Internal Watershed Infarction as an Imaging and Clinical Challenge: a Case Report

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Abstract- We presented the case of a patient with internal watershed infarction with a nonspecific clinical presentation including hemiplegia, hemisensory deficit, and speech disturbance. Neuroimaging and ultrasound diagnostic procedure are important tools for diagnosis of these rare ischemic events that count for about 6% of all strokes. Specific therapy is mandatory for the diagnosis of watershed infarction and different from the therapeutical measures than can be taken for embolic and atherothrombotic strokes. Our patient was a 69-year-old, right-handed Caucasian woman who presented to our facility with acute right side weakness and speech disturbance. He had hypothyroidism, permanent atrial fibrillation, diabetes mellitus and she was hypotensive. She reported dizziness few days before the accident. Imaging studies revealed internal watershed infarction. Therapeutic procedures were taken to restore low cerebral blood flow. Internal watershed infarction is rare (less than 10% of all strokes) but well recognized a clinical feature of stroke. Specific pathophysiology generally is connected with hypoperfusion and hemodynamic mechanisms. Specific therapy is mandatory for these conditions.

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Introduction

Border zone infarcts involve the junction of the distal fields of two non-anastomosing arterial systems (1). Less than 10% of all ischemic strokes involve structures in a deep white matter that lie between medullary arteries arising from the superficial pial plexus and deep penetrating arteries arising from the basal cerebral arteries. This lesion lies in the corona radiate and centrum semiovale adjacent to the lateral ventricles and is called internal watershed strokes (2). Watershed infarcts or border zone infarcts can be divided into external or to embolism related strokes and internal or hypoperfusion/ disrupted hemodynamics related strokes (3). In the case of internal watershed ones, infarction lies in the deep white matter between the two different drainage areas (internal subcortical border zone lies at the junction of the anterior, middle and posterior cerebral artery territories with the Heubner, lenticulostriate, and anterior choroidal artery territories. Pathophysiology of the watershed infarction is still poorly understood but generally is connected to hypoperfusion in the distal region of vascular territories (4). Border zone infarction may be better explained by invoking a combination of two often interrelated processes: hypoperfusion and embolization. Hypoperfusion, or decreased blood flow, is likely to impede the clearance (washout) of emboli (5). Because perfusion is most likely to be impaired in border zone regions, clearance of emboli will be most impaired in these regions of least blood flow. Severe occlusive disease of the internal carotid artery causes both embolization and decreased perfusion. Similarly, cardiac disease is often associated with microembolization from the heart and aorta with periods of diminished systemic and brain perfusion (6). This theory, although it seems reasonable, remains unproven and has been challenged on many accounts. The typical clinical manifestations of syncope, hypotension, and episodic fluctuating or progressive weakness of the hands are also supportive of this theory of hemodynamic failure. Many cases in the past are remained undiagnosed or incorrectly diagnosed

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because of not so typical clinical presentation and the lower sensitivity of diagnostic imaging of the deep white matter (7). Novel methods of neuroimaging and functionally advanced techniques (e.g. diffusion and perfusion magnetic resonance imaging, multi-scan computed tomography, positron emission tomography, transcranial Doppler ultrasonography today brings more light to this dark area of cerebrovascular disease (8). Although lacunar infarctions can mimic watershed strokes, brain imaging usually resolves that problem. Leucoacariosis that have similar imaging features does not have a same clinical picture, so it is not difficult to differentiate these two entities. Specific therapy is aimed to restore disrupted cerebral perfusion (9).

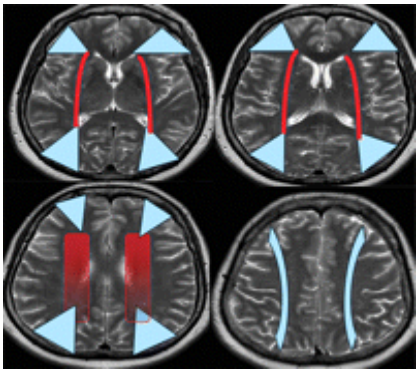

Figure 1. Color overlays on axial T2-weighted magnetic resonance (MR) images of normal cerebrum show probable locations of external (blue) and internal (red) border zone infarcts.

