CORTICAL OR SUPERFICIAL OCCIPITAL ISCHEMIC SYNDROMES OF THE POSTERIOR CEREBRAL ARTERY (PCA)

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Abstract: *Posterior cerebral artery ischemic syndromes are a type of posterior circulation syndromes that present a distinct spectrum of neurological findings, visual, behavioral and oculomotor abnormalities without prominent motor dysfunction. Clinical symptoms can often occur due to occlusions of the closer segments of the basilar artery and its branches, especially the posterior cerebral artery. In this paper, the clinical characteristics of the PCA occlusion are taken into consideration, their imaging findings and management strategies are discussed.*

INTRODUCTION

Ischemic stroke occurs when local blood flow is suddenly limited by vascular occlusion. Rapid resumption of blood flow is essential to save brain tissue.(1) The mechanism of the stroke involving the territory of the posterior cerebral artery: embolization of the heart, aortic arch, vertebral artery or basilar artery; intrinsic atherosclerotic disease; vasospasm (migraine attacks are related to the preferential involvement of the PCA). The causes are: internal carotid stenosis, coagulation abnormalities, vasculitis, sympathomimemic drugs, metabolic disorders.(2,3,4)

The clinical approach to stroke in PCA territory is no different from stroke with another location. A simple three-step algorithm can be applied.

First, it is important to confirm the diagnosis of stroke. If the patient is seen within 3 hours, thrombolytic therapy (e.g. intravenous tissue plasminogen activator [tPA]) is considered.

Second, PCA stroke syndrome should be confirmed after localization of the lesion.

Three, careful and efficient investigations are necessary to determine the mechanism or cause of stroke.

Based on these three steps, one can decide the right preventative strategies. In practice, these three steps are taken almost simultaneously. PCA stroke phenomenology is according to the neuroanatomy and vascular needs. PCA syndromes can be roughly divided into those involving the middle brain, thalamus, occipital cortex, medial temporal lobe, occipitoparietal cortex, and combinations thereof.(3,4)

Due to the diversity of PCA syndromes, a complete neurological examination is required. When thrombolytic therapy is considered, there will be an examination focused on the major areas of neurological dysfunction: level of consciousness, language, half-attention, visual field, ocular movements, inferior cranial nerves, primary motor functions, sensory impairment and ataxia. The NIH Stroke scale is recommended as a model in the patient selected for thrombolytic therapy.

Physical examination should also include cardiological examination and atherosclerosis: cardiac valvular disease, signs of atrial fibrillation and signs of hyperlipidemia (e.g., corneal arch, xanthomata tendency). Often, in PCA stroke syndromes, many of the traits occur concurrently:

Thalamic paramedian infarction: This syndrome, resulting from bilateral medial thalamic infarction, is part of the differential diagnosis of delirium and coma. Patients are often in a coma or agitation, and they may or may not have hemiplegia or hemisensory decline. Occasionally, the nucleus of the cranial nerve III (3) is involved. The prognosis for a good functional recovery is modest due to severe memory dysfunction. The syndrome can result from a "basilar top" arterial embolus. Percheron's artery may be involved.(5)

Pure hemisensory loss: Infarction of the ventral posterolateral nucleus of the thalamus results in hemisensory loss. This is one of the well-described lacunar syndromes. Usually, the vessel involved is a thalamogeniculate branch. The related disorder is Dejerine-Roussy syndrome, in which the fast resolving of hemiparesis and hemiataxia let the patient with hemisensory disorder with paroxysmal pain at the affected site.(4)

Loss of visual field: A general rule of thumb for examining the visual field is that the farther posterior injury is, the more it leads to the loss of the visual field. Bilateral infarcts of the occipital lobes produce varying degrees of cortical blindness depending on the extent of the lesion.(6) Patients often manifest Anton syndrome, a condition in which they believe they can see when they cannot. Patients describe the objects even though they do not see them, erroneously and they neglect this error. Another intriguing phenomenon is vision darkening - although it is about a cortical blindness, patients may respond to movement, lighting or darkening of the surroundings.

