On the Edge of the Brain: The Border Zones Infarcts!!

Border zone or watershed infarcts are ischemic infarcts that occur at characteristic locations at junctions of either two main arterial territories or distal perfusion sites within an arterial territory. They constitute around 10 per cent of all ischemic infarcts[1] and are well described in the literature. Their pathophysiology remains controversial, with possible multiple mechanisms contributing to their development. Hemodynamic impairment as an underlying pathology had been widely accepted in the past as the cause of these infarcts either due to severe stenosis in large arteries, especially internal cerebral artery (ICA), or acute hypotensive episodes like in shock or cardiopulmonary bypass. Micro embolism is also believed to contribute to the same from the heart or narrowed arteries.[2] Caplan suggested that hemodynamic compromise and micro embolism act together in border zone infarcts.[3] In a study by Yong *et al*.,[1] 120 patients with border zone stroke were identified and divided into internal border zone (IBZ) and cortical border zone (CBZ) territories. Their analysis revealed that IBZ patients had a higher degree of either middle cerebral artery (MCA) or ICA, whereas concomitant small infarcts were seen more commonly in CBZ infarcts. The rosary pattern is the most sensitive indicator of hemodynamic failure, as mentioned by Maddula *et al*.[4] Clinical deterioration in the first seven days occurred more commonly in IBZ infarcts, and they had poorer outcomes at 3 months. Association with intracranial atherosclerotic disease (ICAD) and impaired blood flow, impaired clearance of emboli, and perfusion delay across a stenotic artery predisposes to the same.[5-7] Whereas external border zone infarcts are more frequently embolic, IBZ infarcts are more commonly hemodynamic due to hypoperfusion. IBZ infarcts are more commonly located at corona radiata and centrum semi-ovale and have a close association with ICAD. They are typically described between lenticulostriate and superficial perforators of the MCA territory. Peculiar symptomology is usually uncommon in stroke caused by borderzone infarcts, and these patients may present with recurrent syncope, limb shaking transient ischemic attacks (TIA), and retinal ischemia. Syncope denotes widespread bilateral cerebral hypoperfusion at the onset of stroke. Limb shaking TIA occurs due to hypoperfusion in the area of basal ganglia which are often involved in the pathogenesis of these strokes. Recognition of characteristic patterns of these infarcts can drive management and investigations. It is also important to evaluate possible sources of embolism including atrial fibrillation, recent myocardial infarction, prosthetic valve, sick sinus syndrome, dilated cardiomyopathy, and patent foramen ovale. These infarcts were also described in patients with COVID-19 infections who were critically ill and who do not wake up without any clear-cut period of sustained hypotension and hypoxia and had these infarcts on their MRI and had considerably delayed recovery.[5]

This issue of the Annals of the Indian Academy of Neurology has an interesting Indian study on border zone infarct. The authors of the study studied 52 out of 400 patients who presented in the study pattern with border zone infarcts. 62% had a CBZ infarct.[8] An interesting feature of their study was that 69.2 per cent showed symptoms of progression more in CBZ infarcts, and 25% had a history of syncope or presyncope. Also, most of these infarcts were not found to be disabling. Around 60% of patients had ICA stenosis, with a higher percentage having ICA stenosis (53%) and a smaller number (7%) having MCA stenosis. No significant difference was found between ICA/MCA stenosis between CBZ versus IBZ infarcts. However, the major limitation of the present study is that CT angiography brain was not performed in all the cases, which may result in under-reporting of ICAD.

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