Case Report

A Mismatch Between the Abnormalities in Diffusion- and Susceptibility-Weighted Magnetic Resonance Imaging May Represent an Acute Ischemic Penumbra with Misery Perfusion

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Susceptibility-weighted imaging (SWI) has recently attracted attention for its ability to investigate acute stroke pathophysiology. SWI detects an increased ratio of deoxyhemoglobin to oxyhemoglobin in cerebral venous compartments, which can illustrate cerebral misery perfusion with a compensatory increase of oxygen extraction fraction in the hypoperfused brain. In this study we make the first case report of blunt cervical trauma leading to a stroke, demonstrating the disparity between diffusion-weighted imaging (DWI) and SWI changes, or DWI-SWI mismatch, in the acute ischemic brain. The area of mismatch between a smaller DWI cytotoxic edema and a larger SWI misery perfusion in our patient matured into a complete infarction with time. The DWI-SWI mismatch may signify the presence of an ischemic penumbra, and provide information about viability of the brain tissue at risk of potential infarction if without early reperfusion. **Key Words:** Diffusion-weighted imaging—susceptibility-weighted imaging—ischemic-penumbra—misery-perfusion—brain—magnetic resonance imaging.

A mismatch in the ischemic brain tissue between a smaller diffusion-weighted (DW) magnetic resonance imaging (MRI) cytotoxic edema and a larger perfusion-weighted imaging (PWI) hypoperfusion is considered to represent an ischemic penumbra.[1] The brain tissue within the DW imaging (DWI)-PWI mismatch would develop into infarction over time if without adequate early reperfusion.

Susceptibility-weighted imaging (SWI) has recently attracted attention for its ability to investigate acute stroke pathophysiology, including a state of metabolic compensation with misery perfusion.[2,3] SWI exploits the magnetic susceptibility effects caused by local in-homogeneities of the magnetic field. The paramagnetic property of deoxyhemoglobin generates SWI hypo-intensity, depending on the intravascular blood oxygen level. In the hypoperfused brain tissue, the oxygen extraction fraction increases via the metabolic compensation mechanism to rescue the brain cells.[4] As a result, the ratio of deoxyhemoglobin to oxyhemoglobin increases in the venous compartments, which leads to a remarkable magnetic susceptibility difference of the veins from the surrounding ischemic brain tissue. This brain region at risk of infarction exhibits significant hypo-intense veins on SWI.[2,3]

We report the first neuroradiological illustrative case of blunt cervical trauma leading to internal carotid artery

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(ICA) occlusion. The MRI study of the patient in the acute stage demonstrated a cerebral ischemic change of DWI-SWI mismatch: a discrepancy between a smaller DWI cytotoxic edema and a larger SWI misery perfusion.

Case Report

A 54-year-old man was injured by a factory machine. A slow-moving round compressor crushed the man at his left neck and at the right parietal of his head. Immediately after, he was safely rescued. However, his colleagues found him to be in a state of confusion, and took him to a local hospital. The head and neck computed tomographic (CT) scans on arrival at the hospital showed no marked abnormality. He had no neurological deficits but the Glasgow Coma Scale was E4V4M6. The mild consciousness disturbance was considered a result of brain concussion. Eight hours after the left neck blunt trauma, a moderate right hemiparesis developed. He was brought to our hospital by ambulance. The head CT scans on admission showed no apparent abnormality indicating traumatic or ischemic lesions. However, CT angiography showed left cervical ICA occlusion (Fig 1A). Our neuroradiological intervention team performed digital subtraction angiography with an intention of subsequent intravascular recanalization procedure. The patient was diagnosed as having a cervical ICA dissection with thrombosis extending into the intracranial cavernous portion of the left ICA at 11 hours after the trauma (Fig 1B-D). Additional intravascular treatment was not recommended because of the risk of cerebrovascular hemorrhagic complications. DWI immediately after digital subtraction angiography showed a left laminar hyper-intensity, signifying an acute ischemic change of cytotoxic edema