Unilateral infarction produces homonymous hemianopia. Macular saving is commonly encountered in the occipital lobe infarction due to PCA occlusion. Probably the saving of the macula is caused by the collateral vascular flexion of the occipital pole from the posterior branches of the middle cerebral artery (MCA) and preservation of the optical radiation, however the bilateral representation of the macular vision as well would have been suspected. Infarction of the lateral geniculate nuclei can produce hemianopia, quadrantanopia, or sectoranopia. The vascular supply is the following: the anterior choroidal artery supplies the posterior hilum and the anterolateral area and the posterior choroidal artery supplies the

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rest. Occlusion of the posterior choroidal artery may produce a syndrome distinct of hemianopia, hemidisesthesia and memory disorder due to lateral geniculate infarction, fornix, dorsomedial and posterior pluvinar thalamic nucleus.

Visual anomalies: There is an abolition of the voluntary or reflex vertical vision (tested by oculocephalic and caloric maneuvers and Bell phenomenon). Isolated paralysis of looking up or down appears less rarely. In addition, hyperconvergence or convergence spasm can be seen in an attempt to conjugate lateral or vertical view. A sign of the failure of the ocular abduction can be seen with eyes resting in a hyperconvergent manner. This phenomenon is called so because it does not occur due to dysfunction of the sixth nerve. Unilaterally or bilaterally, the Collier sign (lifting and retraction of the upper eyelids) may accompany eye oscillations. The oblique deviation of the eyes may reflect lesions of the middle and medullary cerebellar peduncle. Internuclear ophthalmoplegia can rarely occur after mesencephal ventral tegmentum involvement. Pupils may remain small and exhibit transient light reactions or may be eccentrically positioned (corectopia iridis) and often this change is also seen as transient.(7)

Visual agnosia: This refers to a lack of recognition or understanding of visual objects or construction. It is a disorder of the superior cortical function.(3,4) The strict diagnosis of visual agnosia requires intact visual acuity and language functioning. Most patients have bilateral lesions, retaining the visual cortex, but the visual information is disconnected or disconnected for the affected parts of the visual association cortex with reference to visual memory. The patient with visual agnosia can recognize the object, can identify the keys by palpation or hearing (shaking them); but not by sight. True visual agnosia was divided into aperceptive and associative subtypes. In aperceptive visual agnosia, patients cannot name the objects, draw objects from memory, or identify or match objects. However, they can see and avoid obstacles and detect subtle changes in bright light.

In associative agnosia, patients can draw, indicate or match objects but they cannot name them. They can see them in order to model and reproduce them in the drawing, yet they do not recognize the identity of the objects.(4)

Balint syndrome: It occurs typically in degenerative diseases but can also occur in case of a bilateral parieto-occipital infarction, most at the border between PCA and MCA.(8) It is about a triad: simultaneous visual agnosis, optic ataxia and apraxia of insistent gaze. Simultaneous visual agnosia implies an inability to examine a scene and integrate the parts into an interpretation. A patient can identify the specific parts of a scene but he/she cannot describe the whole picture. Optical ataxia involves a decrease in hand-eye coordination; reaching or performing a motor task with a lower visual tracking. Finally, persistent apraxia of insistent vision is a name mistake describing a supranuclear deficiency in the ability at the beginning of a task on command.

Facial knowledge disorders: Prosopagnosia refers to an inability to recognize faces. Typically, this deficit results from bilateral lesions of the lingual and fusiform gyrus; however, cases of non-dominant hemispheric lesions have been reported resulting in prosopagnosia. Usually, it does not happen in isolation and other unknown objects coexist. Selfprosopagnosia, or the inability to recognize one's face in a painting or mirror, may occur in a subject of this syndrome.

Palinopsia, micropsia and macropsia: These are illusory phenomena because they are pathophysiologically changing. They may represent an excess of activity and are traditionally treated with anticonvulsants. Palinopsia describes the persistence of the visual image for several seconds in partial hemicampus blindness. Micropsy and macropsy describe situations where objects appear smaller or larger than expected.

Reading disorders (alexia, dyslexia): Pure alexia may result from infarction of the dominant occipital cortex. Words are treated as if they were from a foreign language. Patients may retain the ability to formulate a word and its meaning if they are read letter by letter or if they copy the letters with their hand. Patients can then learn to read slowly, letter by letter, unable to integrate word groups. Classical alexia without agraphia was described by Dejerine in the 19th century. In his study, he points out the lesion of the left occipital cortex and also the splenium infarction of the corpus callosum that disconnects the fibers from the right occipital lobe reaching the angular gyrus. Less often, the dominant hemisphere, the posterior temporal lobe is supplied by the PCA. The lesion results in Wernicke's aphasia with association of dyslexia and right hemianopia due to concomitant left occipital infarction.

