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Case Report

Dural lymphoma misdiagnosed as subdural hematoma following head trauma after an episode of syncope ☆

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ABSTRACT

Primary dural lymphoma is a rare subtype of primary central nervous system lymphoma. Primary dural lymphoma may be radiologically misdiagnosed as it shares similar imaging characteristics with several pathologies, including meningiomas and subdural or epidural hematomas. We present a patient who was originally diagnosed with a subdural hematoma following a syncopal episode on computed tomography. Follow-up magnetic resonance imaging of the brain demonstrated heterogeneously enhancing dural-based mass overlying the left frontoparietal convexity associated with bidirectional dural tails, suggestive of a malignant meningioma. Neurosurgical histopathology revealed marginal zone B-cell lymphoma. This case represents the potential difficulty in diagnosing primary dural lymphoma, especially in the setting of uncertain clinical history and obscured imaging features.

Introduction

Marginal zone B-cell lymphoma (MZBCL) is an uncommon subset of primary central nervous system lymphoma (PCNSL). As the name suggests, PCNSL is restricted to the central nervous system (CNS) – brain, leptomeninges, cranial nerves, spinal cord, and the intraocular compartments. MZBCL is classified as a mature B-cell neoplasm of extranodal marginal zone lymphoma of mucosa-associated lymphoid tis-

Abbreviations: MZBCL, Marginal zone B-cell lymphoma; PCNSL, Primary central nervous system tumor; CNS, Central nervous system; ED, Emergency department; PCP, Primary care physician; MR, Magnetic resonance; CT, Computed tomography; FLAIR, Fluid-attenuated inversion recovery; DWI, Diffusion-weighted imaging; ADC, Apparent diffusion coefficient; ^{18}F-FDG PET/CT, 2-deoxy-2-[fluorine-18]fluoro-D-glucose integrated with computed tomography; PDL, Primary dural lymphoma; SPECT, single-photon emission computed tomography.

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sue. MZBNL appears to arise from the meninges and is therefore sometimes misdiagnosed as meningioma or an intracranial bleed [1]. We present a patient who presented to the emergency department (ED) with a radiologic findings suggestive of a subdural hematoma following loss of consciousness who was later diagnosed with pathology-proven MZBCL.

Case presentation

A 70-year-old immunocompetent female with no significant past medical history presented to the ED after stating she experienced a sudden episode of loss of consciousness approximately 3-4 weeks ago. The patient became unresponsive and later lethargic for 15 to 30 minutes according to the husband who witnessed the event. The patient denied head trauma but reported an episode of bowel incontinence. Approximately two weeks after the event, the patient visited her primary care physician (PCP) since she continued to experience mild and persistent lightheadedness and dysgraphia. The PCP ordered magnetic resonance (MR) imaging of the brain at an outside hospital which revealed a subacute subdural collection over the left cerebral convexity measuring at least 1.5 cm associated with a 2-3 mm rightward midline shift and vasogenic edema. The PCP recommended the patient go to the ED for further workup.

At presentation in the ED, the patient denied headache, neck or back pain, vision or hearing changes, changes in speech, numbness or weakness, vertigo, nausea/vomiting, palpitations, fevers, or chills. On physical examination, the patient was hemodynamically stable and afebrile with mild right pronator drift but otherwise neurologically intact. Laboratory evaluation of complete blood count, basic metabolic panel, and coagulation tests did not reveal any abnormalities. Neurosurgery was consulted and recommended a computed tomography (CT) scan of the brain.

CT scan of the brain without contrast (Fig. 1) demonstrated a fluid collection within the left frontoparietal convexity measuring 1.6 cm in its greatest dimension, suggestive of a subdural hematoma. Left frontoparietal vasogenic edema was present with subsequent sulcal effacement and approximately a 3 mm of rightward midline shift. The basal cisterns and foramen magnum remained patent. No depressed calvarial fractures were noted, but a left frontoparietal scalp swelling was present, consistent with a scalp hematoma.

