Border-Zone Infarcts and the Inside Story!

Border-zone infarcts (BZIs) occur at the juncture of two arterial territories and comprise almost 10% of all brain infarctions. It includes cortical/external border zone infarcts (CBZs) and deep/internal border-zone infarcts (IBZs).

CBZ are classically wedge-shaped or gyri form [Figure 1]. It involves the following areas: 1) area between the frontal horn of the lateral ventricle to the frontal cortex (ACA/MCA watershed), 2) area between the parieto-occipital cortex to occipital horn of the lateral ventricle (MCA/PCA watershed), 3) the subcortical white matter of vertex in parallel to subfalcine area of brain (in profound diffuse hypoperfusion), 4) parieto-occipital region posterior to lateral ventricle (triple water shed zone where MCA, ACA, and PCA converge).

IBZ [Figure 1] are recognized when they are at least three, each having at least three millimeters in diameter and present next to the lateral ventricles in corona radiata or centrum semiovale commonly called a string of pearls.[1] The watershed areas ranging between lenticulostriate, perforating medullary, recurrent artery of Heubner and the ACA, MCA, and PCA territories are the internal border-zone areas.

The etiology of BZI is still under evaluation but the consensus is that internal BZI is because of hemodynamic challenges due to proximal stenosis and decreased flow due to low cardiac output, whereas cortical BZI is related to embolization (including fat and air embolism) either from the heart or atherosclerotic plaques in large vessels.[2]

BZI may have subtle symptoms and, if ignored, patients may land up with life-threatening events because of further embolization or impaired flow state. So, knowledge of typical clinical presentations is important. For example, visual field defects are more common than usual motor, language, and sensory deficits in posterior BZI. Bilateral limb weakness, limb shaking movement disorder, retinal ischemia, transient loss of consciousness and even seizure (in case of cortical involvement) are hallmarks in these vascular events.[3]

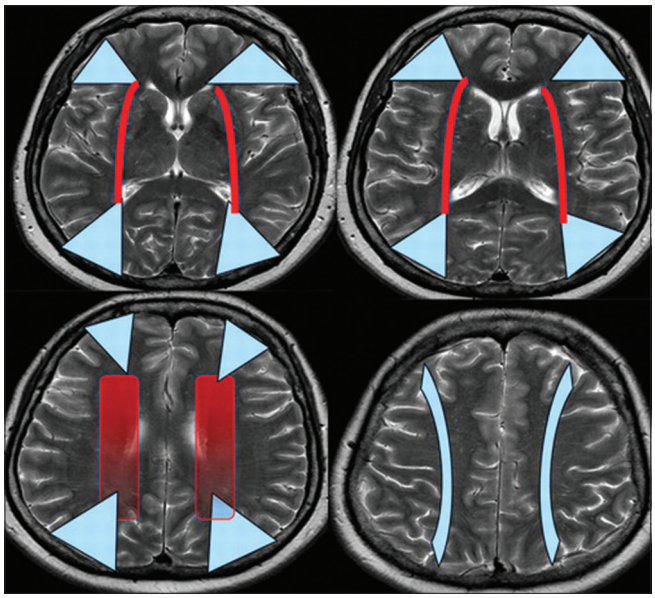

Figure 1: Border-zone infarcts: cortical/external border-zone as blue and deep/internal border-zone infarcts as red

Thus, all patients of syncope/pre-syncopal events/history of orthostatic dizziness, or event precipitated by over-treatment of blood pressure lowering, should be under suspicion for border-zone infarcts. These events might be a harbinger of havoc and a patient at any point in time may land with a catastrophic stroke. We should also remain vigilant about vascular events post-cardiac surgery and post-cardiac arrest, where stroke both because of a hemodynamically compromised state and embolism are possible. This is especially important as sometimes they may occur as in-house strokes and carry significant medico-legal importance.

Preferably, a CT-Angiogram from the aortic arch to the vertex needs to be done as it may reveal a compromised arterial tree. In selected cases, a plaque morphology study (using a dedicated contrast MRA with vessel wall imaging) may be necessary when there is a recurrent vascular event in the same vascular territory without a hemodynamic compromised state. Transcranial Doppler may help us in special cases when understanding the pathophysiology of recurrent embolization and intracranial hemodynamics is important. Brain collateral circulation plays an important role in protecting against border-zone infarcts by redistribution of blood. Anomalies of the circle of Willis, a major contributor to collateral flow, may increase the risk of border-zone infarcts. The importance of detailed cardiac evaluation, including TEE, prolonged cardiac rhythm monitoring, and in special situations, cardiovascular MRI, may be necessary to rule out the cardiac source of embolism.

Recognition of types of BZI is important because they carry huge prognostic and management implications. Patients with IBZ infarcts are noted to have worse hospital courses and remain disabled. In contrast, patients with CBZ infarcts may have a benign clinical course. Hence, more aggressive care is needed in patients, including endovascular therapy, to improve vascular insufficiency in IBZ infarcts/ischemia. Whereas, medical therapy is sufficient for most CBZ infarct cases.[4]

The strategic sites of involvement in BZI, like an anterior portion of the caudate nucleus head, insular cortex, and outer part of globus pallidus contribute significantly to stroke symptomatology. Studies have already proved that lesion topography (besides lesion volume) are important prognostic marker. From the area of ischemia, we can understand the involvement of particular vessels like lenticulostriate arteries, and this information may

help us decide for or against acute endovascular therapy.[5] Thus, knowledge of these strategic sites of involvement in border-zone infarction or ischemia (which are often supplied by arteries that behave like end-arteries) is important for treatment purposes. However, we need to appreciate that there is wide variation among persons in the anatomical distribution of these vessels and their area of supply, so our treatment strategy and the final outcome may vary accordingly.

Sometimes, it is difficult to differentiate between infarcts secondary to small vessel disease and border zone infarcts. It is important both for management reasons and for prognosis as well. MRI findings of rosary pattern of white matter changes, a smaller number of microbleeds, and markedly impaired perfusion are in favor of BZI. While the evidence of lacunar infarcts, and symmetrical white matter hyperintensities and presence of micro-bleeds with no significant perfusion deficit suggests the diagnosis of small vessel disease.[6]

Identification of these patients of BZI may help us provide the following treatment strategies and make future plan:

1. Start medical treatment in advance like antithrombotic(s), high-dose statin. We may modify antihypertensive(s) by individualizing the blood pressure goal (maybe more lenient towards the upper limit of blood pressure in intracranial or extracranial vascular stenosis) keeping in mind the hemodynamic compromised state and ruling out other contraindication(s) for doing the same.
2. Modify the lifestyle of patients as avoidance of precipitating factors and optimizing hydration status in patients with hemodynamically compromised state like in patients with significant intracranial/extracranial stenosis besides selecting them for surgical/endovascular intervention (keep them hydrated as much as tolerated if no contraindication).

This is how we can project our future management from a border-zone infarct in imaging and decrease the incidence of future vascular events.

The present study gives us an excellent idea about the clinical and radiological characteristics of BZI in this part of the world.[7] More studies like these will aid in treating this vascular event with more confidence.

Debabrata Chakraborty

Department of Neurology, Apollo Multispeciality Hospitals, Kolkata, West Bengal, India

Address for correspondence: Dr. Debabrata Chakraborty, 64/4A/9, Beliaghata Main Road, Kolkata, West Bengal - 700 010, India.
E-mail: drdchakraborty1980@gmail.com

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