

Imaging Spectrum of Periventricular Lesions of Brain in Adults

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ABSTRACT

Evaluation of periventricular lesions of the brain on magnetic resonance imaging is always difficult because the cause of these lesions varies considerably. the correct analysis of these lesions, especially the exact location, the characteristics of the signal on different sequences, the enhancement pattern makes it possible to identify a correct cause of these lesions. In this article we will identify a various common and a few uncommon diseases presenting as peri-ventricular white matter lesions in which imaging findings can allow. This is a literature review of peri-ventricular lesions of brain seen in adults with illustrating figures from radiology department at Hassan II^d University hospital in Fez, Morocco.

Keywords: Adults, Brain, Imaging, Peri-ventricular, Spectrum.

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INTRODUCTION

Evaluation of periventricular lesions of the brain on magnetic resonance imaging is always difficult because the cause of these lesions varies considerably. the correct analysis of these lesions, especially the exact location, the characteristics of

the signal on different sequences, the enhancement pattern makes it possible to identify a correct cause of these lesions. In this article we will identify a various common and a few uncommon diseases presenting as periventricular white matter lesions in which imaging findings can allow the radiologist to limit the differential diagnosis.

VASCULAR LESIONS

Lacunar Infarcts

Lacunar infarcts are cavitating small vessel deep infarcts less than 1.5 cm [1]. They are commonly asymptomatic [2]. For a long time, lacunar infarctions were considered to be secondary to an intrinsic disease of the wall of small vessels, called "lipohyalinosis", Related to hypertension and diabetes hypertension and diabetes. However, this "lacunar hypothesis" is controversial because lacunar infarcts concerned 50% of normotensive subjects [1]. Lacunes are now believed to result

from focal ischemic infarction secondary to thrombi or emboli of small vessels in a context of diffuse atherosclerotic narrowing[3]. MRI is used as the diagnostic gold standard for acute and chronic lacunar infarctions. Acute lacunes appear as small lesions of the deep white matter with restriction of diffusion, while chronic lacunes are hyper-intenses on T2 and hypo-intenses with higher signal intensity than CSF on FLAIR images (**Fig.1**). A common differential diagnosis includes Virchow-Robin space, which signal is identical to that of cerebrospinal fluid on all MRI sequences [1].

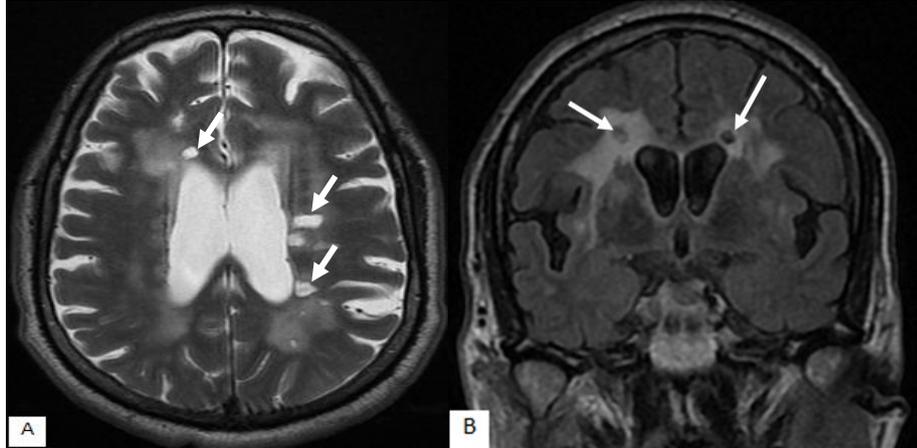


Fig 1. Axial T2w (A) and FLAIR (B) images of a 63 year old man showing multiple chronic lacunar infarcts which are hyper-intense on T2w and hypointense on FLAIR (arrows). Note also the presence of T2w/FLAIR hyper-intensities in the periventricular white matter due to age related changes

Virchow Robin Spaces

Perivascular or Virchow-Robin spaces (VRSs) are perivascular spaces that surround the perforating arteries that enter the brain -usually bilateral and symmetrical-. Lenticulostriate arteries entering the basal ganglia and perforating medullary arteries that enter the cortical grey matter are the most common

sites. They are also found in subinsular region, dentate nuclei and cerebellum [4]. On MRI the perivascular spaces often appear as clusters of round to oval fluid filled cysts with smooth margins, characteristically not exceeding 5 mm diameter. The perivascular spaces follow CSF signal on all pulse sequences (Fig. 2).

