Unusual localization and clinical presentation of primary central nervous system extranodal marginal zone B-cell lymphoma: A case report

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Abstract. Primary central nervous system (CNS) extranodal marginal zone B-cell lymphoma (MZBL) is a rare low-grade non-Hodgkin lymphoma, characterised predominantly by small B cells, plasma cells, monocytoid cells and scattered large immunoblasts. Primary CNS MZBL is a slow-growing tumour that remains localised and is characterised by an excellent clinical prognosis. The present study describes the case of a 48-year-old HIV-negative female patient with a history of head trauma 1 year prior, who presented with worsening neurological symptoms and a magnetic resonance imaging finding of a ~3-cm extra-axial mass within the left lateral ventricle. From histopathology and immunohistochemistry, the lesion was diagnosed as a CNS MZBL; as no other primary lesions were found, the base of the choroid plexuses of the left lateral ventricle was considered the primary site. To the best of our knowledge, the current case is the first study to report on primary CNS MZBL arising in this anatomical site and paves the way for further studies on the role of chronic inflammation (in the present case resulting from trauma) in the pathogenesis not only of primary CNS MZBL but also of lymphoma in general. Additionally, this report could serve as a starting point for studies analysing the role of meningothelial cells in the pathogenesis of primary CNS MZBL.

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Introduction

Marginal zone B-cell lymphoma (MZBL) represents about 7% of all non-Hodgkin lymphomas and is the third most common subtype after diffuse large B-cell lymphoma and follicular lymphoma (1). Extranodal primary MZBL originates from the mucosa-associated lymphoid tissue of several organs, and the stomach is the most frequent site of extranodal involvement (2). Rarely, primary extranodal MZBL may develop in non-mucosal-associated tissue: in this context, central nervous system (CNS) involvement is an extremely rare event. According to the literature data, primary CNS MZBL is usually localised within dura matter and manifests a better prognosis compared to other primary CNS lymphoma subtypes: in fact, most CNS lymphomas are high-grade diffuse large B-cell lymphomas displaying aggressive behaviour, while primary CNS MZBL is characterised by an indolent course and remains localised for a long period of time (2). However, the diagnosis of such an entity is tricky, given the disease rarity, and most cases are initially misdiagnosed in the clinical and on imaging. In fact, several pathologies share overlapping radiological characteristics: meningioma represents the main differential diagnosis, accounting for about 30% of all intracranial tumours (3), but aspergillosis, sarcoidosis, metastases, chloromas, gliomas and schwannomas must also be considered (2). For this

reason, histological examination is essential to correctly and definitively diagnose primary CNS MZBL. In fact, it is mainly characterised by the presence of small B-cell lymphocytes admixed with plasma cells, monocytoid cells, and scattered large immunoblasts (4). B cells are positive for CD20 and show clonal rearrangements of IgH and *k* without expressing CD23, Bcl-6, cyclin D1, CD5, and CD10 (5,6). Tumour cells are also positive for Bcl-2, and 50% of cases are CD43 positive (7). The presence of a low proliferation index confirms the indolent nature of such a neoplasm, and this finding is typical of such pathology.

Multiple genetic abnormalities have been detected in MZBL: chromosomal alterations, most often trisomies of chromosome 3, are occasionally detected (5,8), inactivation of *TNFAIP3* (common in cases with plasmacytic differentiation) and activating *NOTCH2* mutations accompanied by inactivating *TBL1XR1* mutations (common in cases with monocytoid morphology) (9). Here, the first case of a primary CNS extranodal MZBL is presented, arising at the base of the choroid plexuses of the left lateral ventricle.

Case report

In July 2021, a 48-year-old HIV-negative female without a significant medical history (except for a head injury 1 year earlier) presented at the Hospital University Polyclinic 'G. Martino' (Messina, Italy) after having worsening headaches, nausea, vomiting, and dizziness. Neurological examination and routine laboratory tests did not reveal any abnormalities.

The previous year, she underwent a computerised tomography (CT) scan, which showed neither intracranial expansive lesions nor haemorrhages (after a traumatic accident), while the MRI performed after the worsening symptoms revealed an about 3-cm extra-axial mass within the left lateral ventricle with contrast enhancement (Fig. 1).

Clinically, the most likely diagnoses were represented by an ependymoma, a choroid plexus papilloma, an intraventricular meningioma or a metastasis, and less likely by an inflammatory process or a lymphoma.

