Cerebral Neoplasms

Edmond A. Knopp and Girish M. Fatterpekar

A mass-like lesion in the brain always makes us consider the possibility of an underlying tumor. We then assess the imaging pattern in order to establish an appropriate tumoral differential diagnosis. While this approach often works, a mass lesion can sometimes simulate a tumor. Identification of such a tumor mimic is essential since it can significantly influence further management. This review article will focus on imaging features of brain tumors and tumor mimics. Considering the exhaustive list of tumors (intra-axial, calvarial/dural based, sellar based, pineal region based, and intraventricular), we will limit our discussion to intra-axial tumors.

Intra-axial Brain Tumors

Astrocytic Tumors

Pilocytic Astrocytoma

Pilocytic astrocytoma is a WHO grade I well-circumscribed, slow-growing tumor seen more commonly in children and young adults. Common locations in children include the optic pathway, hypothalamus, cerebellum, and brain stem. Common locations in adult include thalamus and basal ganglia.

Characteristic imaging findings: A cystic-appearing lesion with an intensely enhancing mural nodule with minimal to no surrounding edema is often seen [1]. The intense enhancement reflects the prominent vascularity known to be associated with these lesions. Hemorrhage and calcification are uncommon. It should be noted that visual pathway and hypothalamic lesions are more solid in appearance and can show patchy enhancement. Involvement of subarachnoid space can be seen in pilocytic astrocytoma and should not make one think of a malignant transformation.

Best sequence(s) to evaluate pilocytic astrocytoma: Post-contrast T1WI/MPRAGE.

Pilomyxoid Astrocytoma

Pilomyxoid astrocytoma is a WHO grade I tumor which can be considered to be a histologic variant of pilocytic astrocytoma. As the name suggests, it has a markedly myxoid matrix which is not seen in the classic pilocytic astrocytoma. The tumor demonstrates a more aggressive behavior pattern than a typical pilocytic astrocytoma and recurs more often. It is more commonly seen in the pediatric population. Favored location is in the hypothalamic region.

Characteristic imaging findings: Pilomyxoid astrocytoma consistent with its myxoid matrix is seen as a hypointense lesion on T1WI, which appears hyperintense on long TR sequences and demonstrates moderate enhancement [2]. Hemorrhage, calcification, necrosis, and edema are uncommon.

Best sequence(s) to evaluate pilomyxoid astrocytoma: FLAIR/T2WI and post-contrast T1WI/MPRAGE.

Pleomorphic Xanthoastrocytoma

Pleomorphic xanthoastrocytoma is a WHO grade II tumor, seen in children and young adults. Often seen in the supratentorial compartment, the temporal lobe is a favored location.

Characteristic imaging findings: A cystic-appearing lesion with a mural enhancing nodule is seen. Oftentimes, the mural enhancing nodule is cortical based extending superficially up to the leptomeningeal surface (Fig. 1) [3, 4]. Hemorrhage, calcification, and surrounding edema are uncommon.

Best sequence(s) to evaluate pleomorphic xanthoastrocytoma: Post-contrast T1WI/MPRAGE.

Diffuse Astrocytoma

Diffuse astrocytomas are WHO grade II tumors, often seen in adults in the third to fifth decade of life. Characterized by

E.A. Knopp
Radiology, Zwanger-Pesiri Radiology, 150 East Sunrise Highway, Suite 208, Lindenhurst, NY 11757, USA
e-mail: eknopp@zwangerpesiri.com

G.M. Fatterpekar (✉)
Radiology, NYU School of Medicine, 660 First Avenue, New York, NY 10016, USA
e-mail: Girish.Fatterpekar@nyumc.org

J. Hodler et al. (eds.), *Diseases of the Brain, Head and Neck, Spine 2016–2019: Diagnostic Imaging*, DOI 10.1007/978-3-319-30081-8_1