Colored vision disorders (achromatopsia, dyschromatopsia): Lesions of the lingual gyrus in the inferior occipital lobe may cause color perception disorders. Tests like Ishihara reveal a deficit on the board. Colors can be described as gray. This deficiency usually occurs in the contralateral visual field and is called "hemiachomatopsia". A similar problem is color anomia, also called color agnosia, in which patients can perceive and match colors but cannot associate them with their proper name. This disorder was also explained by an interrupted model.

Memory (amnesia): Infarction of the median temporal lobe, fornix, or medial thalamic nucleus may result in permanent anterograde amnesia. Although, traditionally, bilateral infarction has been considered necessary for amnesia, memory functions may be lateralized so that infarction of left-sided structures may have a longer lasting impact on verbal function. Frequently older patients have recent memory deficiency due to unilateral PCA infarction. Recent imaging in patients with transient global amnesia has demonstrated diffusion-weighted lesions in unilateral temporal lobes resulting in temporary amnesia.

Motor dysfunction: When the blood flow to the cerebral peduncles originates in the perforations of the P1 segment, the infarction may occur resulting in hemiplegia or hemiparesis. The clinical syndrome is not different from capsular infarction, but often includes concomitant hemianopsia due to occipital lobe involvement. The syndrome can mimic a vast infraction of MCA.(3,4)

After an evaluation of the PCA syndrome, the next step is to determine the mechanism of stroke. One approach is to start with the heart and proceed rostrally.(9) Cardioembolism is the best known cause of the PCA stroke.

The emboli can form in the heart due to multiple basic conditions. The most known cause is atrial fibrillation. The stroke due to atrial fibrillation can be prevented with long-acting anticoagulants.(10) Other possibilities include a mural thrombus on a hypokinetic segment (e.g., post-myocardial infarction, dilated cardiomyopathy, atrial septal aneurysm, bacterial endocarditis, Libman-Sachs endocarditis, cardiac prosthetic valves, paradoxical embolism through patent foramen ovale and atrial deficit).(10) Mitral valve prolapse and mitral valve failure are likely to be stable risk factors in the event of a stroke.

Embolism due to atheroma of the aortic arch: Due to the occurrence of transesophageal echocardiography, examination and quantification of atheromatous aortic arch disease were possible. The thickness of the plaque greater than 4 mm and / or the presence of the mobile thrombus are predictive of stroke.

Disease of the proximal vertebral artery: Stenosis of

the origin of the vertebral artery is often found in patients with ischemia of the posterior circulation Analogous to narrowed internal carotid artery, stenosis can lead to embolism from artery to artery.

The vertebral arteries are uniquely prone to dissection in the V3 segment or distally as they pass the C1 arch, penetrate hard and enter the large foramen. Dissection may occur spontaneously or is related to minor or major trauma. The mechanisms of PCA stroke secondary to the dissection of the vertebral artery are thought to be due to the formation of the embolus or thrombus around the rupture of the intima.

The usual treatment of the stroke secondary to dissection is anticoagulation; however, no solid evidence exists to support this practice

Vertebral arteriosclerotic disease (9): Caplan studied 10 cases of stroke due to arterioembolism from atherosclerotic vertebral arteries. Frequently, the cerebellum was affected, and the infarction of the PCA territory was also recorded

Disease of the basilar artery. The emboli moving to the posterior circulation can stop at the basilar top, producing ischemia of both PCA territories, thalamus and middle brain.

PCA disease: Intrinsic PCA stenosis due to atherosclerosis is quite rare. However, it is a recognized cause of the stroke

Vasospasm and migraine: Migraine usually affects the posterior circulation. The clinical manifestations of an aura, such as Scintillating scotomas, may show the spread of Leão's depression along the occipital cortex. The angiography of the migraine patients shows a pattern in the form of a bead of vasospasm. Migraine attack most often occurs in the occipital lobes.(1,3,4)

Other diagnostic considerations:

Immediate triggering should alert the physician to the vascular nature of the disorder. Hypoglycaemia, venous infarction, carotid disease and seizure (1,4) are other issues that should be taken into consideration.

ikkRegarding the *differential diagnosis:* - chronic pain syndrome – lacunar stroke - medium stroke - migraine - multiple sclerosis - motor stroke; subarachnoid haemorrhage - subdural hematoma - systemic lupus erythematosus - vascular diseases and rehabilitation of small vessels.(4)

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