In November 2021, the patient was admitted to the Division of Neurosurgery of the Hospital University Polyclinic 'G. Martino' (Messina, Italy), where she underwent total resection of the lesion. At intraoperative macroscopic examination, it appeared as a whitish mass with vascularised areas and a hard-friable consistency (Fig. 2). After the excision, the lesion shrank into multiple fragments that were fixed in 10% neutral formalin for 24-36 h at room temperature, embedded in paraffin at 56˚C, and then cut into 5 μm thick serial sections for routine haematoxylin/eosin histological staining and Congo red stain. Moreover, parallel sections were cut and mounted on silane coated glass for immunohistochemistry, then dewaxed in xylene and rehydrated in graded ethanol. The immunohistochemical procedure was performed using the automated Ventana BenchMark ULTRA platform with Cell Conditioning 1 for 64 min, pre-peroxidase inhibition, and primary antibody incubation for 16 min at 37˚C. The OptiView DAB IHC Detection Kit (Ventana Medical Systems, Inc.) was used to detect protein expression of the primary antibodies shown in Table I. Finally, all slides were counterstained with Hematoxylin II (Ventana Medical Systems, Inc.) and Bluing Reagent (Ventana Medical Systems, Inc.) for 4 min at room temperature. To ensure the reliability of the results of the immunohistochemical reactions, external positive and negative controls were run according to the manufacturer's instructions. Of note, immunohistochemical analyses were performed on parallel sections from the same paraffin-embedded tissue.

Microscopic examination showed a diffuse proliferation of lymphoplasmacellular elements within the fragments of a choroid plexus rich in fibrosclerotic tissue, also with the presence of amorphous material deposits (Fig. 3A-D). Three main pathological pictures represented the main diagnostic hypotheses: first of all, a chronic/reactive post-traumatic inflammatory process, rich in plasma cells; secondly, a hereditary transthyretin amyloidosis, which usually has an adult-onset and is characterised by the deposition of a misfolded transthyretin produced by the choroid plexus and its consequent accumulation in the leptomeninges (10); lastly, an IgG4-related disease. However, the negative Congo red stain and non-specific positivity of plasma cells for IgG and IgG4, together with normal serum IgG4 levels, ruled out the latter two diagnostic hypotheses (IgG and IgG4 immunohistochemical staining is shown in Fig. S1). A post-traumatic inflammatory process represented the only plausible diagnostic hypothesis, but the morphology and distribution pattern of the inflammatory elements were not univocally attributable to such a process. In fact, the lymphoplasmacellular infiltrate was mainly represented by monomorphic small B lymphocytes with a marginal growth pattern and marked plasma cell differentiation (Fig. 3A-D). Immunohistochemical examination revealed positivity for CD20 and Bcl2 in small B lymphocytes (Fig. 3E and F) and CD138 positivity (Fig. 3G) with *k* clonal restriction in plasma cells (Fig. 3H).

CD23, Bcl-6, cyclin D1, SOX-11, CD5 and CD10 were all negative (Fig. S2; immunoreactivity for each antibody is reported in Table I) and the ki-67 proliferation index was very low (5-10%, Fig. 4). On this basis, a lymphoproliferative process represented the only possible diagnosis, both a CNS localisation of a systemic disease or a primitive CNS lymphoma. Of note, the cytological analysis of cerebrospinal fluid and bone marrow biopsy with flow cytometry and cytogenetics showed no evidence of lymphomatous infiltration (data not shown), and CT and positron emission tomography (PET) scans of the thorax, abdomen and pelvis confirmed the absence of systemic disease (Fig. S3 shows the negative PET scan of the thorax, abdomen and pelvis). Therefore, it was a primitive CNS lymphoma. Differential diagnoses included a small lymphocytic lymphoma/chronic lymphocytic leukaemia (CLL/SLL), lymphoplasmacytic lymphoma, follicular lymphoma, mantle cell lymphoma and marginal zone lymphoma. CLL/SLL was unlikely due to the absence of peripheral blood or bone marrow involvement and negativity for CD5 and CD23. Similarly, in the absence of bone marrow involvement, negative PET scan and no clinical history of Waldenstrom's macroglobulinemia with the absence of both *MYD88* mutation and clonal circulating IgM by serum protein electrophoresis and immunofixation electrophoresis (data not shown), lymphoplasmacytic lymphoma involvement was unlikely. Moreover, the lack of an overt morphological follicular appearance and CD10 negativity made follicular lymphoma unlikely.