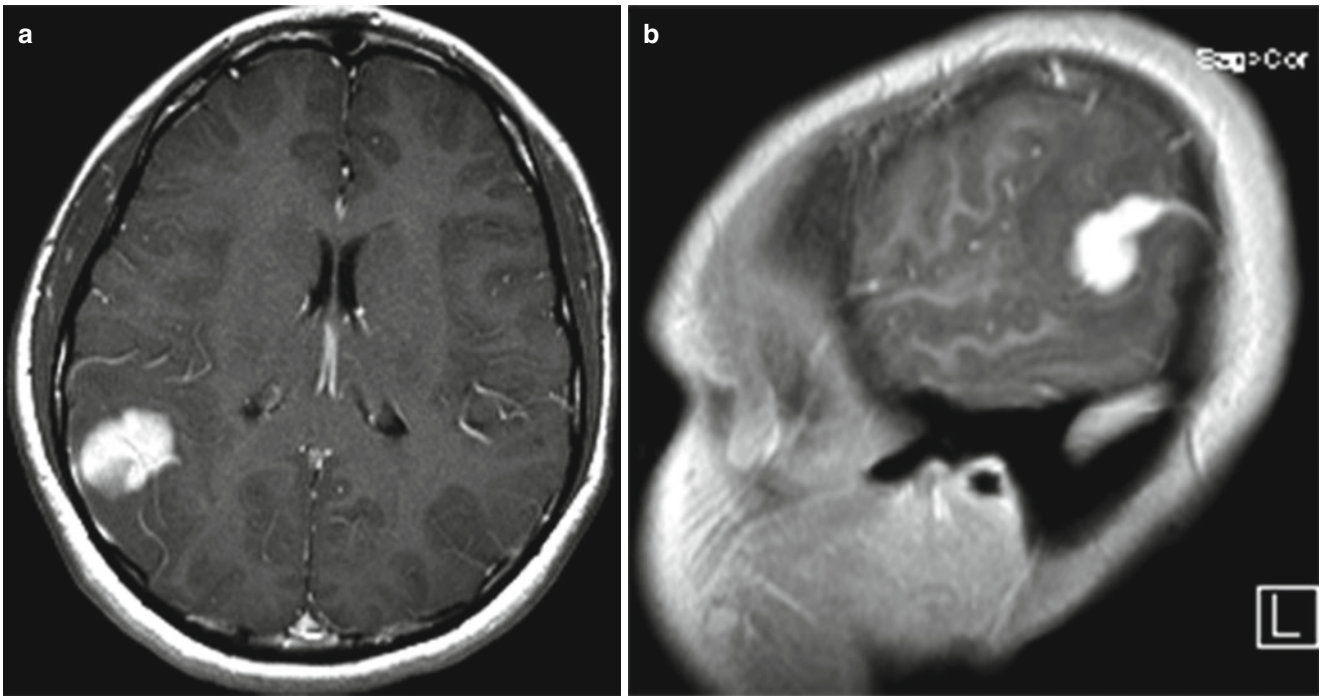

Fig. 1 Contrast-enhanced (**a**) axial and (**b**) sagittal MPRAGE images demonstrating an intensely enhancing nodule with surrounding edema in the left posterior temporal region. The nodule is seen to abut the leptomeningeal surface. Diagnosis: pleomorphic xanthoastrocytoma

slow growth, there is variable infiltration of adjacent brain structures. Malignant degeneration or degeneration into anaplastic astrocytoma can sometimes occur.

Characteristic imaging findings: An ill-defined mass hypointense on T1WI and hyperintense on T2WI, extending to and expanding the cortex, is seen. Absent to minimal contrast enhancement is seen [5]. The lack of high cellularity correlates with lack of diffusion restriction. There is no increased perfusion seen helping distinguish it from an anaplastic astrocytoma (WHO grade III) or glioblastoma (WHO grade IV). Hemorrhage, calcification, and tumoral necrosis are not seen.

Note: Brain stem gliomas, often seen as diffuse pontine lesions, are WHO grade II tumors. These tumors are most often seen in the pediatric population.

Best sequence(s) to evaluate diffuse astrocytoma: FLAIR for extent and perfusion to help distinguish from high-grade tumors.

Anaplastic Astrocytoma

Anaplastic astrocytoma is a WHO grade III tumor, often seen in adults in the third to fifth decade of life. It is defined as a diffuse astrocytoma with focal or dispersed anaplasia.

Characteristic imaging findings: Conventional imaging features are highly similar to those of diffuse astrocytoma. The presence of increased perfusion (likely reflecting neoangiogenesis) helps distinguish anaplastic astrocytoma from diffuse astrocytoma [6].

Best sequence(s) to evaluate anaplastic astrocytoma: FLAIR for extent and perfusion to help establish increased rCBV.

Gliomatosis Cerebri

Gliomatosis cerebri is a WHO grade III tumor, most commonly seen in adults in the third to fifth decade of life. It is defined as a diffusely infiltrating astrocytic tumor, involving three or more lobes. Extension across the corpus callosum and into the infratentorial compartment is common.

Characteristic imaging findings: Ill-defined infiltrative mass lesion involving the cortex and the white matter with associated mass effect and contiguously involving more than three lobes is seen [7, 8]. Extension across the splenium of corpus callosum and into the infratentorial compartment is often seen. Typically, minimal to no enhancement is noted. Hemorrhage, necrosis, and calcification are not seen.

Best sequence(s) to evaluate gliomatosis cerebri: FLAIR.

Glioblastoma

Glioblastoma is a WHO grade IV tumor, the most malignant neoplasm of the group of diffuse astrocytic tumors. It is the most common primary intra-axial brain tumor and contributes to approximately 50–60 % of all astrocytic tumors. In adults, most such tumors are seen in the supratentorial compartment; in the pediatric population, though considered an uncommon tumor, the brain stem is a favored site. Primary

glioblastomas typically develop in older individuals (sixth decade of life), whereas secondary glioblastomas derived from low-grade or anaplastic astrocytomas are seen in younger patients (fourth decade of life).

Characteristic imaging findings: An irregularly marginated, peripherally enhancing, centrally necrotic lesion with variable surrounding edema is seen. Diffusion restriction can be seen from the solid enhancing component of the lesion. Facilitated diffusion is seen from the necrotic component of the lesion. Foci of susceptibility suggestive of hemorrhage and neoangiogenesis are often seen. Increased rCBV from the solid enhancing component of the tumor is seen on perfusion-weighted imaging (Fig. 2) [9].

Best sequence(s) to evaluate glioblastoma: Contrast-enhanced T1WI/MPRAGE, diffusion-weighted imaging, and perfusion imaging.

Oligodendroglial and Oligoastrocytic Tumors

Oligodendroglioma

Oligodendroglioma is a WHO grade II tumor derived from oligodendroglia or from glial precursor cells. It is most commonly seen to involve adults in the third to fourth decade of life. Most such tumors are seen in the cerebral hemispheres, frontal lobes being the most common location.

Characteristic imaging findings: Infiltrative tumors with poorly defined margins. Closer inspection often demonstrates expansion of the involved cortex. Calcification (appreciated on gradient-echo or susceptibility-weighted imaging and still better on CT) is common. Variable degree of enhancement is seen [10–13]. Minimal edema can be seen. Small cysts and hemorrhage can be seen. Increased rCBV is noted on perfusion imaging and unlike diffuse astrocytomas does not suggest a high-grade (WHO grade III or IV) tumor.

