Acute Confusional State with Posterior Cerebral Artery Infarction: A Challenge in the Diagnosis of Acute Stroke at Bedside

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Infarctions in the territory of the posterior cerebral arteries (PCA) represent 5-10% of all strokes in the general population[(1)]. Anatomically, two main territories of vascular supply from the PCA can be differentiated. Proximal and deep PCA territory includes the paramedian mesencephalon, and medial, posterolateral thalamus supplying by the P1 and P2 segments of the PCA; and the superficial PCA territory includes the occipital, temporo-parietal lobes and hippocampus supplying by P3 and P4 segments[(2)]. According to the report by Cals et al.[(3)], isolated superficial territory of the PCA infarction, including combinations of the areas supplied by the calcarine, temporo-occipital, parieto-occipital, and temporal arteries, are uncommon, representing less than a third of all PCA infarctions. The most common clinical sign of pure superficial PCA infarction was visual field defect (96%); the second common clinical signs are neuropsychological deficits and higher order visual dysfunction including memory impairment, dysphasia, alexia with or without agraphia, visual neglect, visual agnosia, color anomia and prosopagnosia (58%). Interestingly, agitated confusion was reported in this study, although with a rather low incidence (7%).

Acute confusional states and agitated delirium are among the most common psychopathologic disorders in the elderly. However, they are rarely reported as a major symptom in the course of cerebral infarctions. It is known that agitated confusional states can be seen in patients with right middle cerebral artery infarction[(4)]; also infarction of the PCA can cause agitated delirium, particularly when it involves the dominant or bilateral hemispheres[(5,6)]. Destruction or disconnection of dominant hemisphere neocortex from limbic structures, resulting in impairment of focal attention, loss of linguistically organized memory, and/or disruption of temporal sequencing may be responsible for this syndrome [(5)]. In this issue, Shih et al.[(7)] present their findings of a hospital based study which aimed to identify the possible anatomic sites and risk factors for the development of confusion or delirium in patients with PCA infarction. They found that confusion or delirium tends to be developed in patients with left or bilateral PCA infarct, and the medial occipito-temporal gyri, especially the left side involvement; is the pivotal factor for the development of confusion or delirium; higher prevalence of diabetes mellitus was also observed.

Now, thrombolytic therpy using recombinant tissue plasminogen activator is effective in acute ischemic stroke if administered within 3 hours of onset (Level 1 evidence). So, “time is brain” and any patient who is

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suspected having acute stroke must be taken to hospital rapidly, assessed quickly and accurately, and promptly sent for the appropriate investigation. However, stroke is a clinical diagnosis, one of the barriers to delivery of thrombolysis for acute stroke is the uncertainty of diagnosis[8]. Therefore, improving the ability of distinguishing between stroke and stroke mimics based on history and neurological examination is very important. According to the report by Hand et al.[9], decreased level of consciousness predicted a mimic of stroke. On the contrary, from the report of Shih et al.[7], around 48.3% of patients with PCA infarct had symptoms of confusion/delirium. The recognition of acute confusional states from PCA infarcts and its distinction from other causes of confusion (substance intoxication or withdrawal, metabolic encephalopathy, or dementia) is thus very important because of the different diagnostic, prognostic and therapeutic implications.

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