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Cognitive problems related to vertebrobasilar circulation

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Abstract: Neurodegenerative disorders are characterized by decreased regional cerebral blood flow. Supporting this concept, both cognitive training exercises and physical activity promote blood flow increase and correlate with healthy cognitive aging. The terminal branches of the posterior circulation supply blood to areas of the brain, such as the thalamus, hippocampus, occipital lobe, and cerebellum, involved with important intellectual functions, particularly recent memory, visual-spatial functioning, and visuomotor adaptations. Amnesia and visual agnosia may be a complication of not only posterior circulation infarctions but also vertebrobasilar insufficiency (VBI) without accompanying structural infarcts. The cognitive impairment may be a manifestation of transient attacks and may persist beyond resolution of symptoms related to ischemia. Early recognition of cognitive deficits in the VBI patient is important because several recent reports show stent placements or medical treatment may improve cognition.

Key words: Cognition, dementia, vertebrobasilar insufficiency, amnesia, agnosia, aging

1. Introduction

Alzheimer disease (AD) is a neurodegenerative disorder characterized by gradual onset, progressive deterioration, and decreased regional cerebral blood flow (1). The degree of severity of extracranial vessel stenosis is accompanied by differential rates of transient ischemic attacks, and hypoperfusion leads to suboptimal supply of nutrients (2). Additionally, chronic stroke patients show hypoperfusion associated with cognitive deficits but without accompanying structural infarcts as indicated by T1- or T2-weighted scans (2,3). Similarly, changes in the white matter of posterior regions occur in the patients with memory impairments and correlate with neuropsychological tests (4). These results suggest that functional areas receive sufficient blood supply to sustain the tissue viability but not enough to support cognitive or neurological functioning (5). Because of the relationship between perfusion and cognition in older adults, whether hyperperfusion can serve as a compensatory mechanism against the cognitive decline seen in normal aging remains unclear (6). In this regard, both cognitive training exercise and physical activity are known to promote increased blood flow, which is correlated with healthy cognitive aging (6,7).

The posterior circulation of the brain includes the vertebral arteries, the basilar artery, and the posterior cerebral arteries and their branches. Posterior circulation related to cognitive decline is associated with lesions in the occipital lobe extending into the parahippocampus or the splenium (8,9). Patients with isolated occipital lobe lesions demonstrated a decline in memory performance, but injuries involving the splenium or posterior ventral temporal lobe in addition to the occipital lobe led to more diverse neuropsychological impairments, including visuospatial, executive, language-related, and memory functions in addition to attention (8). Park et al. showed that the severity of cognitive impairment was independent from left hemisphere lesion location, sex, age, educational level, or the time between stroke and the mini-mental status examination (MMSE) assessment. Only the lesion volume was negatively correlated with MMSE score (8). In a newly published report, Gücüyener et al. demonstrated that cerebral blood flow of posterior circulation decreased in patients with AD (10). Nishia et al. reported that verbal memory impairment, language disturbances dominated by anomia and word-finding difficulty, and apathy were observed in patients with lower regional cerebral blood flow in the thalamus, anterior temporal lobe, inferior parietal lobule, and occipital lobe of the left hemisphere (11). The authors proposed that the Papez circuit disruption at the mammillothalamic tract and possibly thalamomedial temporal disconnection were responsible for memory impairment and the thalamoanterior temporal disconnection was associated with language disturbances (11).

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Aging is associated with subcortical microangiopathy and elderly people may have impaired metabolic activation of cerebral blood flow to the posterior regions of the brain. Improved posterior circulation or vertebral artery perfusion resulted in good cognition (12,13). Performing angioplasty to improve posterior circulation may also improve cognition in elderly patients (14). Posterior cerebral artery (PCA) blood flow velocity (BFV) significantly increases during word stem completion and visual search tasks (15,16). Healthy elderly subjects tend to have overall greater increases in BFV during cognitive tasks (16). Damage in the posterior cerebral artery region results in semantic but not agnostic deficits (15). High-level visual deficits are more frequent than previously reported following PCA strokes affecting the ventral visual cortex (17). In addition, categorized deficits such as prosopagnosia, pure alexia, topographic agnosia, and/or deficit in various body-related tasks have been attributed to lesions involving the PCA region. In this article, we discuss the cognitive deficits following PCA strokes, especially agnostic visual disorders, and the anatomical-clinical correlations.

Extracranial vertebrobasilar artery thrombo-occlusive diseases may cause repetitive transient ischemic episodes, which are defined as a brief episode of neurologic dysfunction caused by a focal disturbance of brain ischemia without imaging evidence of infarction and, less frequently, brain-stem or cerebellar infarction (18). Vertebrobasilar insufficiency (VBI) is mainly a disease of elderly people, like dementia syndromes. The diagnosis of VBI is clinical and the cause is usually atherosclerosis of posterior circulation. Transient global amnesia is a manifestation of transient ischemic attack in the vertebrobasilar arterial system (19). Vertebrobasilar calcification is a risk factor for thalamic infarction; thus, evaluation of posterior circulation is important because transient neurological attacks due to vertebrobasilar ischemia precede posterior circulation strokes (20). Visual field defect is the main clinical symptom in cortical and deep infarct of the PCA; however, sensorial, motor, and neuropsychological deficits occur mostly in patients with cortical deep infarcts involving the thalamus (21). Moftakhar et al. reported that many patients with cervicocerebral stenosis have cognitive dysfunction and that anterior and posterior circulation revascularization with stent placement improves cognition, although cognitive improvement after stent placement in the posterior circulation is less clear (22).

