VASCULAR LESIONS MOSTLY AFFECT EMOTIONAL REGULATION

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It has been known for a long time that acute stroke can also affect mood. However, determinants of post-stroke depression (PSD) have been strongly debated between proponents of a neuroanatomical model, assuming that PSD is causally related to the localization of ischemic lesions and supporters of a psychological model, maintaining that PSD is a result of the psychosocial adjustment required by the disease.

Human studies have suggested a relationship between PSD and lesions in various locations including the left frontal lobe, bilateral frontal cortex, right hemisphere and left anterior and right posterior areas, resulting in disrupted cortical noradrenergic pathways or a failure to upregulate serotonin receptors. However, many studies have not been able to replicate these results and have reported no association between PSD and stroke location suggesting psychological and other biological factors may be more important determinants of PSD.

Late onset depression has been described in association with clinical or neuroimaging evidence of chronic cerebrovascular disease, in particular white matter changes. This has led to the concept of vascular depression and the description of a specific clinical pattern for this entity, including the presence of vascular risk factors (hypertension, atrial fibrillation, hyperlipidemia), evidence of other vascular pathology (carotid bruit, angina, history of stroke or myocardial infarction), a lack of prior personal or family history of depression and a poor response to therapy. The exact type, location and severity of vascular lesions that can lead to depression has yet to be determined but the association with white matter lesions, basal ganglia vascular pathology and cortical or subcortical infarcts has been well described. Underlying pathophysiological hypotheses have implicated increased platelet activation (increased platelet factor IV and thromboglobulin in depression), high catecholamine concentrations (leading to hypertension and hyperlipidemia), increased frequency of risk associated behaviours (smoking, poor adhesion to therapy) in depressed states.

Although further studies are needed to increase our understanding of vascular depression and its underlying mechanisms, this key concept could provide novel research avenues and open the way for new preventive strategies based on the control of vascular risk factors.