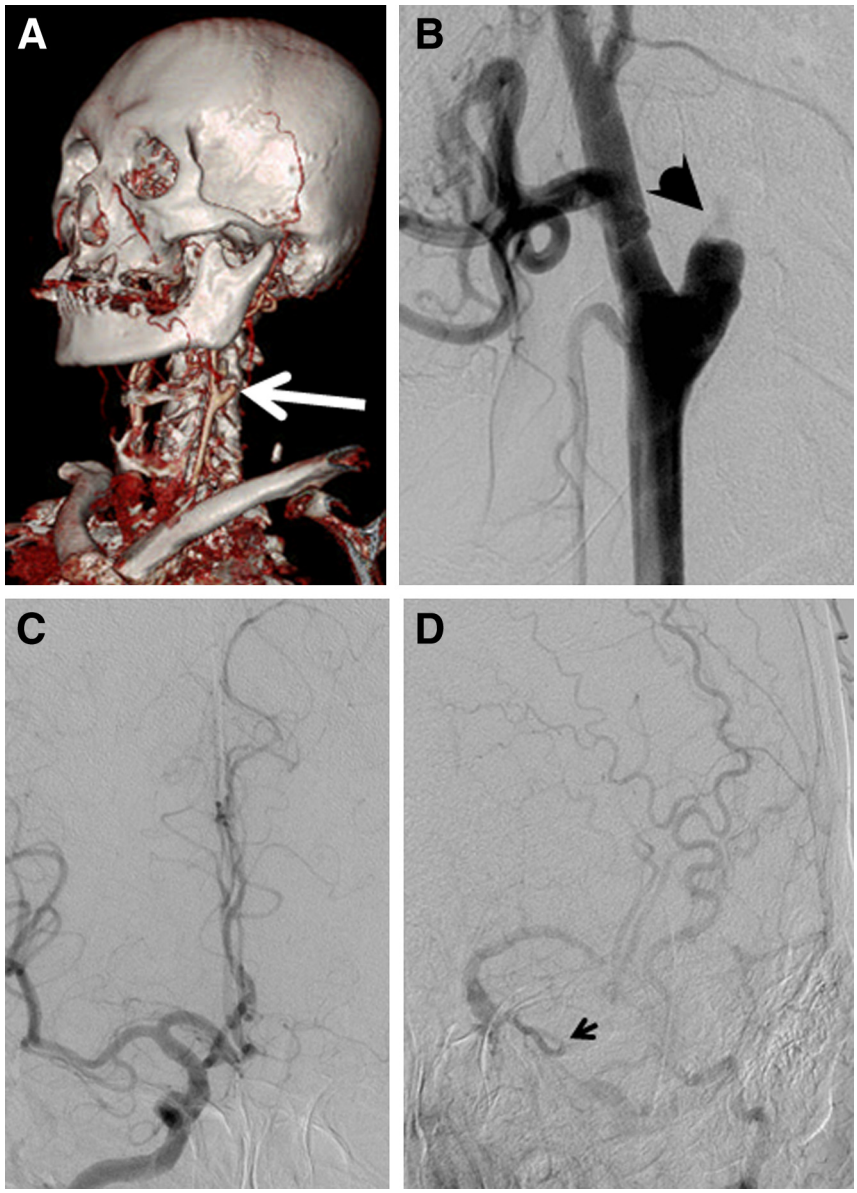

Figure 1. *Computed tomographic angiography on admission showed left cervical internal carotid artery (ICA) occlusion (large arrow)* (A). *Digital subtraction angiography on admission suggested presence of left cervical ICA wall dissection with retention of contrast medium in possible false lumen (arrowhead)* (B). *Right ICA supplies no apparent cross flow to left ICA circulation* (C). *Left ICA-middle cerebral artery system appears via ophthalmic arterial collateral flow (small arrow)* (D).