Radiation injury

- Unfortunate mimic of recurrent glioma
 - Late delayed injury: usually months after treatment
 - Irregular enhancement, necrosis, mass effect
- Pseudoprogression: XRT+temodar, earlier (<12 weeks)
- Timing
 - Acute (during or shortly after radiation)
 - Subacute/early delayed (up to 12 weeks after radiation)
 - Late (months to years after completion of radiation)

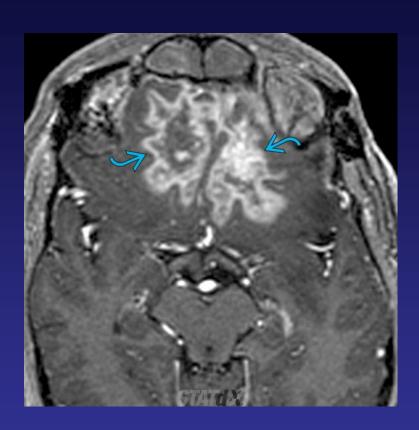
Imaging

- Radiation injury: Mild vasogenic edema to necrosis
- Radiation necrosis: Irregular enhancing lesion(s)
 - MRS: Markedly ↓ metabolites, ± lactate/lipid peaks
 - Perfusion MR: ↓ rCBV compared with tumor
- Leukoencephalopathy: T2 white matter (WM) hyperintensity, spares subcortical U fibers
- Mineralizing microangiopathy: Basal ganglia, subcortical WM Ca⁺⁺, atrophy
- Necrotizing leukoencephalopathy: WM necrosis
- **PRES**: Posterior circulation subcortical WM edema

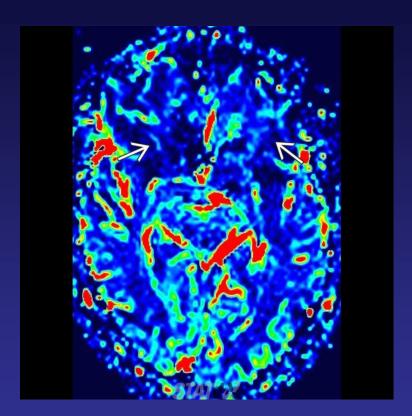
Pathology

- Radiation-induced vasculopathy: Varying degrees of stenosis or occlusion
- Radiation-induced vascular malformations: Capillary telangiectasias ± cavernous malformations
- Radiation-induced parenchymal injury: Radiation necrosis
- Radiation-induced tumor (i.e., sarcoma)
- Mineralizing microangiopathy: Common with chemotherapy and radiation, appears ≥ 2 yrs after radiation
- Necrotizing leukoencephalopathy: Combined radiation and chemotherapy, progressive disease

Radiation Necoris



Axial T1WI C+ MR in a patient with esthesioblastoma 6 months post radiation shows masses (cyan curved arrow) in the inferior frontal lobes with a spreading wavefront enhancement pattern.



MR perfusion rCBV map in the same patient does not show any significant increase in the relative cerebral volume (white solid arrow) in the regions of enhancement.

These findings are typical of radiation necrosis.

Radiation necrosis can occur months to years after radiation therapy. More than 85% of cases occur within 2 years.

General Features

- Radiation necrosis in brain occurs months to years following RT for CNS or head and neck cancers.
- Radiation necrosis: Usually seen 2-32 mos after therapy; 85% of cases occurring within 2 yrs
- Rarely follows RT for vascular lesions (e.g., AVMs)
- Incidence of radiation necrosis in setting of CNS or head and neck radiotherapy has been estimated as 3-24%
- Risk factors
 - Age
 - Radiation volume, radiation dose, radiation fraction size
 - Concurrent chemotherapy
 - Pretreatment cognitive dysfunction
 - Increasing survival time following RT

