Review Article

Imaging of Central Nervous System Lymphoma Spectrum Disorders: Review Article

Piyapittayanan S, MD[1], Cheunsuchon P, MD[2], Chawalparit O, MD[1]

[1] Department of Radiology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand

[2] Department of Pathology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand

Tumors of lymphoid tissue involving central nervous system (CNS), or so-called CNS lymphoma spectrum disorders, consist of many sub-types. Pathologic classification of the tumor ranges from lympho-proliferative disorders to malignant neoplasms. The neuro-imaging diagnosis of CNS lymphoma is crucial and challenging because a variety of imaging appearances of CNS lymphomas may mimic several other disorders such as other tumors, infections, demyelinating diseases, and ischemic strokes. The present article reviewed the variety of imaging appearances of CNS lymphoma spectrum disorders.

Keywords: Lymphoma, Lymphoid tissue, Central nervous system

J Med Assoc Thai 2019;102(10):1140-8
Website: http://www.jmatonline.com
Received 1 May 2019 | Revised 1 Jul 2019 | Accepted 5 Jul 2019

The lymphoma spectrum disorder of the central nervous system (CNS) is a broad group of disorders that range from non-malignant to highly malignant. The malignant lymphomas consist of primary CNS lymphoma (PCNSL), metastatic lymphoma, primary dural lymphoma, intravascular lymphoma, lymphomatosis cerebri (LC), and mucosa-associated lymphoid tissue (MALT) lymphoma. The lympho-proliferative conditions include lymphomatoid granulomatosis (LG), sentinel lesions of PCNSL, and post-transplant lympho-proliferative disorder (PTLD). These lesions constitute a spectrum ranging from polymorphic proliferation of lymphoid or plasma cells to monomorphic proliferation of atypical lymphoid cells indistinguishable from lymphoma. In general, non-malignant and malignant lymphomas cannot be differentiated by imaging characteristics either by mass like or contrast enhancing lesion. Therefore, a pathological diagnosis is needed. Lymphomas demonstrate a variety of imaging features that may mimic other diseases such as other tumors, infections, demyelinating diseases, or strokes, which makes diagnosis challenging. Whenever neuroimaging suggests a lymphoma, corticosteroid treatment is contra-indicated before a biopsy. The reason is that steroids disrupt cellular morphology, causing significant shrinkage of the tumor size, a decrease in the degree of peritumoral edema, and even interference with the histopathologic diagnosis, all within a very short time interval. This phenomenon has been referred to as 'vanishing lymphoma'[1,2]. In the present article, the authors reviewed the varied imaging features of the CNS lymphoma spectrum disorders.

Correspondence to:

Chawalparit O.

Department of Radiology, Faculty of Medicine Siriraj Hospital, 2 Wang Lang Road, Bangkoknoi, Bangkok 10700, Thailand.

Phone: +66-2-4197086, **Fax**: +66-2-4127785

Email: orasa.cha@gmail.com

Primary CNS lymphomas

The precise etiology of transformed lymphocytic tumors occurring in the brain is currently unknown as the CNS lacks lymphatic and lymphoid tissues[3]. However, the perivascular space (PVS) may be the site of early entry into the CNS as B-cells have been detected in cerebral PVSs[4]. Furthermore, some lymphomas are associated with viral infections, especially the Epstein-Barr virus (EBV). Because EBV-positive B lymphocytes are more frequent in HIV-infected brains than normal ones, there is a predisposition for CNS lymphoma among AIDS patients[4,5].

Most PCNSLs are mature B-cell lymphomas

How to cite this article: Piyapittayanan S, Cheunsuchon P, Chawalparit O. Imaging of Central Nervous System Lymphoma Spectrum Disorders: Review Article. J Med Assoc Thai 2019;102:1140-8.

Table 1. Imaging differences in PCNSL between immunocompetent and immunocompromised patients

	Immunocompetent patient	Immunocompromised patient
Number of lesions	Common solitary lesion Multiple lesions in 20% to 40%	Often multifocal lesions
Enhancement pattern	Homogeneous	Common irregular and ring enhancement
Calcification, hemorrhage, necrosis	Rare	Common necrosis Calcification and hemorrhage may be seen and more frequent than in immunocompetent (Figure 3)

Data from references[6,7,14,15]

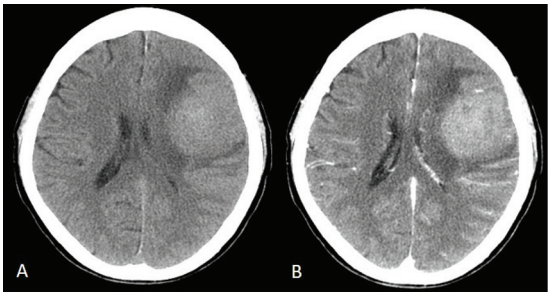

Figure 1. PCNSL. (A) Axial non-contrast CT scan demonstrates iso- to slightly hyperdense mass at left frontal lobe and enhancement after contrast medium administration (B).

