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Acute Reversible Posterior Encephalopathy Triggered by Sheath Removal Pain After Coronary Angiography: A Case Report

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Case Report

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Abstract

Background When acute neurological symptoms appear after coronary angiography, contrast-induced encephalopathy (CIE) and posterior reversible encephalopathy syndrome (PRES) are key differential diagnoses. Yet the role of acute peri-procedural pain as a potential trigger has received little attention.

Case presentation A 77-year-old man with hypertension and stage 3 chronic kidney disease developed severe pain during radial sheath removal three hours after coronary angiography. Within seconds, he lost consciousness and had generalized tonic-clonic seizures. Blood pressure rose from 136/70 mmHg to 160/90 mmHg. CT perfusion imaging showed hypoperfusion in the left temporal and bilateral occipital lobes, with relatively preserved cerebral blood volume and cerebral blood flow, and prolonged mean transit time and time-to-peak. A second seizure occurred later that evening, without a clear pain trigger, suggesting a persistent process with a tendency for spontaneous recurrence. The patient recovered completely within four days with conservative management and remained symptom-free at six-month follow-up. CIE and PRES could not be definitively diagnosed because MRI and EEG were not performed.

Conclusions This case suggests that acute peri-procedural pain may trigger acute reversible posterior encephalopathy. The temporal association—measured in seconds—between pain and neurological symptom onset is exceptionally rare. Clinicians should recognize the potential value of peri-procedural pain management in preventing such complications.

Introduction

Acute ischemic stroke and intracerebral hemorrhage are the first considerations when acute neurological symptoms appear after coronary angiography. Once these emergent and potentially fatal entities have been ruled out, contrast-induced encephalopathy (CIE) and posterior reversible encephalopathy syndrome (PRES) become important differential diagnoses. Both conditions can present with seizures, altered consciousness, and posterior circulation perfusion abnormalities. Iodinated contrast media themselves can also induce PRES. Their clinical and radiological features overlap considerably, making differentiation particularly challenging [1]. Diagnosis of both conditions relies on MRI and EEG [2, 3], which are frequently unobtainable in acute settings because of patient agitation or the need for continuous monitoring.

In this case, however, a more striking clinical feature emerged: neurological symptoms appeared within seconds of severe pain during sheath removal. This temporal association—measured in seconds—suggests that acute pain stress itself may have served as the trigger, rather than a delayed toxic effect of the contrast agent. To date, peri-procedural pain as a trigger for acute reversible posterior encephalopathy remains poorly recognized. We report a case of acute reversible posterior encephalopathy that developed within seconds of severe pain during sheath removal after coronary angiography, focusing on the potential mechanisms by which pain may act as a trigger and the clinical implications of this observation.

Case Presentation

Chief Complaint and Medical History

A 77-year-old man was admitted with a one-month history of recurrent chest tightness. The symptoms were provoked by exertion and relieved by rest or sublingual nitroglycerin, and were accompanied by shortness of breath and palpitations. Occasional anterior chest pain was noted, without radiation. His medical history included hypertension of more than eight years' duration (peak 150/90 mmHg), a 60-pack-year smoking history, chronic obstructive pulmonary disease, and stage 3 chronic kidney disease (eGFR 54.9 mL/min/1.73m²). He had not been taking non-steroidal anti-inflammatory drugs, ACE inhibitors, or angiotensin receptor blockers before admission.

Admission Evaluation

Admission electrocardiogram showed sinus rhythm without significant abnormalities. Echocardiography revealed mild mitral and tricuspid regurgitation. Chest CT showed changes consistent with chronic bronchitis and emphysema. Laboratory findings were as follows: white blood cell count 4.95 × 10⁹/L, hemoglobin 128 g/L, platelets 202 × 10⁹/L; serum creatinine 118.5 μmol/L, eGFR 54.9 mL/min/1.73m²; potassium 4.17 mmol/L, sodium 144.4 mmol/L, chloride 106.8 mmol/L; high-sensitivity troponin I <1.50 pg/mL; NT-proBNP 514.00 pg/mL. Coagulation profile and infectious disease screening were unremarkable. Given the recurrent chest tightness in the setting of advanced age, long-standing hypertension, and heavy smoking, with poor response to outpatient therapy, unstable angina was suspected, and diagnostic coronary angiography was scheduled.

