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CONTRAST-INDUCED NEUROTOXICITY FOLLOWING MECHANICAL THROMBECTOMY FOR ACUTE LEFT MCA OCCLUSION: A REVERSIBLE RADIOLOGICAL MIMIC OF INTRACRANIAL HAEMORRHAGE

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ABSTRACT

Introduction: Neurotoxicity due to contrast extravasation and disruption of the blood–brain barrier is a rare event in neuroendovascular procedures called contrast-induced neurotoxicity (CIN). Although a correct diagnosis may be difficult after mechanical thrombectomy, radiologically, CIN may be similar to haemorrhage or malignant oedema.

Case description: A 59-year-old male with a history of hypertension, paroxysmal atrial fibrillation and recent non-ST-elevation myocardial infarction presented with acute ischemic stroke secondary to distal left middle cerebral artery occlusion. There was successful reperfusion with mechanical thrombectomy. The immediate post-procedure computed tomography scan revealed hyper attenuation of the left frontotemporal gyral with oedema, but no evidence of haemorrhage, which matched with the diagnosis of CIN. Rapid healing of hyper attenuation and evolution to a stable infarct was observed on serial computed tomography scans obtained over 24-48 hours. The patient needed prolonged ventilatory support, which was provided by a tracheostomy and there was no haemorrhagic transformation.

Conclusion: This case serves as an example of a benign radiological mimic after thrombectomy, namely CIN. Understanding its imaging appearances and the need for serial CT imaging helps to prevent misdiagnosis and unnecessary discontinuation if anti-thrombotic treatment. There is no evidence of self-limitation and there is support for management of CIN.

KEYWORDS

Contrast-induced neurotoxicity, mechanical thrombectomy, gyral hyper attenuation, cerebral oedema, stroke imaging

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LEARNING POINTS

- Mechanical thrombectomy can also cause rare and reversible side effects of contrast-induced neurotoxicity (CIN) that can mimic immediately post-procedure computed tomography (CT) scan findings of intracranial bleeding.
- Non-contrast CT imaging is crucial to rule out malignant cerebral oedema or haemorrhagic transformation of the stroke lesion, especially in the case of serial imaging, to avoid unnecessary interventions.
- In patients with cardioembolic stroke and high-risk, antithrombotic therapy can be continued at the time of recognition of CIN.

INTRODUCTION

For acute ischemic stroke due to large-vessel occlusion, mechanical thrombectomy has been a dramatic change in care with significant improvement in functional outcome over medical therapy alone[1]. The use of endovascular stroke therapy is becoming more widespread and the complications of the procedure, including iodinated contrast, have come to light.

Contrast-induced neurotoxicity (CIN) or contrast-induced encephalopathy is a rare but significant event, with a typical radiological appearance and transient neurological deterioration. CIN is thought to be caused by disruption in the blood–brain barrier from ischemia–reperfusion injury and endothelial trauma, as well as from the osmotic effect of the contrast media[2]. In clinical practice, CIN can present with confusion, seizures, focal deficits or coma. Immediately after procedure, non-contrast computed tomography (CT) imaging may reveal hyper attenuation of the gyri and cerebral oedema and can closely mimic subarachnoid haemorrhage, intraparenchymal bleeding, or malignant oedema[3].

It is important to differentiate between the two types of bleeding, to avoid inappropriate surgical treatment of the brain or inappropriate withholding of antithrombotic medications in those who are at risk for cardiac embolism. Differentiation is most easily made by serial non-contrast CT scan: although the CIN can resolve within 24-48 hours, haemorrhage may remain or progress[4].

This case highlights the typical radiological progression of CIN after mechanical thrombectomy and the need for clinical-radiological correlation in acute stroke.

CASE DESCRIPTION

The patient was a 59-year-old Iranian male, retired, with hypertension, paroxysmal atrial fibrillation and recent non-ST-elevation myocardial infarction, who presented with sudden onset neurological deficits consistent with a left middle cerebral artery (MCA) territory stroke. He used to be independent, never smoked and reported never using alcohol or drugs.

At arrival his blood pressure was 152/89 mmHg. Non-contrast CT scan showed evidence of acute ischemic changes with no evidence of haemorrhage *(Fig. 1)*.

On October 15th, 2025, the patient had CT angiography which showed occlusion of the left distal middle cerebral artery (MCA), and the patient was taken for urgent mechanical thrombectomy. Reperfusion was successful.

