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REVIEW ARTICLE

Pseudotumoral brain lesions: MRI review

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Abstract Single or multiple space-occupying lesions on brain MRI, with or without contrast enhancement and/or perilesional oedema, evoke a neoplastic origin. However, a multitude of non-neoplastic disorders can simulate cerebral neoplasia. In this review, we will discuss the MRI characteristics of non-neoplastic disorders that can mimic cerebral neoplasia. Distinguishing MRI characteristics are discussed for each of these non-neoplastic disorders.

Keywords Tumoral · Pseudotumoral · Tumefactive · Lesion · MRI

Introduction

Discussing so-called pseudotumoral brain lesions presumes a clear definition of what is called "tumoral". Unfortunately, the radiological term "tumoral aspect" is not well defined. It rather simply refers to the imaging features frequently seen in cerebral neoplasia, namely one or more space-occupying lesions with or without contrast enhancement, perilesional oedema, or central necrosis.

The most frequently encountered parenchymal cerebral neoplasias in adults are neuroepithelial tumors (e.g. astrocytoma, oligodendroglioma, glioblastoma) and metastatic tumors. CNS lymphomas on the other hand typically manifest with little or no mass effect and are generally homogeneous lesions involving the white matter and/or the basal ganglia with strong, diffuse, and homogeneous contrast-enhancement. In immunocompromised patients with CNS lymphomas, only peripheral enhancement can be seen.

In this review, we will discuss the characteristics on standard MRI of non-neoplastic disorders that can mimic cerebral neoplasia. Although often useful, more advanced MRI techniques (e.g. MRI spectroscopy, perfusion-weighted imaging) will not be discussed in detail.

At the end of the review, a short discussion will concern "chameleons", i.e. neoplastic lesions presenting without the typical imaging features of neoplasia that may be confused with more innocuous lesions.

Table 1 shows the typical MRI characteristics of the different entities discussed in this article.

Demyelinating inflammatory lesions

Multiple sclerosis and other idiopathic inflammatory demyelinating disorders

Multiple sclerosis (MS) is characterized by the presence of multiple T2-/FLAIR-hyperintense lesions in the infratentorial, periventricular, deep and/or juxtactortical white matter as well as in the spinal cord [1]. Acute MS lesions typically present with gadolinium-enhancement. MS lesions may mimic neoplasia, especially when lesions are large (often with a mass effect), and/or with contrast-enhancement. In such cases, the terms "tumefactive" or "pseudotumoral" lesion(s) are often used [2, 3]. Lesions are generally larger than 20 mm and hypointense on T1-weighted imaging. Mild

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Table 1 MRI characteristics in different disorders presenting with pseudotumoral lesions

Disorder	U/M	Mass effect	Gadolinium	Gadolinium shape	T1	T2/ FLAIR	Low ADC	Low T2* signal	Localisation	Other suggestive radiological features
MS	U/M	−/+	−/+	Open ring	→/↓	↑	(+)	(+) perilesional		Spinal cord, optic nerve
ITDL	U	−/+	+	−	↓	↑	(+)	−	PV, CC, JC	
ADEM	(U)/ M	−/+	(−)/+/++	−	→/↓	↑	(+)	−	BG, T	Spinal cord
SS	U	−/(+)	−/(+)	−	→/↓	↑	−	−	Cervicomedullary	
NMOSD	U/M	−/(+)	−/+	Cloud-like, ependymal	→/↓	↑	−	−	Periependymal	Spinal cord, optic nerve
Sarcoidosis	U/M	−/(+)	(−)/+/++	−	→/↓	↓/↑	−	−		Leptomeningeal enhancement, spinal cord
NB	U/ (M)	−/+	−/+	−	→/↓	↑	−	−	Midbrain, BG, T	
CLIPPERS	M	−/+	+/++	Punctuate, curvilinear	→/↓	↑	−	−	Pons, cerebellum	
CAA-I	U/M	−/+/ (++)	−/(+)	−	→/↓	↑	−	+ (associated CAA)	Corticosubcortical	
Infarction	U/M	−/+/ (++)	−/+	Cortical	↓	↑	+	(+) H	Vascular territories	Arterial occlusion
ICH	U/ (M)	+/++	−/+/(++)	−	↓/↑	↓/↑	+ mixed	+	BG, T, lobar	
PCNSV	U/M	−/+	(−)/+	−	→/↓	↑	(+) I	(+) H		
CVT	U/M	−/+	−/+	−	↓/↑ (H)	↑	(+) I/H	(+) H	Subcortical	Veinous thrombosis
AVM	U	−/+	+/++	−	↓	↓FV/↑	−	+		Dilated veins
Radionecrosis	U/ (M)	+/++	−/+/++	−	↓/↑ (H)	↑	−	(+)	Site max irradiation	
Leukodystrophy	U	–	−/+	−	↓	↑	−	−	Large, symmetrical WM	
Abscess	U/M	−/+/ (++)	+	Closed ring	↓	↑	+	−		
Amyloidoma	U/ (M)	−/(+)	−/+	Linear	↓/↑	↓/↑	−	−	WM	
Gummata	U/M	−/+	+	Homogenous	↓/↑	↑	−	−	Cortex	
PML	U/M	−/(+)	−/(+)	−	↓	↑	(+) rare	(+)	WM	

