faciobrachial monoparesis is presumed to be secondary to lesions involving Heubner artery (a proximal perforating branch from anterior cerebral artery) or lateral lenticulostriate artery(branch of middle cerebral artery) Occlusion of these perforating branches causes small vessel stroke. Here we report a case of faciobrachial monoparesis secondary to border zone infarcts.

72years old female patient presented to us with complaints of weakness of right upper limb and deviation of angle of the mouth to left side from past 2days. On examination she was conscious, oriented, had right UMN facial palsy, pronator sign in right upper limb, weakness of small muscles of right hand. Motor power in right lower limb and in left upper and lower limbs were normal. Rest of the neurological and other system examination were within normal limits. MRI-brain revealed borderzone infarcts in left middle cerebral artery(MCA) – posterior cerebral artery and left MCA – anterior cerebral artery territory. In the view of borderzone infarcts we suspected large vessel disease, MR-ANGIOGRAM revealed atherosclerotic changes in both internal carotid artery(ICA), more so at cavernous portion of left ICA and both M1 segments of both middle cerebral arteries. Carotid-vertebral aretery Doppler revealed soft plaque causing 50% stenosis of left common carotid artery. Blood investigations revealed dyslipidaemia (increased low density lipoprotein, triglycerides and total cholesterol). Cardiac evaluation including echocardiography were normal. She was started on antiplatelets, atorvastatin and physiotherapy.

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Discussion:

Lacunar infarction refers to infarction following atherothrombotic or lipohyalinotic occlusion of a small artery (30–300 μm) in the brain. The term small vessel stroke denotes occlusion of such a small penetrating artery . small vessel strokes account for 20% of all strokes. Hypertension and age are the principal risk factors. The MCA stem, the arteries comprising the circle of Willis (A1 segment, anterior and posterior communicating arteries, and P1 segment), and the basilar and vertebral arteries all give rise to 30 to 300μm branches that penetrate the deep grey and white matter of the cerebrum or brainstem. Each of these small branches can occlude either by atherothrombotic disease at its origin or by the development of lipohyalinotic thickening causing small infarcts that are referred to as lacunes

Recovery from small vessel strokes tends to be more rapid and complete than recovery from large-vessel strokes.[1]

Cerebral infarcts in border zones were first discussed in 1883 [2] and were defined as ischemic lesions in an area between two neighbouring vascular territories [3]. These territories can be further classified into two broad categories as (a) external (cortical) or (b) internal (subcortical) border zones. Border zone infarcts constitute approximately 10% of all cerebral infarcts [4].

The external or cortical border zones are located at the junctions of the anterior, middle, and posterior cerebral artery territories. The internal or subcortical border zones are located at the junctions of the anterior, middle, and posterior cerebral artery territories with the Heubner,

lenticulostriate, and anterior choroidal artery territories.

The mechanism of external border zone infarction has been widely debated. Many studies have documented hemodynamic abnormalities in the anterior watershed or frontal cortical border zone. However, in many recent studies, no evidence of such hemodynamic impairment was found [5]. In other studies, substantially fewer severe stenoses or occlusions of major vessels than border zone infarcts were found [6]. The cerebral or carotid vessels may appear entirely normal or show mild or moderate narrowing without hemodynamic compromise. Isolated cortical border zone infarcts may be embolic in nature and are less frequently associated with hemodynamic compromise. Microemboli from the heart or atherosclerotic plaques in major arteries may preferentially propagate to cortical border zones, which have lower perfusion than other areas of the vasculature, and, thus, a limited ability to wash out these emboli. Many patients with cortical border zone infarcts have concomitant smaller cortical infarcts. These findings support the hypothesis that an embolic mechanism plays a crucial role in the pathogenesis of external border zone infarcts.

The pathophysiology of small vessel stroke and borderzone infarcts are different and hence the management strategies. By our case report we want to convey that same clinical syndrome of faciobrachial monoparesis could be due to varied pathology, hence thorough evaluation of patient helps in better management and outcome.

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P-Value

P-value is a statistical value that details how much evidence there is to reject the most common explanation for the data set. It can be considered to be the probability of obtaining a result at least as extreme as the one observed, given that the null hypothesis is true. In chemical engineering, the p-value is often used to analyze marginal conditions of a system, in which case the p-value is the probability that the null hypothesis is true.

The null hypothesis is considered to be the most plausible scenario that can explain a set of data. The most common null hypothesis is that the data is completely random, that there is no relationship between two system results. The null hypothesis is always assumed to be true unless proven otherwise. An alternative hypothesis predicts the opposite of the null hypothesis and is said to be true if the null hypothesis is proven to be false.