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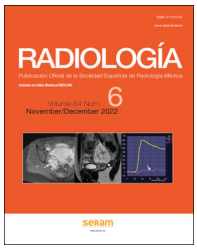

UPDATE IN RADIOLOGY

Atypical presentations of primary lymphoproliferative processes in the central nervous system

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Abstract Central nervous system (CNS) involvement by lymphoproliferative disorders is rare and associated with a poor prognosis. CNS involvement can be exclusive, primary or appear in a secondary manner as part of a systemic process. The spectrum of involvement that we encounter is varied and neuroimaging plays a key role in diagnosis. This article reviews less commonly presented patterns of primary involvement of CNS lymphoproliferative processes such as lymphomatosis cerebri, intravascular lymphoma or dural lymphoma. We will also review other atypical lymphoproliferative processes associated with Epstein-Barr virus (EBV) infection, such as lymphomatoid granulomatosis (LG) or post-transplant lymphoproliferative disorders (PTLD). An awareness of these patterns of presentation and less frequent entities will allow us to make earlier diagnoses and establish treatments that will lead to better prognoses for patients.

PALABRAS CLAVE
Granulomatosis linfomatoide;
Linfoma;
Infecciones por virus de Epstein-Barr;
Imagen por resonancia magnética;
Diagnóstico por imagen

Presentaciones atípicas de los procesos linfoproliferativos primarios del sistema nervioso central

Resumen La afectación del sistema nervioso central (SNC) por trastornos linfoproliferativos es infrecuente y se asocia con un mal pronóstico. El daño en el SNC puede aparecer como una afectación exclusiva o primaria de la enfermedad o aparecer de una manera secundaria dentro de un proceso sistémico. El espectro de lesiones que nos podemos encontrar es variado y la neuroimagen desempeña un papel clave en el diagnóstico. En este trabajo nos centramos en revisar los patrones de presentación menos frecuentes en la afectación primaria de los procesos linfoproliferativos del SNC como son la linfomatosis cerebral, el linfoma intravascular o el linfoma dural. También se revisarán otros procesos linfoproliferativos atípicos asociados

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a la infección del virus de Epstein-Barr (VEB) como son la granulomatosis linfomatoide (GL) y el trastorno linfoproliferativo asociado al trasplante (TLP). El conocimiento de estos patrones de presentación y de estas entidades menos frecuentes, nos van permitir hacer un diagnóstico precoz e instaurar tratamientos que favorezcan un mejor pronóstico.

Introduction

Central nervous system (CNS) lymphomas are the second most common primary brain tumours after gliomas. The most common histological type is diffuse large B-cell lymphoma (DLBCL). The radiological characteristics of primary lymphomas are widely known and described in the literature, with details of the most common associated radiological features.[1,2] However, in addition to the typical presentation of primary CNS lymphoma (PCNSL), there are other more atypical and far less common forms, which include lymphomatosis cerebri, intravascular lymphoma and dural lymphoma. There are also other lymphoproliferative processes of the CNS associated with Epstein-Barr virus (EBV) infection, with clearly defined histological criteria, such as lymphomatoid granulomatosis (LG) and post-transplant lymphoproliferative disorders (PTLD),[3] which originate from a proliferation of usually EBV-positive large B cells.

This review describes the radiological findings and scenarios in which we may encounter these atypical or rare processes, with the aim of being able to detect them and make an early diagnosis. This would make it possible to start treatments which might improve the prognosis, given that in the majority of cases, the poor prognosis associated with these conditions is related to a late diagnosis.

Lymphomatosis cerebri

Lymphomatosis cerebri (LC) is a rare clinical-radiological manifestation of PCNSL which, in most cases, corresponds to the DLBCL histological subtype. It is characterised by diffuse and extensive infiltration of the brain parenchyma, without formation of well-defined masses or lesions. The term is no longer recommended to identify this form of presentation of lymphoma, as it is not considered a distinct histological subtype of PCNSL, but rather a radiological pattern of involvement.[4] However, this traditional term is still used in the literature as it describes the form of presentation in imaging studies very well. Its prevalence is highest in immunocompetent middle-aged and older adults and it is slightly more predominant in males. Clinically, the most common presenting symptoms are rapidly progressive dementia, behavioural changes and gait disturbances.[5,6] Its poor prognosis is partly due to a delay in diagnosis from clinical onset to histopathological confirmation, as due to its rarity pathology confirmation is necessary.[7]

In MRI studies, on FLAIR and T2-weighted sequences, it appears as a nonspecific pattern of extensive areas of hyperintensity in the subcortical white matter, poorly defined and with minimal associated mass effect. The involvement is usually bilateral and asymmetric, and extends to deep areas of the grey matter and brain stem with typical extension through the corticospinal tracts (Fig. 1). It does not usually show diffusion restriction, although it may occasionally be present. In post-contrast sequences, most lesions do not show contrast uptake, but there may be patchy and linear enhancement foci that reflect the perivascular nature of lymphomas.[3] In the early stages of the disease, areas of uptake are subtle or non-existent, but as the disease progresses and infiltration increases, areas of enhancement become more common due to further deterioration of the blood-brain barrier. In advanced stages, larger nodular solid areas with intense contrast uptake may therefore appear[6,8] (Fig. 2). Differential diagnosis includes diffuse glioma with a gliomatosis pattern, with extensive growth affecting multiple brain lobes, progressive multifocal leucoencephalopathy, infectious processes, vasculitis, and some toxic-metabolic disorders.[6,8]

