

## Texas Society of Neuroradiology (TSNR)

### Excerpta Abstract

2026 Annual Meeting – Dallas, TX

February 21–22, 2026

## Recognizing SMART Syndrome: Imaging Evolution, Pitfalls, and Differentiation from Recurrence

Junaid Kalair, MD Houston Methodist Hospital

Steve Fung, MD Houston Methodist Hospital

### Clinical History

A 62-year-old man with remote history of left temporooccipital glioblastoma treated by resection, temozolomide, and high-dose radiation therapy (60 Gy) 19 years earlier presented with new dysarthria, confusion, and falls. CT showed chronic posttreatment changes with extensive encephalomalacia in the left temporoparietooccipital region without acute abnormality. MRI showed new unilateral linear and micronodular leptomeningeal and cortical enhancement, diffuse mild cortical DWI hyperintensity with equivocal diffusion change confined to the prior radiation field without mass effect.

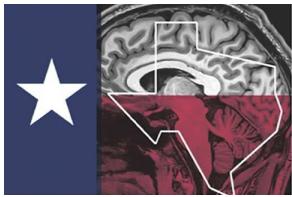
CSF cytology and spinal MRI were negative for malignancy or infection. During admission, the patient had recurrent focal seizures, and interim MRI demonstrated small acute left occipital infarcts distinct from the enhancing regions. Over subsequent weeks, the leptomeningeal and cortical enhancement intensified with associated swelling. MR perfusion and spectroscopy were inconclusive. Multidisciplinary review favored SMART syndrome (stroke-like migraine after radiation therapy) over tumor recurrence or infectious meningitis. Corticosteroids and verapamil led to radiographic resolution and clinical improvement.

### Imaging Findings

Initial MRI: Extensive encephalomalacia with predominantly white-matter volume loss involving left temporal, parietal, and occipital lobes from prior resection and chemoradiation therapy. Superimposed new leptomeningeal and cortical enhancement, diffuse mild cortical DWI hyperintensity with equivocal diffusion change involving left temporal, parietal, occipital, and insular regions coinciding with prior radiation field without mass effect.

Follow-up MRI: Increasing extent of leptomeningeal and cortical enhancement involving left temporal, parietal, occipital, insular, and to lesser degree frontal lobes, accompanied by increasing diffuse cortical thickening with T2 hyperintensity. Persistent diffuse mild cortical DWI hyperintensity in the same distribution with superimposed more pronounced DWI hyperintensity with restricted diffusion from small acute left occipital ischemic infarctions that developed in the interim.

Post-treatment MRI: Near-complete resolution of leptomeningeal and cortical enhancement, confirming reversibility. Subcortical restricted diffusion and gyriform enhancement in left occipital region corresponding to evolving subacute-ischemic infarction and vascular dysregulation related to SMART syndrome.



## Texas Society of Neuroradiology (TSNR)

### Excerpta Abstract

2026 Annual Meeting – Dallas, TX

February 21–22, 2026

### Discussion

SMART syndrome is a delayed neurovascular complication of cranial irradiation resulting from radiation-induced endothelial injury, autoregulatory failure, and transient breakdown of the blood-brain-barrier (1). The consequence is episodic cortical hyperexcitability and reversible vasogenic edema that can occur years to decades after treatment (2).

Proposed pathways include endothelial dysfunction, impaired vasoreactivity, cortical spreading depression, and regional perfusion instability. These processes overlap with migraine and seizure physiology (3,4). The vascular and metabolic shifts explain the transient nature of the clinical symptoms and imaging abnormalities.

When new unilateral cortical or leptomeningeal enhancement develops within a prior radiation field, diagnosis should focus on:

1. Diffusion and perfusion: lack of restricted diffusion or hyperperfusion argues against disease recurrence.
2. Confinement: enhancement confined to the irradiated territory supports SMART.
3. Reversibility: resolution on follow-up imaging confirms the diagnosis and prevents unnecessary biopsy.

Treatment remains supportive with corticosteroids and calcium-channel blockers to reduce cortical edema, stabilize the endothelium, and restore autoregulatory balance (1,2).

### Teaching Point

- SMART syndrome may occur months to decades after cranial irradiation.
- Imaging hallmark: reversible unilateral or asymmetric cortical and leptomeningeal enhancement within the radiation field.
- Assessment of diffusion, perfusion, and reversibility differentiates SMART from recurrence or infection.
- Recognition prevents unnecessary biopsy and guides appropriate management.

### References

- [1] Black DF, Bartleson JD, Bell ML, Lachance DH. SMART syndrome: stroke-like migraine attacks after radiation therapy. *Cephalalgia*. 2006;26(9):1137-1142. doi:10.1111/j.1468-2982.2006.01184.x



## Texas Society of Neuroradiology (TSNR)

### Excerpta Abstract

2026 Annual Meeting – Dallas, TX

February 21–22, 2026

[2] Black DF, Morris JM, Lindell EP, Krecke KN, Worrell GA, Bartleson JD, et al. Stroke-like migraine attacks after radiation therapy (SMART) syndrome is not always completely reversible: a case series. *AJNR Am J Neuroradiol.* 2013;34(12):2298-2303. doi:10.3174/ajnr.A3602

[3] Zheng Q, Yang L, Tan LM, Qin LX, Wang CY, Zhang HN, et al. Stroke-like migraine attacks after radiation therapy syndrome. *Chin Med J (Engl).* 2015;128(15):2097-2101. doi:10.4103/0366-6999.161393

[4] Ota Y, Nemoto M, Gyoten T, Liao E, Shah G, Capizzano AA. Comprehensive update and review of clinical and imaging features of SMART syndrome. *AJNR Am J Neuroradiol.* 2023;44(6):626-639. doi:10.3174/ajnr.A7859

### Figures

