# Gadolinium-Based Contrast Agent Extravasation Mimicking Subarachnoid Hemorrhage After Electroconvulsive Therapy

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## Key words

- Contrast extravasation
- Electroconvulsive therapy
- Magnetic resonance imaging
- Subarachnoid hemorrhage

#### Abbreviations and Acronyms

CT: Computed tomography ECT: Electroconvulsive therapy FLAIR: Fluid-attenuated inversion recovery MRI: Magnetic resonance imaging SAH: Subarachnoid hemorrhage

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# **INTRODUCTION**

Electroconvulsive therapy (ECT) is a safe method that has been applied for many years in medical treatment–resistant depression treatment. Intracranial bleeding is seen rarely in this procedure.<sup>1</sup> Subarachnoid hemorrhage (SAH) occurs in the subarachnoid space of the brain, usually of arterial origin and rarely of venous origin.<sup>2</sup> Here we report the case of a patient with extravasation of gadolinium-based contrast into the subarachnoid space mimicking SAH after ECT.

# **CASE PRESENTATION**

ECT was planned in a 70-year-old male patient who was followed up in our hospital due to medical treatment—resistant depression. Pre-ECT intravenous contrast-enhanced magnetic resonance BACKGROUND: Electroconvulsive therapy (ECT) is a safe method that has been applied for many years in medical treatment – resistant depression treatment. In this case report, contrast extravasation due to deterioration of the blood-brain barrier after ECT mimicking subarachnoid hemorrhage (SAH) on magnetic resonance imaging (MRI) is discussed.

CASE DESCRIPTION: A 70-year-old male patient who underwent ECT presented with sulcal hyperintensities suggesting subarachnoid hemorrhage on fluid-attenuated inversion recovery sequence of MRI obtained after ECT. However, there was no evidence to suggest SAH on nonenhanced brain computed tomography.

CONCLUSION: It should be kept in mind that patients may have contrast extravasation due to deterioration of the blood-brain barrier after ECT, and other alternative methods should be used for the diagnosis of SAH in these patients, not MRI.

imaging (MRI) was obtained to exclude organic disorders in the patient before 4 hours from ECT, and this MRI was evaluated as normal (Figure 1). One day after ECT was administered, the patient developed a headache. However, no features were found in the neurologic examination and laboratory findings of the patient. Noncontrast MRI was then performed to exclude a possible acute pathology in the patient after 23 hours ECT. On MRI, from sulcal hyperintensities were seen in the fluidattenuated inversion recovery (FLAIR) sequence, suggesting SAH (Figure 2). However, because of the lack of clinical evidence suggesting SAH in the patient, nonenhanced brain computed tomography (CT) was applied to support the diagnosis after 3 hours from second MRI. There was no evidence to suggest SAH on CT (Figure 3). To exclude SAH exactly, lumbar puncture was also performed. It was proved that there was no SAH on lumbar puncture. Hence the hyperintensities in FLAIR were thought to be due to the extravasation of gadolinium-based contrast into the subarachnoid space as a consequence of deterioration of the blood-brain barrier after ECT.

### **DISCUSSION**

In this case report, contrast extravasation due to ECT-induced deterioration of the blood-brain barrier after ECT mimicking SAH on MRI is discussed. Contrast extravasation in the brain has been previously reported on both CT3 and MRI.4,5 However, these studies were more focused on the contrast extravasation seen in intracerebral hemorrhage. A case report by Kothari et al<sup>6</sup> presented a patient with extravasation of gadoliniumbased contrast agent into the subdural space mimicking spinal subdural hematoma after lumbar puncture. To the best of our knowledge, there is no case report in English literature that presents MRI of gadolinium-based contrast extravasation into the subarachnoid space mimicking SAH after ECT.

The incidence of subarachnoid hemorrhage is 10-16/100,000, but this rate increases with age. Subarachnoid

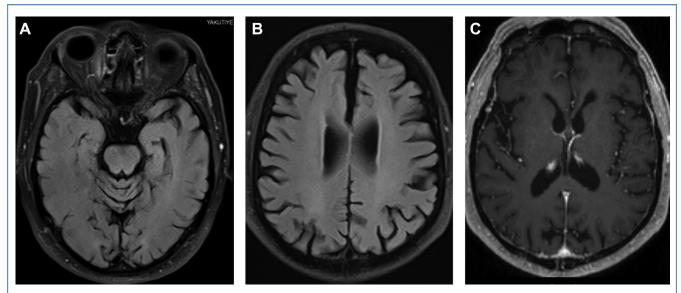


Figure 1. Fluid-attenuated inversion recovery (A and B) and postcontrast (C) magnetic resonance imaging obtained before electroconvulsive therapy