Given the CT findings, a follow up MR imaging of the brain with intravenous gadolinium contrast (Fig. 2) was ordered which demonstrated a heterogeneously enhancing dural-based mass overlying the left frontoparietal convexity with extension and effacement of the adjacent sulci measuring approximately 8.2 × 1.6 × 4.8 cm (anteroposterior by transverse by craniocaudal). There was thickening and enhancement of the dura extending anteriorly and posteriorly along the frontoparietal convexity. The mass was seen invading into the overlying calvaria. There was redemonstration of sulcal effacement and rightward midline shift secondary to the frontoparietal edema. The basal cisterns and foramen magnum remained patent. Subtle osseous remodeling of the overlying calvarium was present along with variable fluid-attenuated inversion recovery (FLAIR) signal hyperintensity and heterogenous contrast-enhancement. The mass demonstrated hyperintensity on diffusion weighted imaging (DWI) and hypointensity on apparent diffusion coefficient (ADC). Given these imaging findings, the mass was concerning for a malignant meningioma.

Neurosurgery brought the patient to the operative room for a left-sided craniotomy with tumor resection/debulking. Areas with extensive tumor infiltration were avoid to prevent underlying brain ischemia secondary to devascularization of the pial vessels. Neuronavigation was used to confirm the extent of the tumor within the resection margin. The resected portions of the brain were sent for pathological examination. Staining with hematoxylin and eosin showed diffuse infiltration of the dura by numerous atypical small lymphocytes with irregular, angulated, or twisted nuclear contours, punctate chromatin, small or inconspicuous nucleoli, and cleared-out cytoplasm that suggested a monocytoid appearance. The infiltrate was monotonous throughout most of the specimen, and a few large-scale lymphoid architectural features were apparent apart from some areas which contained follicles. Immunohistochemical stains revealed atypical lymphocytes that were strongly and diffusely positive for CD20 and BCL2 and negative for CD10, BCL6, and cyclin D1. The BCL6 stain revealed several variably sized germinal centers that were negative for BCL2. Stains for CD3, CD5, and CD43 marked abundant of background T cells. Ki-67 positivity was 80%-90% within the germinal centers and was <5% elsewhere. Follow-up polymerase chain reaction testing of the specimen demonstrated positive monoclonal immunoglobulin heavy chain and immunoglobulin κ light chain gene rearrangements indicating the presence of a monoclonal B cell lymphoproliferation.

MR imaging of the brain with contrast (Fig. 3) demonstrated interval post-surgical changes and resection with of the heterogeneously enhancing left dural-based mass with small residual enhancing tissue in the margins of the surgical cavity and underlying left cortical sulci. Hyperintense signal in the underlying frontoparietal parenchyma with associated mass effect and a 4 mm rightward midline shift at the level of the septum pellucidum was noted.

The patient was seen by radiation oncology and neurooncology. A 2-deoxy-2-[fluorine-18]fluoro-D-glucose integrated with computed tomography (^{18}F-FDG PET/CT) was negative for systemic disease. A bone marrow biopsy was normal. The patient was diagnosed with stage 1E dural-based MZBCL. The patient received rituximab weekly for 4 weeks followed by radiotherapy (3000 cGy in 20 fractions). The patient did well with her treatment and remains disease-free approximately 1 year following the initial presentation.

Discussion

PCNSL, which is most commonly diffuse large B-cell lymphoma, is an extranodal non-Hodgkin's lymphoma. Primary dural lymphoma (PDL) is a rare subtype of PCNSL. PCNSL rarely affects immunocompetent patients and represents approximately 3% of intracranial neoplasms [2]. It is more commonly seen in immunocompromised patients, such as acquired im-