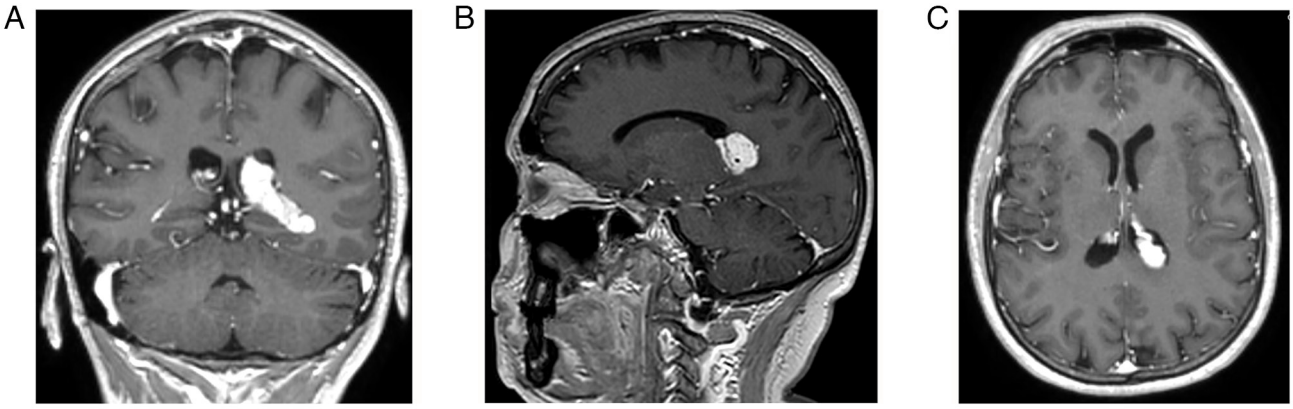

Figure 1. Magnetic resonance imaging images showing a ~3-cm extra-axial mass within the left lateral ventricle with contrast enhancement in (A) coronal, (B) sagittal and (C) axial planes.

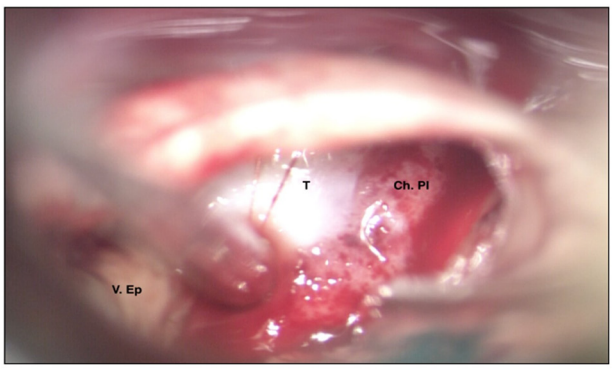

Figure 2. Intraoperative photo of the tumour, appearing as a whitish lesion with vascularised areas and a hard-friable consistency. T, tumour; V. Ep., epidural vein; Ch. Pl., choroid plexus.

Similarly, negativity for cyclin D1, SOX-11 and CD5 ruled out mantle cell lymphoma. Therefore, a primary CNS MZBL arising at the base of the choroid plexuses of the left lateral ventricle represented the only possible diagnosis. In this diagnostic challenge, considering the unusual anatomical site for the localisation of such a lymphoproliferative process, a polymerase chain reaction (PCR) analysis of B lymphocyte clonality was performed. A monoclonal population of B lymphocytes with a monoallelic rearrangement of the *IGH* gene, FR2/JH, FR3/JH fragments and the *k* gene was identified by testing for immunoglobulin heavy chain (IGH) and kappa light chain gene (IGK) rearrangement using the PCR developed by the collaborative European BIOMED-2 Concerted Action Study Group. The monoallelic rearrangement was typically characterised by the presence of a single peak (11), as in the case of the IGH gene, FR2/JH fragment (Fig. S4), in which the neoplastic elements showed a dominant fluorescent peak indicative of a clonal population with identical PCR fragment sizes at ~128 bp. Therefore, molecular studies corroborated the diagnosis of a primary CNS MZBL with *k* plasma cell differentiation. However, we currently do not have access to all PCR result graphs since they are no longer visible in our digital archive, and this issue can represent a limitation of our study. Nonetheless, all missing PCR results can be retrieved by our hospital informatics staff upon reasonable request. After the diagnosis, the patient underwent standard clinical treatment for primary CNS extranodal MZBL, receiving whole-brain external beam radiotherapy with a total dose of 24 Gy, and follow-up CT (shown in Fig. S5) and MRI (not shown) showed the disappearance of the gross disease (neither a mass effect nor an abnormal enhancement was evident). She has also undergone a strict follow-up to exclude treatment-related toxicities, particularly regarding head and neck irradiated structures. Specifically, thyroid function tests showed no signs of hypothyroidism (data not shown). There were also no signs of treatment-related neurotoxicity (like impaired cognitive function), xerostomia from parotid irradiation and cataracts, or dry eye from orbital irradiation. Currently, she has been alive and without symptoms for 12 months up to the date of this report. The patient performed all follow-up examinations privately, and therefore these were not available to us at the moment of writing the manuscript. However, they have been viewed by the neurosurgeons who managed her (MC and FA). The corresponding author could request all the missing examinations from the patient and provide them to anyone who asks upon reasonable request.