Coronary Angiography and Peri-Procedural Management

No pre-procedural head CT was performed. On day 3 of hospitalization, coronary angiography was performed via the right radial artery. Given the stage 3 chronic kidney disease, intravenous hydration with normal saline at 1 mL/kg/h was initiated 12 hours before the procedure and continued for 12 hours afterward. During the procedure, approximately 30 mL of the low-osmolar non-ionic contrast agent iohexol (350 mg I/mL) was administered. Angiography revealed no significant coronary stenosis.

Neurological Event

Three hours after angiography (at 13:35), during radial sheath removal, the patient experienced severe pain at the puncture site and, within seconds, lost consciousness and developed generalized tonic-clonic seizures lasting approximately two to three minutes. Cardiac monitoring revealed blood pressure elevation from a baseline of 136/70 mmHg to 160/90 mmHg, with heart rate increasing from 60 to 70–110 beats/min. Neurological examination revealed unconsciousness, rightward eye deviation, horizontal nystagmus, sluggish pupillary light reflexes, and increased muscle tone. The Glasgow Coma Scale (GCS)

score was 3 (E1V1M1, during the seizure phase), and the National Institutes of Health Stroke Scale (NIHSS) score was estimated at 22.

Imaging Studies

Emergency non-contrast head CT and CT perfusion (CTP) were performed. An additional 40 mL of iohexol was administered to complete the CTP study. CTP revealed hypoperfusion in the left temporal and bilateral occipital lobes, with normal or mildly decreased cerebral blood volume and cerebral blood flow, and prolonged mean transit time and time-to-peak (Tmax > 6 s in the left temporal lobe) (Figures 1 and 2). Non-contrast head CT showed no hemorrhage or definite infarct. MRI could not be performed because the patient was in a post-ictal state with agitation and required continuous monitoring; after clinical recovery, the patient declined further imaging.

Follow-Up Laboratory Studies

Random blood glucose was 9.2 mmol/L; potassium 3.80 mmol/L, sodium 142.8 mmol/L, chloride 106.4 mmol/L; creatinine 116.9 μmol/L, eGFR 54.9 mL/min/1.73m² (consistent with baseline); white blood cell count 6.69 × 10⁹/L, hemoglobin 117 g/L, platelets 191 × 10⁹/L; high-sensitivity C-reactive protein <0.50 mg/L; high-sensitivity troponin I <1.50 pg/mL. No metabolic emergencies requiring urgent intervention were identified.

Treatment and Outcome

At approximately 14:00 (about 25 minutes after onset), the patient's consciousness partially recovered, and GCS improved to approximately 11 (E3V3M5), with NIHSS decreasing to approximately 8. Paroxysmal nausea and vomiting developed and were treated with metoclopramide 10 mg intramuscularly. At 20:50 that evening, the patient experienced a second episode of loss of consciousness with upward gaze deviation and incontinence; monitoring revealed a blood pressure of 151/90 mmHg and a heart rate of 70–110 beats/min. At 22:00, emergency repeat non-contrast head CT showed no new infarction or hemorrhage in the previously hypoperfused regions. The patient received continuous intravenous hydration and supportive care, without corticosteroids, mannitol, antiepileptic drugs, or antihypertensive agents. Neurological symptoms resolved completely by post-procedure day 4. At regular follow-up, the most recent assessment (6 months post-procedure) revealed no neurological abnormalities.

Discussion

Pain as a Trigger: The Core Observation

The most striking clinical feature of this case is the onset of neurological symptoms within seconds of severe pain during sheath removal. This temporal association—measured in seconds—suggests that acute pain stress may have acted as the trigger, rather than a delayed toxic effect of the contrast agent. Peri-procedural pain as a trigger for acute reversible posterior encephalopathy has rarely been reported.

Pain stress may trigger posterior encephalopathy through the following mechanisms:

(1) Sympathetic activation and blood pressure fluctuation. Acute pain can activate the sympathoadrenal axis, leading to catecholamine release and abrupt blood pressure elevation. The posterior circulation (vertebrobasilar system) has relatively sparse sympathetic innervation and is particularly vulnerable to hyperperfusion injury from blood pressure fluctuations, which explains the anatomical basis for the posterior predilection of PRES [2]. In the context of impaired cerebrovascular autoregulation due to chronic kidney disease and long-standing hypertension, acute blood pressure fluctuations may exceed the autoregulatory threshold, leading to hyperperfusion and blood-brain barrier disruption in the posterior circulation.