Non-contrast CT scan at the time showed hyper attenuation of the gyri and an area of cerebral oedema in the left frontotemporal cortex, but no haemorrhage or midline shift *(Fig. 2)*. These findings were considered to

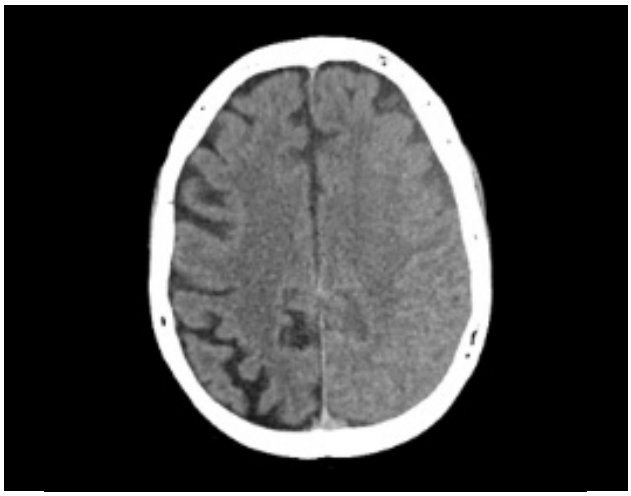


Figure 1. *Baseline CT scan: Pre-thrombectomy non-contrast CT brain demonstrating acute ischemic changes without the evidence of intracranial hemorrhage or cortical hyper attenuation.*

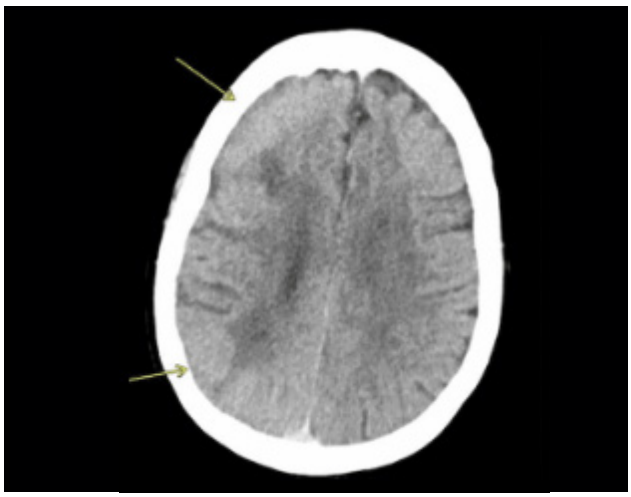


Figure 2. *Post-thrombectomy CT scan: Immediate post-procedural CT scan showing right hemispheric cortical/gyral hyper attenuation (arrows), consistent with contrast extravasation and blood-brain barrier disruption in contrast-induced neurotoxicity.*

be compatible with CIN. A 24-hour follow-up CT scan showed significant reduction in hyper attenuation and evolving infarction in the left parietotemporal region without haemorrhage or hydrocephalus *(Fig. 3)*. The resolution of hyper attenuation was nearly complete on a 48-hour CT scan *(Fig. 4)*, showing the transient nature of CIN.

The patient had neurologic impairment and aspiration was a concern. Prolonged mechanical ventilation was required. Tracheostomy was performed October 20th, 2025. Chest X-rays revealed mild cardiomegaly and slight pleural effusion.

Considering the presence of atrial fibrillation and recent myocardial infarction, antiplatelet and anticoagulant agents were started for secondary prevention. Osmotic therapy and/or decompressive surgery were not indicated as there was no evidence of malignant oedema on the imaging.

As of October 24th, 2025, the patient continued to rely on a tracheostomy and was in the intensive care unit. There was no haemorrhagic transformation of the infarct.

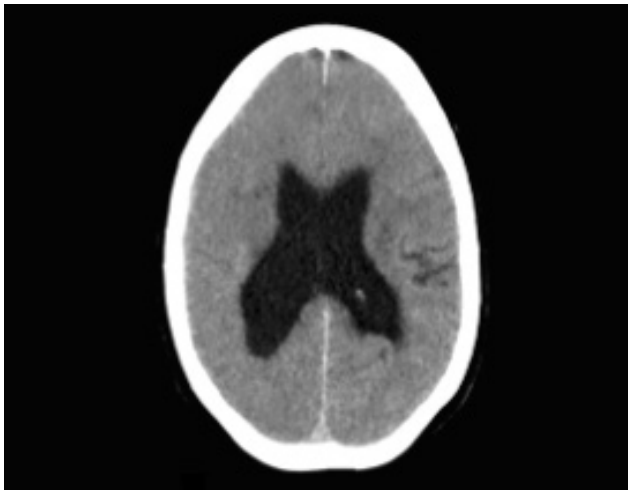


Figure 3. *Follow -up CT scan: Repeat CT scan after 24 hours showing interval reduction in cortical hyper attenuation and enhancing cerebral edema.*