Risk factors

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Definitions

- Radiation-induced brain injury (RIBI): Variety of imaging findings that result from radiation therapy
 - **Acute** (during or shortly after radiation), **early delayed** (typically 1-6 months after radiation), and **late delayed** (6 months to years after completion of radiation)
 - Radiation-induced vascular injury
 - Radiation-induced vasculopathy, radiation-induced vascular malformations (RIVMs), and mineralizing microangiopathy
 - Radiation-induced parenchymal injury
 - Radiation necrosis, combination of vascular & parenchymal injury with necrosis of tumor &/or normal brain parenchyma
 - Radiation-induced leukoencephalopathy denotes white matter (WM) injury, necrotizing leukoencephalopathy (NLE)
 - Radiation-induced neoplasms
 - Stroke-like migraine attacks after radiation therapy (SMART) syndrome
- Chemotherapeutic agents related toxicity to CNS: Drug-related toxic leukoencephalopathy, posterior reversible encephalopathy syndrome (PRES)

Radiation injury

- Edema
- Arteritis
- Radiation necrosis
- Leukoencephalopathy
- Mineralizing microangiopathy
- Necrotizing leukoencephalopathy
- Radiation-induced tumors

Nuclear medicine

Radiation necrosis

- Radiation necrosis and recurrent brain tumor both show mass effect and contrast enhancement on CT
 and MR
- High-grade tumors have more F-18 FDG uptake than low-grade tumors
- Generally hypometabolic, F-18 FDG should not accumulate in necrotic tissue
 - Low uptake favors radiation necrosis or low-grade tumor
 - Sensitivity 75% and specificity 81% for F-18 FDG PET/CT in differentiating radiation necrosis from recurrent tumor

Pseudoprogression

- Subacute treatment-related effects that can mimic tumor progression
- Early findings, 2-6 months following RT that eventually resolve
- F-18 FDG uptake is not increased in postoperative period and is not affected by steroid therapy

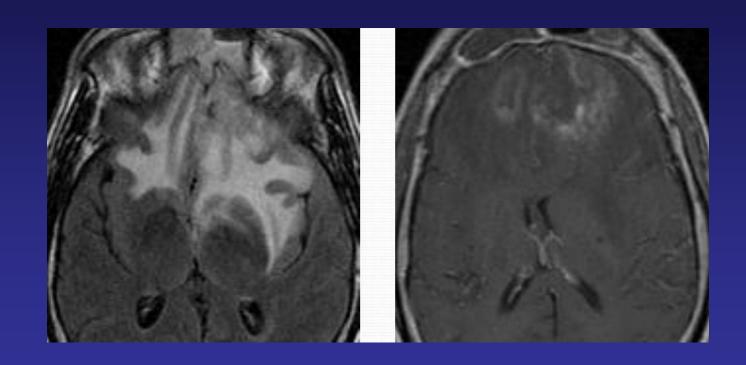
Recurrent tumor

- Tumor cells accumulate F-18 FDG
- High uptake favors tumor recurrence or radiation necrosis
- High-grade primary tumors and metastases are more F-18 FDG-avid than lower grade gliomas
- Conversion to higher grade glioma suggested by level of hypermetabolism
- Small tumors < 6 mm may be undetectable by F-18 FDG PET/CT

Imaging

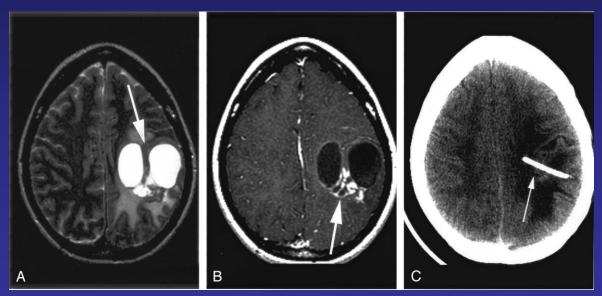
- F-18 FDG PET/CT has high negative predictive value
- Lesions with no F-18 FDG uptake are likely radiation necrosis (or low-grade tumor recurrence)
- Thallium can be used