(2) Endothelial dysfunction. Chronic kidney disease itself can lead to endothelial dysfunction and increased blood-brain barrier permeability. Uremic toxins such as indoxyl sulfate are endothelial-toxic and can damage brain endothelial cells; in CKD mouse models, increased blood-brain barrier permeability has been associated with memory impairment and aryl hydrocarbon receptor activation [4]. This renders CKD patients less tolerant of blood pressure fluctuations, such that blood-brain barrier disruption may occur even under milder stress.

(3) Additive effects of peri-procedural stress. Surgical procedures can lead to significant elevation of endothelial glycocalyx shedding markers (Syndecan-1), endothelial injury markers (sTM), and inflammatory cytokines (IL-6), and the degree of elevation correlates with postoperative complication rates [5]. Although coronary angiography is a minimally invasive procedure, the peri-procedural stress response can still cause endothelial dysfunction, and pain stress may further exacerbate this process.

Notably, reports of peri-procedural PRES are increasing. PRES can occur after various surgical procedures, and its triggering mechanisms are mostly related to peri-procedural blood pressure fluctuations and endothelial dysfunction [6]. The unique aspect of this case is that the trigger was not the surgical procedure itself, but acute pain stress from a postoperative nursing procedure (sheath removal), with symptom onset measured in seconds—markedly different from the several days typically reported for postoperative PRES.

Differential Diagnosis

Acute ischemic stroke or transient ischemic attack. Given the patient's advanced age and multiple vascular risk factors, this required priority exclusion. However, symptoms began to improve within approximately 25 minutes without persistent focal neurological deficits, which is inconsistent with the typical evolution of acute cerebral infarction. Although CTP showed perfusion delay, repeat CT revealed

no new infarction, supporting the reversibility of the perfusion abnormality. PRES itself is an important stroke mimic, and its clinical and radiological features can overlap substantially with acute ischemic stroke, potentially leading to misdiagnosis and unnecessary thrombolysis [7].

Cerebral microembolism. Embolic events typically present with focal deficits in a single vascular territory. Computational modeling studies have shown that medium-sized emboli preferentially distribute to the posterior cerebral artery and posterior middle cerebral artery territories, whereas small emboli distribute uniformly throughout the cerebral circulation [8]. The bilateral occipital and left temporal hypoperfusion observed in this case is inconsistent with the typical distribution of a single embolus and cannot explain the complete recovery over several days.

Metabolic encephalopathy. Although renal insufficiency predisposes to metabolic encephalopathy, blood glucose, electrolytes, and eGFR showed no significant abnormalities after the episode, and the mildly elevated ammonia level (51.0 μmol/L) was far below the threshold typically required to cause consciousness disturbance [9]. The diagnosis of metabolic encephalopathy requires exclusion of structural causes, and its imaging findings are usually nonspecific.

Post-ictal state. Post-ictal states typically last minutes to hours and present with confusion, lethargy, and headache. While this could explain the initial consciousness disturbance, it cannot explain the persistent perfusion abnormalities on CTP or the recovery course lasting several days. In patients with PRES, consciousness disturbance may be caused by non-convulsive status epilepticus, which requires EEG for differentiation [2].

Interpretation of the Second Episode

Approximately seven hours after the first episode, the patient experienced a second seizure without a clear pain trigger. The most reasonable interpretation is that this represented continuation of the same acute reversible posterior encephalopathy process, rather than a new, independent event requiring a separate explanation.

Regardless of whether the case is ultimately classified as PRES or CIE, the shared pathological substrate is vasogenic edema in the posterior circulation, blood-brain barrier disruption, and increased neuronal excitability. Once initiated, this pathological process does not resolve immediately after the first seizure. Specifically:

Cerebral edema and blood-brain barrier disruption persist: After the first episode, the posterior perfusion abnormalities on CTP did not immediately normalize, and the repeat CT that evening could only exclude new infarction and hemorrhage—it could not confirm resolution of cerebral edema. Until edema resolves, cortical excitability remains elevated.

Seizure susceptibility remains elevated: Seizures in PRES and CIE are often not single events but recur within the first 24–48 hours. Literature reports that approximately 60%–70% of patients with PRES experience seizures, and a subset present with recurrent seizures [10].

Blood pressure remained elevated during the second episode (151/90 mmHg). Although the magnitude was modest, in the setting of impaired cerebrovascular autoregulation, it may have constituted a new minor fluctuation sufficient to provoke a seizure in an already hyperexcitable cortex.