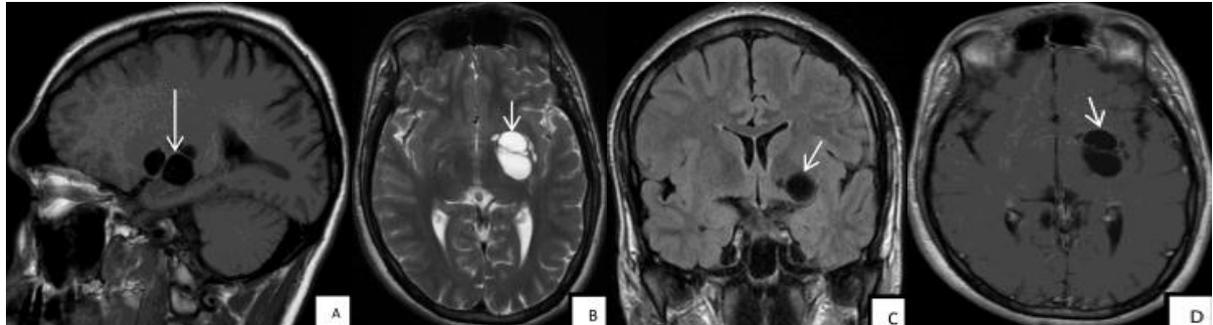


Fig 2. 22 years old woman with seizures. Sagittal T1 (A), Axial T2 (B), coronal FLAIR (C) and Contrast-enhanced T1-weighted images (D) show a purely intra-parenchymal fluid lesion without enhancement, located at the anterior left perforated space(arrow): enlargement of the Virchow-Robin spaces.

CNS Vasculitis

Cerebral vasculitis are a heterogeneous group of CNS diseases that include inflammation of the walls of variable-sized blood vessels; with or without necrosis[5]. Symptoms of cerebral vasculitis may include focal neurological deficit, psychiatric symptoms, and cognitive impairment. When the cerebral symptoms are part of a systemic disorder, the diagnosis may be easier[6]. MRI is the most commonly used imaging modality in the diagnosis of cerebral vasculitis T2-weighted images allow to detect ischemic or gliotic lesions as

multiple small hyperintensities in the deep white matter and grey matter. FLAIR images (Fig3) increase detection of subarachnoid hemorrhage and ischemic deep white matter lesions. Contrast-enhanced T1-weighted images may show enhancement of the leptomeninges or the thick vessel walls. Enhanced intra-parenchymal lesions can also be seen. DW MR imaging (Fig3) allows us to distinguish acute lesions from chronic ischemic brain lesions[7]. Digital subtraction angiography (DSA) can detect vessel irregularities due to alternating areas of stenosis [7].

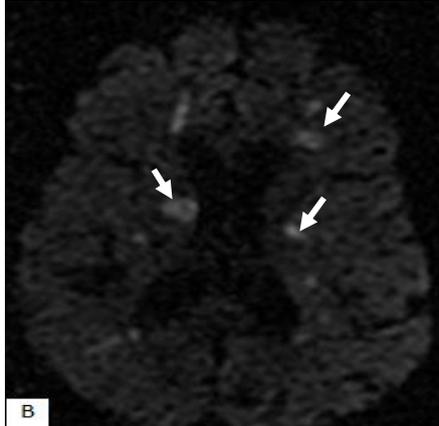


Fig 3. 22 years old man with focal neurological deficit. Axial FLAIRWI (A) shows multiple, nonspecific, small hyperintensities in the deep white matter with restricted diffusion on DWI(B) (arrows).

CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy)

CADASIL is an autosomal dominant vascular disorder. Clinical presentation of CADASIL may include transient ischemic attack (TIA) and stroke, dementia, migraine with aura, primary epilepsy, and psychiatric disorder as depression and psychosis [8, 9]. MRI is the investigation of choice, often demonstrating three types of lesions in patients with CADASIL. The first type is increased signal intensity in T2W and FLAIR images within the periventricular region and in deep white matter, but less frequently in superficial white matter. The most commonly affected regions are the frontal, parietal,

and anterior temporal white matter and the external capsule. Several studies have suggested that both anterior temporal lobe and external capsule involvement allow to differentiate CADASIL from other forms of small-vessel disease (Fig. 4). [8, 9]. The second type is Lacunar infarcts in CADASIL which are located within the semi oval center, thalamus, basal ganglia and pons [10]. Another findings are cerebral micro-hemorrhages found in 45% (range 25-70%) of cases without a characteristic distribution [11]. They are depicted as focal areas of signal loss on T2 imaging, increasing in size in T2*- weighted gradient echo planar images (“blooming effect”), with a diameter smaller than 10 mm [12].

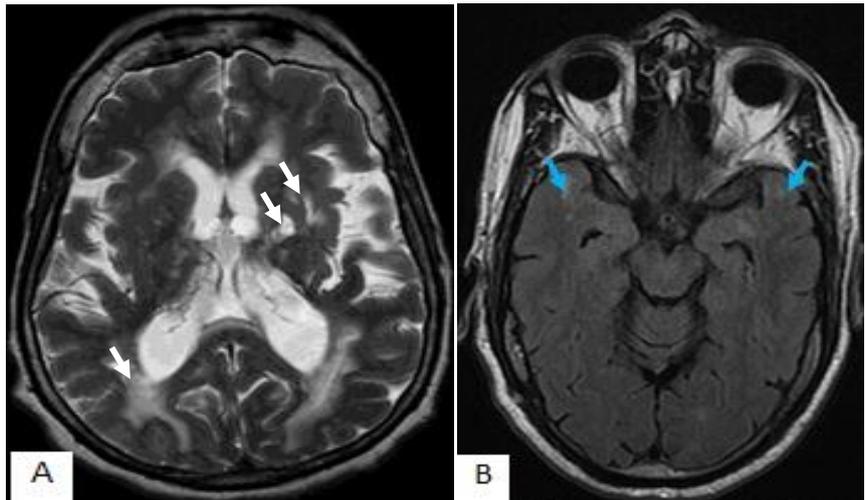


Fig 4. CADASIL in a 59 year old male patient who presented with right hemiplegia. Axial T2 WI (A) shows abnormal diffuse hyper-intensities and chronic lacunar infarcts in periventricular white matter and left external capsules (white arrows), axial FLAIR WI (B) shows increased signal intensity in the anterior part of the temporal lobes (blue arrows)

DEMYELINATING LESIONS

Multiple Sclerosis

Multiple sclerosis (MS) is a chronic, inflammatory, autoimmune disease involving the central nervous system. It is characterized by demyelination and axonal damage. Multiple sclerosis affects young and middle-age adults, but also affects older people[13]. According to the McDonald criteria for MS, the diagnosis requires objective evidence of lesions disseminated in time and space. MRI can show multiple lesions in at least two regions among peri-ventricular, juxta-cortical, infra-tentorial and spinal cord (dissemination in space), some of which can be clinically occult, and MRI can show new lesions on follow up scans (dissemination in time)[14]. The characteristic peri-ventricular lesions of MS are hyperintensities on T2w and FLAIR images. They are ovoid, localized at right angles to the ventricles and are called Dawson fingers (Fig. 5).[13,14].

On T1-weighted imaging (T1WI), the acute lesions of MS are usually iso-intense to the normal white matter, however, if a chronic tissue lesion or severe inflammatory edema occurs, they may be hypointense. The accumulation of hypointense lesions (called black holes) may correlate with disease progression and disability[13]. Contrast-enhanced T1WI is used in daily practice for measuring inflammatory activity of MS in Vivo. In the acute inflammatory phase, MS lesion may show homogeneous nodular, ring or horse shoe like pattern of enhancement, and that can last from days to weeks[13]. Other common locations of Multiple Sclerosis lesions are corpus callosum, subcortical region, U-fibers, temporal lobes, optic nerves, brainstem, cerebellum, and spinal cord. This pattern of involvement is uncommon in other diseases[2, 14].