Radiation necrosis



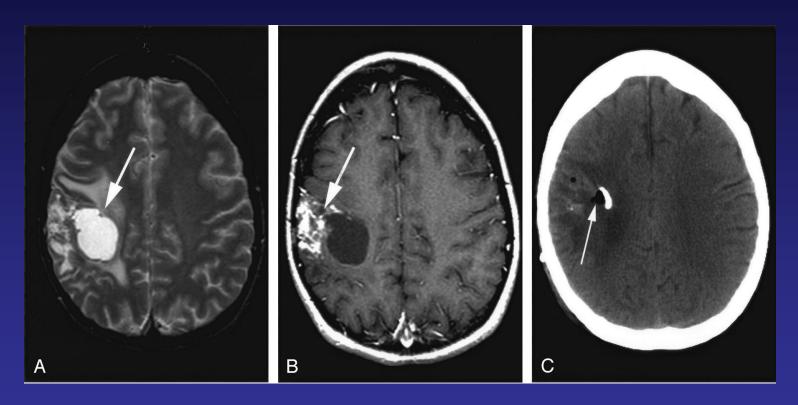
Tumefactive cysts

- Late complication (years) of AVM radiosurgery
- Thin enhancing wall, ± adjacent heterogeneous enhancement



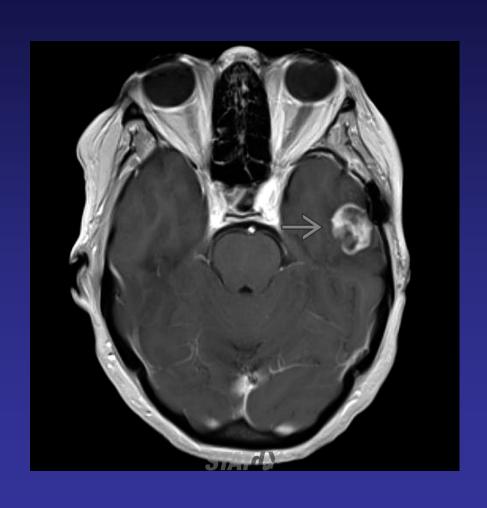
- MR image obtained 40 months after AVM ablation.A,
- T2-weighted axial image showed a 5-cm complex, multiloculated cyst in the left frontoparietal region with vasogenic edema and local mass effect (arrow).
- B, Contrast-enhanced T1-weighted axial image shows nodular parenchymal enhancement (arrow).
- C, CT after successful cystoperitoneal shunt placement shows decompression of the cysts.

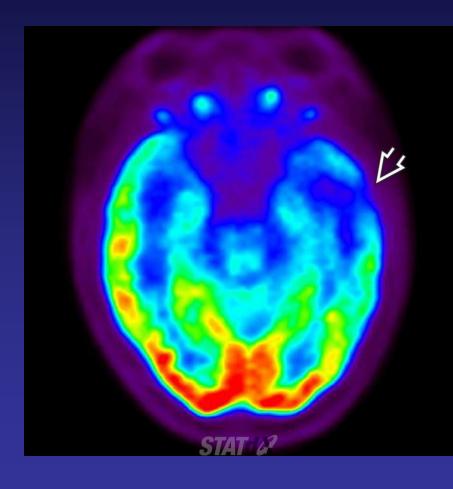
Tumefactive Cyst



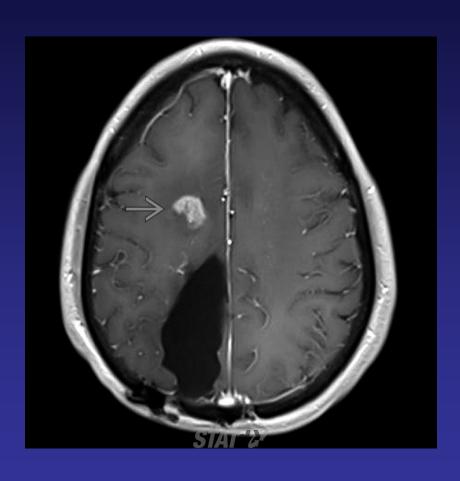
- Radiation therapy-induced cyst 89 months after AVM treatment.A,
- T2-weighted axial image shows a 3-cm cyst in the frontoparietal region with vasogenic edema (arrow).B, Contrast-enhanced
- T1-weighted axial image shows nodular contrast enhancement superficial to the cyst (arrow).C, Postoperative CT showed successful cyst decompression (arrow) after cystoperitoneal shunt placement.