Intravascular lymphoma

Intravascular lymphoma (IVL) is a rare subtype of extranodal lymphoma characterised by intravascular proliferation of clonal B lymphocytes within small vessels with no invasion of adjacent tissues in most cases. IVL can affect any organ, but the CNS and skin are the most often affected. It can develop at any age, but is most common between the ages of 60 and 70.[3,9] The signs and symptoms are variable and derive from thrombotic phenomena caused by the intravascular proliferation of lymphocytes in different organs, with a rapidly progressive course in many cases.[9] Fever and skin lesions are the most common symptoms, while lymphadenopathy is rare. Due to the wide variety of clinical manifestations, it is unlikely to be suspected clinically, which explains why in most cases the diagnosis is made *post mortem*. Biopsy of affected organs such as the skin, even if they do not show visible lesions, is the method of choice for definitive diagnosis.[10]

The imaging findings are derived from the damage caused by the obstruction of the vessels, which is why most of the time nonspecific ischaemic lesions appear in undefined vascular territories, in the form of hyperintense lesions in T2-weighted sequences with restricted diffusion. Parenchy-

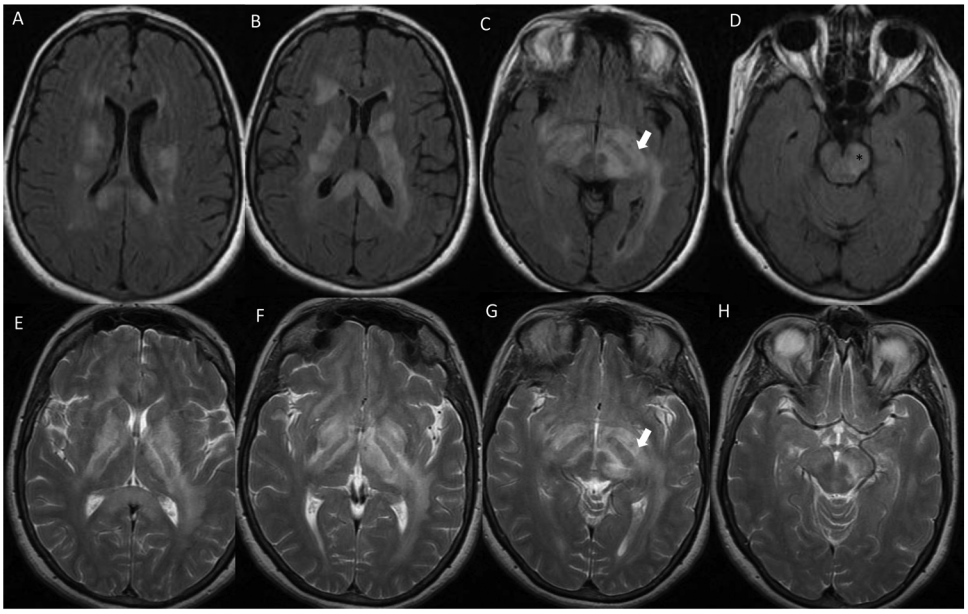

Figure 1 MRI study showing extensive areas of hypersignal in supratentorial white matter in T2-weighted (E–H) and FLAIR (A–d) sequences, with extension through the cerebral peduncles (arrow) to the infratentorial region and infiltration of the brain stem (*). The pattern of involvement corresponds to a LC with a histological diagnosis of DLBCL.