U unifocal, *M* multifocal, *T1* T1-weighted imaging *T2* T2-weighted imaging, *ADC* attenuated diffusion coefficient map, *MS* multiple sclerosis, *ITDL* isolated tumefactive demyelinating lesion, *ADEM* acute disseminated encephalomyelitis, *NMOSD* neuromyelitis optica spectrum disorder, *NB* Neuro-Behçet, *CLIPPERS* chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids, *CAA-I* cerebral amyloid angiopathy inflammation, *ICH* intracerebral hemorrhage, *PCNSV* primary CNS vasculitis, *CVT* cerebral venous thrombosis, *AVM* arteriovenous malformation, *PML* progressive multifocal leukoencephalopathy, *PV* periventricular, *CC* corpus callosum, *JC* juxtacortical, *BG* basal ganglia, *T* thalamus, *CAA* cerebral amyloid angiopathy, *I* in case of brain infarction, *H* in case of brain hemorrhage, *FV* flow void, *WM* white matter

to moderate perilesional oedema can be encountered. Contrast-enhancement is typical of the open-ring type, as opposed to the closed-ring enhancement frequently seen in high grade neoplasms (Fig. 1). Peripheral restricted diffusion can be observed on diffusion-weighted imaging in some of the lesions, sometimes resulting in a cerebral infarction-like aspect [4]. Tumefactive demyelinating lesions can occur in isolation and are called isolated tumefactive demyelinating lesions (ITDL) (Fig. 2) [5–7]. About one third of the patients with an ITDL develop MS over time.

These idiopathic atypical demyelinating lesions often show no or only mild mass effect. Other atypical inflammatory demyelinating syndromes of the CNS include acute disseminated encephalomyelitis, neuromyelitis optica spectrum disorders (discussed below), Schilder's disease, Balo's concentric sclerosis, and Marburg's MS (not discussed in this review) [8].

An isolated demyelinating lesion involving the brainstem, the cervicomedullary junction or the upper cervical spinal cord is often referred to as solitary sclerosis (SS) [9]. SS lesions typically do not show contrast enhancement or perilesional oedema, but may evoke a neoplasm because of their solitary character.

Acute disseminated encephalomyelitis

Acute disseminated encephalomyelitis (ADEM) is a monophasic post-infectious/vaccination disorder. Radiological features overlap with MS. However, callosal and periventricular lesions are less frequent and thalamic and basal ganglia lesions more frequent in ADEM than in MS [10]. In ADEM, lesions are most often multiple and typically contrast-enhancing (punctuate, diffuse, closed-ring, open-ring, or peripheral). Tumefactive lesions can be seen, with often less mass effect than expected for the size of the lesion [8].

Neuromyelitis optica spectrum disorders

Recent studies in neuromyeltis optica spectrum disorders (NMOSD) showed that cerebral MRI abnormalities are far more frequent than previously reported [11]. The periventricular white matter is the most frequently involved area followed by the corpus callosum (probably explained by the high expression of aquaporin-4 protein along the ventricular surface). Contrast-enhancement can be observed with "cloud-like" parenchymal enhancement, "pencil-thin" ependymal enhancement, and/or leptomeningeal enhancement around the brainstem (Supplementary Fig. 1). Occasionally, large pseudotumoral lesions can be observed [12–14].