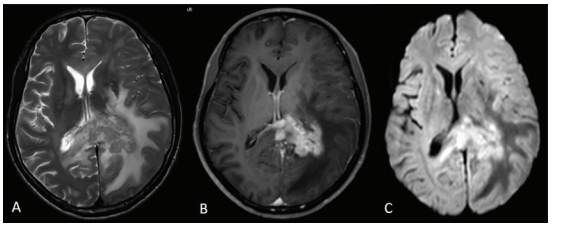

Figure 2. PCNSL in immunocompetent patient. (A) Axial T2WI shows iso- to hypointense signal of lesion at splenium of corpus callosum and left periventricular region. (B) Axial T1WI-GD shows homogeneous contrast enhancement. (C) Axial DWI shows restricted diffusion of lesion.

with diffuse large B-cell lymphoma while T-cell lymphomas are rare[6]. PCNSLs account for up to 6% of all primary CNS tumors. They are defined as tumors originating in the brain, leptomeninges, spinal cord, or the eyes, without evidence of them elsewhere, at the primary diagnosis[7-9]. Consequently, the initial imaging evaluation for the diagnosis of PCNSLs includes computed tomography (CT) scanning of the chest, abdomen, and pelvis, as well as an ultrasound of the testes, and optional FDG-PET scanning[10]. PCNSLs can occur in both immunocompetent and immunocompromised patients; however, immunocompromised patients, including those with AIDS, immunodeficiency, autoimmune diseases, and organ transplants, are at increased risk of developing a PCNSL[6,11]. As an intraocular lymphoma may be associated with a PCNSL, an ophthalmologic examination is necessary for all patients, which may affect treatment planning[12,13]. PCNSLs typically present with a focal parenchymal mass and have a tendency to abut the ependyma, the meninges, or both. Common locations include the cerebral hemispheres, periventricular white matter, deep gray matter, corpus callosum, subependymal region, and areas adjacent to other CSF spaces[14]. The typical imaging features of

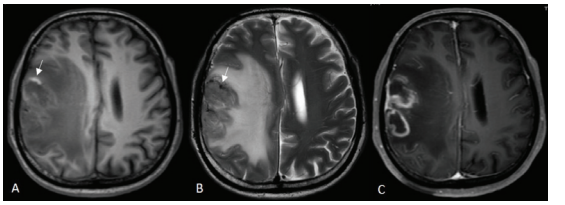

Figure 3. PCNSL in immunocompromised patient. (A) Axial T1WI demonstrates isointense lesion at right fronto-parietal lobe with some area of hemorrhage with hyperintense foci (arrow). (B) Axial T2WI shows lesion isointense to gray matter with some area of hemorrhage with hypointense foci (arrow). (C) Axial T1WI-GD shows two rim enhancing lesions.

PCNSLs correlate with hypercellularity and a high nuclear-to-cytoplasmic ratio. They also depend on the immune status of the patient. The imaging findings of both immunocompetent and immunocompromised patients may be different and are summarized in Table 1.

On non-contrast CT scans, the lesions are usually hyperdense because of their high cellularity and nucleus-to-cytoplasmic ratio (Figure 1).

On magnetic resonance (MR) imaging, most

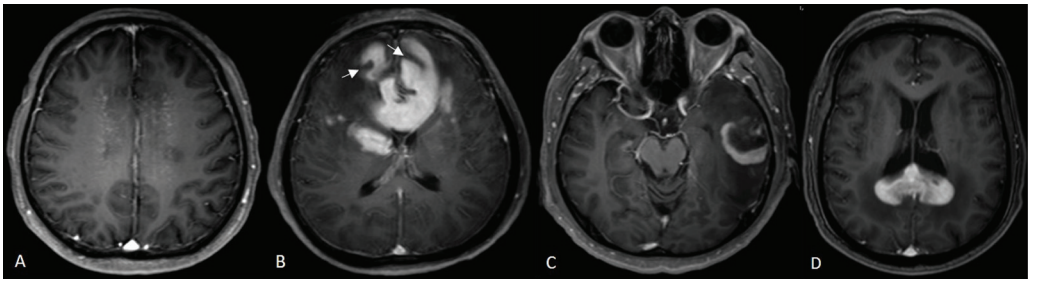

Figure 4. PCNSL. (A-D) Axial T1WI-GD, (A) punctate and linear enhancement, tracking along perivascular space at bilateral centrum semiovale, (B) 'Notch sign' at bilateral frontal lobes (arrows), (C) open ring enhancement at left temporal lobe, (D) butterfly lesion involving bilateral splenium of corpus callosum.