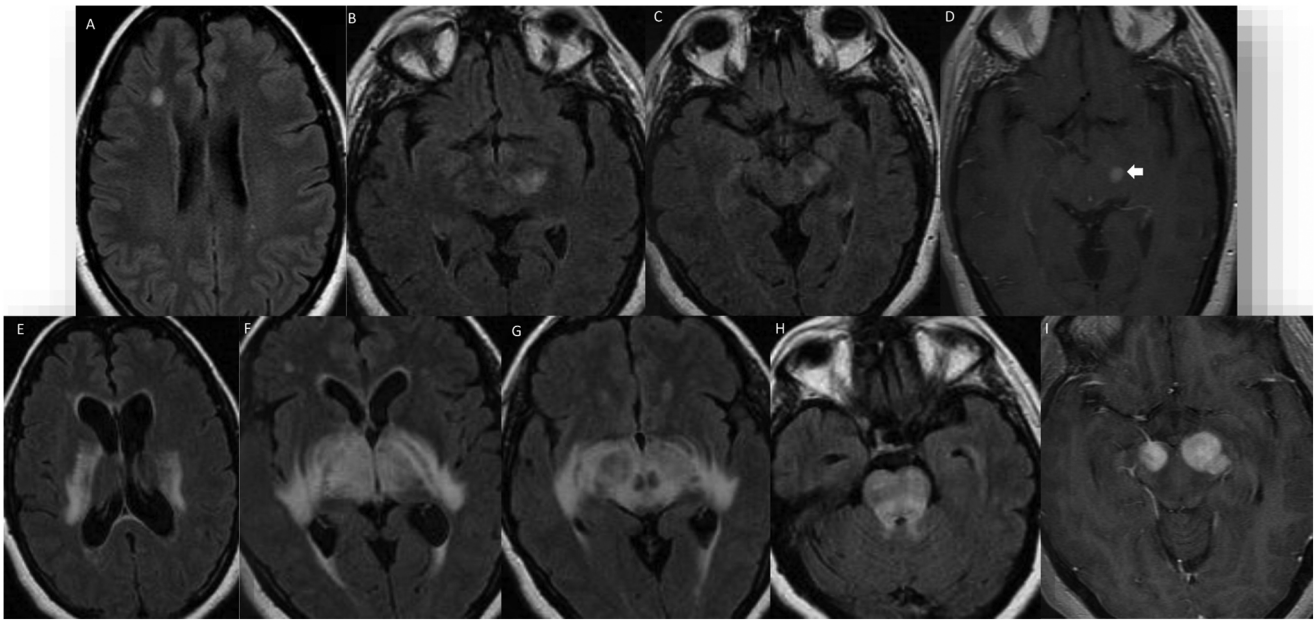

Figure 2 Forty-one-year-old woman with a neurological history of over a year with right faciobrachial paresis. MRI images in the top row (A–D) show the first MRI with hyperintense lesions in FLAIR sequences, non-specific, with extension through the left cerebral peduncle to the midbrain where a small millimetre-sized nodular uptake focus is identified (arrow). Follow-up MRI one year later, bottom images (E–I), shows a clear worsening consisting of signal hyperintensity in T2-weighted and FLAIR sequences affecting both internal capsules and corona radiata and extending through the posterior arm of the internal capsules and through both cerebral peduncles to the entire midbrain. The white matter of both temporal lobes in contact with the ventricular horn is also affected, as well as both optic tracts. In contrast-enhanced sequences there is intense solid uptake of nodular morphology in both cerebral peduncles. Brain biopsy confirmed DLBCL.

mal enhancement after contrast administration is variable, with subtle areas of linear, punctate or more extensive enhancement within areas of ischaemia (Fig. 3). There may be petechial and parenchymal haemorrhages.[9] Although this pattern of ''infarct-like'' lesions is the most common, other patterns have been described on MRI studies including non-specific white matter lesions with patchy and/or diffuse involvement, presence of meningeal enhancement,