There are some newly reported studies about the relationships between cognition and transient problems. Guyomard et al. showed that transient ischemic attack (TIA) patients had cognitive impairment compared with controls without vascular risk factors at the time of diagnosis (23). The studies examining the causal relationship between vascular risk factors and cognitive functions showed that diabetes was an independent correlate of visual-spatial dysfunction while hypertension and hypercholesterolemia were related to memory dysfunction (24,25). The cognitive impairment may be a manifestation of TIA and may persist beyond resolution of symptoms related to ischemia (26,27). As a result, it was obvious that transient attacks were related to cognitive deficits, although both of the studies mentioned above were not detailed and included anterior and posterior circulation cases.

2. Main symptoms of posterior circulation

Visual field loss is the most common reported sign. Agnosia is another problem that may be seen in patients with posterior circulation insufficiency. Agnosias are disorders of recognition, and objects, faces, and colors cannot be separately distinguished (28). Visual agnosia can be as high as 8.5% in isolated infarctions in the superficial region of the PCA (29). Agnostic visual or cognitive disorders following PCA infarcts are in fact more frequent than previously reported and several battery tests, such as the Wechsler Adult Intelligence Scale, Wechsler Memory Scale, Birmingham Object Recognition Battery, Visual Object and Space Perception Battery, Benton Facial Recognition Test with measure of accuracy and response times, and Warrington Recognition Memory Test, are required to identify such deficits (17,30,31). The visual long-term memory associated with human faces is maintained by the neural networks between the fusiform face area (FFA) and the fusiform gyrus, and posterior cerebral artery strokes usually result in the loss of human face recognition (prosopagnosia) and, sometimes, in category-specific object agnosia (32,33). The anterior temporal cortex as well as the FFA may be the sites where facial memory is stored (34). The right fusiform gyrus is the most important visual memory processing/storage site, while the left fusiform gyrus functions as a complex interface between the visual and lingual brain (35). Conversely, acquired prosopagnosia is usually associated with bilateral or right-sided lesions of the occipital or temporal lobes. In rare cases, after left-sided lesions in left-handed subjects, prosopagnosia is attributed to a reversed hemispheric specialization for face processing (36). White matter fibers link the occipital and the FFA and damage to this pathway may result in altered facial recognition (37). Other etiologies of facial recognition problems include carbon monoxide poisoning, temporal lobectomy, encephalitis, neoplasm, right temporal lobe atrophy, trauma, Parkinson disease, and AD (30). Not only a damaged brain but also hypoperfusion without any lesion, i.e. VBI, results in facial recognition problems (38).

3. Anatomy of posterior circulation related cognition

When evaluating regions related to cognition problems in posterior circulation hypoperfusion/stroke, there are 3 main areas. First is the hippocampus, located in the limbic system, an area important in consciousness and remembering. The hippocampus, the temporal lobes, and the structures of the limbic system that are connected are essential for the consolidation of long-term memory. The neural circuit that arises from the hippocampus via the fornix, mamillary body, and posterior cingulate cortex and then projects back to the hippocampus is known as the Papez or Delay–Brion circuit (Figure) and plays a central role in memory (39). Acute amnesia of vascular origin appears in lesions of PCA, thalamic arteries, and the anterior choroidal artery (33,40). Amnesia can present after unilateral ischemic stroke in the PCA and anterior choroidal artery regions (41). Sudden memory loss with prolonged cognitive deterioration, initially resembling a transitory global amnesia-like episode, might be caused by ischemic stroke in the hippocampal region (42). Similar to AD, patients reveal an anterograde episodic amnesia with an inability to retain new information, and short-term memory is clearly affected, although access to old memories remains relatively intact (40–42). A careful neuropsychological examination is necessary to detect resulting memory deficits in patients with hippocampus infarction.

The second main area is the thalamus, a connection center that regulates cortical activity. The thalamus is part of the Papez circuit and various structures of the limbic system exert their influence on the hippocampus and the temporal lobe via the hippocampal/mammillothalamic tract (39,43). The thalami of the human brain obtain their blood supply from the first segment of the posterior cerebral artery (39,43). Neuropsychological deficits occur mostly in patients with deep cortical infarcts involving the thalamus (21). The unique characteristics of thalamic damage may consist of not only memory dysfunction but also hypophonia, time disorientation, and apathy (43). Clinical observations have documented that the thalamus participates in a great variety of cognitive functions and mental activities, including memory, language (dysphasia), perception, and emotion.