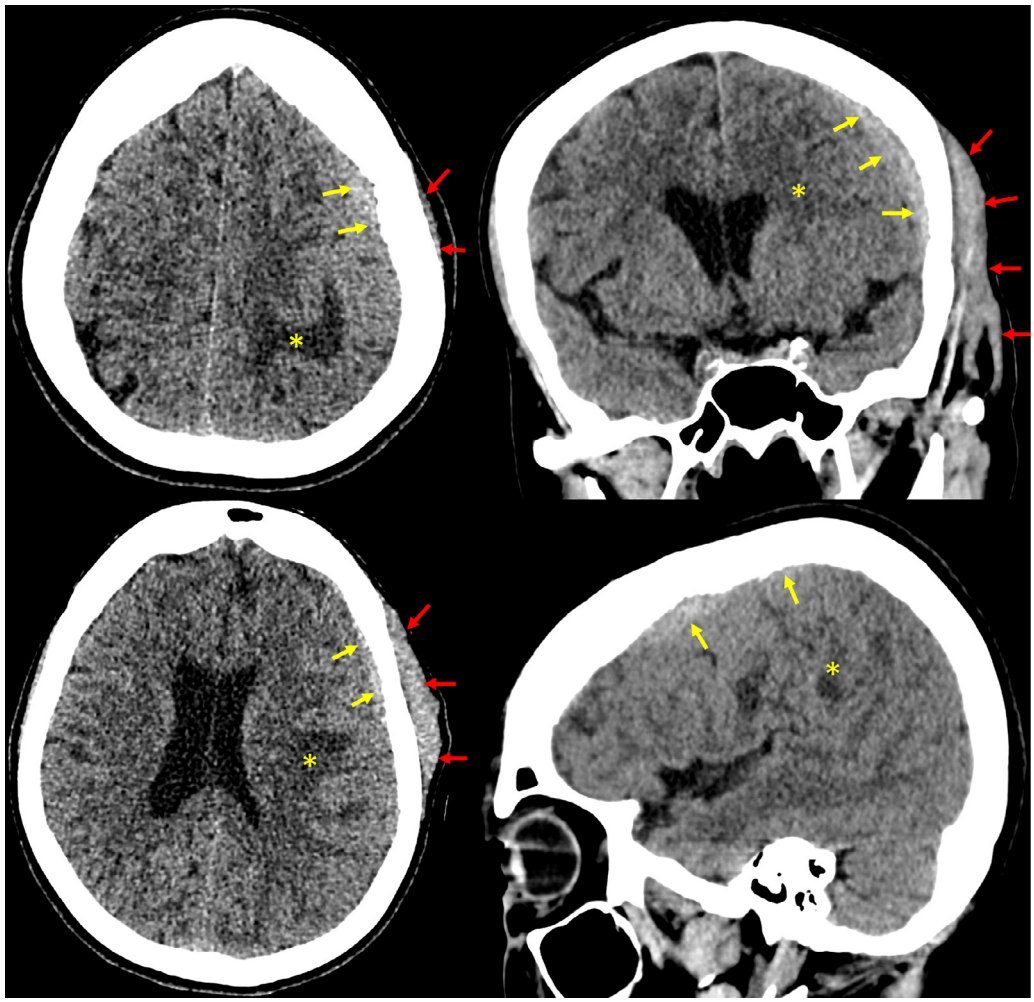

Fig. 1 – CT scan of the brain without contrast at presentation demonstrating a left frontoparietal convexity (yellow arrows) consistent with findings suggestive of a subdural hematoma with associated vasogenic edema (yellow asterisk). Scalp swelling due to a hematoma is seen overlying the left frontoparietal scalp (red arrows).

mune deficiency syndrome or solid organ transplant recipients [3,4]. MZBCL of the dura in an immunocompetent patient is an even rarer phenomenon [4].

PCNSL may have a variable presentation with most patients reporting to be symptomatic at initial presentation. Most frequently, reported symptoms are secondary to tumor mass effect, such as headaches, cranial nerve deficits, limb weakness or dysesthesia, gait instability/vertigo, word-finding difficulty or confusion, or a painless subcutaneous mass [5]. Our patient initially had a syncopal episode which may be attributed to seizure activity given her episode of concurrent bowel incontinence. The mass effect on the left parietal lobe may be the cause of the patient's peristent lightheadedness and dysgraphia.