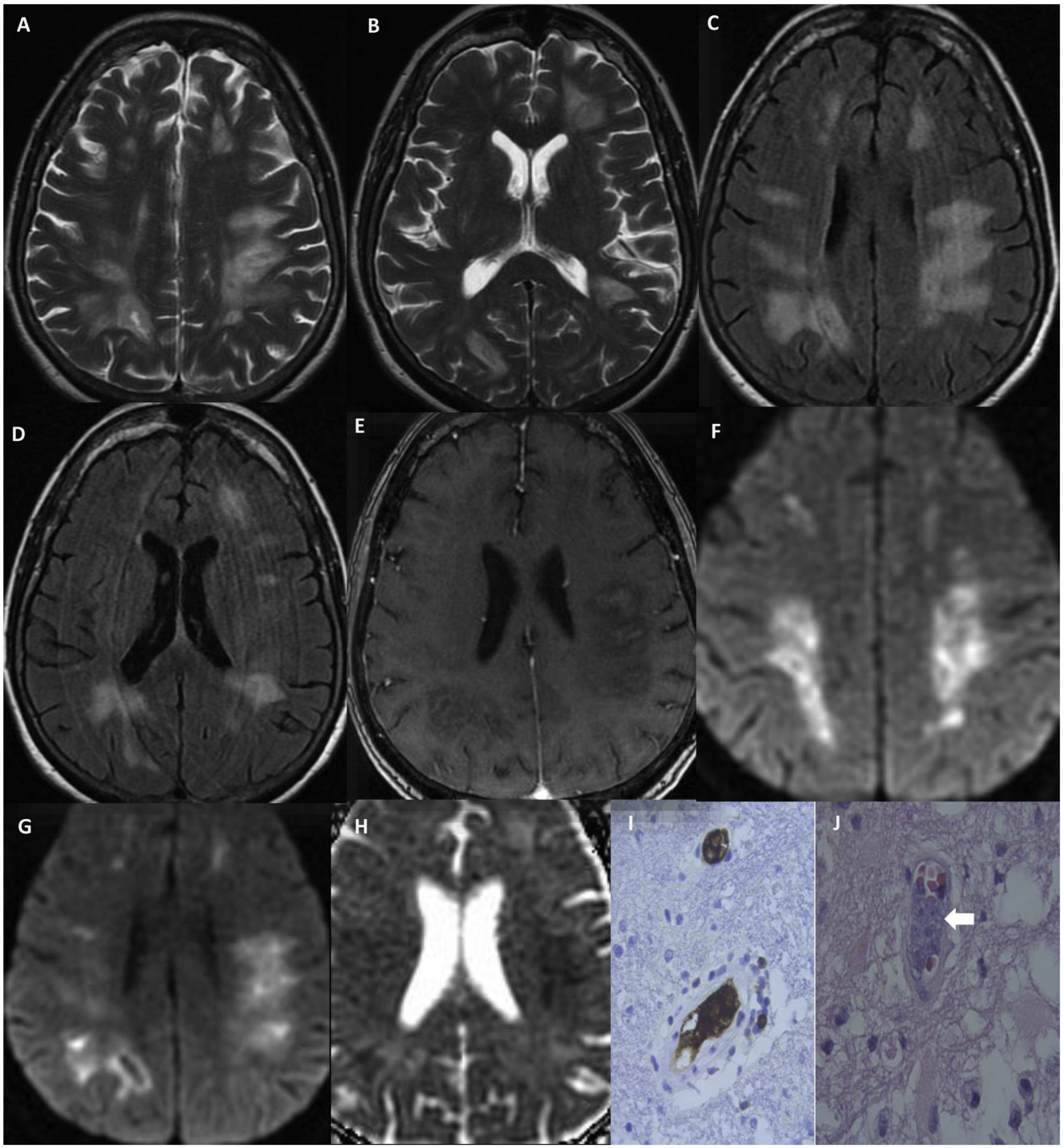

Figure 3 Fifty-year-old woman with progressive gait disturbance associated with subacute onset cognitive impairment of one and a half months' duration. Diagnosed with intravascular lymphoma type DLBCL after brain biopsy. MRI shows multiple ''infarct-like'' lesions affecting the white matter of both convexities with diffusion restriction (F–H) and subtle uptake in some of the lesions (E). The AP images (J) show neoplastic proliferation in the lumen of the small vessels in the form of clusters of atypical cells (arrow) and in the immunohistochemical study (I), the tumour cells express CD45 corresponding to the B phenotype.

mass-like lesions and hyperintense lesions on T2-weighted sequences in the pons. As these findings do not usually exist simultaneously and none of them in isolation is specific, diagnosis is difficult, leading to late diagnosis due to the need for histological confirmation in most cases.[11]

Dural lymphoma

Dural lymphoma (DL) is a very rare subtype of PCNSL belonging to the low-grade marginal zone B-cell lymphomas with a more indolent clinical course.[12] DL originates in the dura