The third main area is the cerebellum. A cerebellar lesion can impair visuospatial abilities with different characteristics depending on the side of the lesion (44).

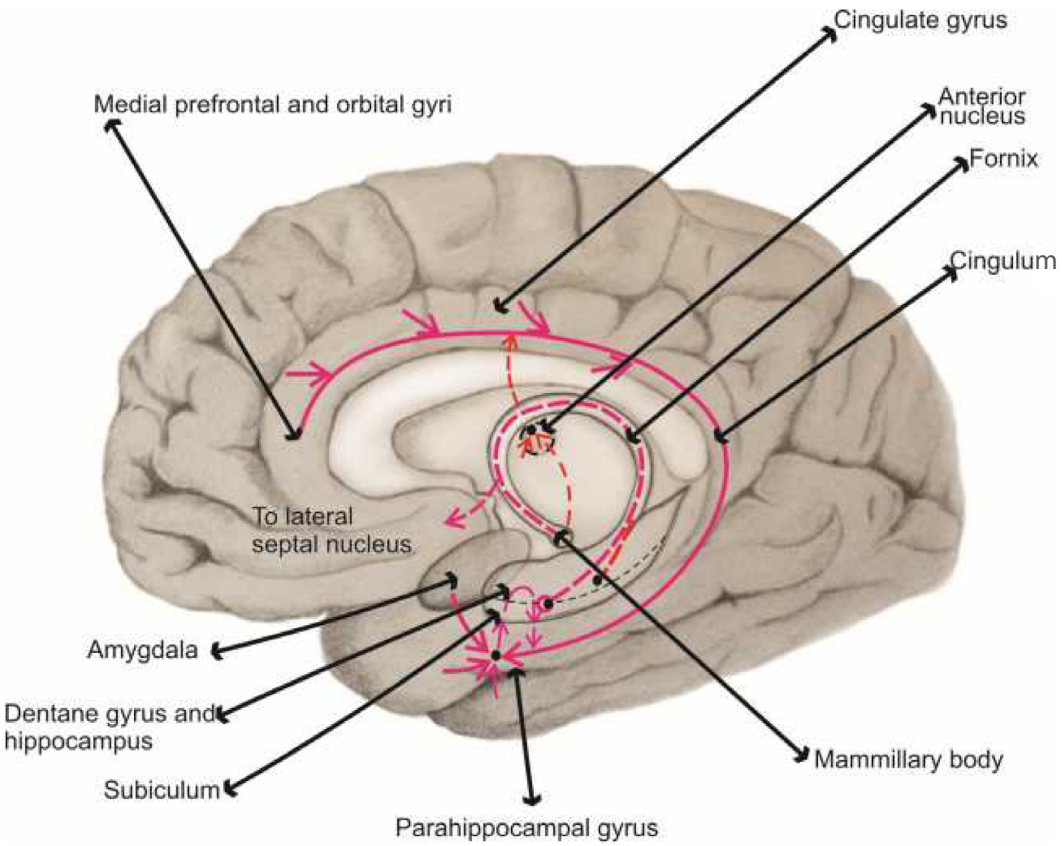

Figure. The neural circuit that arises from the hippocampus via the fornix, mamillary body, and posterior cingulate cortex and then projects back to the hippocampus (by Sadi Çağdır).

The patients with left and right cerebellar lesions were impaired in solving visuospatial tasks and had specific deficits. Subjects with left-sided lesions processed fewer items but correctly, while subjects with right-sided lesions processed more items but with less precision (45). Werner et al. confirmed the importance of the cerebellum for visuomotor adaptation (45). While adaptive improvement was impaired in both posterior inferior cerebellar artery (PICA) and superior cerebellar artery (SCA) patient groups, visuomotor recalibration appears to be located within the SCA region, with lobules V and VI being particularly important (21,45). Cerebellar patients present a variety of deficits particularly in the areas of decision-making, reasoning, and performance (46,47). PICA lesions but not SCA lesions resulted in cognitive and affective deficits, indicating a dominant role for posterior cerebellar regions in cognitive and affective processing (48). Patients with cerebellar infarcts exhibited significantly lower neuropsychological performances as compared to controls, without any obvious difference between the different vascular cerebellar regions. The deficit pattern observed in isolated cerebellar infarcts highlights the nonmotor functions of the cerebellum and functional relationship between the cerebral cortex and the cerebellum (49).

4. Conclusion

Both anterior and posterior circulation are important in cognition. Brain stem or cerebellar symptoms are more prominent in posterior circulation strokes or transient ischemic processes; thus, cognitive deficits including memory, language, perception, emotion, or visual agnosia tend to be underdiagnosed. Evaluation of posterior circulation by radiological investigations is important because transient neurological attacks due to transient attacks precede posterior circulation strokes in patients with vascular risk factors. Whether optimal management of modifiable risk factors such as hypertension would prevent decline in cognitive function in VBI patients needs further investigation. Routine cognitive assessment in TIA work-up is recommended in order to lessen cognitive dysfunction in these patients with TIA.

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