Progressive multifocal leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is a rare demyelinating infection of the CNS caused by the John Cunningham-virus [15, 16]. MRI shows asymmetric predominantly subcortical white matter lesions with irregular borders, often involving the frontal lobe (Supplementary Fig. 2). Classically, the absence of mass effect and gadolinium

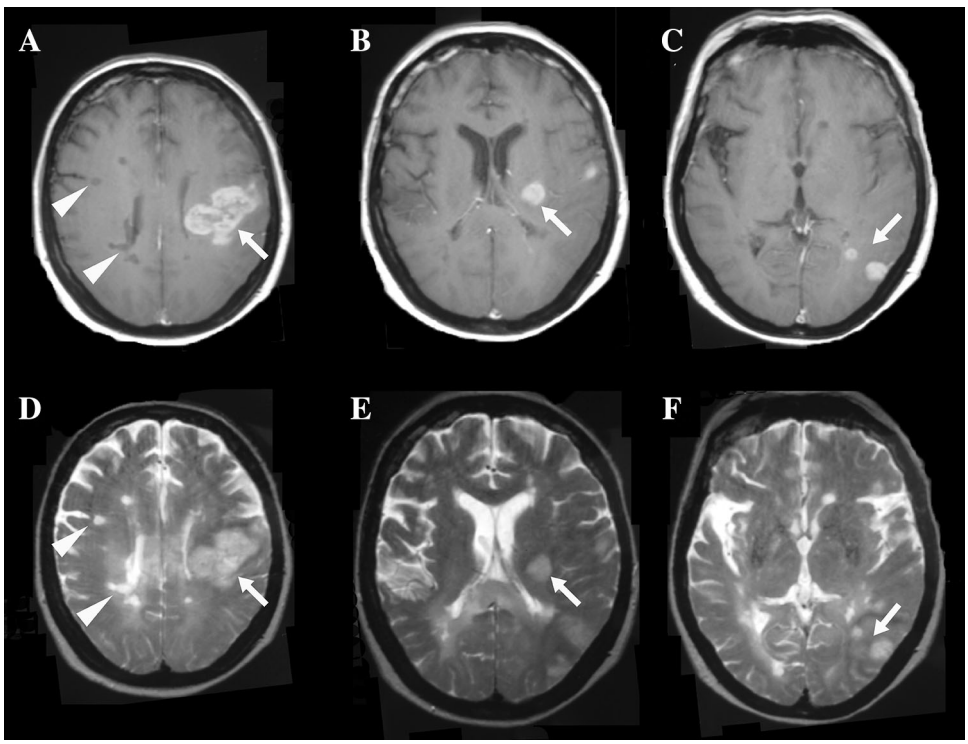

Fig. 1 MRI of an MS patient showing multiple relatively large pseudotumoral enhancing lesions on gadolinium enhanced T1-weighted imaging (*arrows*, **a**–**c**) in the absence of perilesional oedema. These lesions are also seen on T2-weighted imaging (*arrows*, **d**–**f**), together with more classical MS lesions (seen as hyperintensities on T2- and as hypointensities on T1-weighted imaging, *arrowheads*)

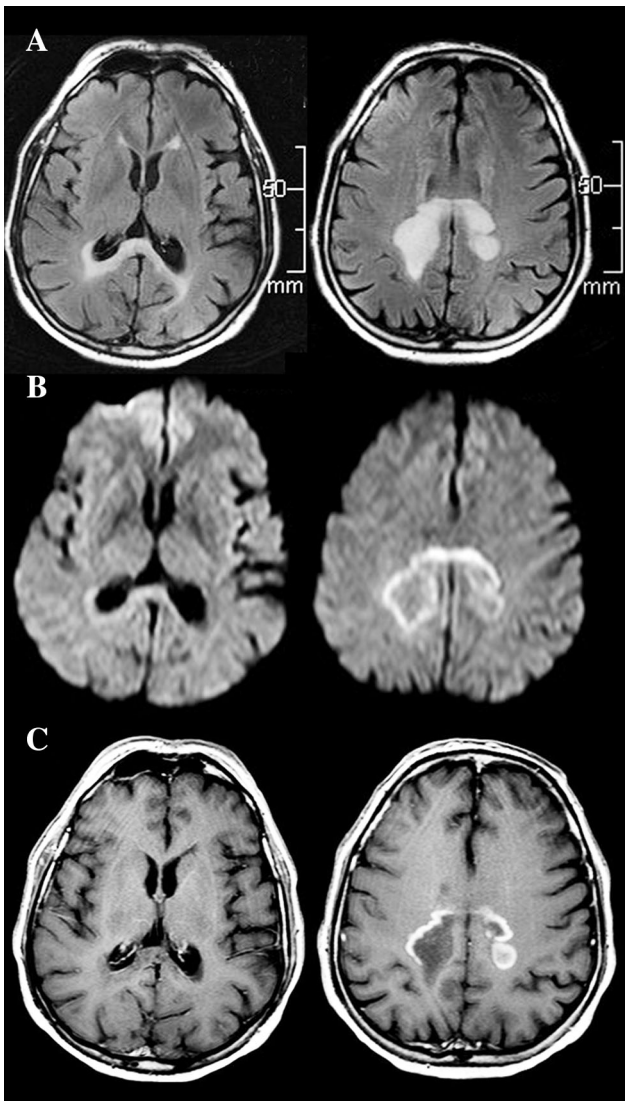

Fig. 2 MRI of a patient with a bilateral periventricular lesion (involving also the splenium of the corpus callosum) ITDL, hyperintense on FLAIR sequences (**a**), presenting with hyperintense borders on DWI (**b**), and with peripheral enhancement on gadolinium-enhanced T1-weighted imaging (**c**)