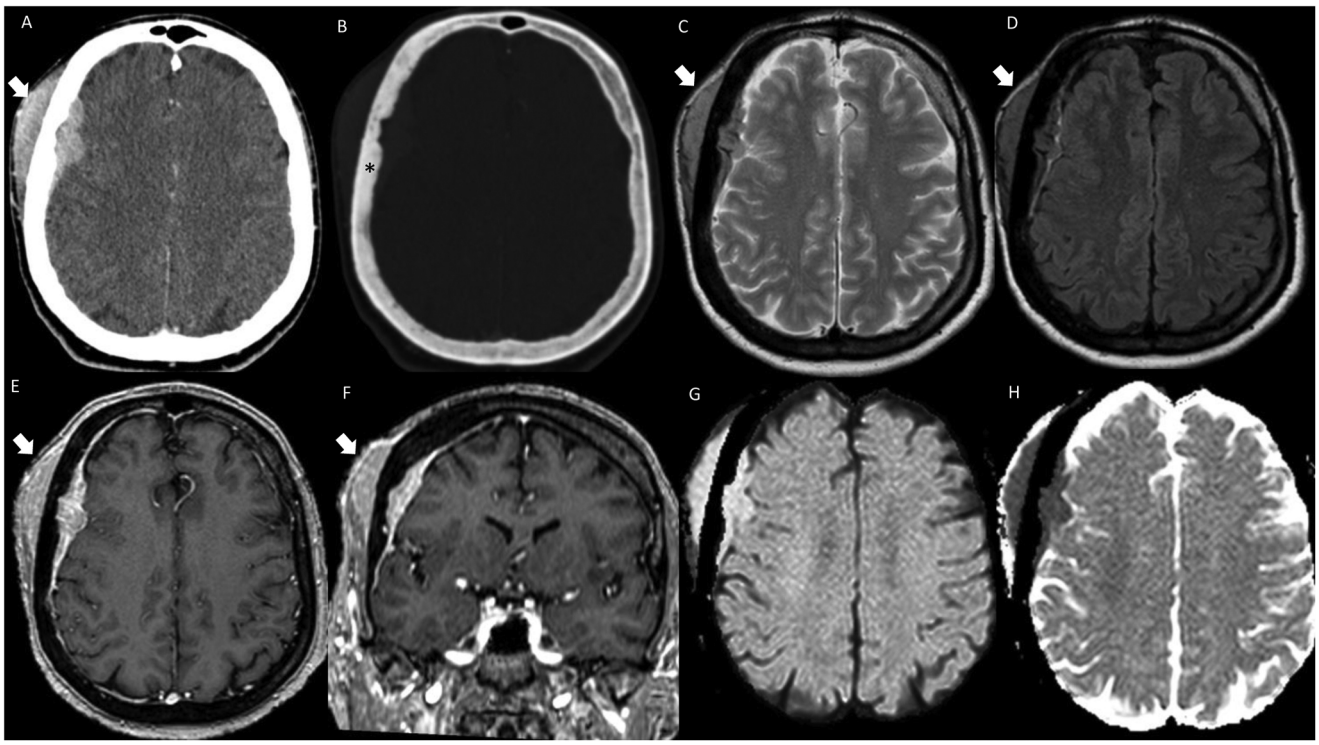

Figure 4 Dural MALT lymphoma. CT and MR images identify a right parietal extra-axial lesion with a wide dural implant base and marked T2 hypointensity (C), showing diffusion restriction (G and H) and intense contrast uptake (E and F). In the CT image with bone window (B), the skull bone is almost normal, only a subtle sclerosis is identified (*), although there is a large tumour component outside the diploe (arrow).

mater and occurs in the absence of parenchymal or systemic disease. Otherwise, if there is disease at other sites, we need to rule out dural involvement being a consequence of dissemination of a systemic lymphoma, and not a primary lymphoma, with a worse prognosis.[13]

On imaging, they appear as single or multiple extra-axial masses with a normally homogeneous and intense contrast uptake. In T2-weighted sequences they show marked hypointensity and they often show restriction in diffusion-weighted sequences (Fig. 4). All these findings make it very difficult to differentiate it from meningioma, which is the most common dural lesion in adults. In DL, unlike meningioma, it is common to find abundant oedema and/or the presence of infiltration of the adjacent brain parenchyma. In addition, there may be bone infiltration or transdiploic extension, in the form of a mass that invades the diploë without causing bone destruction and extends to both sides of the diploë.[1] In contrast, hyperostosis predominates in meningioma.

Lymphomatoid granulomatosis

Lymphomatoid granulomatosis (LG) is a rare angiocentric and angiodestructive lymphoproliferative disease characterised by the presence of EBV-positive B-cells, first described in 1979 by Liebow et al.[14] EBV is closely linked to its origin, and it is postulated that the development of the disease is caused by deficient immune surveillance despite the fact that patients lack a known primary immunodeficiency. There is a large overlap with other EBV-associated lymphoproliferative disorders in immunocompromised patients.[15] With the new WHO classification of haematolymphoid tumours, the diagnosis of LG has become a condition exclusive to immunocompetent patients, as the presence of an immunodeficiency makes it necessary to consider a polymorphic lymphoproliferative disorder subtype conditioned by immunodeficiency, different from LG.[1,15] It is most common in middle-aged adults, between the ages of 40 and 60, with a male predominance of 2:1.[3] The presence of pulmonary involvement is a prerequisite for the diagnosis, which radiologically appears as multiple bilateral cavitated nodules of variable size and predominantly in the middle and lower lung fields. Concomitant organ involvement is in the CNS (40%), skin (34%), kidney (19%) and liver (17%).[15] It is classified into three grades according to the extent of necrosis and the number of EBV-positive B-lymphocytes. Grades 1 and 2 are considered indolent processes and can be managed with corticosteroid therapy, while grade 3 is equivalent to DLBCL and requires aggressive treatment with chemotherapy and/or radiotherapy.[9] Patients with CNS involvement develop confusion, dementia, ataxia, paresis and seizures.[15]

In MRI studies, the form of presentation is highly variable, most often the presence of multiple hyperintense parenchymal lesions in FLAIR/T2-weighted sequences in the hemispheric white matter, deep grey matter and brainstem.[3] They usually show annular, punctate and/or linear enhancement, reflecting the angiocentric nature and perivascular infiltration of these processes (Fig. 5). Another less common pattern is the presence of larger single or multiple infiltrative lesions, often with irregu-

