THE ROLE OF PERFUSION CT IN THE ACUTE DIAGNOSIS OF SMART SYNDROME

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INTRODUCTION

SMART syndrome (Stroke-like Migraine Attacks after Radiation Therapy) is a rare late complication of cerebral radiotherapy, characterized by reversible migraine attacks, transient focal neurological deficits and seizures. Acute phase imaging → MRI cortical enhancement and T2-FLAIR hyperintensity.

Perfusion imaging data are still limited and inconsistent

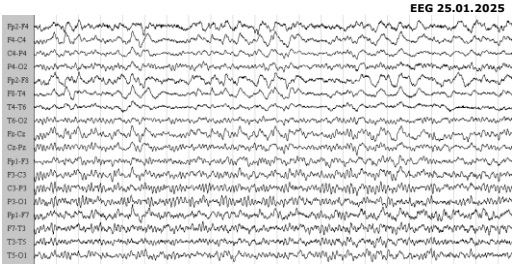
- ↑ **CBV e** ↑ **CBF** corticale during attacks
- initial ↓ **CBF** suggesting impaired cerebrovascular autoregulation
- transition from hypo to hyperperfusion during symptom resolution.

CASE REPORT

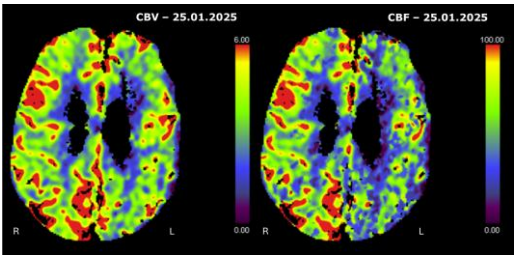
31-year-old man.
→ 18 yo resection of multicentric supratentorial medulloblastoma (EVD + chemo + **radio**) with sequelae blindness, hearing loss (cochlear implant), and panhypopituitarism
→ since 23 yo **transient episodes** of left-sided hemiparesis and speech disturbances (SMART syndrome)

→ 31 yo: **relapse** of left-sided hemiplegia lasting 30 min

- **brain CTP**: right hemisphere hyperperperfusion (MRI not possible due to cochlear implant artifact)
- simultaneous **EEG**: depressed right cortical activity without epileptiform abnormalities

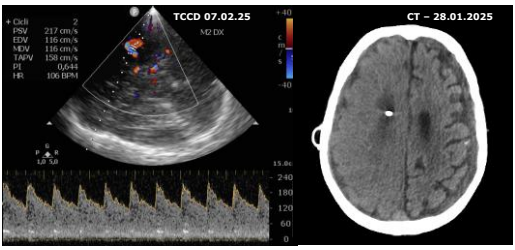


30 sec/pg - 10 uV/mm, HF 30, TC 0.1



→ required intensive care and endotracheal intubation for a lower respiratory tract infection

- follow-up **brain CT** showed right hemispheric edema
- **EEG** remained stable
- **TCCD** increased FV with turbulences at M2 frontal branch of right MCA, later progressively reduced (PSV max 217 c/s, then 140 c/s; n.v. 78-108 c/s)
- **iv steroid therapy** » resolution of the edema



Outcome: near-complete recovery of motor function on the left side.

CONCLUSIONS

Perfusion CT can be helpful in the diagnosis of acute neurological deficits in patients with a history of cranial radiotherapy. In this case, the hyperperfusion did not correlate with neuronal hyperexcitability, ruling out status epilepticus as the underlying mechanism, but rather transient dysregulation of cerebral autoregulation. This suggests that hyperperfusion alone should not be interpreted as a marker of ictal activity and that EEG is essential for accurate interpretation.

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