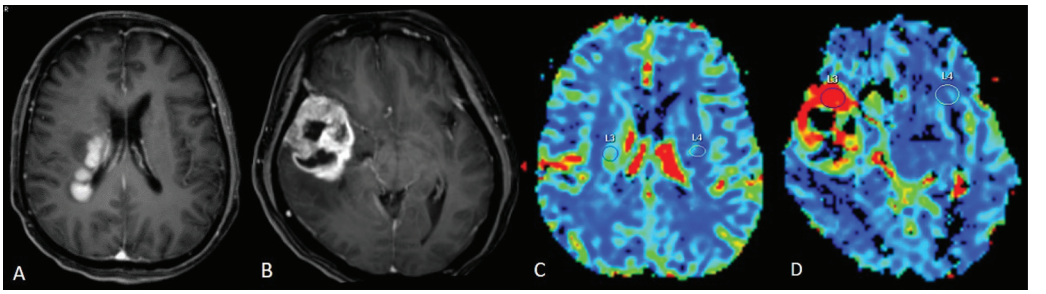

Figure 5. PCNSL vs. GBM. (A) Axial T1WI-GD shows homogeneous enhancing mass at right periventricular region in PCNSL. (B) Axial T1WI-GD shows thick irregular rim enhancing mass at right temporal lobe and pathologically proven GBM. (C) DSC perfusion MR in PCNSL shows rCBV ratio at enhancing mass=2.19. (D) DSC perfusion MR in GBM shows rCBV ratio at rim enhancing portion of mass=10.92.

lesions are typically isointense to hypointense, relative to the gray matter on T1WI, and isointense to hypointense on T2WI (Figure 2). Perilesional edema is usually present. Nearly all tumors show moderate to marked enhancement after administration of a contrast agent, which typically appears as a homogeneous enhancement in immunocompetent patients (Figure 2), but as a heterogeneous enhancement, often with a rim, and with cystic or necrotic portions, in immuno-compromised patients (Figure 3). Linear enhancement at the margins of the lesion, tracking along the PVS, is highly specific for PCNSL[7,16] (Figure 4). When a tumor occurs in a subcortical location, it infiltrates along the cortex, which probably reflects the soft, infiltrative nature of the tumors. Furthermore, the specific enhancing patterns in the subcortical location of those tumors are an open-ring enhancement and a 'notch sign', which is a deep, abnormal depression at the tumor margin[11,15,17] (Figure 4). The open-ring enhancement pattern is typically described in brain demyelination; however, when present, the ring is usually thinner and more uniform than those in cases of PCNSLs. A tumor involving both sides of the genu or splenium of the corpus callosum is referred to as a butterfly pattern, which is commonly the characteristic sign of a PCNSL (Figure 4). The main differential diagnosis for a mass centered at the corpus callosum is glioblastomas (GBMs). Nearly all GBMs show hemorrhage, necrosis, and heterogeneous enhancements, which are unlike lymphomas.

Advanced MR imaging has been used to improve the diagnostic accuracy for PCNSLs. On diffusion-weighted imaging, the dense cellularity of the tumor decreases the relative size of the extracellular space and consequently restricts the random movement of water molecules. The tumor appears hyperintense on diffusion-weighted imaging, with corresponding areas of hypointensity on apparent diffusion coefficient (ADC) maps. A decreased ADC value is suggestive of increased tumor cellularity. The ADC value of PCNSLs is often lower than those of high-grade gliomas. On MR perfusion imaging, the PCNSLs tend to have a relatively lower perfusion compared to high-grade gliomas due to the lack of tumor neovascularization in PCNSLs (Figure 5). MR spectroscopy reveals an

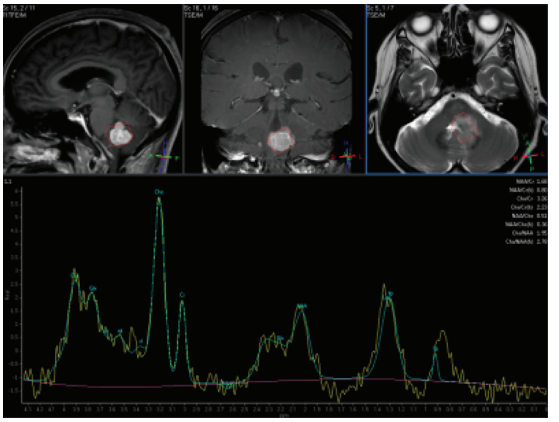

Figure 6. PCNSL. Single voxel technique MRS of enhancing mass at periventricular region of 4th ventricle shows increased Cho peak, Lid/Lac peak but decreased NAA peak that represents tumor spectrum.

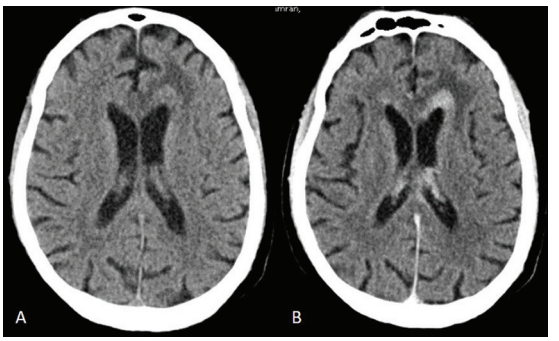

Figure 7. CNS metastasis from testis diffuse large B-cell lymphoma. (A) Axial non-contrast CT demonstrates slightly hyperdense lesion along wall of both lateral ventricles. (B) Axial contrast enhanced CT shows diffuse subependymal contrast enhancement along bilateral lateral ventricles.

increased Cho/Cr ratio and a lipid peak that represents the tumor spectrum (Figure 6).