Diagnosis is often delayed as PCNSL may resemble other pathological conditions such as subdural hematomas (on nonenhanced CT) or en plaque meningiomas (on enhanced CT) [5–10]. Most patients will receive neuroradiologic imaging shortly after presentation given their neurologic symptomatology. CT of the brain will commonly reveal a single or multifocal irregularly- or crescent-shaped, extra-axial, vividly enhancing, high-density mass compared to the brain parenchyma [5,6,11,12]. The hyperdensity is often attributed to the highly cellular nature of the lesions [12]. There is often an associated hypodense area adjacent to the lymphoma which represents vasogenic edema [5,6,11,12].

MR imaging of the brain corroborates the CT findings of an irregular or crescent-shaped, extra-axial mass [5,12]. These masses may also be lobulated in appearance [12,13]. In addition, the mass often appears as a broad-based dural attachment following dural reflections, most notably along the falx cerebri, tentorium cerebelli, and parasellar regions [10]. Lesions avidly enhance with intravenous contrast. The mass is seen as isointense to grey matter on T1-weighted imaging and isointense to hypointense on T2-weighted imaging [5,12]. Most dural lymphomas displace the cortical gray matter inwardly and are often associated with an adjacent T2-weighted/FLAIR signal hyperintensity suggestive of vasogenic edema. DWI sequences often reveal restricted diffusion secondary to high cellularity. The mass may contain a dural tail, which is seen as enhancement and thickening of the dura [5,13]. Bone and leptomeningeal infiltration has been reported.

