Student's Corner
Letter to the Editor

Reformulating the Lacunae Hypothesis

Madam, Stroke is the third leading cause of death and the leading cause of permanent disability in the world. Most of these strokes occur in developing countries.[1] Pakistan reports one of the highest incidences of Lacunar infarcts worldwide (43% of all ischaemic strokes as compared to the 10% reported in western figures).[2] Comparative studies have also noted a higher frequency of Lacunar infarctions in South East Asian countries.

Since their detailed description by Fisher, lacunar strokes are known to have particular clinical presentations, etiologies and prognostic implications. The "lacune hypothesis" states that lacunes are caused by a combination of hypertension and characteristic vascular lesions involving single perforating brain arteries.[1] This hypothesis has made its way to general practice in Pakistan where at times no detailed evaluation of a patient is necessary when a diagnosis of "lacunar infarction" is made and hypertension is the assumed culprit. Recent studies have shown none or little differences in the prevalence of hypertension in patients with lacunar infarction compared to patients with non-lacunar infarction.[3] It has been proposed that infarcts, under the umbrella term of lacunes, can be subdivided in different categories.[4] These subtypes constitute morphologically, radiologically and clinically distinct entities. Differences in risk factors between these subtypes of lacunar infarcts might support a distinct underlying pathological process.

Multiple, small lacunaes are usually caused by small vessel disease (SVD) and the larger, single ones by atheromatous or embolic perforator occlusion. It is suggested that patients with non-SVD lacunar strokes (associated with hypertension, dementia, leucoariosis and age) have a worse clinical outcome than the SVD lacunar strokes (associated with hypercholesterolaemia, diabetes and myocardial infarction).[4,5] Therefore, some patients with lacunar infarction may require a more aggressive workup to find an etiology for the infarct. This would also provide the possibility of recognizing during life those patients who are at a high probability of developing subcortical vascular dementia.

These unanswered questions need further research keeping newer observations in mind. To decipher the disease mechanisms which are locally pertinent, preliminary strategies may include risk factor profiling of lacunae subtypes. Determining their respective pathophysiology would help in initiating appropriate treatment and prompt further studies. Studies are needed from regions where this is a common disease to elucidate its biologic underpinnings, to uncover novel risk factors, to detail genetic predilection and eventually find appropriate medications. Efforts should be made to understand locally relevant intracranial infarct subtype.

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