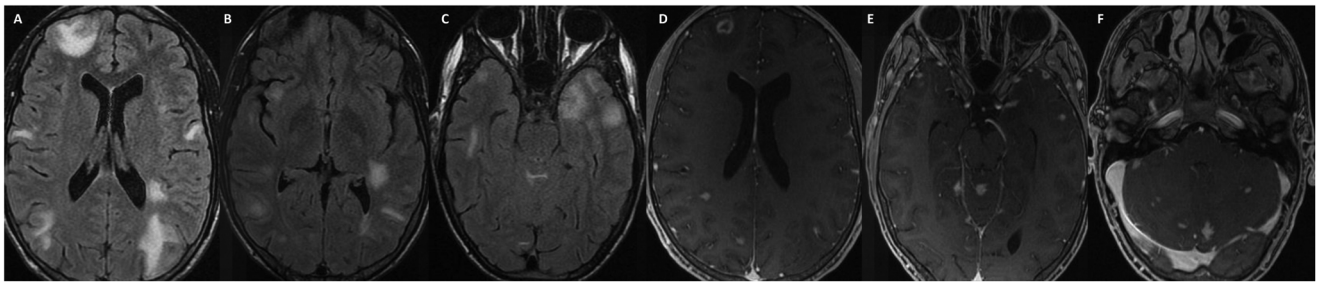

Figure 5 EBV-positive lymphoproliferative process compatible with lymphomatoid granulomatosis grade 2. MRI images show multifocal patchy involvement with hyperintensities in FLAIR sequences (A–C) in the white matter, deep grey matter and infratentorial region. Contrast-enhanced sequences (D and E) show punctate, annular and linear enhancement, indicating angiocentric and perivascular infiltration.

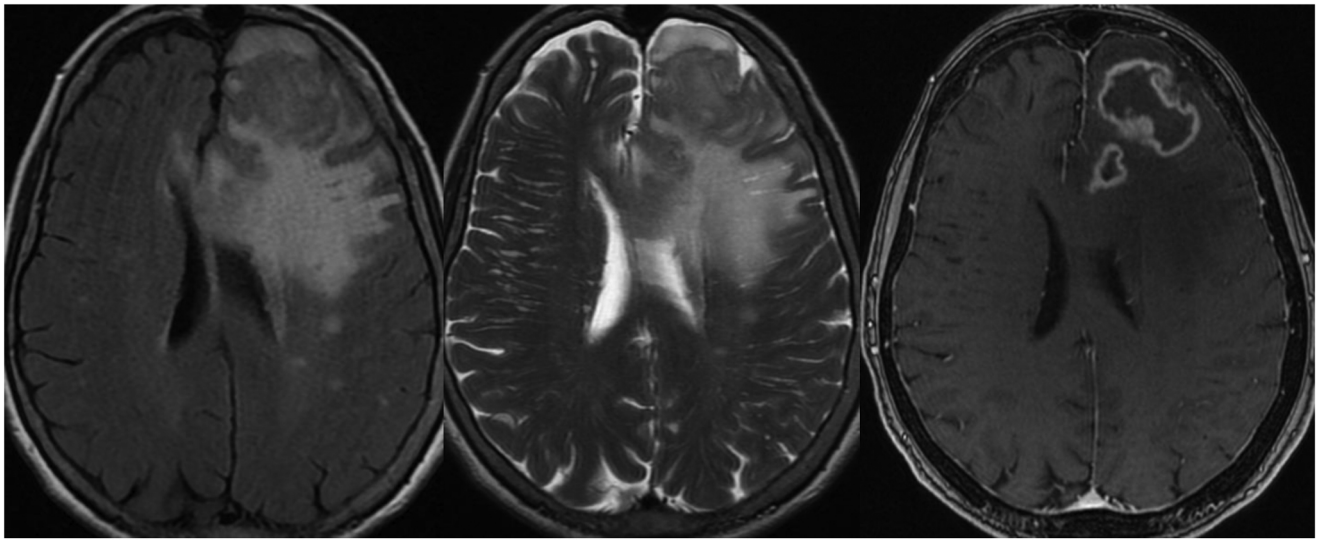

Figure 6 Seventy-year-old man with liver transplantation 10 years earlier, had confusion for previous one and a half months with behavioural changes and gait disturbance with instability. Brain MRI shows two frontal lesions with annular contrast uptake with irregular margins and extensive surrounding vasogenic oedema. Both lesions show marked hypointensity on T2-weighted sequences. After resection of the lesion the histopathology result was a polymorphic EBV-positive post-transplant lymphoproliferative disorder.

lar annular enhancement or solid mass-like uptake.[9] When there is CNS involvement in the form of large infiltrative lesions, it usually corresponds to grade 3 LG, and brain biopsy reveals EBV-positive DLBCL.[1] It is not uncommon to also have leptomeningeal involvement, with this sometimes being the only form of manifestation in the CNS.[9] The differential diagnosis is established with PCNSL, which is often indistinguishable by imaging, and with other diseases such as vasculitis, multiple sclerosis and, in some cases, with gliomas and metastatic lesions.[16] Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) is another condition to be considered in the differential diagnosis. In particular, a close relationship between the two conditions is described based on the hypothesis that CLIPPERS is a pre-lymphoma stage, which may transform into a high-grade lymphoma or lymphoproliferative process, pathophysiologically similar to that occurring in LG.[17] Certain radiological findings have been described that should alert us in patients diagnosed with CLIPPERS, such as gadolinium uptake lesions of unusual size, inhomogeneous uptake or with annular morphology, the presence of subpial, perineural, cortical or leptomeningeal uptake, lesions with mass effect, the presence of extensive signal hyperintensities in T2-weighted sequences, and the lack of response to steroids. The presence of any of these features is alarming and should lead us to suspect a possible transformation to a lymphoproliferative process.[18]