Best sequence(s) to evaluate oligodendroglioma: FLAIR/T2WI, gradient-echo or susceptibility-weighted imaging, and non-contrast CT.

Note: Interval hemorrhage, necrosis, or ring enhancement on follow-up studies should be worrisome for anaplastic transformation of oligodendroglioma.

Oligoastrocytoma

Oligoastrocytoma is a WHO grade II tumor resembling tumor cells in both oligodendroglioma and diffuse astrocytoma. Most tumors are seen to occur in adults, in their third to fourth decade of life [14].

Characteristic imaging findings: Imaging findings demonstrating an overlap between both oligodendroglioma and astrocytoma are seen.

Best sequence(s) to evaluate oligoastrocytoma: FLAIR/T2WI and contrast-enhanced T1WI/MPRAGE.

Neuronal and Mixed Neuronal-Glial Tumors

Desmoplastic Infantile Ganglioglioma

Desmoplastic infantile ganglioglioma is a WHO grade I tumor, often seen in the first 2 years of life. This tumor is typically classified together with the desmoplastic infantile astrocytoma, which differs histologically by its lack of mature neuronal components.

Characteristic imaging findings: A complex tumor, relatively large in size and demonstrating both cystic and solid components, is seen. The solid enhancing component is superficially placed in contrast to the typically deep-seated uni- or multilocular large cyst [4, 15]. For the size of the lesion, only minimal edema is seen. Calcification and hemorrhagic foci are uncommon.

Best sequence(s) to evaluate desmoplastic infantile ganglioglioma: T2WI and contrast-enhanced T1WI/MPRAGE.

Gangliglioma

Ganglioglioma is an uncommon WHO grade I or II tumor. Most tumors are seen in the first 3 decades of life with a peak age of incidence between 10 and 20 years of age. Often seen in the supratentorial compartment, the temporal lobe is a favored site.

Characteristic imaging findings: A cortical-based cystic-appearing lesion with calcification within the temporal lobe is highly suggestive of ganglioglioma. Oftentimes, enhancement is seen [4, 16, 17].

Best sequence(s) to evaluate ganglioglioma: Contrast-enhanced T1WI/MPRAGE, gradient-echo or susceptibility-weighted imaging to look for calcification, and non-contrast CT.

Dysembryoplastic Neuroepithelial Tumor

Dysembryoplastic neuroepithelial tumor is an uncommon WHO grade I tumor, seen most commonly in the pediatric population.

Characteristic imaging findings: Superficially (cortically) located mass lesion demonstrating multiple pseudocysts causing a soap bubble appearance on T2WI is seen. No diffusion restriction, hemorrhage, or calcification is seen. FLAIR typically demonstrates a hyperintense margin along the margin of the cysts. No enhancement is seen [18, 19].

Best sequence(s) to evaluate dysembryoplastic neuroepithelial tumor: T2WI/FLAIR and contrast-enhanced T1WI/MPRAGE.

Embryonal Tumors

Medulloblastoma

Medulloblastoma is a WHO grade IV tumor, most often seen in the pediatric population in the posterior fossa. A second smaller peak occurs in the late second-early third decade of life.