The absence of a pain trigger is not contradictory: The first episode was "pain → abrupt blood pressure elevation → seizure," whereas the second was "persistent edema → cortical hyperexcitability → spontaneous seizure." The triggers may differ, but the underlying disease process is the same.

Therefore, the second episode should not be interpreted as evidence weakening the pain-trigger hypothesis, but rather as an indication that the pathological process is persistent and prone to spontaneous recurrence. This also serves as a reminder that such patients require continuous monitoring, as seizures may recur even without a new trigger.

Diagnostic Uncertainty

Because MRI and EEG were not performed, CIE and PRES could not be definitively diagnosed. The diagnostic cornerstone of PRES is vasogenic edema in the bilateral parieto-occipital subcortical white matter on MRI [2]; the diagnosis of CIE likewise depends on MRI to exclude other etiologies [3]. The absence of these studies precluded definitive diagnosis of either condition. We therefore adopted a descriptive diagnosis: acute reversible posterior encephalopathy following sheath removal pain after coronary angiography, of undetermined etiology.

Importantly, this diagnostic uncertainty does not diminish the clinical value of the core observation—the temporal association, measured in seconds, between pain and neurological symptoms. This observation is independent of the final diagnostic label and provides an important warning signal regarding peri-procedural pain management.

Clinical Implications

This case has the following implications for clinical practice.

First, peri-procedural pain management may have neuroprotective significance. Pain stress may trigger cerebrovascular autoregulatory failure through blood pressure fluctuations. For patients with high-risk features such as chronic kidney disease and hypertension, adequate analgesia before sheath removal (e.g., local anesthesia or oral analgesics) may help mitigate pain-induced blood pressure fluctuations.

Second, vigilance for recurrent seizures is warranted. The second episode in this case suggests that cortical excitability remains elevated during the acute phase and that seizures may occur spontaneously even without a new trigger. Clinicians should maintain continuous monitoring in such patients rather than relaxing vigilance after the first seizure is controlled.

Third, CIE and PRES should be considered first-line differential diagnoses. When acute neurological symptoms occur after coronary angiography, clinicians should not consider only CIE, nor should they abandon diagnostic reasoning because MRI is unavailable. When the trigger is clear, the clinical presentation is typical, CTP shows reversible perfusion abnormalities, and the clinical course is one of complete recovery, a descriptive diagnosis of "acute reversible posterior encephalopathy" is appropriate.

Limitations

The primary limitation of this report is the lack of MRI (particularly DWI, FLAIR, and SWI sequences) and EEG, which precluded definitive diagnosis of CIE or PRES and prevented complete exclusion of acute ischemic stroke, microembolism, or non-convulsive status epilepticus. Acute-phase MRI could not be performed because the patient was in a post-ictal state with unstable vital signs, making safe transport impossible; after clinical recovery, the patient declined further imaging. Repeat CT showing no new infarction or hemorrhage in the previously hypoperfused regions partially compensated for the lack of MRI. PRES is not always completely reversible, and some patients are at risk of long-term seizures, warranting long-term follow-up [10].

Conclusions

We report a case of acute reversible posterior encephalopathy that developed within seconds of severe pain during sheath removal after coronary angiography. The core value of this case lies in the exceptionally rare temporal association—measured in seconds—between pain and neurological symptom onset, suggesting that acute pain stress may serve as a trigger. The second episode suggests that the pathological process is persistent and prone to spontaneous recurrence. Although CIE and PRES could not be definitively diagnosed because MRI and EEG were not performed, this diagnostic uncertainty does not diminish the clinical significance of the core observation regarding pain as a trigger. Clinicians should recognize the potential value of peri-procedural pain management in preventing such complications.

Abbreviations

BBB: Blood-brain barrier; CIE: Contrast-induced encephalopathy; CKD: Chronic kidney disease; CTP: Computed tomography perfusion; eGFR: Estimated glomerular filtration rate; GCS: Glasgow Coma Scale; NIHSS: National Institutes of Health Stroke Scale; PRES: Posterior reversible encephalopathy syndrome; TIA: Transient ischemic attack; Tmax: Time to maximum

Declarations

Ethics approval and consent to participate

This study was approved by the Medical Ethics Committee of Suzhou Hospital of Traditional Chinese Medicine (approval number: 2024□□□032) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for participation in this case report.

