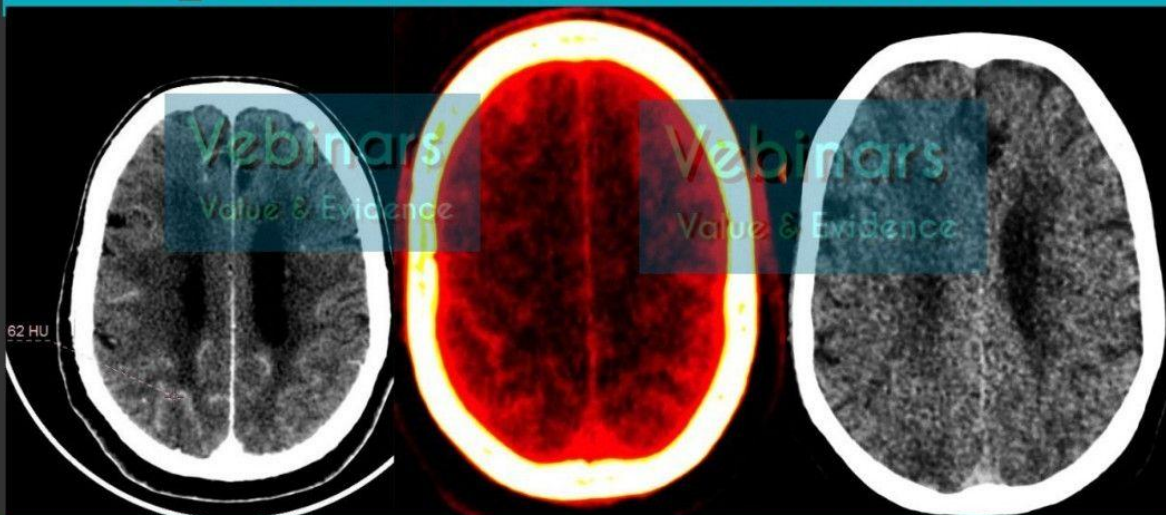


VEBINARS SS_02 Contrast induced Encephalopathy

SS_02 History

- ▶ Sudden onset chest pain- Diagnosed as Inferior wall MI
- ▶ Moderate renal dysfunction
- ▶ Underwent prolonged percutaneous coronary intervention-with approximately 300 ml of contrast
- ▶ Pt had altered sensorium post PCI

SS_02-CONTRAST INDUCED ENCEPHALOPATHY



Plain CT- Sulcal
Hyperdensities

DECT-Iodine overlay map-
Sulcal Hyperdensity

DECT- Virtual Non Contrast
No Hyperdensity

Vebinars
Value & Evidence

CONTRAST

SS_02 CIE: Teaching points and Clinical pearls

CIE: Diagnosis of exclusion

Presentation: Encephalopathy, convulsions, cortical blindness and focal neurological deficits

Prolonged/Difficult interventions requiring large amounts of iodine based contrast material

Altered hemodynamic status

Renal derangement

Role of Imaging in CIE : To exclude CVA-Hemorrhage; Infarction- Secondary to Embolic showers/Hemodynamic compromise/Arterial injury-Dissection

Imaging features: Can be Normal, Cerebral edema, Cortical and subcortical enhancement, Diffuse high densities in the subarachnoid space-mimics SAH(HU/DECT for differentiation)- Confirm with MRI or F/U CT -48 to 72 hours

Management: Conservative-Medical management-Anti edema, neuroprotective measures

Prognosis: Reversible in majority of cases with no long term deficits

SS_02 CIE: Teaching points

Contrast staining following any angiographic procedure using iodine based contrast- happens likely secondary to transient breakdown in BBB

Contrast staining vs SAH

Plain CT - HU often unreliable

DECT - Iodine only and iodine subtracted (virtual non contrast) maps

Iodine overlay map- Hyper attenuation indicates iodine

Iso to hypoattenuation no iodine- Hemorrhage

Interval imaging preferably after 24 hours - Contrast staining disappears, Bleed persists