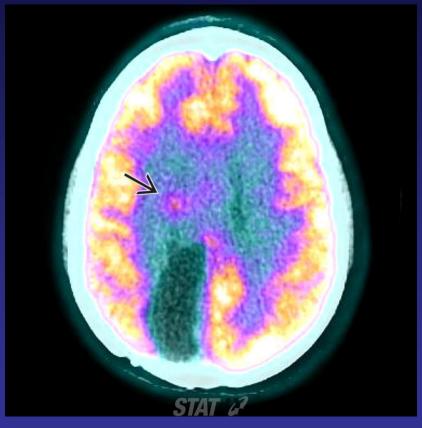
Necrosis

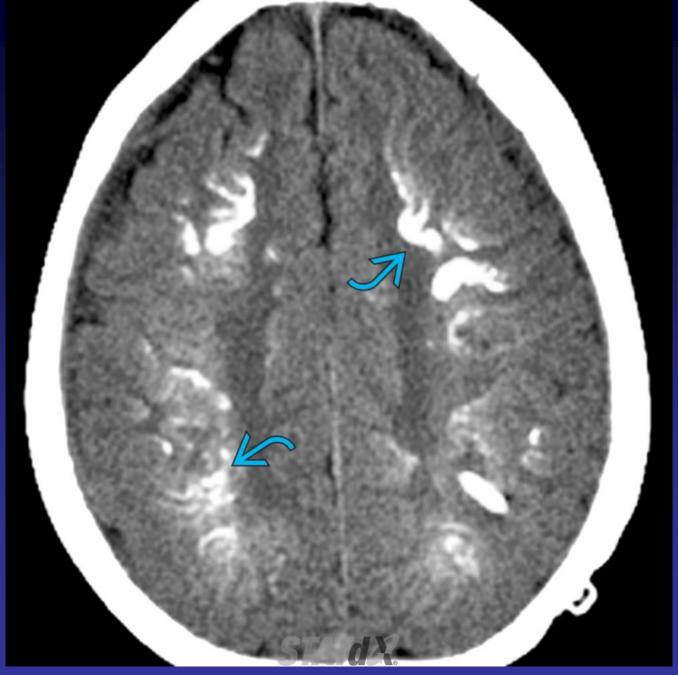




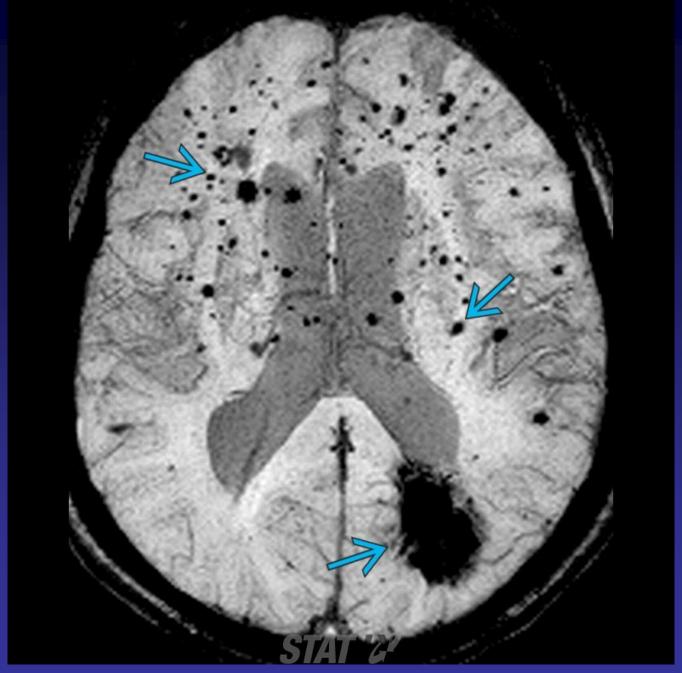
Recurrence







Axial NECT shows extensive calcification in the subcortical white matter (WM) (cyan curved arrow) in a 20-year-old patient. Mineralizing microangiopathy usually results after a combination of <u>radiation therapy and chemotherapy 2 or more years after treatment</u>.



Axial SWI in an adult patient with neurofibromatosis and optic nerve glioma status post radiation therapy in childhood shows innumerable "blooming" hypointense foci (cyan solid arrow) consistent with radiation-induced vascular malformations.