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Original article

Clinical, demographic and neuroradiological features of different types of cerebral border zone infarcts in North-west India

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ABSTRACT

Background: Whether there are different pathogenic mechanisms for different types of cerebral border zone infarct is still not clear as most of previous studies had conflicting results. Furthermore there is no data available for Indian population.
Objectives: To study the demographic, clinical and radiological profile of different types of border zone infarct (BZI) patients in North-west India.
Methods: We prospectively analyzed 672 consecutive patients with ischemic stroke. Among them 86 border zone infarct patients were selected and further categorized as external border zone infarct (EBI), internal border zone infarct (IBI) and mixed border zone infarct (MBI) based on diffusion-weighted imaging characteristics. Baseline patient characteristics, clinical courses, and neuroradiological features were compared between different types.
Results: We identified 31 patients with EBI, 21 with IBI and 34 with MBI. When compared with the EBI patients, IBI and MBI patients had significant middle cerebral or internal carotid artery stenosis (*P*-0.008). Concomitant small cortical infarcts were more seen with EBI. More patients in IBI group had early clinical deterioration and poor outcome at 1 month after stroke than in EBI group.
Conclusions: IBI are mainly caused by hemodynamic compromise, whereas embolic mechanism responsible for the genesis of EBI. Patients with IBI had worse hospital course and poor outcome than EBI patients. Different therapeutic approaches may be required for different types of border-zone infarcts to improve outcome.

1. Background

Border zone or watershed infarcts are ischemic lesions that occur between two neighbouring non-anastomosing main arterial territories [1]. Border-zone infarcts (BZI) constitute approximately 10% of all brain infarcts [2] and are broadly divided into two groups [1] external border zone infarct (EBI; or cortical border zone) [2] internal border zone (IBI; or subcortical border zone).

Pathophysiology of border zone infarct has not been completely understood. Most of previous reports [3,4] concluded that hemodynamic compromise is a major cause of border zone infarct while some other studies [5,6] suggested that embolization is implicated in pathogenesis of border zone infarct. Few studies [7,8] revealed that both hemodynamic compromise and embolization are involved in development of border zone infarct.

Regarding pathophysiology, inconsistent results were most likely due to the fact that earlier studies either included both EBI and IBI into a single group or focused only on one type. Very few studies [9] tried to find out the differences in mechanism of EBI and IBI but the results have been variable. In addition, mixed border zone infarct (MBI) patients in whom both EBI and IBI are present, have never been enrolled in these studies. Furthermore there is no such type of study in Indian population; therefore we planned for this study to confirm whether there is a different pathogenic mechanism for EBI and IBI in Indian population.

In the present study, we analyzed clinical and neuroradiologic characteristics and difference in prognosis of patients with different types of BZI.

2. Methods

2.1. Type of study

Present study is a hospital based prospective, observational study done from September 2015 to August 2017.

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2.2. Inclusion and exclusion criteria

Patients with acute ischemic stroke who presented within 48 h of onset and MRI brain diffusion-weighted imaging (DWI) revealed cerebral border zone infarct, were included in the study. Patients with recurrent stroke, infarct associated with hemorrhage, cerebellar infarct, associated metabolic or septic encephalopathy were excluded.

2.3. Study protocol

All patients had undergone a complete evaluation including medical history, vascular risk factors, routine blood tests, stroke scales, electrocardiogram (ECG), echocardiogram and vascular studies. CT or MR angiogram of brain and neck vessels was done to evaluate extracranial and intracranial vascular status. Potential sources of cardioembolism (PSCE) were identified by ECG, 2Dimensional-echocardiogram (and blood culture, Holter monitoring as needed), if patients had any of the following: recent myocardial infarction (<3 weeks), dilated cardiomyopathy, acute bacterial endocarditis, mitral stenosis, prosthetic valve replacement, atrial fibrillation, sick sinus syndrome or patent foramen ovale.

Basic clinical characteristics of every patient were assessed (NIHSS score) [10]. Score was checked serially at 1, 3 and 7 days after admission according to NIHSS protocol. NIHSS score was calculated by same examiner to eliminate the inter-observer bias. On the seventh day clinical course was defined as [1]: improved (if NIHSS score decreased by >2 points) [2], stable (if NIHSS score decreased by 2 points) [3], deteriorated (if NIHSS score increased after admission). The outcome was assessed by Modified Rankin Scale [10] at 1 month after stroke onset and considered poor if the Modified Rankin Scale (MRS) value >3. Although use of both NIHSS and MRS score require proper training and these scales also have their own limitations, we have used these scales in our study as they are standardized, well accepted and widely used in clinical practice.