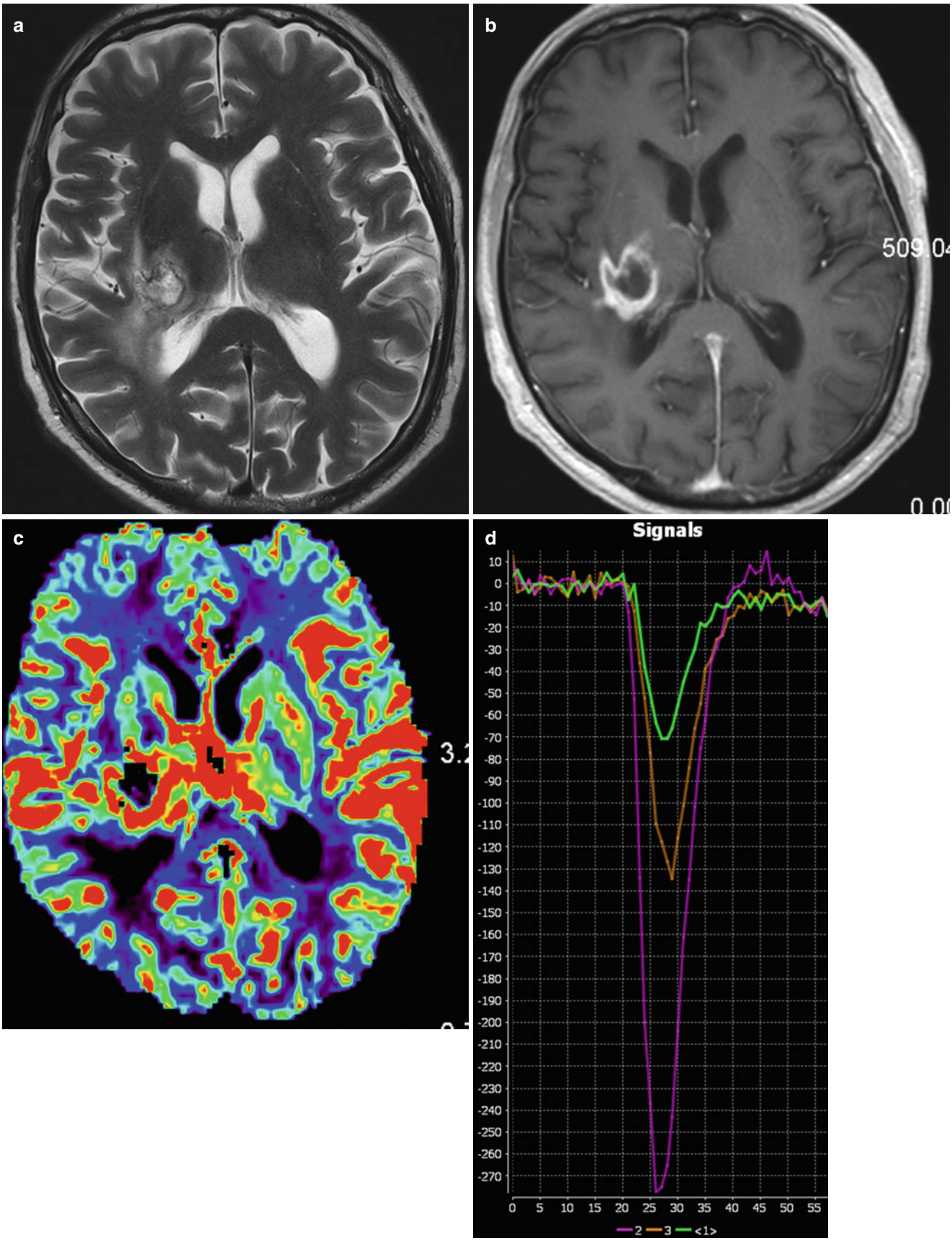

Fig. 2 (**a**) Axial T2WI and (**b**) contrast-enhanced axial T1WI demonstrate a peripherally enhancing centrally necrotic lesion in the right corona radiata. (**c**) DSC perfusion imaging demonstrates increased perfusion from the peripheral rim of the lesion. Also, there is a suggestion of increased perfusion even in the adjacent non-enhancing white matter. (**d**) Perfusion maps demonstrate markedly increased rCBV (compared to the green curve correlating to contralateral normal-appearing white matter), with values corresponding to 7.81. Diagnosis: glioblastoma

Characteristic imaging findings: Midline, posterior fossa masses arising from the roof of the fourth ventricle, displacing the fourth ventricle ventrally, and demonstrating homogenous diffusion restriction and enhancement are radiologic features of a classic medulloblastoma. Metastatic foci seeding the subarachnoid space within the intracranial compartment and in the spine can be seen [20, 21].

Best sequence(s) to evaluate medulloblastoma: Diffusion-weighted imaging and contrast-enhanced T1WI/MPRAGE.

Note: The reader is also encouraged to read about the desmoplastic medulloblastoma seen in young adults which presents more laterally, sometimes close to the cerebellopontine angle cistern, and exhibiting cysts. Part of this tumor can demonstrate diffusion restriction. Enhancement is only minimal. Imaging features of desmoplastic medulloblastoma are therefore distinct from those of classic medulloblastoma.

Primitive Neuroectodermal Tumor and Atypical Teratoid-Rhabdoid Tumors

These tumors are typically seen in infancy. In fact, atypical teratoid-rhabdoid tumor is the # 1 diagnosis to consider in a new born with an intracranial mass [22]. Also, in the first 2 years of life, a mass lesion in the brain demonstrating diffusion restriction and enhancement should strongly suggest the diagnosis of primitive neuroectodermal tumor [23].

Best sequence(s) to evaluate primitive neuroectodermal tumor: Diffusion-weighted imaging, FLAIR, and contrast-enhanced T1WI/MPRAGE.

Other Intra-axial Brain Tumors

Primary CNS Lymphoma

Primary CNS lymphoma is of the non-Hodgkin's type. It is more commonly seen in the fifth to sixth decades of life. Predisposing factors include immunodeficient states such as the AIDS population and other immunocompromised settings such as in transplant patients. It can sometimes also be seen in immunocompetent patients. The imaging appearance for both these substrata of patients is different.

Characteristic imaging findings: Immunocompromised patients: Periventricular region is a favored site. A peripherally enhancing centrally necrotic lesion with surrounding edema is seen. No diffusion restriction is seen from the centrally necrotic component of the lesion. Contiguous subependymal spread is commonly seen. Multiple lesions can be seen. Cortical-subcortical lesions can be seen. Hemorrhage and calcification are uncommon.

Immunocompetent patients: Basal ganglia, thalami, and periventricular white matter are favored locations. Solitary, solid-appearing lesion, demonstrating diffusion restriction and homogenous contrast enhancement, is seen (Fig. 3). Necrosis is occasionally seen [24]. Increased rCBV on perfusion imaging is seen. However, the rCBV values are typically < 4.0, unlike in glioblastoma where they can be higher.

Best sequence(s) to evaluate primary CNS lymphoma: DWI, FLAIR, and contrast-enhanced T1WI/MPRAGE.

Ependymoma

Ependymoma typically is seen in the pediatric population as a posterior fossa (4th ventricular) tumor. However, when it occurs in the adult population, it is seen more often as an intraparenchymal tumor. Heterogenously enhancing lesion is seen. Calcification is common. It is a difficult diagnosis to make considering the nonspecific imaging features and the rarity of its occurrence.

Metastases

Approximately 60 % of new intracranial tumors reported every year are metastatic tumors. Common primary sites include the lung and breast. Other common metastatic tumors to the brain include melanoma and gastrointestinal tumors. Imaging findings are nonspecific and include nodular deposits, large solid enhancing tumors, and peripherally enhancing centrally necrotic lesions. Hemorrhage can be seen. Surrounding edema is often seen. Calcification is uncommon. Some imaging pearls: New enhancing infratentorial tumor in an elderly patient is most likely a metastatic tumor. Also, multiple enhancing lesions at the gray-white matter interface in an appropriate clinical setting are most likely metastatic foci.