Post-transplant lymphoproliferative disorders

Post-transplant lymphoproliferative disorder (PTLD) of the CNS is a rare disorder caused by immunosuppression following solid organ or stem cell transplantation.[19,20] It is a group of lymphoproliferative diseases occurring in 0.5%–10% of patients, depending on the type of transplantation.[21] It represents a spectrum of processes that includes non-destructive PTLD (plasmacytic hyperplasia, follicular hyperplasia and mononuclear infection), polymorphic PTLD and monomorphic PTLD,[15] which correspond to the vast majority of cases of DLBCL.[19,22] The latest WHO classification (5th ed. of the Classification of Haematolym-

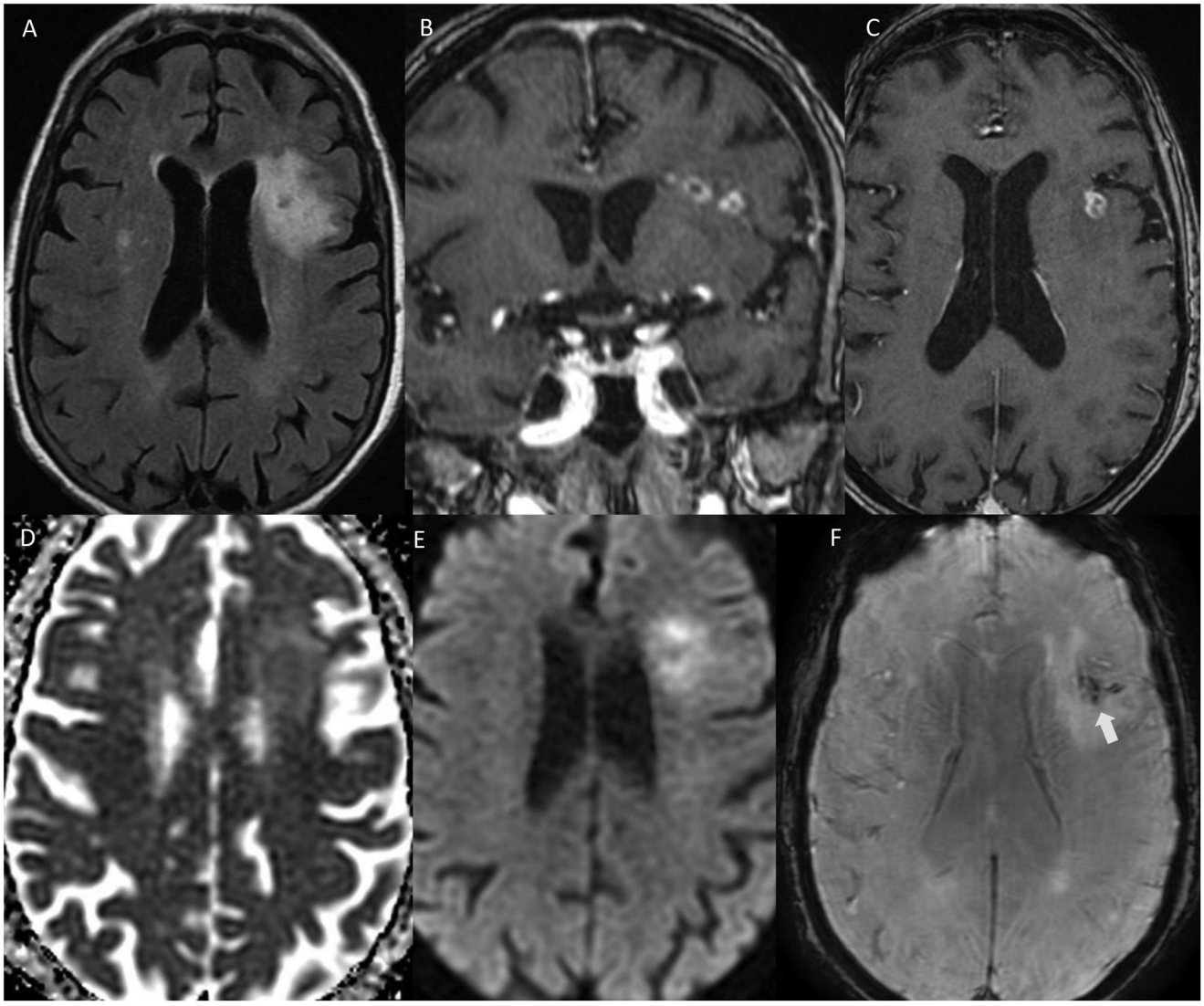

Figure 7 Woman with renal transplant 16 years ago who developed neurological symptoms. Brain MRI confirms left frontal brain involvement with extension into the periventricular region in the form of annular lesions with peripheral diffusion restriction (D and E) and perilesional oedema. The SWI sequence (F) shows a discontinuous annular punctate pattern at the margins of the lesion (arrow). Histopathology study confirmed a transplant-associated EBV-positive lymphoproliferative process.

phoid Tumours) has introduced important changes affecting the classification of immunodeficiency-associated lymphoproliferative disorders. In previous classifications these disorders were distinct entities and were classified according to their association with primary immunodeficiencies, HIV immunosuppression, post-transplant immunosuppression and iatrogenic immunodeficiencies. However, in this new classification, they are included in the group of ''Lymphoid proliferations and lymphomas associated with immune deficiency and dysregulation''.[23] However, in the literature, PTLD continues to be maintained as a distinct entity that includes different lymphoproliferative processes deriving from post-transplant immunosuppression.[21] There is a lower incidence in kidney and liver transplant patients (1%–5%) and a higher incidence in intestine, lung and multiorgan transplant patients (5%–20%).

