Etiologic Subtypes of Watershed Infarcts

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Background: Two types of watershed infarcts (WI) are recognized. Internal WI are usually attributed to either severe stenosis in large arteries or acute hypotensive events, whereas external WI are thought to be caused by embolism. The aim of this study was to determine the etiologic background and prognosis of external and internal WI in our patients. *Methods:* We reviewed the medical records and diffusion-weighted images of the patients who were admitted to our stroke unit with acute ischemic stroke between January 2012 and November 2014. The demographics, clinical features, radiologic investigations, and other etiologic tests of the patients with internal or external WI were recorded. We determined etiologic stroke subtypes according to the automated Causative Classification System. *Results:* Fifty-three patients with WI were detected in our registry. Twenty-two (41.5%) of them were women. The mean age was 69 ± 12.8 (33-98) years. Twenty-one (39.6%) patients had external WI: 7 (33.3%) of them had large-artery atherosclerosis (LAA), 8 (38.1%) patients had cardioembolism, 3 (14.3%) patients had stroke due to other causes (vasculitis; n = 3), and etiologic subtype was undetermined in 3 patients (14.3%). Thirty-two (60.4%) patients had internal WI: 21 (65.6.%) of them had LAA, 5 (15.6%) patients had cardioembolism, 3 (9.4%) patients had stroke due to other causes (aneurysm; n = 1, hypercoagulability due to chronic myeloid leukemia; n = 1, vasculitis; n = 1), and etiologic subtype of 3 (9.4%) patients remained cryptogenic. LAA was significantly associated with internal WI (P = .024). Hypertension was more common in patients with internal WI (P = .035). *Conclusions:* In this series, cardioembolism was the most common etiologic subtype in the patients with external WI, whereas internal WI were significantly associated with LAA. Uncommon causes should also be investigated in cryptogenic patients. **Key Words:** Ischemic stroke—watershed infarct—etiology—prognosis.
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Border-zone or watershed infarcts (WI) are ischemic lesions located at the junction between 2 main arterial territories.[1] These lesions constitute approximately 10% of all brain infarcts.[2] Border-zone distribution of infarction has traditionally been attributed to hypoperfusion related to reduced blood flow in areas between major hemispheric vascular territories. Two types of WI are recognized. Internal (subcortical) WI usually appear as multiple small infarcts in a rosary-like pattern parallel to the lateral ventricle in the centrum semiovale or corona radiata.[3] The external (cortical) WI are usually wedge-shaped and located in the cerebral cortex between the territories of anterior, middle, and posterior cerebral arteries.[4,5]

Internal WI are believed to be caused mainly by hemodynamic compromise secondary to severe stenosis or occlusion of paroxysmal craniocervical arteries and/or hypoperfusion under the circumstance of cardiac arrest or systemic hypotension, whereas external WI are usually attributed to embolization from the heart or

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atherosclerotic plaques in large arteries. Severe stenosis or occlusion of major cerebral arteries was found to be substantially fewer in external WI.[6] In the patients with both external and internal WI, the most probable mechanism of stroke seems to be hemodynamic impairment.[3,6] Patients with external WI have a more benign clinical course and a better prognosis than those with internal border-zone infarcts.[4]

In previous studies, WI usually have been reported in stroke patients with carotid occlusive disease, with a cardioembolic source, or with severe cerebral hypoperfusion during carotid endarterectomy, systemic hypotension, cardiac arrest, or surgery.[2,6-14] However, several cases with acute WI due to idiopathic hypereosinophilic syndrome, parasitic infections (eg, schistosomiasis), toxicity due to cyclosporine treatment, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), and malignancy-related coagulopathy have also been reported previously.[15-23] However, etiologic subtypes and prognosis of WI have not been studied systematically in the patients with acute ischemic stroke.

The aim of this study was to determine the etiologic subtypes and prognosis of external and internal WI in our stroke registry.