Discussion

Extranodal MZBL arises from mucosa-associated lymphoid tissue and has been described in association with specific chronic inflammatory processes, both infectious and autoimmune, such as within gastric mucosa colonised by *H. pylori* and within lacrimal glands in Sjögren syndrome (3,4). However, the CNS is devoid of mucosa-associated lymphoid tissue, and the exact pathogenesis of primary CNS MZBL is poorly understood (12). In most cases, primary CNS MZBL is not directly linked to any infectious or autoimmune condition and is, in fact, most frequently diagnosed in immunocompetent middle-aged females, consistent with our patient's presentation (12). The possible mechanisms underlying the pathogenesis of CNS MZBL could be ascribed to an implantation metastasis of undetected or vanishing meningeal MZBL (13,14), a localisation of an undetected primary extracranial MZBL, or chronic inflammation. This last condition could derive from a direct chronic antigenic stimulation of the dura, for example, from a long-standing history of

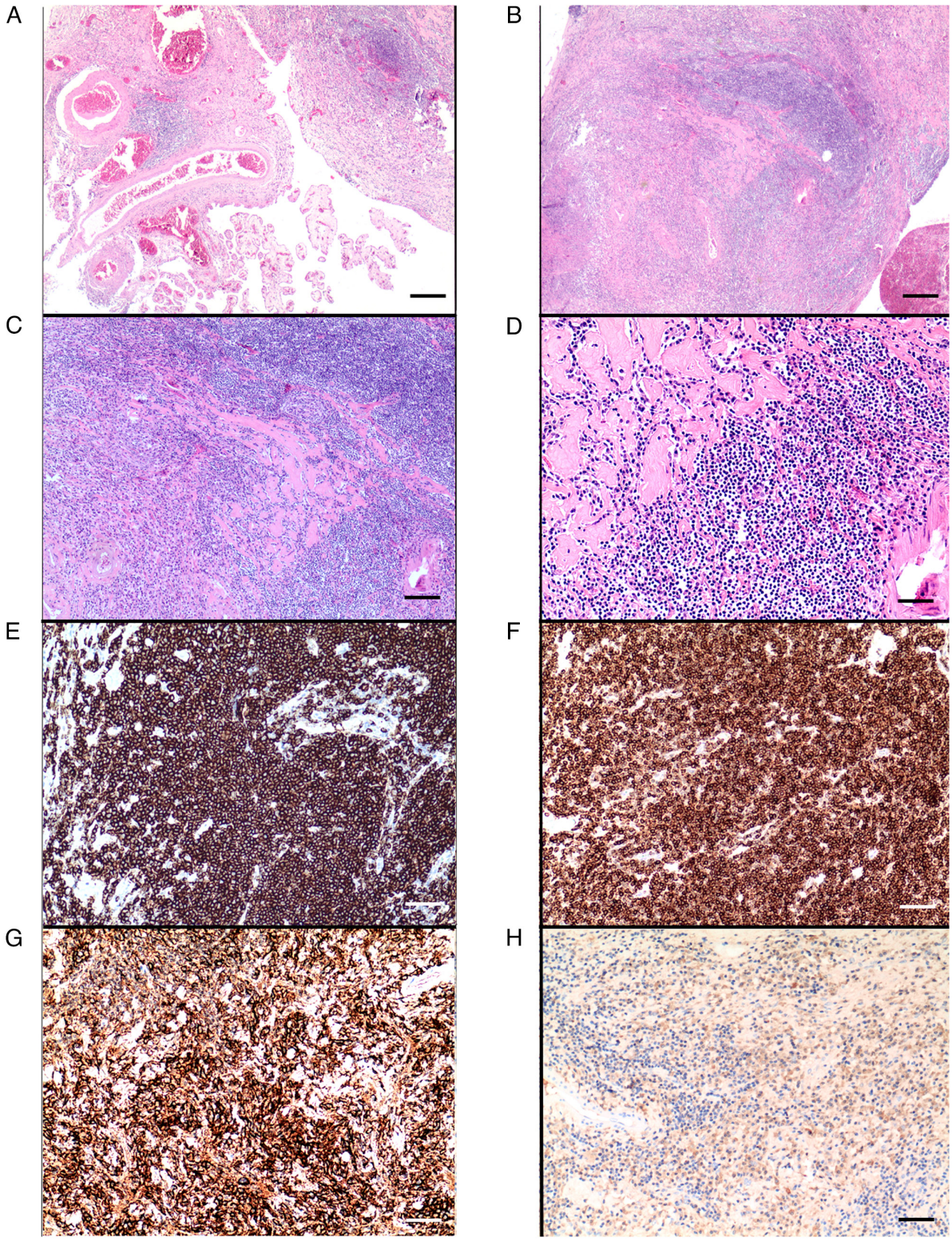

Figure 3. (A-D) Haematoxylin and eosin staining showing (A) fragments of a choroid plexus (x20; scale bar, 420 μm) infiltrated (B) by a diffuse proliferation of lymphoplasmacellular elements with a marginal growth pattern (x20; scale bar, 420 μm), intermingled (C) with fibrosclerotic tissue (x40; scale bar, 210 μm; the same area is shown at different magnifications in B and C). (D) Small monomorphic B lymphocytes, fibrosclerotic bands and deposits of amorphous material are particularly evident at higher magnification (x100; scale bar, 84 μm). (E-H) Immunohistochemical staining showing a neoplastic B-lymphocyte population with diffuse positivity for (E) CD20 (x100; scale bar, 84 μm) and (F) Bcl2 (x100; scale bar, 84 μm). The marked plasma cell differentiation is evidenced by (G) CD138 positivity (x100; scale bar, 84 μm) with (H) k clonal restriction (x100; scale bar, 84 μm).