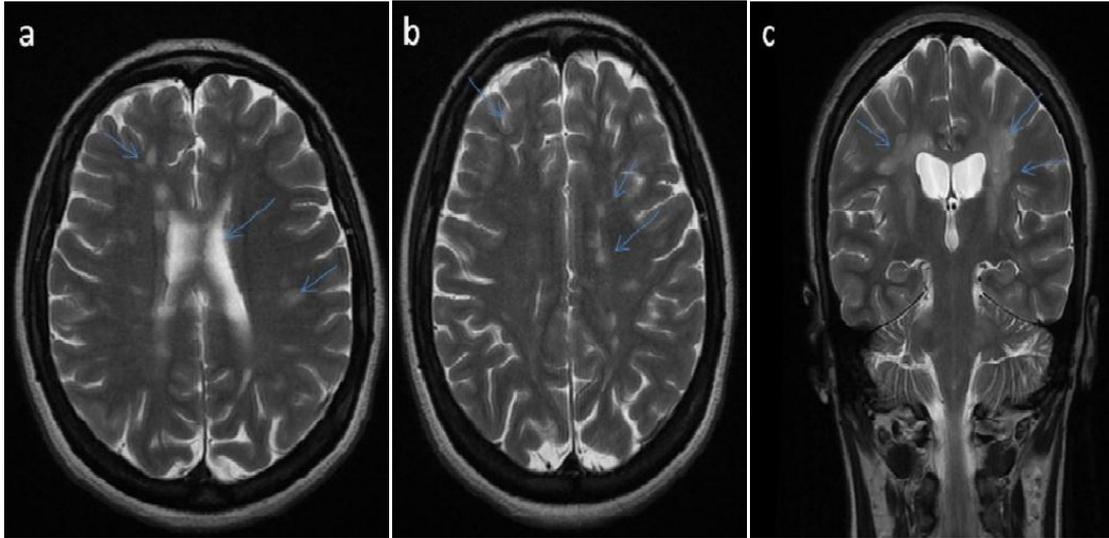


Fig 5. Axial (a,b) and coronal (c)T2-weighted images showing typical ovoid peri-ventricular and juxta-cortical hyperintense Multiple Sclerosis lesions.

INFECTIOUS LESIONS

Toxoplasmosis

Cerebral toxoplasmosis, is an opportunistic infection caused by the parasite *Toxoplasma gondii*. It typically affects patients with HIV/AIDS. Clinical presentation of neurotoxoplasmosis commonly include headache which is often accompanied by altered mental status, fever and seizures[15]. On unenhanced CT images, cerebral toxoplasmosis usually appears as solitary lesion or multiple hypo to iso-attenuating lesions which are usually localized in basal ganglia, corticomedullary

junction, white matter, and periventricular regions. Surrounding vasogenic edema and mass effect are common. Calcification may be seen after therapy. On contrast-enhanced CT images, these lesions may have nodular or ring enhancement which is typically thin and smooth; but No enhancement can also be seen[15, 16]. These lesions are hypointense on T1- weighted MR images, with peripheral hyperintensity, a feature that can differentiate them from lymphoma. On T2-weighted and FLAIR images the lesions usually have mixed intensities. On diffusion-weighted

images, restricted diffusion within the central portion of the lesions (a finding in pyogenic abscesses) is uncommon. On contrast-enhanced T1-weighted images, lesions often demonstrate ring enhancement or nodular enhancement (Fig 6). [15, 16]. The most common differential diagnosis of

cerebral toxoplasmosis in immunocompromised individuals is lymphoma. Compared to toxoplasmosis, lymphomas are locally infiltrative, show diffusion restriction, occur more commonly in periventricular region and show butterfly pattern on imaging [15].