Consent for publication

Written informed consent for publication of the clinical details was obtained from the patient. The consent form is available for review by the Editor of this journal.

Availability of data and materials

All data supporting the findings of this case report are contained within the manuscript.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

X.Z. is the first author of this manuscript. X.Z. and J.Y. diagnosed and treated the patient. X.Z. and W.Z. collected and analyzed the data, performed the literature search, and drafted the manuscript. J.Y. conceived the study, critically revised the manuscript, and provided final approval. All authors read and approved the final manuscript.

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Figures

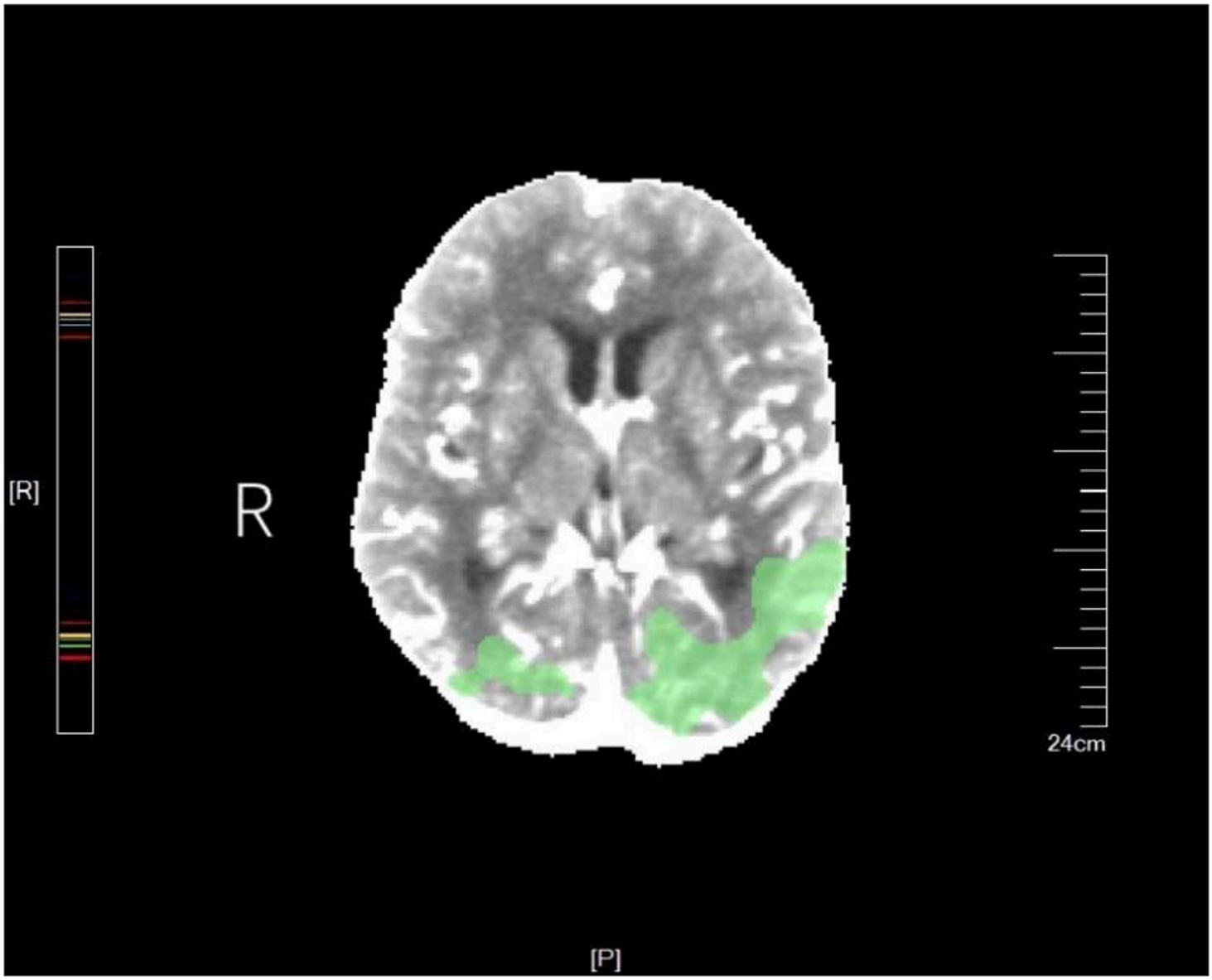


Figure 1

Computed tomography perfusion (CTP) imaging showing hypoperfusion in the left temporal and bilateral occipital lobes, with normal or mildly decreased cerebral blood volume and cerebral blood flow.

