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Case Studies

A Case of Contrast Leakage Mimicking Intraventricular Hemorrhage in a Patient with Intravenous Thrombolysis

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> Contrast leakage on non-enhanced brain computed tomography (CT) is a common phenomenon after diagnostic or interventional cerebral angiography in patients with acute ischemic stroke. Contrast leakage is known to be related to the loosening of the blood-brain barrier. Sometimes, the contrast leakage on nonenhanced brain CT has been mistaken for intracranial hemorrhage. Differentiating the contrast leakage from the intracranial hemorrhage on non-enhanced brain CT is a very important issue, because subsequent treatment after intravenous thrombolysis (IVT) or intra-arterial thrombolysis would depend on the result of brain CT after thrombolysis. Recently, we experienced a case of contrast leakage mimicking intraventricular hemorrhage in a patient with IVT. The contrast leakage into the lateral ventricles after IVT is not a common phenomenon. We describe here our case. **Key Words:** Tissue plasminogen activator—contrast media—intracranial hemorrhage—magnetic resonance imaging.

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Background

Contrast leakage on non-enhanced brain computed tomography (CT) is a common phenomenon after diagnostic or interventional cerebral angiography in patients with acute ischemic stroke.¹ Contrast leakage is known to be related to the loosening of the blood–brain barrier. Sometimes, the contrast leakage on non-enhanced brain CT is mistaken for intracranial hemorrhage. Differentiating the contrast leakage from the intracranial hemorrhage on non-enhanced brain CT is a very important issue, because subsequent treatment after intravenous thrombolysis (IVT) or intraarterial thrombolysis would depend on the result of brain CT after thrombolysis. Recently, we experienced a case of contrast leakage mimicking intraventricular hemorrhage in a patient with IVT. The contrast leakage into the lateral ventricles after IVT is not a common phenomenon. To our knowledge, this is the first report of contrast leakage into the lateral ventricles after IVT. We are trying to interpret the unusual phenomenon through review of previous literatures.

Case

A 66-year-old man with hypertension presented with a sudden-onset left hemiparesis. Neurological examination revealed dysarthria, left-sided facial palsy, hemiplegia, and hemineglect. The patient's score in the National Institutes of Health Stroke Scale was 7. His blood pressure was 130/

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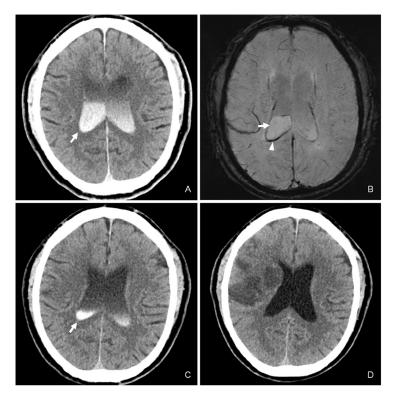


Figure 1. Immediate computed tomography (CT) after intravenous thrombolysis shows the fluid collection with hyperintensity (arrow) in the dependent portion of both lateral ventricles (A). The fluid collection (arrow) is not demonstrated as a dark signal, and a thin low signal (arrowhead) is observed along with the rim of the right lateral ventricle on susceptibility-weighted imaging (B). The hyperintensity (arrow) in lateral ventricles is rapidly decreased on the second followup CT (C). The final CT shows no hyperintensity in lateral ventricles (D).

80 mm Hg. Initial non-enhanced brain CT revealed no intracranial hemorrhage. CT angiography revealed M1 occlusion of the right middle cerebral artery. IVT was started 100 minutes after symptoms onset. Alteplase of 58.5 mg was infused intravenously over 1 hour according to standard regimen (0.9 mg/kg). His score in the National Institutes of Health Stroke Scale improved from 7 to 4 after IVT. Immediate non-enhanced CT after IVT showed fluid collection with hyperintensity in the dependent portion of both lateral ventricles (Fig 1, A), which had a mean Hounsfield unit (HU) of 66 (range from 41 HU to 84 HU). Although intraventricular hemorrhage was suspected based on the finding of the immediate non-enhanced CT, susceptibilityweighted imaging (SWI) confirmed that the intraventricular hyperintensity on the CT was not the intraventricular hemorrhage (Fig 1, B). A thin low signal was observed along with the dependent portion of the right lateral ventricle

on SWI (Fig 1, B). The second follow-up CT was performed 6 hours after the initial CT. The fluid collection with hyperintensity was rapidly decreased on the second follow-up CT (Fig 1, C). The third follow-up CT was performed 24 hours after the initial CT, which showed that the hyperintensity on the initial CT almost completely cleared (Fig 1, D). Immediate magnetic resonance angiography after IVT showed no recanalization of the right M1 occlusion (Fig 2, A). Follow-up magnetic resonance angiography 7 days after IVT showed partial recanalization of the right M1 occlusion (Fig 2, B). In the meantime, the patient's neurological status was kept stable.

Discussion

In this case, the unexpected hyperintensity in the lateral ventricles on brain CT turned out to be not an intraven-

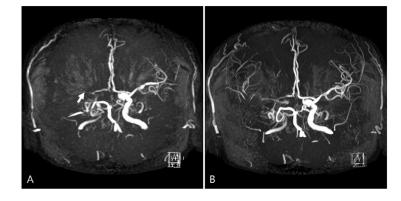


Figure 2. Immediate magnetic resonance (MR) angiography after intravenous thrombolysis shows no recanalization (arrow) of the right M1 occlusion (A). Seven days after intravenous thrombolysis, follow-up MR angiography shows partial recanalization of the right M1 occlusion (B).

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