Metastatic lymphomas

The secondary CNS involvement by systemic lymphomas typically manifests in the form of leptomeningeal spreading, similar to leptomeningeal metastases from any cause[6,7] (Figure 7). Approximately one-third of patients present with parenchymal lesions occurring as single or multiple enhancing masses. The imaging appearances are quite similar to the findings for PCNSLs. Parenchymal metastases from systemic lymphomas can be accompanied by leptomeningeal metastases, thus raising clinical suspicions of CNS metastases[7]. However, there are no differences in the neuroimaging appearances of primary and secondary CNS lymphomas. Therefore, a systemic evaluation of a patient with a CNS lymphoma is required to differentiate between the two entities.

Primary dural lymphomas

A primary dural lymphoma is a rare sub-entity of PCNSLs arising from the dura mater. It is usually a low-grade B-cell marginal-zone lymphoma. Neuroimaging reveals single or multiple extra-axial masses, and a diffuse enhancement following contrast medium administration that strongly mimics meningioma. Other imaging findings include a dural tail, adjacent parenchymal vasogenic edema, hyperostosis, and bone erosion, all of which share the imaging features of meningioma. The most common location is the cerebral convexities, but the falx, tentorial, and sellar or suprasellar regions can be involved[8,18].

Intravascular lymphomas

An intravascular lymphoma, also called angiotropic large cell lymphoma or malignant angioendotheliomatosis, is an extremely rare type of large B-cell lymphoma. It is characterized by the aggressive proliferation of neoplastic lymphoid cells within blood vessel lumens, particularly in small vessels and capillary beds, and the subsequent occlusion of the small vessel lumens. This lymphoma usually occurs in a multisystemic disease, which can be found in any organ. CNS and skin are the most frequently affected organs (referred to as the Western form). The liver, spleen, and bone marrow are involved to a much lesser extent, causing multiorgan failure, including hepatosplenomegaly, pancytopenia, and hemophagocytic syndrome (referred to as the Asian form)[2,19]. Diagnosis is often difficult because of a variable presentation, non-specific constitutional symptoms, and a lack of pathognomonic neuroimaging features. The multiplicity of presentations includes fever, stroke, transient ischemic attacks, non-focal neurological deficits, seizure, dementia, subacute encephalopathy, and cutaneous manifestations. Various imaging features have been described but are non-specific. The MR imaging findings include multifocal hyperintensity on T2-weighted images in subcortical and deep white matter, blooming signal foci on gradient echo images, restrictive diffusion on diffusion weighted images, and variable contrast enhancements such as spots, dots, lines, meningeal, ring-like and, less commonly, or mass-like[20] (Figure 8). These findings may mimic small vessel ischemic strokes or CNS vasculitis[19,21]. Hence, a

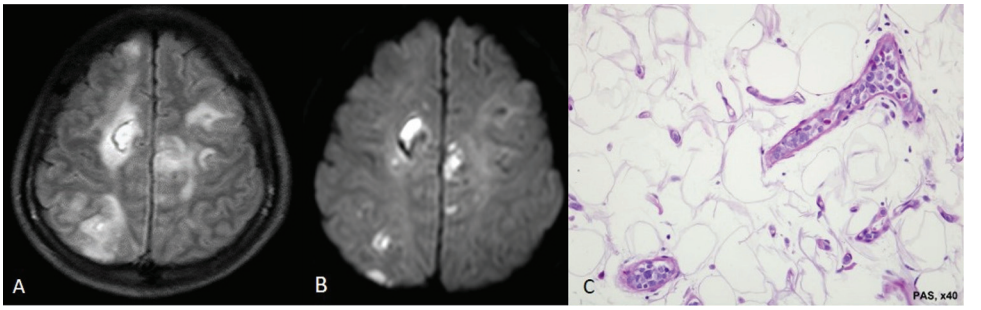

Figure 8. Intravascular lymphoma. (A) Axial FLAIR shows multiple discrete hyperintensity of lesions at both fronto-parietal regions. (B) Axial DWI demonstrates restricted diffusion of lesions that mimic small vessel stroke and CNS vasculitis. (C) Small vessels in subcutaneous tissue contain medium to atypical large lymphoid cells in its lumen. The immunohistochemical study shows that tumor cells are positive for CD20 in its lumen (not shown).