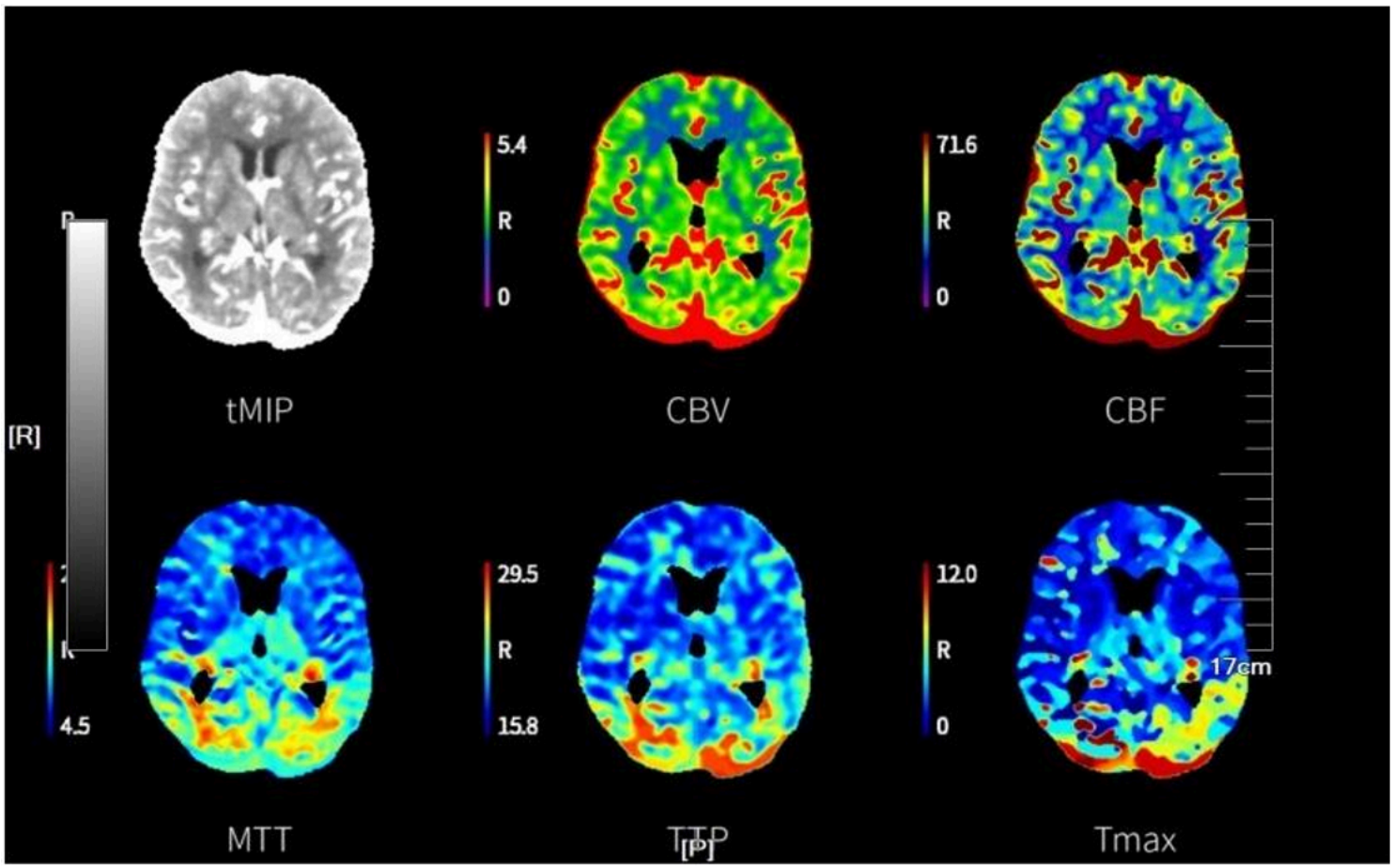


Figure 2

Computed tomography perfusion (CTP) imaging showing prolonged mean transit time and time-to-peak (Tmax > 6 s) in the corresponding regions, indicating perfusion delay without evidence of irreversible infarction.