Methods

The medical records of 488 consecutive patients who were admitted to the stroke unit with acute ischemic stroke from January 2012 to November 2014 were retrospectively evaluated. Magnetic resonance imaging scans of all patients were reviewed by 2 investigators (S.R. and V.Y.). Patients with external and internal WI were selected based on their lesion location on diffusion-weighted imaging (DWI). The subcortical infarcts that appear in a rosary-like pattern arranged in a linear fashion parallel to the centrum semiovale or corona radiata have been recorded as internal WI. The patients with a single infarct at the same location were not included in the study. External WI are defined as wedge-shaped or ovoid infarcts located between the anterior, middle, or posterior cerebral arteries. The patients were divided into 2 groups: group 1 included patients with external WI and group 2 included patients with internal WI (Fig 1). The patients with internal WI in association with external WI were also included into group 2 based on the previous studies suggesting a similar mechanism for both patient groups.

Patient demographics and medical risk factors, including history of hypertension, diabetes, hyperlipidemia, atrial fibrillation, congestive heart failure, coronary artery disease, previous transient ischemic attack, previous stroke, and the National Institutes of Health Stroke Scale (NIHSS) scores at admission, were collected using a standard data collection form and entered into an institutional database. Hypertension was defined as blood pressure 140/90 mm Hg or more on repeated measurements or prior use of antihypertensive medication, diabetes mellitus as fasting blood glucose level 126 mg/dL or more on repeated measurements or the use of medications to lower blood glucose, and atrial fibrillation by previous history or if detected on ECG or Holter. Coronary artery disease included any history of angina, myocardial infarction, or coronary revascularization.

A single rater (M.H.S.) determined etiologic stroke subtypes using the automated Causative Classification System (CCS, available at https://ccs.mgh.harvard.edu).[24] The CCS subtypes included supra-aortic large-artery atherosclerosis, cardioaortic embolism, small-artery occlusion, other causes, and undetermined causes. Etiologic workup included vascular imaging studies, such as carotid Doppler ultrasonography, computerized tomography angiography, magnetic resonance angiography, or digital subtraction angiography, transthoracic

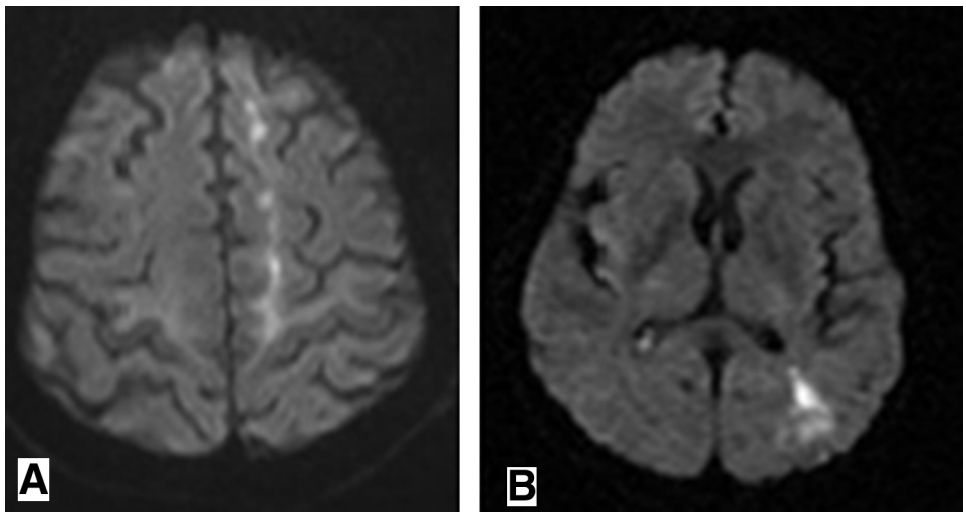

Figure 1. *Axial diffusion-weighted images show internal watershed infarct (A) and external watershed infarct (B).*

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