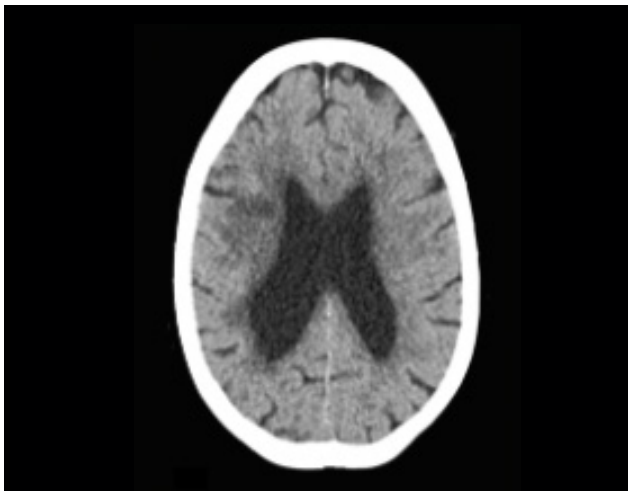


Figure 4. *Follow-up CT scan: Follow-up CT scan after 48 hours showing near-complete resolution of prior cortical hyper attenuation, supporting the diagnosis of transient contrast-induced neurotoxicity.*

DISCUSSION

Neuroendovascular procedures, especially mechanical thrombectomy for an acute ischemic stroke (AIS), are associated with a rare but emerging complication termed CIN. Although uncommon, its incidence is reported to be between 0.3 and 4.7%, it is a diagnostic challenge due to a close radiological resemblance with intracranial haemorrhage or malignant cerebral oedema[1,2]. CIN is thought to be from transient disruption of the blood–brain barrier due to ischemia–reperfusion injury, endothelial trauma from any device manipulation and the osmotic properties of iodinated contrast agents[3]. The mechanisms permit extravasation of contrast into the cortex and subarachnoid space where it has direct neurotoxic and osmotic effects. Risk factors include several patient-related issues (e.g., hypertension, diabetes, chronic kidney disease, prior cerebrovascular disease) and procedural issues (e.g., high contrast dose, long intervention, multiple passes with thrombectomy). The patient's long-term hypertension and endothelial stress from reperfusion were factors that may have contributed to CIN in this case.

Radiologically, CIN usually presents as gyral/ cortical hyper attenuation on immediate post procedure non contrast CT scan with sulcal effacement and cerebral oedema. These results can be similar to subarachnoid haemorrhage, contrast staining and/or haemorrhagic transformation of an infarct[5]. The distinguishing characteristic is, however, the evolution of the images over time. A true haemorrhage will last for several days to weeks and can progress to hematoma formation or mass effect[6]. CIN will resolve or significantly improve in 24-48 hours when the contrast is excreted from the brain parenchyma.

In this patient, the imaging findings were typical for CIN, with marked hyper attenuation immediately post thrombectomy followed by significant attenuation at 24 and near-complete resolution at 48 hours with eventual consolidation to a stable infarct without haemorrhagic transformation. This radiological course is similar to published series which demonstrated that the majority of regions of contrast extravasation evolve into infarction instead of hematoma and are not clinically significant[7].

The clinical implications of identification of CIN versus haemorrhage are significant. Inappropriate avoidance of antithrombotic therapy due to misinterpretation of transient contrast-related hyper attenuation as bleeding is especially problematic in patients with high cardioembolic risk (e.g., atrial fibrillation and recent myocardial infarction)[3,5]. For this patient, recognition of the CIN was correct and secondary stroke prevention was initiated in time, reducing the danger of the patient being unnecessarily at risk of stroke during drug therapy. Radiologically, CIN is reversible, but there is a wide range of clinical outcomes associated with CIN. Transient neurologic worsening, seizures, or encephalopathy occur in some patients, while a subset of patients requires mechanical ventilation. However, the poor outcomes are frequently due to the

severity of the underlying stroke and not due to the CIN itself[8]. This patient's prolonged ventilatory dependence and requirement for a tracheostomy is a reflection of the severity of the large MCA infarct, not the transient contrast related changes.

The significance of clinical context and serial neuroimaging is highlighted in this case, to prevent misdiagnosis and unwarranted interventions[9,10]. Advanced imaging, like dual-energy CT, is capable of distinguishing iodine from blood with high accuracy, but is not always available, so repeated non-contrast CT scans are the most practical and widely available imaging test. The self-limiting and benign nature of CIN as seen here emphasizes the importance of increased awareness among clinicians involved with acute stroke care. With the increasing use of mechanical thrombectomy, awareness of CIN and its typical imaging timeline will be critical to avoid misdiagnosis, for an appropriate approach to treatment, and for the best possible results.

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