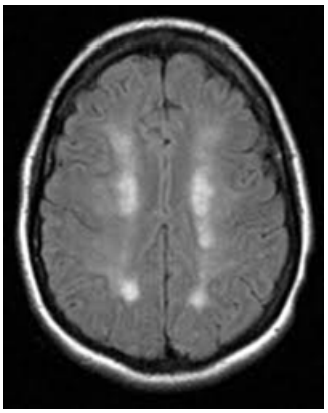

Figure 2. MRI brain, T1, transversal

Case Report

Our patient was a 69-year-old, right-handed Caucasian woman who presented to our facility with acute right-sided motor and sensory weakness with speech disturbance. She was eupneic and afebrile. Her blood pressure was 100/60mmHg on the time of admission to hospital and 105/56 mmHg on day 1. Her heart rhythm was irregular, with no murmurs or other pathological phenomena. Breathing sounds were normal. Distal artery pulses were normal, and there was no edema. Neurology examination showed a near complete right-sided weakness (arm 0/5, leg 1/5) with positive ipsilateral Babinski sign, right sided sensory deficit including all sensory modalities, right sided facial weakness and significant speech disturbance. She also had isochoric pupils with normal photoreaction. She was treated for hypothyroidism for more than 10 years, and she knew about sustained atrial fibrillation. Few years ago she was operated for deep venous thrombosis and after that she constantly take anticoagulant therapy (INR was in the therapeutic rang, e.g., 2.2mmol/l). Four months before the stroke, she was operated for pancreas carcinoma after she had post-operational diabetes mellitus; she had to take oral antidiabetic drugs. Few days before the stroke, she was complaining of dizziness and lightheadedness. ECG revealed sustained atrial fibrillation confirmed by 24 hour ECG recording as basic heart rhythm. Laboratory results were remarkable including an INR value of 2.3mmmol/l. CT scan was taken on day 1, about 6 hours after admission to hospital and showed hypodense lesion in deep white matter of left cerebral hemisphere that was described as lacunar by the radiologist on duty. Doppler ultrasound was taken on the day and revealed no hemodynamic significant stenosis in both internal carotid artery systems. On day 3, we took MR scan of the brain (University hospital Split, 1.5 T, Siemens) included T1, T2, FLAIR images and also perfusion and diffusion sequences. Only these images revealed true nature of stroke and showed subacute lesion in a deep white matter that was in the internal watershed region. In spite of all our efforts to restore cerebral blood pressure, she regained normal blood pressure on day 4 -122/84 mmHg) and sustained with normal blood pressure above values of 120/80 mmHg. Her neurological deficit was stable, and there was no improvement even after physical therapy started during day 4.

Discussion

Internal watershed infarction is a rare but serious type of stroke which can lead to permanent disability and sometimes to death (10). Pathophysiology is not fully understood, and this condition is often neglected in favor of embolic and atherothrombotic strokes (11). Because of different pathophysiology, therapy is a different and most important factor is arterial blood pressure (12). Systemic hypotension leads to low flow infarction in the

zones that lie between great vascular territories. In the case of internal watershed infarction that its territories lie between anterior, middle and posterior cerebral artery territories with the Heubner, lenticulostriate and anterior choroidal artery territories (13). Embolic events usually are correlated to atrial fibrillation, and atherothrombosis (14), mainly correlated to arterial hypertension; diabetes and atherosclerosis could be considered cofactor for this event, but essential role in this type of cerebrovascular disease is low flow in border vascular zone (15). To select the most appropriate methods for managing these infarcts, it is important to understand the underlying causal mechanisms (16). Internal border zone infarcts are caused mainly by hemodynamic compromise, whereas external border zone infarcts are believed to result from embolism but not always associated with hypoperfusion (17). Low perfusion can exist in patients with absolute arterial systemic hypotension or the ones with chronic arterial hypertension in whom drop of blood pressure even to normal values lead to impairment of cerebral perfusion because of disturbance of cerebral auto-regulation (18). Various imaging modalities have been used to determine the presence and extent of hemodynamic compromise or misery perfusion in association with border zone infarcts, and some findings (e.g., multiple small internal infarcts) have proved to be independent predictors of subsequent ischemic stroke (19). A combination of several advanced techniques (e.g.,, diffusion and perfusion magnetic resonance imaging and computed tomography, positron emission tomography, transcranial doppler ultrasonography) can be useful for identifying the pathophysiologic process, making an early clinical diagnosis, guiding management, and predicting the outcome (20,21). The essential therapeutic procedure is to restore low cerebral perfusion as soon as is possible so different strategies must be done immediately in opposite to embolic and atherothrombotic cerebral infarction (22).

Internal watershed infarction is the rare but serious type of stroke with a unique localization and pathophysiology. This particular case highlights the importance of clinical skills and imaging procedure to confirm the diagnosis and direct therapeutical measures to ones that can prevent disastrous disability. Internal (subcortical) border zone infarcts, which typically appear in a linear rosary like pattern in the centrum semiovale, are caused mainly by hemodynamic compromise. Sometimes, we can overlook these rare events by simple CT scan that is commonly used in emergency departments, so another advanced imaging techniques such as diffusion and perfusion MR imaging, PET, perfusion CT, and transcranial Doppler US must be performed to detect associated hemodynamic compromise, and guiding disease management. Different therapeutic approaches may be required to prevent early clinical deterioration in patients with different types of border zone infarcts.

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