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Letter to the Editor

Pathophysiologic classification of stroke territory (PCST)

Abstract

Pathophysiologic Classification of Stroke Territory (PCST) was designed for the categorization of brain infarctions. It is based on the neuroimaging and etiology of stroke. PCST provides an impression about mechanism of stroke in clinical practice.

Keywords: Stroke; Pathophysiology; Classification

1. Introduction

The identification of the cause of stroke is critical in research and clinical practice. Many ischemic stroke subtyping systems have been developed for use in randomized clinical trials or epidemiologic studies [1,2]. Syndromic subtyping systems rely on recognizing findings patterns patient's history and physical examination [3]. The etiologic classifications of stroke have direct influence in management of the stroke patients. Due to this practical importance numerous etiologic subtyping systems have been developed for clinical trials and stroke registries [1–4].

We searched for all studies describing pathophysiologic classification of brain infarction using the following keywords into MEDLINE (OVID and PUBMED): [Pathophysiology] and [Classification] and [Stroke] and [Criteria] and [Territory], with the final search performed in February, 24, 2009. Surprisingly, no pathophysiologic classification was found for ischemic stroke. The PCST was developed as an academic tool for categorization of brain infarction by a stroke neurologist and it approved in the scientific committee of Khorasan Association of Neurologists in 2009.

Every stroke subtyping system has some limitations and advantages. Presumed stroke subtype diagnosis guides both clinical evaluation and treatment decisions. Most of these classifications have been developed for specific research projects in specialist institutions, a quite different situation from routine clinical practice [4]. Attributing an infarct to a particular pathogenesis purely on the basis of its site and size is often incorrect. A complete diagnostic workup is required, as the presenting clinical syndromes are usually not distinctive enough to infer the cause. All of the mechanisms could lead to temporary (TIA) or permanent (infarction) tissue injury. For optimal management of the stroke patient, the physician must identify the mechanism of stroke. Because it is not always possible to be absolutely certain of the true mechanism, the clinician often must consider the possibility of more than one mechanism such as atherothrombosis end cardioembolism and must evaluate for each [5]. The frequency of cardioembolic sources for brain ischemia depends on diagnostic investigations of the patients, what lesions are accepted as potentially emboligenic and the specific diagnostic criteria for heart disease [6].

2. Pathophysiology of cerebral ischemia

Low systemic perfusion pressure could be pathophysiology of diminished flow to the brain tissue. Cardiac pump failure affects the brain bilaterally in borderzone regions of vascular territories [7]. Asymmetric effects can result from pre-existing sever stenotic vascular lesions causing asymmetrical distribution of underperfusion [7]. The ischemia occurs mainly between major arterial territories because this is where perfusion pressure is likely to be most attenuated. There are variations between individuals in borderzone areas, and they may even change with time in the same individual [7]. Two pathophysiologic mechanisms could cause

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stroke in patients with sever ICA stenosis. The first mechanism is artery to artery embolism causing intracranial arterial occlusion and subsequent perfusion failure [7]. The second mechanism is distal flow insufficiency with borderzone infarction. Both mechanisms may even be operative in the same patient in different episodes [7]. In embolism, material formed elsewhere within the vascular system lodges in a vessel and blocks the blood flow. Emboli can block any artery depending on the size of the embolic material. The material arises proximally from the heart, major arteries and systemic veins. Cardiac sources of embolism include the heart valves, endocardium, clots and tumors within the atrial or ventricular cavities. Artery-to-artery emboli are composed of clot, platelet clumps and fragments of atherosclerotic plaques that break off from the proximal vessels [8]. Clots from systemic veins travel to the brain through right to left heart shunts such as atrial septal defect or a patent foramen ovale, a process termed paradoxical embolism [8]. Infrequently air, fat, bacterial clumps, tumor cells and injected drugs enter the vascular system and embolize to brain vessels [7,8]. Since cortical and lacunar ischemic stroke patients have similar vascular risk profile, the same type of individual, i.e. diabetic or hypertensive develops either small vessel disease and so lacunar infarcts or large vessel atherothromboembolism and so cortical infarction [9]. The difference in site of vascular disease reflects differing genetic susceptibility [9]. However, many atherosclerotic stroke patients have stroke in both of these vascular territories in different times. Perhaps the same individuals are susceptible for atherosclerosis of both vascular territories but one becomes symptomatic before the other [9].