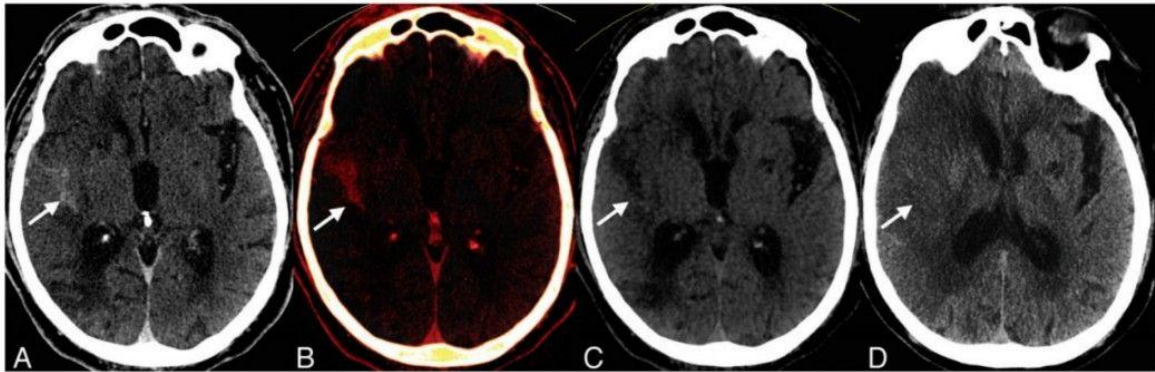


Fig 3. Subarachnoid hyperattenuation due to contrast staining in a 79-year-old man treated endovascularly for an acute stroke in the right MCA territory. *A*, Diffuse hyperattenuation in the right Sylvian fissure (*arrow*) on the SE image corresponds to the hyperattenuation seen on the iodine overlay image (*B*). The lack of hyperattenuation on the VNC image (*C*) suggests contrast extravasation in the Sylvian fissure. This is confirmed by the near-complete washout of the hyperattenuation on the 24-hour follow-up NCCT (*D*).

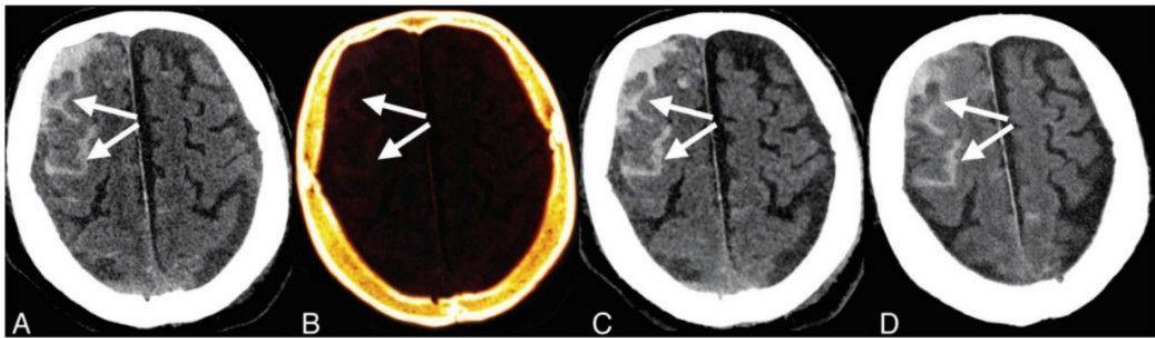


Fig 4. Subarachnoid hyperattenuation due to hemorrhage in a 64-year-old man. *A*, There are foci of sulcal hyperattenuation (*arrows*) on the SE image. *B*, No corresponding hyperattenuation is seen on the iodine overlay image. *C*, VNC image shows identical foci of sulcal hyperattenuation, suggesting subarachnoid hemorrhage that was confirmed by the 24-hour follow-up NCCT (*D*).

Differentiation of Hemorrhage from Iodinated Contrast in Different Intracranial Compartments Using Dual-Energy Head CT. *American Journal of Neuroradiology* June 2012, 33 (6) 1088-1094; DOI: <https://doi.org/10.3174/ajnr.A2909>