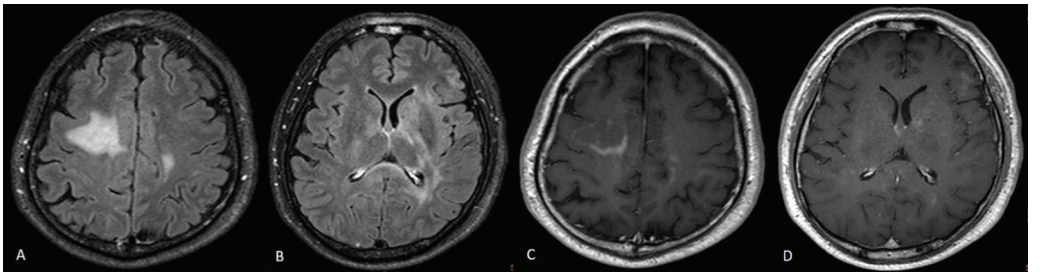

Figure 9. Lymphomatosis cerebri. (A, B) Axial FLAIR show infiltrative multifocal hyperintensity lesion without obvious mass effect at both frontal lobes, both basal ganglia and left temporo-occipital periventricular regions. (C, D) Axial T1WI-GD demonstrate some punctate foci and rim enhancement.

brain tissue biopsy is needed in patients with presumed intravascular lymphoma who have not yet been given a definitive diagnosis. However, an intracranial biopsy is a relatively invasive procedure. In the case of patients with skin lesions, the correct diagnosis is often made from a skin biopsy that reveals tumor cells in the small vessels of the subcutaneous tissue (Figure 8).

Lymphomatosis cerebri

LC is a rare variant of PCNSLs that manifests as a widespread infiltration of the lymphoma cells in the brain parenchyma without focal mass. The common presentations of patients with LC are rapidly progressive cognitive decline, personality change, or gait disturbance[22]. The typical MR imaging appearances reveal diffuse hyperintense on T2WI in the cerebral white matter without a mass effect, and a subsequent extension to involve the basal ganglia, thalamus, and brainstem[23] (Figure 9). The diffuse infiltrative lesion in the brain parenchyma shows variable contrast enhancement but it usually does not show contrast enhancement[24]. These MR imaging features can mimic other conditions, such as gliomatosis cerebri, hypertensive encephalopathy, encephalitis, and demyelinating diseases. Hence, a definitive diagnosis can be established by adequate tissue biopsy sampling[25].

MALT lymphomas

A MALT lymphoma is a rare subtype of marginal zone B-cell lymphomas, and it is characterized by lymphoid proliferation in MALT rather than lymph nodes. The most common location is the stomach, where it is called a gastric MALT lymphoma. However, this subtype of lymphoma can occur in the head and neck regions, such as the salivary glands, thyroid gland, ocular adnexa, dura mater, and pituitary gland[26-28]. A MALT lymphoma is usually associated with infections and autoimmune diseases. A primary CNS MALT lymphoma is a very rare subtype of MALT lymphomas, whose imaging findings have been reported as a dural mass mimicking a meningioma[27]. The ocular adnexal lymphoma is characterized by a tumor within the intra- and extraconal orbital fat, extraocular muscles, lacrimal structures, eyelids, and conjunctiva[29]. A primary intraocular lymphoma

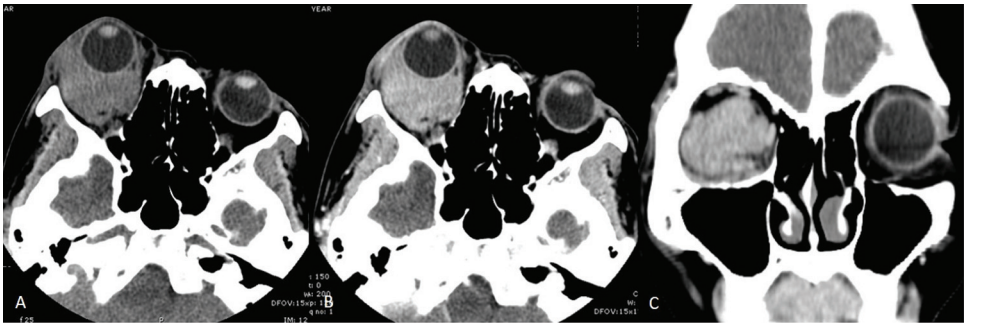

Figure 10. Ocular adnexal MALT lymphoma. (A) Axial noncontrast CT orbits shows isodensity soft tissue mass at ocular adnexa of right orbit. (B, C) Axial and coronal contrast enhanced CT orbits show homogeneous strong contrast enhancement.

is not included in this category, but it is regarded as a subgroup of PCNSLs. The imaging of an adnexal ocular MALT lymphoma shows diffuse infiltration of the ocular adnexa, with homogeneous attenuation and strong contrast enhancement (Figure 10), which share findings with idiopathic orbital inflammatory pseudotumor.