The organs affected by PTLD are usually the lymph nodes, gastrointestinal tract, lungs and liver,[24] with CNS involvement being more rare and occurring in up to 30% of patients with PTLD.[25] Primary CNS PTLD without systemic involvement is extremely rare. However, with the increase in the number of transplants and the introduction of new immunosuppressive drugs, the incidence exclusively in the CNS has increased. In cases of primary CNS PTLD, the disease course is more aggressive than systemic PTLD with higher morbidity and mortality rates.[24] It can develop in the first year after transplantation, but most cases occur several years later. EBV is detected in 90% of cases[20] and primary infection in the first year after transplantation is a risk factor for the development of lymphoproliferative disease in the first year post-transplantation.[19,22]

The pattern of presentation is in the form of intra-axial lesions, multiple in most cases (61%–83%), predominantly supratentorial with a predominance in the periventricular region and the basal ganglia,[3,20] with the posterior fossa being affected in one third of cases. The lesions are usually

ring-shaped, with central necrosis and abundant surrounding oedema[24] (Fig. 6). On T2-weighted sequences the lesions show the typical signal hypointensity associated with the hypercellularity of lymphoproliferative processes.[3,25] The presence of small punctate foci of signal hypointensity with a peripheral distribution on susceptibility sequences, due to associated haemorrhage, are a relatively common finding, as described in some series (Fig. 7). This finding may be fundamental in the differential diagnosis with other lesions, such as fungal abscesses, which show a continuous and linear SWI hypointense halo or with PCNSL where microhaemorrhages are rarer, making the SWI sequence a fundamental tool in the MRI study protocol.[26]

However, the imaging features are non-specific and very reminiscent of brain metastases. Given that there is a high risk of developing tumours and metastases in transplant patients, it is logical to consider metastases as the main differential diagnosis in a transplant patient with multiple brain lesions. In contrast to PTLD lesions, metastases usually occur at the junctions of the grey matter and white matter and in the bordering vascular areas, with involvement of the deep grey matter and periventricular regions being rare. Another tool that can be of help in the differential diagnosis would be the calculation of cerebral blood volume (rCBV) values in perfusion sequences. Brain metastases are usually highly vascular lesions with high rCBVB values, whereas PTLD lesions do not usually have increased perfusion. Although both types of lesions are diffusion restricted, some series have reported lower apparent diffusion coefficient values for lymphoproliferative lesions.[24,26]

Conclusions

PCNSL are rare, accounting for approximately 6% of all primary brain tumours.[3] In the CNS they most often develop in the form of single or multiple solid lesions with conventional and functional MRI features widely described and validated in the literature, meaning that when this type of lesion is found it is suggestive of the diagnosis. However, there are other less common patterns of presentation of PCNSL which, due to their low incidence, are not usually found in MRI studies, hence the difficulty and delay in diagnosis. Knowledge of these less common forms of presentation, including IVL, LC or DL, helps guide us towards an early diagnosis and facilitates management.

There are also other lymphoproliferative processes that make up a spectrum of disorders related to EBV infection which have different therapeutic management from lymphoma. These are primary CNS conditions or secondary conditions that can affect the CNS as part of systemic involvement. These manifestations are restricted to specific clinical contexts and in patients with particular clinical features and/or history, which facilitates diagnosis.

CRediT authorship contribution statement

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Data analysis and interpretation: not applicable.

Review of the cases: Elena Salvador Álvarez, Agustín Cárdenas del Carré, Zhao Hui Chen and Carmen Lechuga Vázquez.

Literature research: Elena Salvador Álvarez, Agustín Cárdenas del Carré, Zhao Hui Chen and Carmen Lechuga Vázquez.

Drafting of the paper: Elena Salvador Álvarez, Amaya Hilario Barrio and Ana Ramos González.

Draft article: Elena Salvador Álvarez, Ana Ramos González, Zhao Hui Chen and Amaya Hilario Barrio.

Critical review of the intellectual content: Amaya Hilario Barrio and Ana Ramos González.

Final approval of the submitted manuscript: Elena Salvador Álvarez, Amaya Hilario Barrio, Agustín Cárdenas del Carré, Zhao Hui Chen, Carmen Lechuga Vázquez and Ana Ramos González.

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Declaration of competing interest

The authors declare that they have no conflicts of interest.

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