2.4. Ethics

Informed consent was taken from all patients for the use of data for research purpose and the study is approved by institutional ethical committee. All investigations were done only for clinical reasons including magnetic resonance imaging (MRI) and computed tomographic angiogram (CTA).

2.5. Imaging protocol

MRI was performed on a 3.0-T Philips Ingenia (Netherlands) scanner using an identical protocol in all patients. The protocols and typical parameters were as follows: Transverse T2WI (Repetition time/Echo delay time: 2500 ms/80 ms); slice thickness / gap for Transverse T2WI (5/1 mm) with a 256 × 256 acquisition matrix. All studies were performed with cardiac gating without using gradient-moment nulling technique. CTA of neck and brain vessels was performed on Philips Ingenity (Netherlands) scanner with an identical protocol in all patients. The protocols and typical parameters were as follows: 128 slices; slice thickness/gap = 0.9 mm/0.45 mm; Pitch rotation time: 0.5 s. 70–90 ml of Non-ionic contrast (Iohexol 300 mg/100 ml) mixed with 30 ml of Normal saline was injected at 4.3–4.5 ml/s. Reconstruction and bony substraction was done automatically with workstation software developed by Philips.

2.6. Imaging analysis

Based on DWI characteristics, type of BZI was determined with the help of expert radiologists who were unaware of patient's clinical status (Fig. 1). EBI defined as hyperintense areas on DWI sequence present at the junctions of the anterior cerebral artery

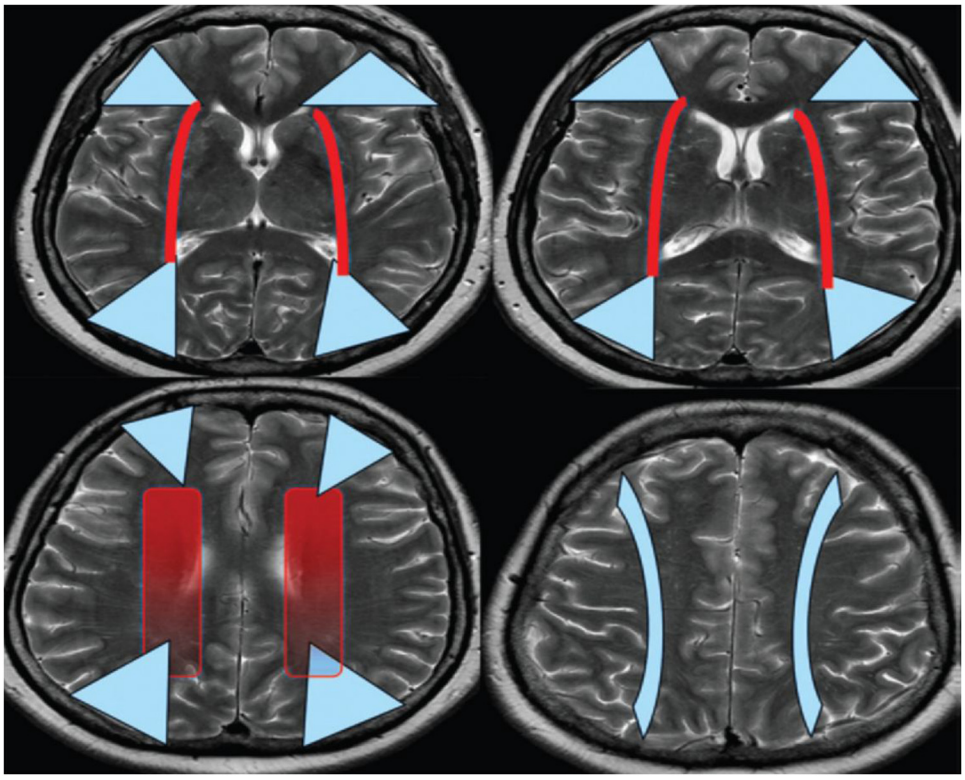

Fig. 1. MRI T2W images axial view showing different types of cerebral border zone infarct. (External (blue) and internal (red) border zone infarct).

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