Lymphomatoid granulomatosis

LG is a lymphoproliferative disorder involving extranodal sites. The lesions are composed of angiocentric and angiodestructive polymorphous lymphoid infiltrates. There are usually small number of EBV-positive B cells on background of reactive T-cells[30]. LG is a multisystemic premalignant lymphomatoid condition. It is defined as an angiocentric, angiodestructive lymphoproliferative and granulomatous disease[31]. This condition is associated with EBV infections and may eventually develop lymphomas of the large B-cell type, whose incidence ranges from 10% to 60%[32,33]. The lung is the most frequent and, in more than 90% of cases, the initial organ of manifestation of LG[32]. However, the skin and CNS are also common organs to be involved, with skin involvement present in approximately 25% to 50% of cases as well as CNS lesions in about 25% to 35% of cases[33]. The neuroimaging findings of LG are non-specific and commonly reveal focal intraparenchymal lesions showing multiple hyperintense foci on a T2-weighted image and a FLAIR image, and multiple punctuate or linear foci of contrast enhancement (Figure 11). These lesions commonly go along white matter, deep gray matter or the brainstem, and appear to reside along the medullary vessels. The other findings include leptomeningeal or cranial nerve enhancement, ring-like enhancement, intracranial mass or enlargement, and intense enhancement of the choroid plexus[32,34]. Lymphocytic-infiltrated vascular walls and variable necrosis in a perivascular distribution are reported on pathology, and are classified into grades I to III on the basis of the number of EBV-positive atypical large B-cells and the size of the necrotizing zone[30].

Sentinel lesions of primary CNS lymphomas

Sentinel lesions of PCNSLs are categorized as a pre-lymphoma state that may reflect an immune response attack against the lymphoma. MR imaging may show hyperintense lesions on T2WI and FLAIR with punctate-curvilinear contrast enhancement along the PVS, predominantly involving the brainstem. The pathology of the lesions demonstrates CD4-positive lymphocytes infiltrating along the perivascular region, sometimes with a demyelination. These lesions can disappear spontaneously or after steroid treatment, and they precede the development of a PCNSL by 1 year or less[35].

Post-transplant lymphoproliferative disorder

PTLD is defined as a lymphoid proliferation or a lymphoma that develops in immunosuppressive recipients of solid organ or bone marrow transplantations. They are best considered as a spectrum of diseases ranging from early EBV-driven polyclonal proliferations, to malignant lymphomas that can be EBV+ or EBV–. The incidence of PTLD is less than 2%. The pathogenesis is T-cell suppressed, leading to B-cells infected with EBV proliferate. The imaging of PTLD is non-specific multifocal discrete contrast enhancing lesions and typically extensive necrosis (Figure 12), which overlap most with those of PCNSLs that arise in immunocompromised patients. Sometimes, it is not always possible to distinguish them from CNS infections, and a definitive diagnosis

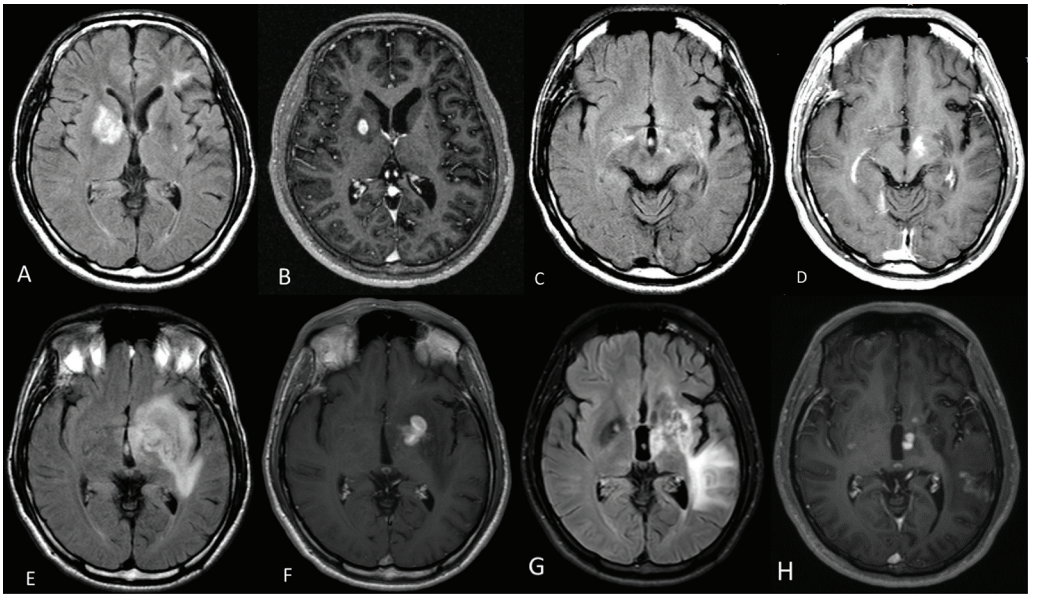

Figure 11. Lymphomatoid granulomatosis. (A, C) Axial FLAIR images show high signal intensity at right basal ganglia. (B, D) Axial T1WI-GD shows enhancing lesions at right basal ganglia and left cerebral peduncle. One year later, the lesion was changed the position to the left basal ganglia which showed high signal intensity on FLAIR image and contrast enhancing lesion. Resolution of the lesion at right basal ganglia is seen as shown on E, F. The stereotactic biopsy was performed for three times and pathological diagnosis showed gliosis. (G, H) From axial FLAIR image and T1WI/GD, the lesion at left basal ganglia has improved but demonstrated the new high T2 and enhancing lesions at left thalamus and left temporal lobe. This patient developed lymphoma later (picture not shown).