enhancement does not evoke a neoplastic lesion (except for rare CNS lymphoma without contrast enhancement). However, the MRI pattern in natalizumab-related PML may be different, presenting more often with cortical involvement and the presence of gadolinium enhancement [16].

Non-demyelinating non-infectious inflammatory lesions

Neurosarcoidosis

Multiple or solitary parenchymal mass lesions or granulomas can be observed in neurosarcoidosis [17–19]. These lesions are often associated with leptomeningeal involvement, and typically show enhancement (Supplementary Fig. 3). Central necrosis is usually absent. Lesions are typically T1 isointense and T2 hypo- or hyperintense. Radiological abnormalities, especially contrast enhancement, improve after immunosuppressive treatment.

Neuro-Behçet

The characteristic neuro-Behçet lesion is a T2/FLAIR hyperintense (and T1 hypo- or isointense) upper brainstem lesion extending (most often unilaterally) into the thalamus and the basal ganglia and showing gadolinium-enhancement (Supplementary Fig. 4) [20, 21]. Restricted diffusion can sometimes be encountered. Lesions typically regress after immunosuppressive therapy. Chronic neuro-Behçet is characterised by small white matter lesions, often without contrast enhancement, that are sometimes difficult to differentiate from MS.

Histiocytosis

Intra-axial brain masses can occur with all types of histiocytosis, often with the presence of gadolinium enhancement [22]. Mass effect and perilesional oedema may be present in association with large lesions [23]. Associated involvement of the meningeal structures and the hypothalamic–pituitary axis is often observed.

Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids

Characteristic abnormalities in chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) are multiple small, punctuate and curvilinear gadolinium-enhancing lesions peppering the brainstem (Supplementary Fig. 5) [24]. Associated lesions in the cerebellum, the spinal cord, and the cerebral hemispheres are relatively frequent. Mass effect is usually absent or only mild. Contrast-enhancing lesions typically improve dramatically (at least transitorily) after corticoid treatment.

Cerebral amyloid angiopathy-inflammation

Two pathological subtypes have been described: one with a perivascular non-destructive inflammatory infiltration (so-called cerebral amyloid angiopathy-related inflammation, CAA-RI), and the second with vasculitic transmural, often granulomatous, inflammatory infiltrate (Aβ-related angiitis, ABRA). Both identities are clinically and radiologically difficult to distinguish and, therefore, often simply called cerebral amyloid angiopathy-inflammation (CAA-I).

In CAA-I, patchy or confluent T2/FLAIR hyperintensities associated with pre-existing CAA-related hemorrhagic manifestations on susceptibility-weighted MRI sequences are typically observed [25]. T2/FLAIR white matter hyperintensities are uni- or multifocal (asymmetric), corticosubcortical or deep, and extending to the immediately subcortical white matter (Fig. 3) [26]. Mass effect is often present in association with large lesions [27]. Parenchymal and/or meningeal contrast enhancement is often observed [28]. T2/FLAIR hyperintensities are often seen in the vicinity of the associated CAA-related hemorrhagic lesions [29]. T2/FLAIR hyperintensities improve dramatically with immunosuppressive treatment.

Non-demyelinating infectious inflammatory lesions

Abscess

Both abscesses and high grade neoplastic lesions can present with space-occupying lesions, contrast (often closed ring) enhancement, and perilesional oedema (Fig. 4). Imaging findings in abscesses vary with the stage of abscess development. The most interesting MRI sequence for differentiating abscess from neoplasia is diffusion-weighted imaging, which generally shows a lack of restricted diffusion in primary or metastatic neoplasm and markedly restricted diffusion in pyogenic abscesses [30]. However, restricted diffusion is often less severe or even absent in non-pyogenic abscesses (e.g. toxoplasmosis). After gadolinium administration, neoplastic lesions often show thick, nodular wall enhancement whereas abscesses often show a thin-walled enhancing rim in the early phase (evolving to enhancement of a thickened wall in the late phase associated with cavity collapse).