untreated HBV (15,16) or HCV (17). One patient with CNS MZBL had a long history of white matter disease with some features of multiple sclerosis (5), and another suffered from a *Chlamydia psittaci* infection (18). Moreover, an association between primary intracranial MZBLs and IgG4 expression was observed by Venkataraman *et al* (8) in a series of 32

Table I. List of antibodies used and their immunoreactivity in our case.

Antibody	Manufacturer	Catalogue number	Clone	Dilution	Immunoreactivity in our case
Bcl2	Ventana	790-4604	SP66	Pre-diluted	Positive in neoplastic small B lymphocytes
CD20	Ventana	760-2531	L26	Pre-diluted	Positive in neoplastic small B lymphocytes
CD138	Ventana	760-4248	B-A38	Pre-diluted	Positive in clonal plasma cells
Kappa	Ventana	760-2514	Polyclonal	Pre-diluted	Positive in clonal plasma cells
Lambda	Ventana	760-2515	Polyclonal	Pre-diluted	Negative in clonal plasma cells
IgG	Ventana	760-2653	Polyclonal	Pre-diluted	Non-specific positivity of plasma cells
IgG4	Ventana	760-4614	MRQ-44	Pre-diluted	Non-specific positivity of plasma cells
Bcl6	Ventana	760-4241	GI191E/A8	Pre-diluted	Negative in neoplastic small B lymphocytes
CD23	Ventana	790-4408	EP3093	Pre-diluted	Negative in neoplastic small B lymphocytes
Cyclin D1	Ventana	790-4508	SP4-R	Pre-diluted	Negative in neoplastic small B lymphocytes
SOX-11	Ventana	760-4868	MRQ-58	Pre-diluted	Negative in neoplastic small B lymphocytes
CD5	Ventana	790-4451	SP19	Pre-diluted	Negative in neoplastic small B lymphocytes
CD10	Ventana	790-4506	SP67	Pre-diluted	Negative in neoplastic small B lymphocytes
Ki-67	Ventana	790-4286	30-9	Pre-diluted	5-10% of neoplastic small B lymphocytes