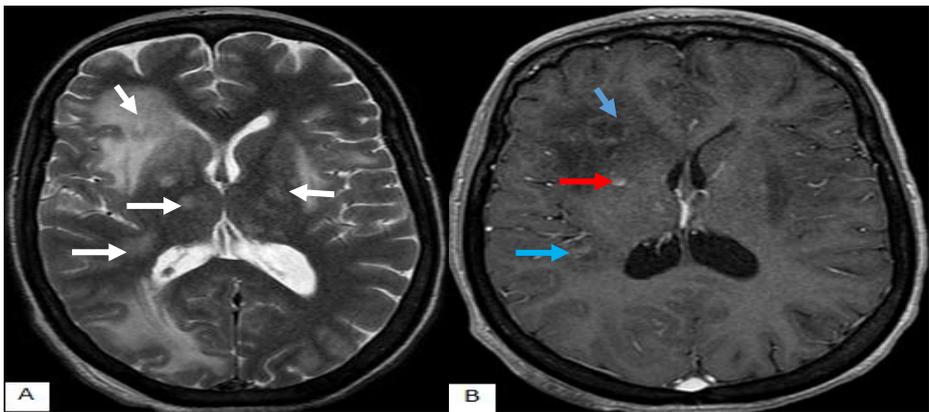


Fig 6. Toxoplasmosis in a 35 year old HIV positive male. Axial T2WI (A) shows multiple hyperintensities with perilesional edema in the periventricular white matter (white arrows) which show ring enhancement (blue arrows) and nodular enhancement (red arrows) on post contrast T1WI (B).

Cryptococcosis

Central nervous system (CNS) cryptococcosis is the third most common cause of CNS infection in AIDS patients which results from infection with the yeast-like fungus *Cryptococcus neoformans*. Clinical presentation is often nonspecific. Patients may present with signs of meningitis or, less frequently, meningo-encephalitis. The imaging findings of cryptococcosis are often minimal. They may consist variably of meningo-encephalitis, intra-ventricular or intra-parenchymal cryptococcomas, gelatinous pseudocysts, or hydrocephalus [18].

The meningeal disease can show leptomeningeal enhancement on contrast enhanced T1-weighted images. Cryptococcomas show low signal on T1-weighted images, high signal on T2 and FLAIR weighted images. On contrast-enhanced T1-weighted images, findings are variable, ranging from no enhancement to peripheral nodular enhancement (Fig 7). Gelatinous pseudocysts are round or oval, appearing similar to cerebrospinal fluid, they are present in the basal ganglia, thalami, midbrain, cerebellum, peri-ventricular regions and tend to give a "soap bubble" appearance [18, 19].

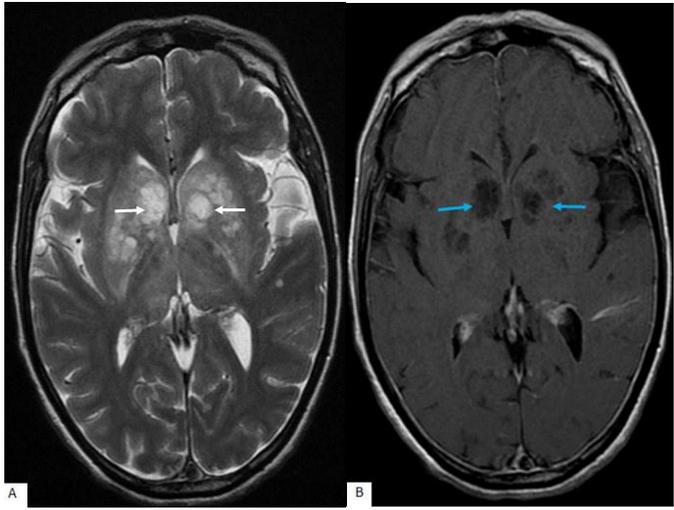


Fig 7. Cryptococcosis in a 35 year old HIV positive male. AxialT2WI (A) shows abnormal focal hyper-intensities in the basal ganglia(white arrows), with no enhancement on post contrast axial T1WI(B) (blue arrows).

Brain abscess

Brain abscess can be widely distributed anywhere in the brain can also affect the periventricular regions, making it difficult to distinguish it from other diseases. Clinical presentation is non-specific with no inflammatory or septic symptoms in several cases. Commonly it may include Symptoms of Increased intracranial pressure, seizures and focal neurological deficits.

