PRACTICAL PEARL



# Dual-Energy CT to Diagnose Pseudoedema in Contrast-Induced Encephalopathy Following Cerebral Angiography

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#### Abstract

*Background* Contrast-induced encephalopathy (CIE) is a syndrome that may be clinically unrecognized and misdiagnosed as cerebral edema.

Methods Case report and review.

Results A 72-year-old woman was admitted for elective endovascular embolization of a 10-mm left anterior communicating artery aneurysm. One hour post-procedure, she acutely developed global aphasia. Emergent head computed tomography (CT) and computed tomographyangiography (CTA) showed high attenuation of the left hemispheric subarachnoid spaces interpreted as hemispheric edema; emergent magnetic resonance imaging revealed left hemispheric punctate infarcts. At 12 h, she developed right hemiparesis and encephalopathy. Repeat CTA and CT perfusion revealed decreased left hemisphere cerebral blood flow and diminutive caliber of distal left middle cerebral artery territory vasculature. Repeated angiography with intra-arterial verapamil and systemic blood pressure augmentation were performed for presumed vasospasm. At 20 h, head CT was concerning for worsening left hemispheric edema, but dual-energy, iodinesubtracting sequences revealed significant contrast extravasation contributing to the appearance of sulcal effacement but without actual edema. Out of concern for blood-brain barrier breakdown from CIE, pressor augmentation was discontinued and the patient gradually improved to full neurological recovery within 72 h of symptom onset.

*Conclusions* Our case is the first known to report the use of dual-energy, iodine-subtracting CT as a diagnostic tool in differentiating between cerebral edema and pseudoedema in CIE.

Keywords Contrast · Encephalopathy · Pseudoedema

## Introduction

We present a case and review of contrast-induced encephalopathy (CIE) and highlight the use of dual-energy iodine-subtracting computed tomography (CT) to differentiate between cerebral edema and "pseudoedema" from subarachnoid space contrast.

## Methods

A case report and subsequent literature review are described. We report a case of CIE in which we used a dualenergy CT scan to make the diagnosis. This type of scanning technique is performed at our institution when trying to rule out intracranial hemorrhage following neuro-interventional procedures or diagnostic angiography [1]. The advantage of this technique is that it allows one to distinguish between high-density contrast and high-density blood products. The dual-energy technique is based on the behavior of various materials (i.e., hemorrhage and iodine) when exposed to X-rays at low and high energy, leading to differences in attenuation. These differences reflect the

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energy and material dependency of Compton and photoelectric effects of iodine and hemorrhage.

Omnipaque 300 contrast was used in both CT and conventional angiography. Somatom Definition (Siemens Healthcare, Forchheim, Germany) was used for dual-energy scanning. This scanner consists of two X-ray tubes and two detectors, mounted on a common gantry and is available at most academic institutions in North America and Europe. In the dual-energy mode, the tube voltage and tube current settings for each X-ray source are set and optimized independently. CT examinations were performed using the following protocol: Tube A at 80 kV, 499 mA and Tube B at 140 kV, 118 mA. Dual-energy postprocessing was performed using dedicated software (Syngo Dual Energy Brain Hemorrhage, Siemens). Images were reconstructed at 1.5-mm section thickness with 0.7mm increments (D30s kernel). The analysis of low- and high-energy images was performed using a three-material decomposition algorithm based on brain parenchyma, hemorrhage, and iodine, producing a VNC and an iodineonly overlay image.

# Results

A 72-year-old woman with type 2 diabetes mellitus underwent elective endovascular flow-diverter embolization of a 10-mm left anterior communicating artery aneurysm. Immediately post-procedure, the patient was normal. One hour later, she developed global aphasia. CT and CT-angiography (CTA) of the head showed normal vasculature and high attenuation of the left hemispheric subarachnoid space interpreted as hemispheric edema. Emergent brain magnetic resonance imaging (MRI) showed scattered, punctate left middle cerebral artery territory FLAIR hyperintense lesions, nearly all of which showed restricted diffusion, which could not solely account for the patient's deficits.

The patient developed right hemiparesis and encephalopathy 12 h post-procedure with gradual worsening that peaked at 24 h. Repeat CTA showed diminutive caliber of the distal left middle cerebral (MCA) artery territory vasculature (Fig. 1). CT perfusion revealed decreased left hemisphere cerebral blood flow (Fig. 2a) with preservation of cerebral blood volume (Fig. 2b). The patient underwent emergent angiography and received 20 mg of intra-arterial verapamil for treatment of vasospasm due to distal left M2 tapering and diminutive M3 branches (Fig. 2c). Blood pressure was augmented using intravenous phenylephrine to a systolic goal of 160 mmHg. However, there was no change in distal left MCA vessel caliber on follow-up CTA. Electroencephalography showed focal moderate to high amplitude and persistent delta slowing on the left, maximally over the fronto-temporal head regions.