Best sequence(s) to evaluate metastases: Contrast-enhanced T1WI/MPRAGE. Perfusion imaging can help distinguish metastatic tumors from primary brain tumors [9].

Tumor Mimics

This category includes multiple etiologies including inflammatory, infectious, and vascular conditions. Also included are normal variants such as Virchow-Robin spaces. Treatment-related changes such as pseudoprogression and pseudoresponse have also been included to complete the discussion. Again, similar to brain tumors, tumor mimics includes an extensive list of underlying etiologies. We will limit our discussion to commonly occurring tumor mimics.

Inflammatory Conditions

While there are multiple etiologies in this subset, we will limit our discussion to demyelinating disease and amyloid angiopathy-related inflammation.

Tumefactive Demyelinating Lesion (TDL)

TDL is one of the most common tumor mimics. In its most classic form, TDL is defined as a single, large (>2.0 cm) lesion in the brain, most often in the periventricular location. There are no other imaging lesions to suggest an underlying demyelinating condition.

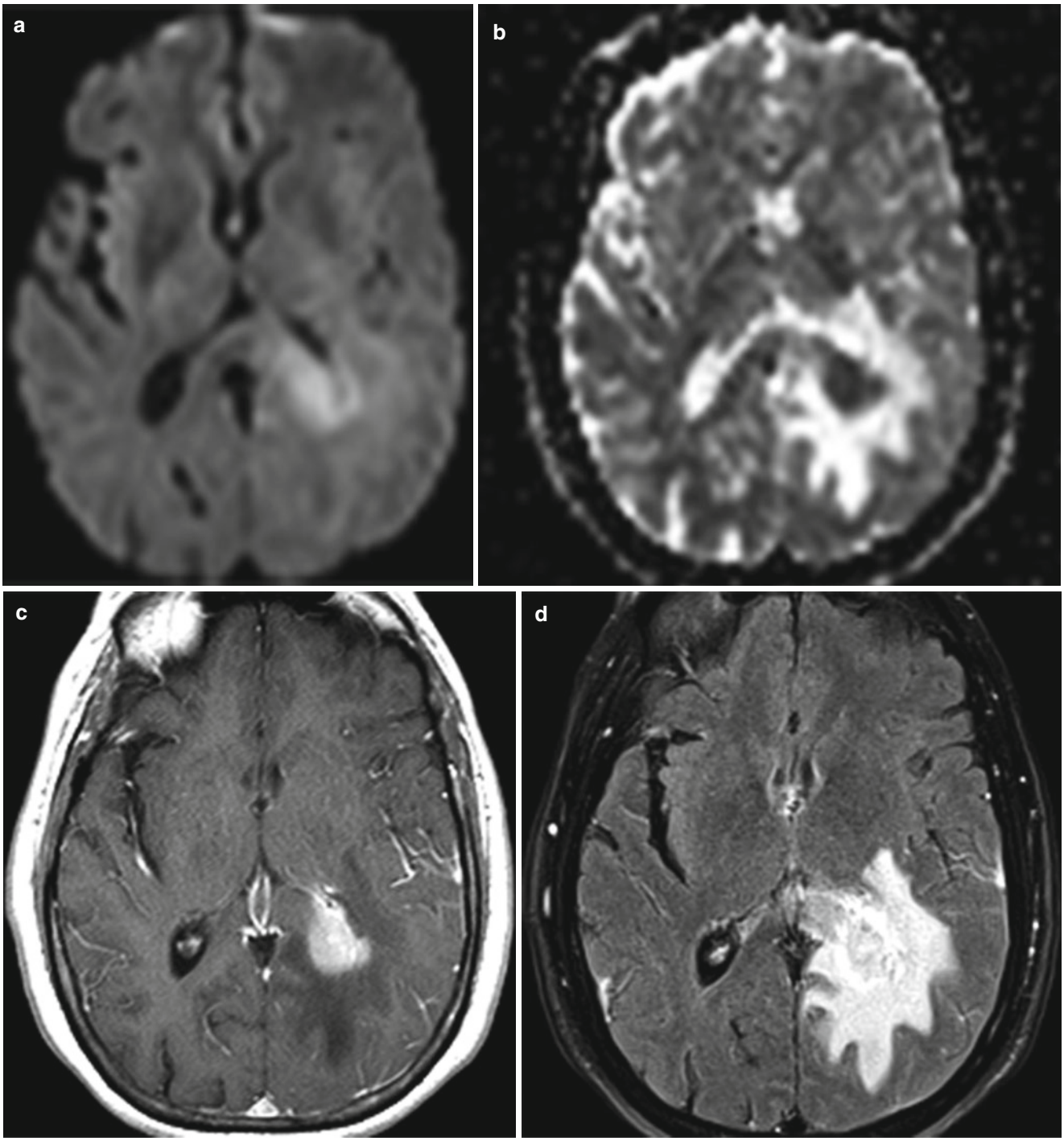

Fig. 3 (**a**) Axial DWI and corresponding (**b**) ADC map confirm diffusion restriction in the left periatrial region and extending into the splenium of corpus callosum. (**c**) Axial FLAIR demonstrates significant surrounding vasogenic edema and mass effect. (**d**) Contrast-enhanced axial T1WI demonstrates homogenous enhancement of the solid-appearing lesion. There is a suggestion of subependymal enhancement. Diagnosis: primary CNS lymphoma

Characteristic imaging findings: Large hypodense lesion on CT which appears hypointense on T1WI and hyperintense on T2WI/FLAIR is seen. Minimal surrounding edema and minimal mass effect, disproportionate to the size of the lesion, is seen. No hemorrhage or calcification is seen. An incomplete ring of enhancement is a hallmark feature of this lesion (Fig 4). This incomplete ring oftentimes corresponds to a band of diffusion restriction. Typically no increased perfusion is seen [25, 26]. In addition on either the perfusion source dataset or a SWI image, venular structures may be seen coursing through the mass lesion.