MRI is the investigation of choice, usually shows lesions located at the junction of the gray and white matter. These lesions are hypointense on T1-weighted MR images, with peripheral hyperintensity, On T2-weighted and FLAIR images the lesions typically are hyper-intense with slightly low signal thin rim[20]. On diffusion-weighted images, high signal is usually present centrally with low ADC values(Fig 8).[21, 22]. On contrast-enhanced T1- weighted images, lesions often demonstrate ring enhancement [22].

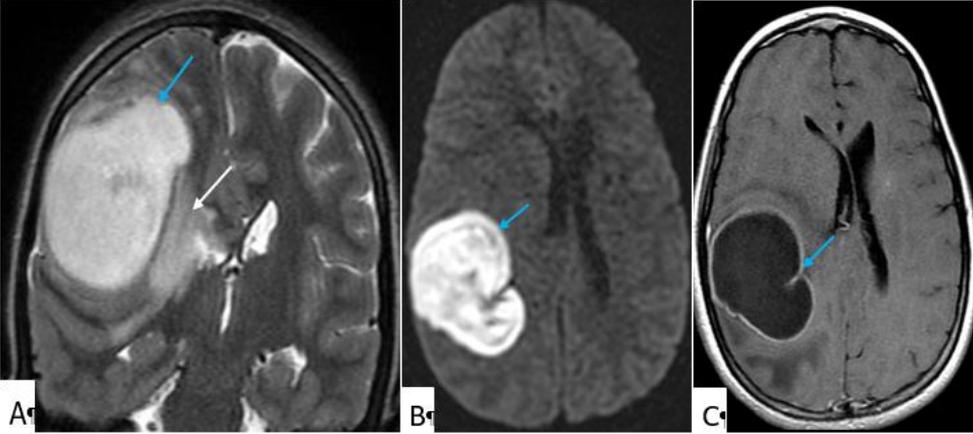


Fig 8. Brain abscess in a 39 year old male patient who presented with right hemiplegia.A, T2-weighted image shows a hyper-intense in the right parietal region (blue arrow)with peri-focal edema(white arrows).B, DW images shows a hyper-intense lesion indicating restricted diffusion due to pus (blue arrow).C, Axial contrast-enhanced T1-weighted image shows a ring-enhancingof the lesion (blue arrow)

TUMOR

Primary central nervous system lymphoma

Primary central nervous system lymphomas (PCNSL) are uncommon tumours, accounting for only 2% of primary brain tumors [23], and most of them are diffuse large B-cell lymphomas [24]. Presentation can be with focal neurological deficits or features of increased intracranial pressure[23]. PCNSL in immuno-competent individuals occur as solitary or multiple lesions usually located at supratentorial white matter (frontal and parietal regions), and the periventricular regions, infiltrating the corpus callosum and the basal ganglia [23]. PCNSL often has a characteristic appearance on both CT and MR imaging reflecting its hypercellularity[23]. CT shows iso to hyperdense lesion while in MRI

the lesion appearing iso to hypo intense on T1-weightedimages and hypointense to gray matter on T2-weighted images and flair sequence. Most lesions show moderate-to-marked contrast enhancement on both CT and MRI. PCNSL lesions demonstrate a restricted diffusion (hyperintense on DWI and hypointense on ADC maps), this makes it possible to differentiate them from other similar lesions, especially high-grade gliomas and metastases (Fig9).[14]. In fact, PCNSL lesions often have more restricted diffusion and lower ADC values That these lesions[23, 25].MR spectroscopy demonstrated elevated lipid peaks combined with high Cho/Cr ratios [25].