Neurosyphilis

Brain gummas in gummatous neurosyphilis are a manifestation of tertiary syphilis, typically located in the cortex, arising from the dura and the pia mater [31, 32]. Cerebral gummas are hypo- or isointense on T1- and hyperintense on T2-weighted imaging. Gadolinium enhancement of the gummas (most often homogeneous) and the adjacent meningeal structures are classically observed. Since neurosyphilis is often seen in immunocompromised (often HIV-positive) patients, other disorders potentially presenting with pseudotumoral lesions may also be evoked (e.g. abscess, lymphoma).

Vascular lesions

Brain infarction

Large brain infarctions often present with mass effect. Superficial gyral contrast enhancement is frequently observed in the subacute phase (Supplementary Fig. 6). In general, the diagnosis of acute infarction is straightforward based on the presence of restricted diffusion. In the case of progressive, recurrent, or chronic brain ischemia, however, lesions often show a mix of acute, subacute, and chronic infarction making diagnosis sometimes difficult (Supplementary Fig. 7). The most useful MRI feature to help in the diagnosis of infarction in these cases is restricted diffusion (at least in a portion of the brain lesion), the lack of mass effect (and rather the presence of atrophy when associated with more chronic ischemia), and the presence of an underlying arterial stenosis/occlusion.

Another difficulty occurs when an infarction is located in a clinically silent area and discovered fortuitously in the subacute or early chronic phase (when an ADC map, showing iso- or hyperintense signal, does not confirm restricted diffusion) in the presence of contrast enhancement and/or mass effect.

Intracerebral hemorrhage

Intracerebral hemorrhage (ICH) can be caused by an underlying neoplasm, but, depending on the timing of imaging, can also be confused with a neoplasm. In case of an underlying neoplasm, the primary lesion can often be suspected when imaging is performed in the (hyper) acute phase due to the presence of strong contrast enhancement, mass effect, and/or perilesional oedema, an atypical ICH location, or lack of evident cause/risk factor for ICH. In the acute phase of ICH, contrast enhancement-except for the so-called 'spot-sign' (i.e. uni- or multifocal contrast enhancement within the ICH, associated with hematoma expansion) usually lacking.

Brain tumors associated with hemorrhage include pituitary adenoma, oligodendroglioma, glioblastoma multiforme, medulloblastoma and metastatic tumors (e.g. malignant melanoma, renal cell carcinoma, choriocarcinoma, bronchogenic carcinoma, and germ cell tumors).

When MRI is performed in the subacute phase, however, ICH might mimic neoplasia, due to the development of reactive contrast-enhancement, progressive perilesional edema and associated mass effect (Supplementary Fig. 8).

Primary CNS vasculitis

In primary CNS vasculitis (PCNSV), multifocal arterial wall irregularities (most often in the distal cerebral arteries) are classically seen on a digital subtraction angiogram, associated with (often multiple) brain infarction and/or hemorrhage. In a minority of PCNSV patients, a gadolinium-enhancing mass lesion (mimicking a neoplasia) can be observed [33–36]. In case of a pseudotumoral lesion, associated infarction/hemorrhage and radiological