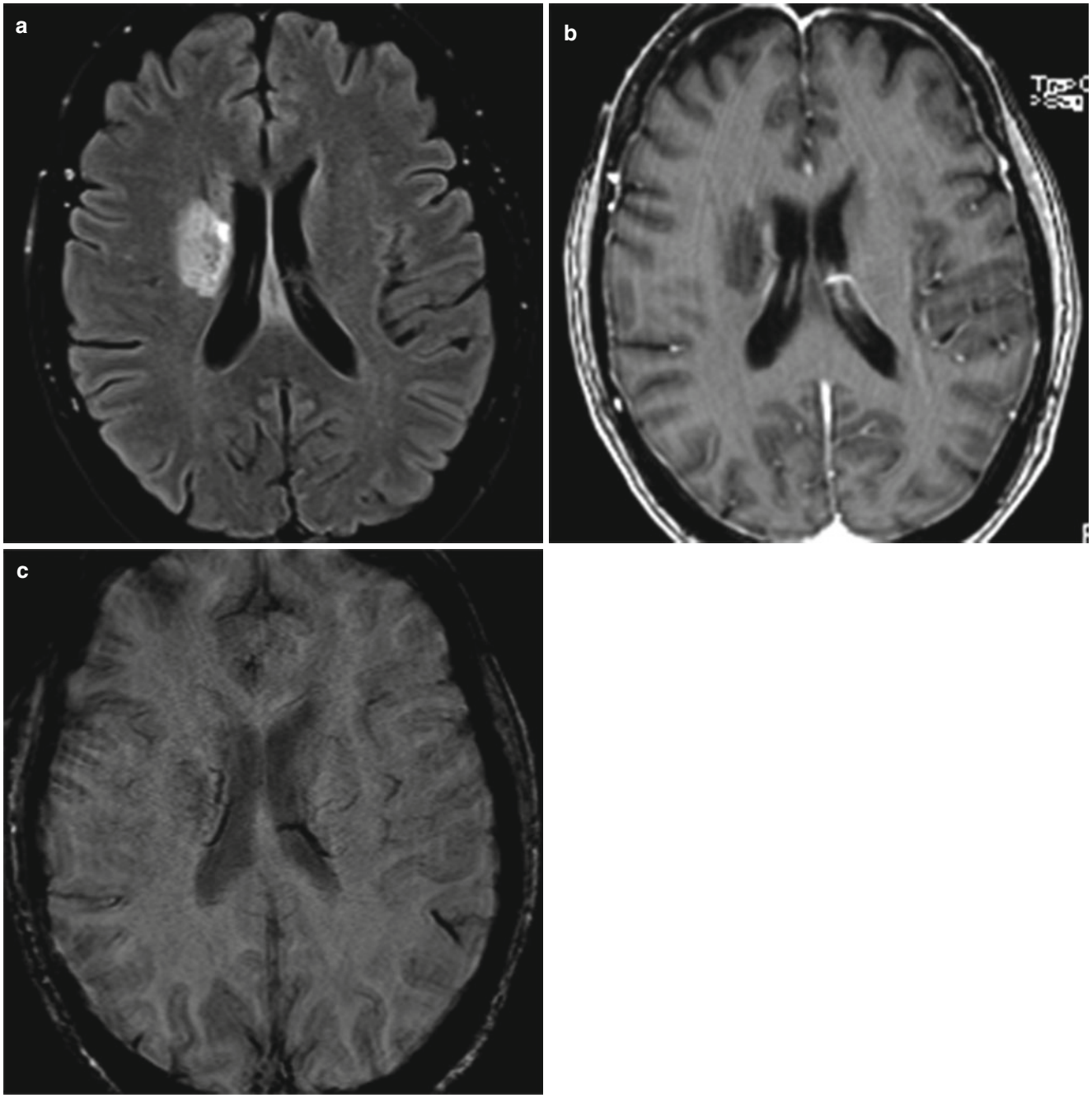

Fig. 4 (**a**) Axial FLAIR demonstrates an area of abnormal signal in the right corona radiata. No surrounding edema is seen. Minimal mass effect is noted. (**b**) Contrast-enhanced axial T1WI demonstrates a peripheral incomplete ring of enhancement. (**c**) Susceptibility-weighted image demonstrates wispy linear susceptibility foci coursing through the lesion. Diagnosis: tumefactive demyelinating lesion

Best sequence(s) to evaluate TDL: T2WI/FLAIR and contrast-enhanced T1WI/MPRAGE.

Amyloid Angiopathy-Related Inflammation

Cerebral amyloid angiopathy is seen in the elderly population. It results from extracellular deposition of amyloid, an amorphous eosinophilic fibrillary protein, in the walls of small- and medium-sized arteries. Occasionally, such deposition causes an inflammatory response in the brain. Patients present with headache, cognitive decline, encephalopathy, seizures, and occasionally focal deficits.

Characteristic imaging findings: Peripherally located foci of susceptibility in an elderly person should raise the possibility of amyloid angiopathy. Focal area of the brain demonstrating FLAIR hyperintense signal in an appropriate clinical setting should suggest amyloid angiopathy-related inflammation. Associated mass effect and subtle enhancement in the overlying leptomeningeal space can be seen [27].