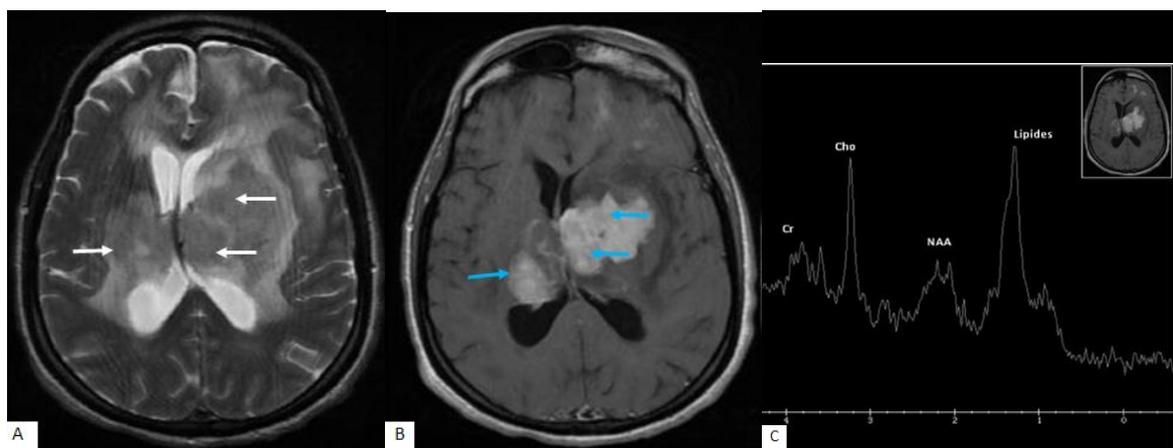


Fig 9. 48 years old man with increased intra cranial pressure .multiple focal lesions in the basal ganglia and frontal left lobe with solid enhancement, associated to peri-ventricular white matter vasogenic edema and mass effect displacing the midline multifocal strong patchy homogeneous enhancement. The single voxel, short Time Echo MRS demonstrates an exaggerated lipid peak in a solid mass. Lipid peaks are typically demonstrated in necrotic lesions, but in solid PCNSL lesions the peak is due to the increased macrophage content. MRS demonstrates also a decreasing value of NAA and elevation of choline.

GLIOMATOSIS CEREBRI

Cerebral gliomatosis (GC) is a rare glial tumor described for the first time in 1938 by Nevin, characterized by diffuse glial cell infiltration of the brain involving more than two cerebral lobes, with preservation of the neuronal architecture[26]. The clinical presentation of GC is polymorphic and non-specific including epilepsy, cognitive disorders, headache, and intracranial hypertension[27]. According to autopsy studies, the invaded areas include the cerebral hemispheres (Affecting the periventricular white matter extended to the cortex), midbrain, pons, thalamus, the basal ganglia, cerebellum, medulla oblongata, and, in less than 10% of cases each, the hypothalamus, optic nerve and chiasm, and spinal cord[28]. CT findings are either normal or show areas of hypo-attenuation with little mass effect [29], with more or less diffuse mass effect and minimal or no enhancement[28].

MR imaging is the radiologic method of choice because it reveals a more extensive involvement of the CNS and subtle changes; affected regions show bilateral poorly defined areas that are hyper-intense on T2- weighted MR images(Fig 10).and iso to hypo-intense on T1- weighted MR images. Lack of enhancement (indicating a preserved blood-brain barrier) and preservation of tissue architecture are usually found. If areas of enhancement are seen, they represent both higher grade tumor and dense tumor infiltration[27, 28]. On perfusion MR, lesions show low/normal relative cerebral blood volume (rCBV), which is correlated with no vascular hyperplasia[30]. On MR spectroscopy, the areas of hyper-intensity on T2-weighted images show an elevation of myoinositol and choline and a decrease in NAA, elevated Cho/Cr and Cho/NAA ratios as well as decreased NAA/Cr ratios of varying degrees (Fig 10).[29]