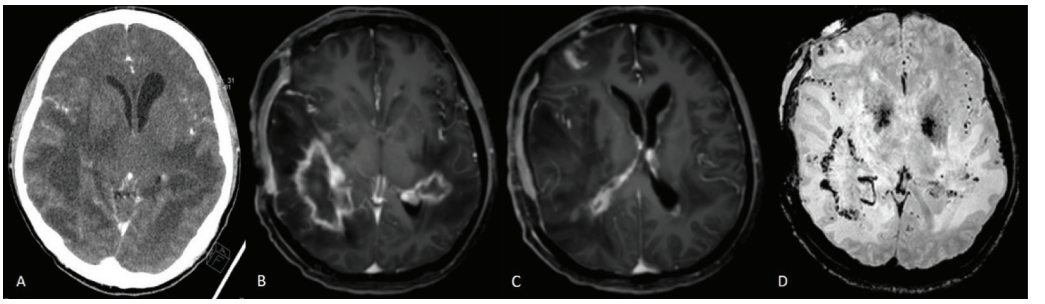

Figure 12. PTLD. (A) Axial contrast enhanced CT shows vasogenic edema areas at posterior bilateral cerebral hemisphere with diffuse brain swelling and midline shifting to the left. (B, C) Axial T1WI-GD shows obviously seen rim enhancing lesions at both temporo-occipital periventricular regions and linear ependymal enhancement along both lateral ventricles. (D) Axial SWI shows multiple foci of microbleeds in these lesions.

is eventually made from a tissue biopsy.

Conclusion

Lymphoid neoplasms have a wide range of imaging presentations and pathologies. The typical or common CT and MR imaging findings can be illustrated for the diagnosis of CNS lymphomas. Sometimes, however, the imaging appearances of intracranial lymphomas are non-specific or share common findings with other diseases, which is why they are called one of the great mimickers. Therefore, tissue biopsies need to be performed to obtain a definitive diagnosis in inconclusive cases.

What is already known on this topic?

The common imaging finding of PCNSL and secondary CNS involvement by systemic lymphomas are already known.

Subtypes of lymphoid tissue tumors involving central nervous system make a variety of imaging appearances and result in difficulty to diagnosis.

What this study adds?

The pathologic classification of the central nervous system lymphoma spectrum disorders ranges from non-malignant to highly malignant.

Sometime, the imaging appearance are non-specific and may mimic several other diseases such as other tumors, infections, demyelinating diseases, and ischemic strokes.