Best sequence(s) to evaluate amyloid angiopathy-related inflammation: Susceptibility-weighted imaging or gradient-echo image, FLAIR/T2WI, and contrast-enhanced T1WI/MPRAGE.

Infectious Etiologies

This primarily includes bacterial, including mycobacterial, and fungal etiologies. Occasionally, parasitic infections can mimic brain tumors.

Abscess

Characteristic imaging findings: A centrally necrotic peripherally enhancing lesion is seen. The central necrotic component demonstrates diffusion restriction due to the inherent viscosity of pus. This diffusion restriction seen from the central necrotic component helps distinguish infection from tumor. There are certain exceptions to the rule which are mentioned below.

Note: Diffusion restriction from mucinous adenocarcinoma metastases can mimic an infection. On the other hand, lack of diffusion restriction from tuberculous abscess can mimic a tumor.

Best sequence(s) to evaluate an abscess: Diffusion-weighted imaging and contrast-enhanced T1WI/MPRAGE.

Encephalitis

Rhombencephalitis or brain stem encephalitis is often associated with infectious or autoimmune disease conditions. Occasionally, it is also associated with paraneoplastic syndromes. In the infectious category, listeria is the most common offending agent.

Characteristic imaging findings: Diffuse abnormal signal involving the brain stem and cerebellum is best appreciated on FLAIR sequences. The abnormal signal when involving the brain stem can mimic the appearance caused by diffuse intrinsic pontine glioma. However, the involvement of the cerebellum (in the presence or absence of involvement of the cerebral periventricular white matter) when seen should suggest the diagnosis of rhombencephalitis. Associated scattered foci of susceptibility reflecting hemorrhage should suggest the diagnosis of listeria encephalitis.

Best sequence(s) to evaluate listeria rhombencephalitis: FLAIR/T2WI, susceptibility-weighted imaging, or gradient-echo imaging.

Vascular Causes

In this basket of vascular causes, we will discuss ischemic and vasculitic processes.

Ischemic processes especially subacute infarction.

Subacute Infarction

Subacute infarction is the classic tumor mimic. The enhancement associated sometimes with subacute infarction is primarily responsible for considering it as a mass-like lesion.

Characteristic imaging findings: The sharply demarcated boundaries of the enhancement area, typically seen to involve a region of arterial branch distribution, should suggest the diagnosis of subacute infarction. The presence of luxury perfusion should also help establish this diagnosis. Also, most such lesions will have a characteristic acute onset of neurologic deficit versus a tumor which has a progressive worsening of focal neurologic deficit.

Best sequence(s) to evaluate subacute infarction: Contrast-enhanced T1WI/MPRAGE and perfusion imaging.

Vasculitic Processes

This will include etiologies such a primary angiitis of central nervous system and Behcet's disease among other vasculitides.

Behcet's Disease

Characteristic imaging findings: Most often the dorsal aspect of the brain stem is involved. Ill-defined hyperintense signal on long TR sequences will be seen. Patchy enhancement is occasionally seen. No diffusion restriction, hemorrhage, or calcification is seen. Imaging features are nonspecific. However, it is the association with characteristic clinical features including aphthous ulcers that helps diagnose this disease condition [28].

Best sequence(s) to evaluate Behcet's disease: FLAIR/T2WI.

Treatment-Related Changes

Pseudoprogression

The Stupp-combined protocol is the standard treatment of care for glioblastoma [29]. Both radiation therapy and temozolomide are toxic to tumor cells. However, at the same time, they incite an inflammatory response in the brain. As a result, the surgical bed on follow-up imaging can demonstrate interval progression in enhancement (due to increased breakdown of blood-brain barrier) and increased FLAIR signal abnormality (inflammatory response). These imaging findings look similar to those seen in tumor recurrence. However, this appearance in fact represents a favorable response to treatment. Hence, though the imaging appearance looks bad, it ideally is not and therefore the term pseudoprogression.

Best sequence(s) to evaluate pseudoprogression: Conventional imaging has no role to play in distinguishing pseudoprogression from tumor recurrence. Advanced imaging can help. Interval decreased rCBV on perfusion imaging

suggests pseudoprogression. In contrast, interval increased rCBV favors tumor progression [30].

Pseudoresponse

Bevacizumab is the standard treatment of care for recurrent glioblastoma. Bevacizumab is an antiangiogenic agent. It stabilizes the blood-brain barrier. Therefore, upon administration of bevacizumab, follow-up imaging often demonstrates reduced enhancement and interval decrease in FLAIR signal abnormality. On conventional imaging, therefore, the imaging findings suggest a good response. However, bevacizumab has no toxic effect on tumor cells. Hence, though imaging suggests a good response, the lack of antitumoral effect in fact allows the tumor to grow along white matter tracts (not visible on conventional imaging) and therefore the term pseudoresponse.

Best sequence(s) to evaluate pseudoresponse: New FLAIR signal abnormality remote from surgical bed should suggest tumor recurrence in a patient with pseudoresponse. Increasing enhancement, increasing FLAIR signal abnormality, and increasing rCBV from the surgical treatment bed also suggest tumor recurrence [30].