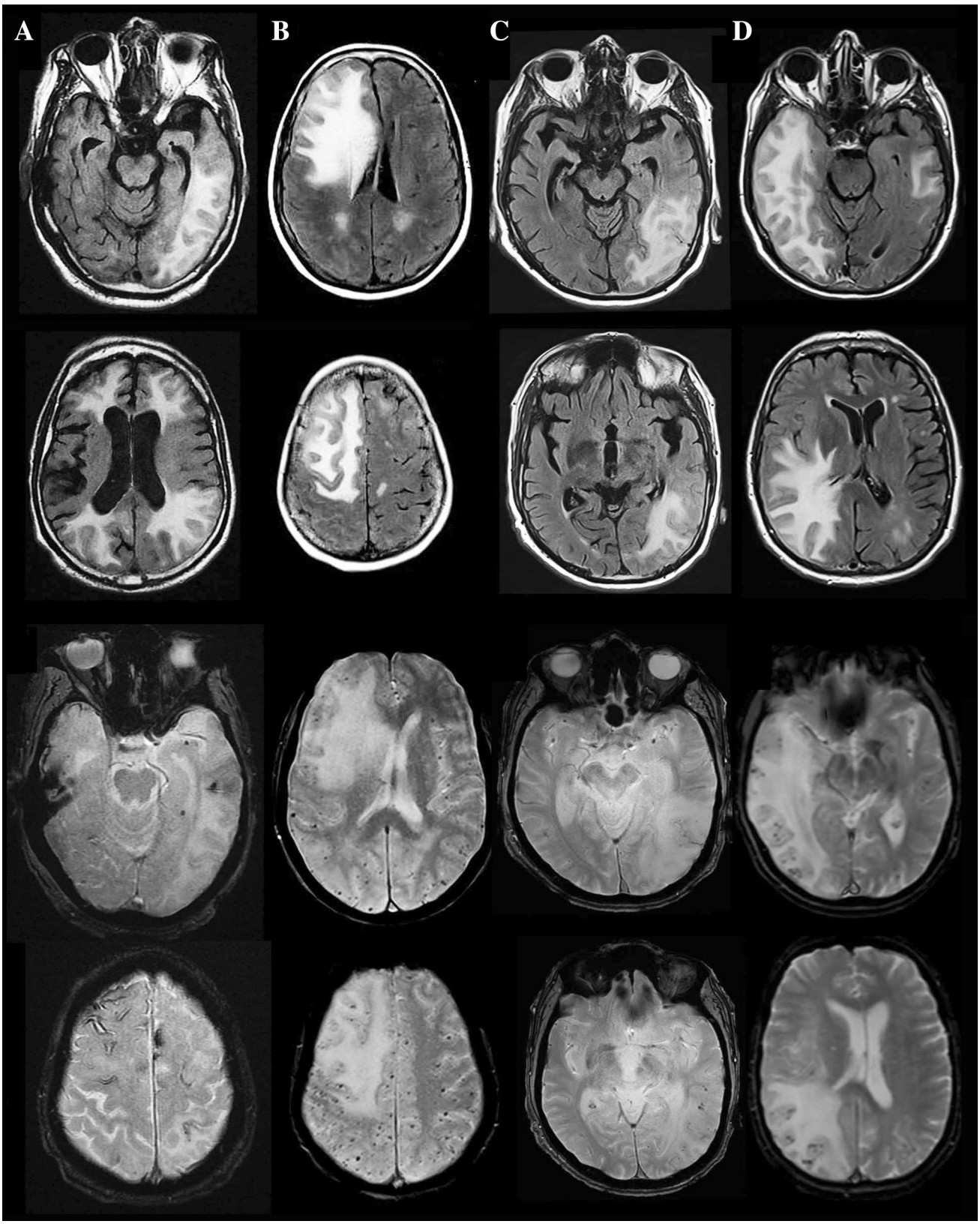

Fig. 3 MRI of 4 different CAA-I patients (with each *vertical row* corresponding to a different patient) showing uni or multifocal corticosubcortical FLAIR hyperintense lesions, often associated with mass effect (two *horizontal upper rows*). On T2*-weighted imaging, associated hypointense cortical superficial siderosis and/or cortical microbleeds can be observed (two *horizontal lower rows*)

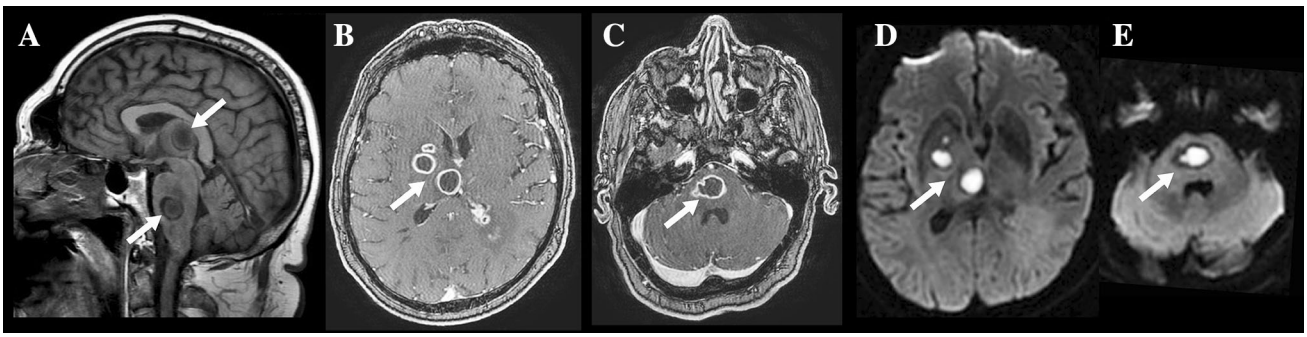

Fig. 4 Multiple staphylococcus epidermidis abscesses can be observed in the basal ganglia and the brainstem, seen as hypointensities on T1-weighted imaging (**a**), with thin-walled closed ring enhancement on gadolinium-enhanced T1-weighted imaging (**b**, **c**), and with restricted diffusion on DWI sequences (**d**, **e**)

improvement under immunosuppressive therapy favour the diagnosis of PCNSV.