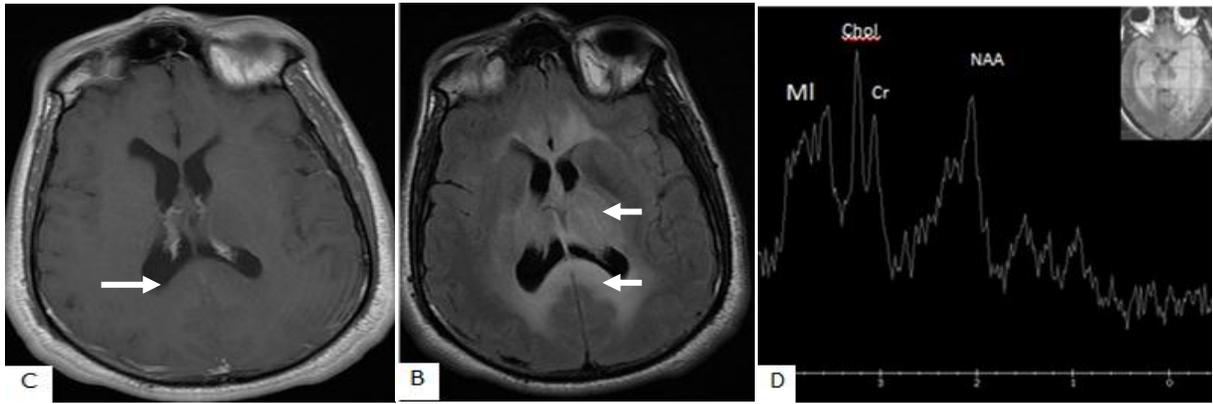


Fig 10. Gliomatosis cerebri in a 45 year old woman. Axial FLAIR WI (A, B) demonstrate a homogeneous infiltration in periventricular white mater and brainstem. Post contrast axial T1WI (C) shows a no enhancement of the infiltration. Single voxel, short time echo MRSpectroscopy shows increased concentration of choline and decreased concentration of N-acetyl aspartate with a Cho / NAA <2, another metabolite whose elevation evokes the diagnosis of GC is Myoinositol. Furthermore there is a lack of lipids which can rule lymphoma origin of tumor infiltration.

GLIOBLASTOMA MULTIFORME

Glioblastoma multiforme is the most common primary brain tumour, it accounts for 12%–15% of all intracranial neoplasms and 50–60% of all astrocytomas. It usually originates from the white matter, and can spread via white matter tracts and perivascular spaces. The lesions are hypo to iso-intense on T1- weighted MR images with central heterogeneous signal (necrosis, intra-tumoural

hemorrhage) while they are hyper-intense on T2-weighted and FLAIR images with surrounding vasogenic edema. The lesions typically show thick irregular ring enhancement surrounding necrosis on contrast-enhanced T1- weighted images (**Fig. 11**). Typical spectroscopic findings include a high peak of choline , as well as reduction in the NAA levels with subsequent increase in the Ch/Cr and Ch/NAA ratios. A marked increase in the peak of lipids/lactate is also observed (**Fig. 11**).

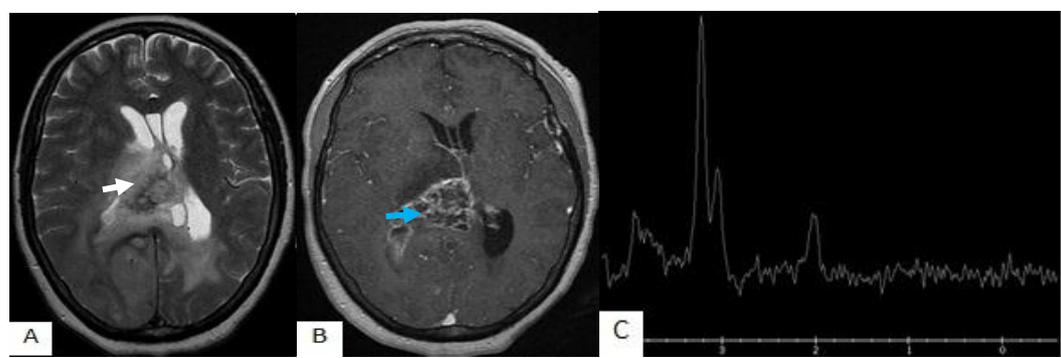


Fig 11. 65 year old male with glioblastoma multiforme. Axial T2WI(A) shows an heterogeneous mass in the splenium of the corpus callosum with perilesional edema (white arrow) . Post contrast axialT1WI (B) shows irregular rim enhancement with necrotic center (blue arrow). a high peak of choline , as well as reduction in the NAA levels with subsequent increase in the Ch/Cr and Ch/NAA ratios.

CONCLUSION

Periventricular lesions are often found in the daily practice of a radiologist. the etiologic spectrum of these lesions is very wide including vascular, infectious, tumoral and inflammatory disease knowing the clinical presentation, and the imaging findings especially on MRI can allow the radiologist to limit the differential diagnosis.

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