References

1. Koeller KK, Rushing EJ (2004) From the archives of AFIP: pilocytic astrocytoma: radiologic pathologic correlation. Radiographics 24:1693–1708
2. Linscott LL, Osborn AG, Blaser S et al (2008) Pilomyxoid astrocytoma: expanding the imaging spectrum. AJNR Am J Neuroradiol 29:1861–1866
3. Crespo-Rodriguez AM, Smirniotopoulos JG, Rushing EJ (2007) MR and CT imaging of 24 pleomorphic xanthoastrocytomas (PXA) and a review of the literature. Neuroradiology 49:307–315
4. Koeller KK, Henry JM (2001) From the archives of AFIP: superficial gliomas: radiologic-pathologic correlation. Radiographics 21:1533–1536
5. Al-Okaili RN, Krejza J, Woo JH et al (2007) Intra-axial brain masses: MR imaging-based diagnostic strategy – Initial experience. Radiology 243:539–550
6. Law M, Yang S, Wang H et al (2003) Glioma grading: sensitivity, specificity, and predictive values of perfusion MR imaging and proton MR spectroscopic imaging compared with conventional MR imaging. AJNR Am J Neuroradiol 24:1989–1998
7. Felsberg GJ, Silver SA, Brown MT et al (1994) Radiologic-pathologic correlation: gliomatosis cerebri. AJNR Am J Neuroradiol 15:1745–1753
8. Spagnoli MV, Grossman RI, Packer RJ et al (1987) Magnetic resonance imaging determination of gliomatosis cerebri. Neuroradiology 29:15–18
9. Cha S, Lupo JM, Chen MH et al (2007) Differentiation of glioblastoma multiforme and single brain metastasis by peak height and percentage of signal intensity recovery derived from dynamic susceptibility-weighted contrast-enhanced perfusion MR imaging. AJNR Am J Neuroradiol 28:1078–1084
10. Jenkinson MD, du Plessis DG, Smith TS et al (2006) Histological growth patterns and genotype in oligodendroglial tumours: correlation with MRI features. Brain 129:1884–1891
11. Koeller KK, Rushing EJ (2005) From the archives of the AFIP. Oligodendroglioma and its variants: radiologic-pathologic correlation. Radiographics 25:1669–1688
12. Engelhard HH, Stelea A, Mundt A (2003) Oligodendroglioma and anaplastic oligodendroglioma: clinical features, treatment, and prognosis. Surg Neurol 60:443–456
13. Giannini C, Burger PC, Berkey BA et al (2008) Anaplastic oligodendroglial tumors: refining the correlation among histopathology, 1p 19q deletion and clinical outcome in Intergroup Radiation Therapy Oncology Group Trial 9402. Brain Pathol 18:360–369
14. van den Bent MJ (2007) Anaplastic oligodendroglioma and oligoastrocytoma. Neurol Clin 25:1089–1093
15. Shin JH, Lee HK, Khang SK et al (2002) Neuronal tumors of the central nervous system: radiologic findings and pathologic correlation. Radiographics 22:1177–1189
16. Castillo M, Davis PC, Takei Y et al (1990) Intracranial gangliogliomas: MR, CT, and clinical findings in 18 patients. AJR Am J Roentgenol 154:607–612
17. Zenter J, Wolf HK, Ostertun B et al (1994) Gangliogliomas: clinical, radiological, and histopathological findings in 52 patients. J Neurol Neurosurg Psychiatry 57:1497–1502
18. Campos AR, Clusmann H, von Lehe M et al (2009) Simple and complex dysembryoplastic neuroepithelial tumors (DNT) variants: clinical profile, MRI, and histopathology. Neuroradiology 51:433–443
19. Stanescu Cosson R, Varlet P, Beuvon F et al (2001) Dysembryoplastic neuroepithelial tumors: CT, MR findings and imaging follow-up: a study of 53 cases. J Neuroradiol 28:230–240
20. Koeller KK, Rushing EJ (2003) Medulloblastoma: a comprehensive review with radiologic-pathologic correlation. Radiographics 23:1613–1637
21. Rumboldt Z, Camacho DL, Lake D et al (2006) Apparent diffusion coefficients for differentiation of cerebellar tumors in children. AJNR Am J Neuroradiol 27:1362–1369
22. Han L, Qiuu Y, Xie C et al (2010) Atypical teratoid/rhabdoid tumors in adult patients: CT and MR imaging features. AJNR Am J Neuroradiol 32:103–108
23. MacDonald TJ, Rood BR, Santi MR et al (2003) Advances in diagnosis, molecular genetics, and treatment of pediatric embryonal CNS tumors. Oncologist 8:174–186
24. Erdag N, Bhorade RM, Alberico RA et al (2001) Primary lymphoma of the central nervous system: typical and atypical CT and MR imaging appearances. AJR Am J Roentgenol 176:1319–1326
25. Cha S, Pierce S, Knopp EA et al (2001) Dynamic contrast-enhanced T2*-weighted MR imaging of tumefactive demyelinating lesions. AJNR Am J Neuroradiol 22:1109–1116
26. Dagher AP, Smirniotopoulos J (1996) Tumefactive demyelinating lesions. Neuroradiology 38:560–565
27. Savoiardo M, Erbetta A, Storchi G et al (2010) Case 159: cerebral amyloid angiopathy-related inflammation. Radiology 256: 323–327
28. Kocer N, Islak C, Siva A et al (1999) CNS involvement in neuro-Behcet syndrome. AJNR Am J Neuroradiol 20:1015–1024
29. Stupp R, Mason WP, van den Bent MJ et al (2005) Radiotherapy plus concomitant and adjuvant temozolomide for glioblastoma. N Engl J Med 352:987–996
30. Fatterpekar GM, Galheigo D, Narayana A et al (2012) Treatment-related change versus tumor recurrence in high-grade gliomas: a diagnostic conundrum – use of dynamic susceptibility contrast-enhanced (DSC) perfusion MRI. AJR Am J Roentgenol 198: 19–26