Twenty hours post-procedure, she continued to have severe neurological deficits that were progressively worsening. A non-contrast head CT was reported to show worsening left hemispheric edema (Fig. 2d). However, dual-energy sequences used to subtract the contrast signal revealed significant contrast extravasation contributing to the appearance of sulcal effacement but without actual brain edema (Fig. 2e, f). Vasopressor augmentation was discontinued and the patient had full neurologic recovery 72 h following symptom onset.

# Discussion

The clinical presentation of CIE can be severe and includes cortical blindness, encephalopathy, seizure, and focal neurological deficits [2]. Although most patients fully recover within 2–3 days, a minority (approximately 15%) die of cerebral edema or has persistent neurologic deficits [2]. Hypertension and renal failure are possible CIE risk factors, but our patient had neither [2].

The typical non-contrast CT finding in CIE is high attenuation of the cerebral sulci that can be misinterpreted as cerebral edema. Our case is the first described instance of using dual-energy, iodine-subtracting CT to differentiate cerebral edema from subarachnoid contrast in CIE. It is important to be aware of this diagnostic pitfall as some previously reported cases of edema on CT resulting from CIE could have actually been pseudoedema, as seen in our case. Our patient received multiple loads of contrast with additional tests because of the initial uncertainty about the reason for her clinical decline. We believe these repeated doses of iodinated contrast likely worsened her neurologic symptoms and perpetuated the disease process. Dual-energy CT can help the clinician make the diagnosis accurately and promptly and thus avoid any additional contrast. Dual-energy head CT is the test of choice when neuro-imaging is needed emergently after exposure to iodinated contrast. If findings do not suggest CIE, then performing a CTA (or angiography) may be suitable for further evaluation. As previous fatal cases of CIE have involved the use of high osmolar contrast agents [2], it is recommended to minimize osmolar load to the extent possible.

Brain MRI imaging findings in CIE have been previously reported. Increased T2 and FLAIR signal changes with facilitated diffusion (ADC hyperintensity) have been described [2–4]. However, punctate foci of restricted diffusion without T2 signal change have also been described [5, 6]. These findings are not in themselves specific or diagnostic. Arterial spin labeling or perfusion MRI studies



Fig. 1 Contrast-induced encephalopathy computed tomography-angiography (CTA). CTA demonstrates attenuation of the cortical vessels of the distal left middle cerebral artery territory relative to the right, presumably due to vasospasm



Fig. 2 Contrast-induced encephalopathy computed tomography (CT) perfusion, conventional angiography, and dual-energy CT. a Cerebral blood flow map demonstrates decreased relative blood flow in the left cerebral hemisphere relative to the right. b However, the cerebral blood volume map is normal. c Repeat conventional angiography revealed distal left M2 tapering and diminutive M3 branches. d Non-

contrast CT shows diffuse sulcal effacement, with mild high density in the sulci. This was concerning for cerebral edema.  $\mathbf{e}$  However, the iodine map from the dual-energy CT demonstrates that the high attenuation in the sulci is actually due to contrast in the subarachnoid space and not edema.  $\mathbf{f}$  Following subtraction of iodine, there is no evidence of cerebral edema in the left hemisphere may have been more informative (and were not performed in our case), but would also take longer to obtain.

Postulated CIE mechanisms include blood-brain barrier (BBB) breakdown from direct contrast chemotoxicity, fluid shifts from hyperosmolality, and endothelin release permeabilizing the BBB [2, 7]. As indicated by our case, diffuse vasospasm with resultant hypoperfusion from contrast-induced vessel wall irritation may be another mechanism.

While some investigators elect for conservative management in CIE, others have utilized dexamethasone and mannitol [2]. Given the angiographic vasospasm, we administered intra-arterial verapamil. It is unclear whether or not this altered the clinical course of CIE in our patient or whether the improvement was simply due to the natural history of the disease. Since there was no appreciable change in vessel caliber following verapamil, we favor the latter.

# Conclusion

CIE is a rare complication of angiography and endovascular treatment of cerebrovascular diseases. Dual-energy, iodine-subtracting CT promptly confirms the diagnosis and avoids further exposure to additional iodinated contrast, which might otherwise exacerbate the disease.

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#### **Compliance with Ethical Standards**

**Conflict of interest** Drs. Pagani-Estévez, Nasr, Brinjikji, Perry, and Fugate have no conflicts of interest to disclose. Funding was not used for this study.

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