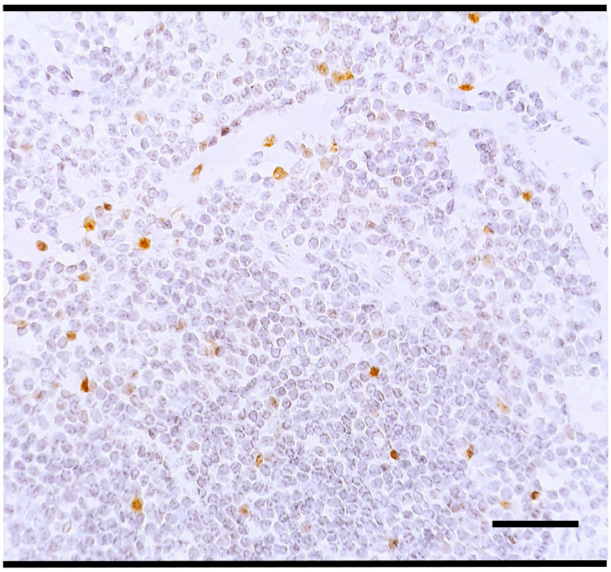

Figure 4. Ki67 immunostaining highlights 5-10% of proliferating neoplastic elements (x400; scale bar, 75 μm).

dural-based MZBLs, a significant subset of which showed IgG4 in light-chain-restricted clonal plasma cells.

In our case, the above-mentioned mechanism of chronic inflammation, consequent to a previous head injury, could have represented the pathogenetic substrate of MZBL onset: in fact, chronic inflammation is associated with the genesis of organised lymphoid tissue, and it has been demonstrated that the combination of persistent BCR triggering, chronic T-cell help and TLR stimulation elicited by chronic inflammation in these ectopically formed lymphoid tissues can be overruled by genetic alterations that guarantee constitutive NF-κB signalling. Thus, cell becomes less dependent on the environmental stimuli, therefore predisposing to MZBL development (19). It has also been hypothesised that meningothelial cells of the arachnoid membrane are comparable to epithelial cells in those organs in which typically MALT lymphomas arise. These cells are ubiquitous in the arachnoid membrane and are mainly concentrated not only in the arachnoid granulations but also in the choroid plexus and subarachnoid cisterns (20). Therefore, this theory could well explain primary CNS MZBLs arising extra-axially (21,22).

To our knowledge, this is the first literature-reported case of a primary CNS MZBL arising at the base of the choroid plexuses of a lateral ventricle; in fact, even the most recent literature data do not report anything similar at this anatomical site (14). Moreover, our case is really peculiar because it is related to a history of previous trauma and chronic inflammation, confirming what has already been demonstrated about the role of chronic inflammation in the pathogenesis of primary CNS MZBLs. Additionally, it could represent a starting point for studies analysing the role of meningothelial cells in the pathogenesis of primary CNS MZBL. Given the singularity of our case, it would have been useful to exploit whole slide imaging (WSI) systems to quickly share histological slides with international experts in the field and also to preserve high-quality images in a digital archive. However, we currently do not have WSI scanners at our Institution, and the absence of images in WSI format represents a limitation of our study. Nonetheless, our experience underlines the therapeutic and prognostic importance of a correct diagnosis of primary CNS MZBL since it is a particularly radiosensitive low-grade disease with an indolent clinical course, an excellent prognosis, and is effectively treatable with localised radiotherapy.

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Availability of data and materials

The datasets used and/or analysed during the present study are available from the corresponding author on reasonable request.

Authors' contributions

VF and MM confirm the authenticity of all the raw data. VF, MM and LML conceptualised the manuscript. CP, ML and AI made substantial contributions to the conception and design of the manuscript. VF wrote the original draft. MC and FA managed the patient. VF, MM, FP and LML participated in the pathological evaluation. CP, ML, MC and FP retrieved data from the literature. FA, GT and GF made substantial contributions to the acquisition of clinicopathological data and their analysis and interpretation. AI, FA, GT and GF reviewed and edited the manuscript. LML, GT and GF supervised. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient's consent for publication

Written informed consent was obtained from the patient for publication of this case report.

Competing interests

The authors declare that they have no competing interests.

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