Venous infarction

Cerebral venous thrombosis (CVT) can be complicated with venous infarction, typically manifesting with a mixture of cytotoxic and vasogenic oedema and/or hemorrhage (Supplementary Figs. 9 and 10). The MRI signal intensity of the venous thrombus itself varies according to the age of the thrombus. CT venography, or TOF or gadolinium-enhanced MR venography are the most frequently used techniques to detect CVT. CVT, due to the presence of infarction/hemorrhage-related mass effect and frequently encountered gadolinium-enhancement, can sometimes mimic neoplasia [37]. Careful examination of the MRI appearance of venous structures and the use of specialized MRI techniques improve the recognition of CVT. The presence of bilateral paramedian radiological abnormalities should evoke the possibility of a CVT of median-located venous structures.

Arteriovenous malformation

An arteriovenous malformation (AVM) can sometimes mimic a neoplasia due to the presence of strong gadolinium enhancement and occasional mass effect. MRI arguments in favour of AVM are the presence of flow voids on T2-weighted imaging (representing fast-flow vessels) (Supplementary Fig. 11). FLAIR hyperintensities surrounding the AVM (representing gliosis) may sometimes be difficult to distinguish from perilesional oedema (in case of suspected neoplastic lesions).

Cerebral cavernous malformation

Multiple cerebral cavernous malformations (often familial due to genetic disease) can mimic hemorrhagic cerebral metastases as they both present with multiple T2* hypointense lesions on MRI (Supplementary Fig. 12). CT can help to distinguish both entities since cavernous malformations often show calcification. On MRI, cavernous malformations often show mixed signal intensity (due to chronic and recent hemorrhage and associated calcification). In contrast with metastasis, cavernous malformations generally do not enhance. Gadolinium enhancement can be sometimes difficult to interpret, however, due to pre-existing T1-hyperintensity caused by subacute blood-breakdown products on unenhanced imaging.

Radionecrosis

Radionecrosis (RN) is an important differential diagnosis to consider in patients with a history of cerebral neoplasm treated with radiation therapy and the presence of a new brain lesion showing gadolinium enhancement. RN typically occurs at the site of the maximum irradiation dose (with the white matter particularly vulnerable to the secondary ischemic consequences of post-irradiation vasculopathy). The type/aspect of gadolinium enhancement in RN is variable. Hemorrhagic changes (seen as decreased signal on T2*-imaging) are often present in RN. The mass effect in RN is often relatively limited for the size of the lesion, provided no extensive vasogenic edema is present (Fig. 5). Overall, the morphological features of RN lesions are very similar to those of a recurrent or progressive tumor. Involvement of the corpus callosum with multiple enhancement sites or subependymal contrast enhancement is in favour of tumor recurrence [38]. Perfusion-weighted MR imaging (PWI) may help to distinguish RN from tumor recurrence since higher levels of neoangiogenesis are present in tumor recurrence than in RN.

In case of cerebral metastasis treated by radiation therapy, a severe increase in lesion volume post-radiation is in favour of recurrence or continued activity (rather than RN, most often showing only a modest increase in volume) whereas the presence of the so-called T1/T2 mismatch (referring to the correlation between the boundaries of the