The small penetrating brain arteries are more often damaged by hypertension than by atherosclerotic process. Increased arterial tension leads to hypertrophy of the media and deposition of fibrinoid material into the vessel wall, a process that gradually encroaches on the already small lumen [10]. Atheromatous plaques, can obstruct the orifices of penetrating arteries [10]. Pathophysiology of thrombosis refers to an obstruction of blood flow due to a localized occlusive process within one or more blood vessels. The lumen of the vessel is narrowed or occluded by an alteration in the vessel wall or by superimposed clot formation [10]. The most common type of thrombotic vascular pathology is atherosclerosis in which fibrinous and muscular tissues overgrow in the subintima and fatty materials form plaques that can encroach on the lumen [10]. Next, platelets adhere to plaque and form clumps that serve as nidi for deposition of fibrin, thrombin and clot leading to formation of an occlusive thrombus [8]. The nidi of loosely adherent platelets and fibrin can break off and embolize distally. Hemorrhage into a plaque leads to acute luminal compromise [8]. Atherosclerosis mainly affects large (i.e. aortic arch) and medium sized arteries at places of arterial branching (i.e. carotid bifurcation and origin of extracranial vertebral artery), tortusity (i.e. carotid siphon) and confluence (i.e. basilar artery) [11]. Intracranial or extracranial arteritis and arterial dissection, fibromuscular displasia, dolicoectasia make luminal clot which blocks the affected artery or lead to distal embolism [11]. Sever vasospasm can lead to decreased blood flow and thrombosis. Aortic atheroma as well as cardiac source of embolism could cause infarcts in multiple vascular territories [7,11].

3. Proposition for Pathophysiologic Classification of Stroke Territory (PCST)

The basis for development of PCST has been comparison of stroke territory based on the neuroimaging and etiology of stroke in the Khorasan Stroke Registry [11] and Khorasan Posterior Circulation Stroke Registry [12]. All of the ischemic stroke patients in these stroke registries underwent a standard battery of diagnostic investigations for detection of stroke etiology [11–14]. Determination of stroke territory was based on a computed tomographic guide to the identification of cerebral vascular territories [15]. The PCST makes a primary view and impression about cause of stroke based on its territorial involvement. We welcome stroke neurologists around the world to help in modification of PCST. The PCST is presented in Table 1.

Table 1
Pathophysiologic Classification of Stroke Territory (PCST).

1. Total Internal Carotid Artery (ICA) territory infarct: ICA atherothrombosis, cardioembolism, ICA dissection all with non-functional anterior communicating artery.
2. Total or large Middle Cerebral Artery (MCA) territory infarct: ICA atherothrombosis or dissective occlusion with functional anterior communicating artery, Cardioembolism, Aortocarotid to MCA embolism with atherosclerotic or dissective pathology, MCA stem atherothrombosis.
3. Deep MCA territory infarct: Cardioembolism, Aortocarotid to MCA embolism involving MCA stem with recanalization or functional pial MCA collaterals.
4. Cortical MCA territory infarct: Cardioembolism, Aortocarotid to MCA embolism.
5. Anterior Cerebral Artery (ACA) territory infarct: Cardioembolism, Aortocarotid to ACA embolism, Vasospasm.
6. Anterior choroidal artery territory infarcts: Cardioembolism, Artery-to-artery embolism.
7. Posterior Cerebral Artery (PCA) territory infarct: Vertebrobasilar arteries to PCA embolism, Cardioembolism.
8. Vertebral Artery (VA) and Basilar Artery (BA) territorial infarcts; brain stem and cerebellum infarctions: Atherothrombosis in distal VA or BA, Cardioembolism, Aortovertebral to distal artertial embolism.
9. Borderzone territory infarcts: Unilateral in sever proximal arterial stenosis with distal flow insufficiency, Bilateral in cardiac arrest.
10. Small deep infarcts in small penetrating arterial territories (Lacunar infarcts): Often atherothrombosis, Artery to artery embolism, Cardioembolism.
11. Ischemia in more than one cerebrovascular territory: Multiple cardioembolic and Aortoembolic processes, Diffuse arterial disease.

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