The findings can be used as a differential diagnosis in the central nervous system disorders because they are great mimickers.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Perini GF, Campregher PV, Santos FP, Hamerschlak N. Primary central nervous system lymphoma: what a neurologist/neurosurgeon should know? Arq Neuropsiquiatr 2013;71:254-7.
2. Giannini C, Dogan A, Salomao DR. CNS lymphoma: a practical diagnostic approach. J Neuropathol Exp Neurol 2014;73:478-94.
3. Kadoch C, Treseler P, Rubenstein JL. Molecular pathogenesis of primary central nervous system lymphoma. Neurosurg Focus 2006;21:E1.
4. Anthony IC, Crawford DH, Bell JE. B lymphocytes in the normal brain: contrasts with HIV-associated lymphoid infiltrates and lymphomas. Brain 2003;126:1058-67.
5. Paulus W. Classification, pathogenesis and molecular pathology of primary CNS lymphomas. J Neurooncol 1999;43:203-8.
6. Slone HW, Blake JJ, Shah R, Guttikonda S, Bourekas EC. CT and MRI findings of intracranial lymphoma. AJR Am J Roentgenol 2005;184:1679-85.
7. Haldorsen IS, Espeland A, Larsson EM. Central nervous system lymphoma: characteristic findings on traditional and advanced imaging. AJNR Am J Neuroradiol 2011;32:984-92.
8. Tang YZ, Booth TC, Bhogal P, Malhotra A, Wilhelm T. Imaging of primary central nervous system lymphoma. Clin Radiol 2011;66:768-77.
9. Yap KK, Sutherland T, Liew E, Tartaglia CJ, Pang M, Trost N. Magnetic resonance features of primary central nervous system lymphoma in the immunocompetent patient: a pictorial essay. J Med Imaging Radiat Oncol 2012;56:179-86.
10. Baraniskin A, Deckert M, Schulte-Altedorneburg G, Schlegel U, Schroers R. Current strategies in the diagnosis of diffuse large B-cell lymphoma of the central nervous system. Br J Haematol 2012;156: 421-32.
11. Mansour A, Qandeel M, Abdel-Razeq H, Abu Ali HA. MR imaging features of intracranial primary CNS lymphoma in immune competent patients. Cancer Imaging 2014;14:22.
12. Ferreri AJ, Blay JY, Reni M, Pasini F, Gubkin A, Tirelli U, et al. Relevance of intraocular involvement in the management of primary central nervous system lymphomas. Ann Oncol 2002;13:531-8.
13. Clarke JL, Deangelis LM. Primary central nervous system lymphoma. Handb Clin Neurol 2012;105:517-27.
14. Haque S, Law M, Abrey LE, Young RJ. Imaging of lymphoma of the central nervous system, spine, and orbit. Radiol Clin North Am 2008;46:339-61, ix.
15. Adachi K, Yamaguchi F, Node Y, Kobayashi S, Takagi R, Teramoto A. Neuroimaging of primary central nervous system lymphoma in immunocompetent patients: comparison of recent and previous findings. J Nippon Med Sch 2013;80:174-83.
16. Go JL, Lee SC, Kim PE. Imaging of primary central nervous system lymphoma. Neurosurg Focus 2006;21:E4.
17. Zhang D, Hu LB, Henning TD, Ravarani EM, Zou LG, Feng XY, et al. MRI findings of primary CNS lymphoma in 26 immunocompetent patients. Korean J Radiol 2010;11:269-77.
18. Iwamoto FM, Abrey LE. Primary dural lymphomas: a review. Neurosurg Focus 2006;21:E5.
19. Hundsberger T, Cogliatti S, Kleger GR, Fretz C, Gahler A, Anliker M, et al. Intravascular lymphoma mimicking cerebral stroke: report of two cases. Case Rep Neurol 2011;3:278-83.
20. Baehring JM, Henchcliffe C, Ledezma CJ, Fulbright R, Hochberg FH. Intravascular lymphoma: magnetic resonance imaging correlates of disease dynamics within the central nervous system. J Neurol Neurosurg Psychiatry 2005;76:540-4.
21. Song DK, Boulis NM, McKeever PE, Quint DJ. Angiotropic large cell lymphoma with imaging characteristics of CNS vasculitis. AJNR Am J Neuroradiol 2002;23:239-42.
22. Kitai R, Hashimoto N, Yamate K, Ikawa M, Yoneda M, Nakajima T, et al. Lymphomatosis cerebri: clinical characteristics, neuroimaging, and pathological findings. Brain Tumor Pathol 2012;29:47-53.
23. Courtois F, Gille M, Haven F, Hantson P. Lymphomatosis cerebri presenting as a recurrent leukoencephalopathy. Case Rep Neurol 2012;4:181-6.
24. Kanai R, Shibuya M, Hata T, Hori M, Hirabayashi K, Terada T, et al. A case of 'lymphomatosis cerebri' diagnosed in an early phase and treated by whole brain radiation: case report and literature review. J Neurooncol 2008;86:83-8.
25. Lee PJ, Berrios I, Ionete C, Smith T. Lymphomatosis cerebri: diagnostic challenges and review of the literature. BMJ Case Rep 2016;2016. pii: bcr-2016-216591.

26. Lee JH, Lee HK, Choi CT, Huh J. Mucosa-associated lymphoid tissue lymphoma of the pituitary gland: MR imaging features. AJNR Am J Neuroradiol 2002;23:838-40.
27. Kamoshima Y, Sawamura Y, Sugiyama T, Yamaguchi S, Houkin K, Kubota K. Primary central nervous system mucosa-associated lymphoid tissue lymphoma--case report. Neurol Med Chir (Tokyo) 2011;51:527-30.
28. Asproudis I, Gorezis S, Charonis GC, Tolis C, Tsanou E, Agnantis NJ. Mucosa-associated lymphoid tissue lymphoma of the lacrimal gland--a case report. In Vivo 2005;19:1105-9.
29. Politi LS, Forghani R, Godi C, Resti AG, Ponzoni M, Bianchi S, et al. Ocular adnexal lymphoma: diffusion-weighted MR imaging for differential diagnosis and therapeutic monitoring. Radiology 2010;256:565-74.
30. Swerdlow SH, Campo E, Harris NL, Jaffe ES, Pileri SA, Stein H, et al. WHO classification of tumours of haematopoietic and lymphoid tissues. Revised 4th ed. Geneva: WHO; 2017.
31. Whelan HT, Moore P. Central nervous system lymphomatoid granulomatosis. Pediatr Neurosci 1987;13:113-7.
32. Patsalides AD, Atac G, Hedge U, Janik J, Grant N, Jaffe ES, et al. Lymphomatoid granulomatosis: abnormalities of the brain at MR imaging. Radiology 2005;237:265-73.
33. Castrale C, El Haggan W, Chapon F, Reman O, Lobbedez T, Ryckelynck JP, et al. Lymphomatoid granulomatosis treated successfully with rituximab in a renal transplant patient. J Transplant 2011;2011:865957.
34. Sohn EH, Song CJ, Lee HJ, Kim S, Kim JM, Lee AY. Central nervous system lymphomatoid granulomatosis presenting with parkinsonism. J Clin Neurol 2007;3:108-11.
35. Taieb G, Duran-Pena A, de Chamfleur NM, Moulignier A, Thouvenot E, Allou T, et al. Punctate and curvilinear gadolinium enhancing lesions in the brain: a practical approach. Neuroradiology 2016;58:221-35.