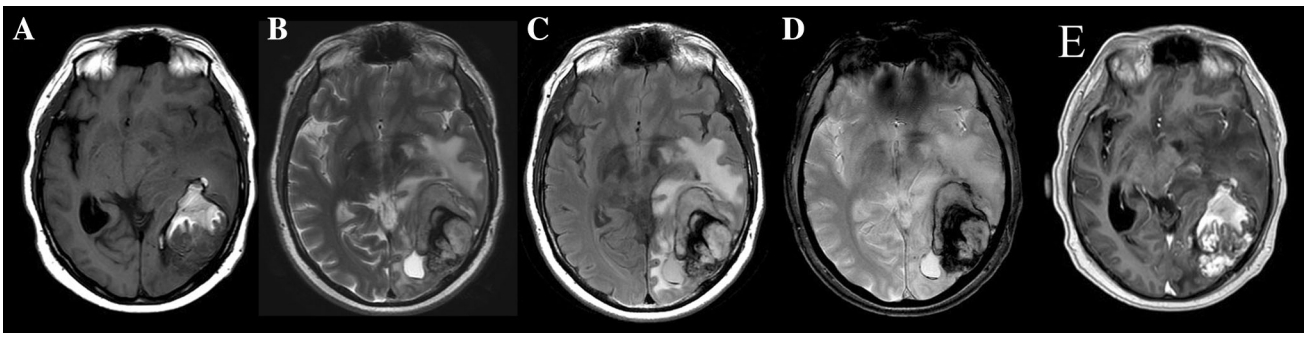

Fig. 5 Radionecrosis several years after gamma-knife treatment for a small cavernomatous malformation seen as a large spaceoccupying temporo-occipital lesion a with mass effect and perilesional oedema (**a** T1-weighted imaging, **b** T2-weighted imaging, **c** FLAIR imaging, **d** T2*-weighted imaging, **e** gadolinium-enhanced T1-weighted imaging). A subacute hemorrhage can be seen as hyperintensity on T1-weighted imaging (**a**) and as hypointensity on T2*-weighted imaging (**d**). Contrast enhancement can be observed in a part of the lesion after gadolinium injection (**e**)

lesion seen on enhanced T1-weighted imaging and T2-weighted imaging, with correlation reflecting tumor recurrence and non-correlation reflecting RN) is in favour of RN.

The most frequently used nuclear medicine imaging technique to distinguishing RN from tumor recurrence/progression is 18F-fluorodeoxyglucose positron emission tomography (FDG-PET) showing decreased uptake in RN and increased uptake in tumor recurrence/progression. False positive FDG-PET results, however, may be observed with abscesses and from inflammation caused by RN [39].

Leukodystrophy

Leukodystrophy is a group of genetic disorders characterized by white matter involvement (typically seen as large bilateral confluent FLAIR hyperintensities in the white matter), classically without mass effect, contrast-enhancement, or perilesional oedema. Rarely, extensive contrast-enhancement can be observed in these white matter lesions [40–42]. For instance, several cases of X-linked adrenoleukodystrophy with severe contrast-enhancement (correlated with disease progression) have been reported [42]. In Alexander disease and Krabbe leukoencephalopathy, gadolinium enhancement has also been reported. In cases of leukodystrophy associated with gadolinium enhancement, the absence of a mass effect and the symmetrical character of the lesions on MRI and the presence of hypometabolism on FDG-PET speak against neoplasm [43].

Amyloidoma

Localized amyloid masses, called amyloidomas, are very rare [44]. Brain amyloidoma can be single or multiple, mainly involve the white matter, and are most often hyperdense on CT. These lesions may mimic neoplasm since gadolinium enhancement (often linear) is often observed in these lesions on MRI. Oedema and mass effect, however, are frequently absent (Supplementary Fig. 13). A mixed signal has been described on T1- and T2-weighted imaging.

Gliomatosis cerebri

In gliomatosis cerebri, diffuse T2/FLAIR-hyperintense white matter infiltration involving two or more lobes with associated brain swelling is seen. Absent (or minimal) enhancement on gadolinium-injected T1-weighted imaging is typical. Involvement of the cortex, corpus callosum, and basal ganglia is often observed. Lesions typically progress over time. In early stage disease, when white matter involvement is often absent, cortically located signal changes are sometimes difficult to distinguish from focas cortical dysplasia, seizure-related cortical signal changes, reversible posterior leukoencephalopathy syndrome, encephalitis, Creutzfeldt Jakob disease, and mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS).

When gliomatosis cerebri is located in the mesial part of the temporal lobe(s), radiological abnormalities can mimic those seen in autoimmune/paraneoplastic or infectious encephalitis.

Non-enhancing brain lymphoma

Most frequently, CNS lymphomas present with homogeneous lesions involving the white matter (and/or the basal ganglia) with strong, diffuse, and homogeneous contrast-enhancement, with no or only discrete mass effect. In immunocompromised patients, enhancement is often peripheral. A minority of CNS lymphoma patients do not show gadolinium enhancement [45, 46]. In these patients, other non-neoplastic disorders often presenting with

confluent predominant white matter lesions in the absence of mass effect and gadolinium enhancement can be evoked, including reversible posterior leukoencephalopathy syndrome, PML, and demyelinating lesions.

Conclusion

Although differentiating neoplastic from non-neoplastic lesion is often straightforward, knowledge of radiological characteristics of more rare pseudotumoral presentations of non-neoplastic lesions may help to distinguish both entities to perform further appropriate examinations and to start treatment without delay when needed.

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Compliance with ethical standards

Conflict of interest We have no conflict of interest to declare.

Ethical standards This review was performed with the approval of the institutional review board of the hospital of CHU Nîmes.

Informed consent Informed consent was obtained from all individual participants included in the review.

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