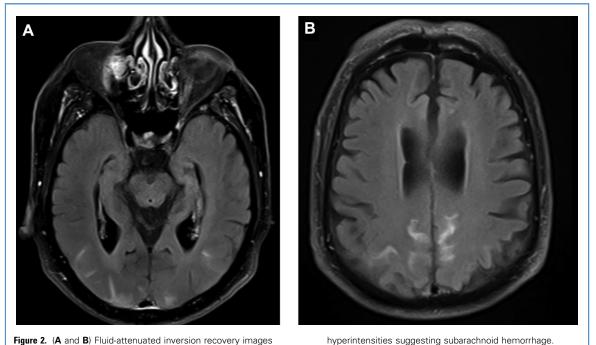
not showing any significant features.

hemorrhages may occur due to trauma, aneurysm, vascular malformations, bleeding disorders, or brain tumors, but 20% of cases have no known cause.<sup>7</sup>

Noncontrast head CT is quite successful in the diagnosis of SAH, although in

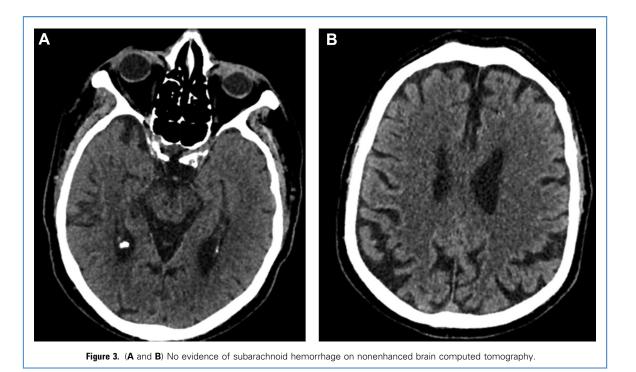
clinically suspected cases, lumbar puncture should be performed. In recent years, it has been reported that MRI (especially FLAIR, proton density, diffusion-weighted imaging, and gradient echo sequences) is successful in the diagnosis of SAH.8

The FLAIR magnetic resonance sequence is conventional imaging technique commonly used for brain imaging. This pulse sequence is used effectively in demonstration of lesions within the cerebrospinal fluid. Failed suppression of



hyperintensities suggesting subarachnoid hemorrhage.

obtained after electroconvulsive therapy showing sulcal



sulcal fluid on cerebral FLAIR imaging is called "hyperintense cerebrospinal fluid."<sup>9</sup> The FLAIR magnetic resonance sequence is a commonly used imaging method for the description of diseases affecting the cerebrospinal fluid such as subarachnoid hemorrhage, leptomeningitis, leptomeningeal metastasis, meningeal carcinomatosis, venous thrombosis, and postocclusive reactive hyperemia.<sup>9-11</sup>

On normal healthy individuals, CT or MRI evidence of the presence of the gadolinium within the cerebrospinal fluid has not been reported previously in the literature. However, it has been reported that there is gadolinium diffusion into ventricles in patients with chronic renal disease.4-12 Some cerebral diseases such as stroke, previous surgery, high-grade glioma, and meningioma can result in blood-brain barrier breakdown. Bozzao et al<sup>13</sup> reported that there is FLAIR magnetic resonance evidence of gadolinium leakage into the subarachnoid space 2-24 hours after intravenous injection of the contrast media. The investigators found that the patient group has sulcal increased intensity in FLAIR sequence, while the controls have no pathologic signal into the subarachnoid space.

Sulcal hyperintensity on FLAIR MRI is associated with frequently acute subarachnoid hemorrhage.<sup>9</sup> Our patient had no clinical findings of the subarachnoid hemorrhage. Because ECT causes breach in the blood-brain barrier, we thought that gadolinium leak into the subarachnoid space is responsible from sulcal high signals.

ECT is a safe treatment method. In a study involving 20,000 patients who underwent ECT procedure, mortality was present in 4 of them. One of these patients died of intracerebral hemorrhage.<sup>14</sup> There are only 2 cases in the literature with intracerebral hematoma after ECT.<sup>1,15</sup>

ECT leads to an increase in blood-brain barrier permeability. This is thought to be due to sudden fluctuations in blood pressure, an increase in cerebral blood flow, and increased vascular permeability during the procedure. Transient brain edema following ECT is also linked to this increase in permeability.<sup>16</sup>

In conclusion, it should be kept in mind that patients may have contrast extravasation due to deterioration of the bloodbrain barrier after ECT, and alternative methods should be used for the diagnosis of SAH in these patients, not MRI.

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**CASE REPORT** 

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