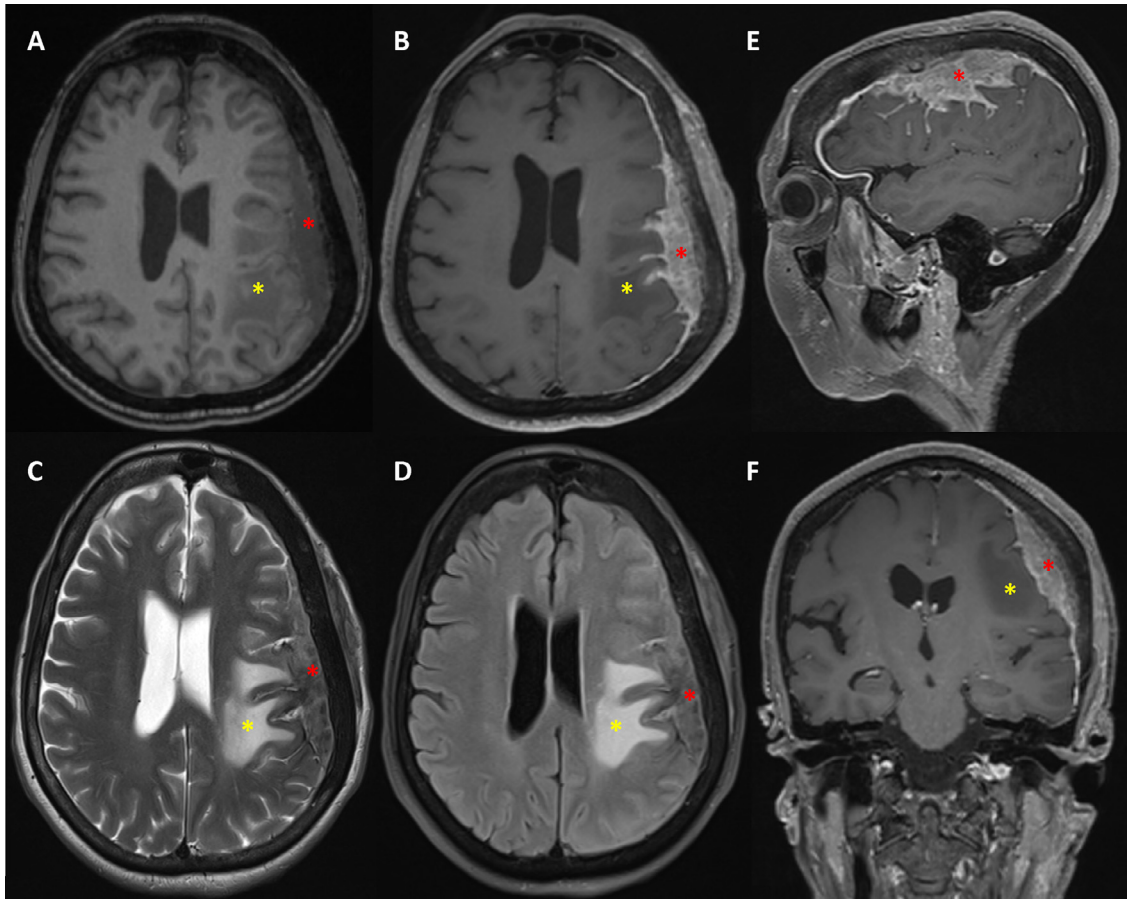

Fig. 2 – MR imaging of the brain with and without intravenous contrast in the axial (A–D), sagittal (E), and coronal (F) planes. Pre- (A) and postcontrast (B) T1-weighted imaging demonstrated a heterogeneously enhancing, dural-based mass overlying the left frontoparietal convexity (red asterisk) with associated vasogenic edema (yellow asterisk). T2-weighted imaging (C) and FLAIR (D) demonstrated a dural-based mass that is isointense to grey matter with a more conspicuous appearance of the adjacent vasogenic edema. Sagittal (E) and coronal (F) planes again redemonstrate the heterogeneously enhancing dural-based mass (red asterisk) extending anterior and posteriorly along the left frontoparietal convexity.

Bone involvement is manifested by reduced signal intensity in the diploë while leptomeningeal involvement may be seen as enhancement of an irregularly contoured shape in the adjacent sulci forming a focal mass or plaque-like thickening of the dura mater [14–17].

The differential diagnosis may be broad for a dural-based mass, including a subdural or epidural hematoma, malignant meningioma, dural/meningeal metastasis, solitary fibrous tumor, gliosarcomas, leiomyosarcomas, or neurosarcoidosis [12,15]. Characteristics that aid in differentiating PDL from meningiomas include a relatively flat body of the mass, long dural tails, absence/rare intratumoral calcification, absence of overlying skull hyperplasia/hyperostosis, and lobulation of the mass [7,10]. Also, the presence of an indistinct ("fuzzy") brain-tumor interface suggests PDL over a meningioma [18]. On DWI, the high cellularity of lymphomatous lesions will often restrict diffusion more than meningiomas and metastatic lesions [19]. MR spectroscopy has also been shown to be useful in that raised lipid/lactate peaks with a high choline/creatine ratios are more characteristic of lymphomatous lesions [10,20]. On perfusion-weighted imaging, PCNSL lesions typically have lower relative cerebral blood volume compared to other intracranial tumors, including meningiomas [10,21,22]. PDL lesions are more metabolically active and show avid tracer uptake on ^{18}F-FDG PET/CT compared to meningiomas [7,23]. Carbon-11 methionine PET has also shown that there is a larger area of brain involvement than standard contrast-enhanced MR imaging. This suggests carbon-11 methionine PET may be more sensitive to detect subtle leptomeningeal disease or to evaluate treatment response or recurrence [24]. While some of the listed PDL characteristics are shared with metastatic lesions or glioblastomas, they are helpful in differentiating PDL from meningiomas [7,19].

Another radiologic modality that can be useful in diagnosing PCNSL, as well as differentiating it from intracranial infectious lesions, is single-photon emission CT (SPECT) imaging. In immunocomptent patients, PCNSL retains higher levels of iodine-123 N-isopropyl-p-iodoamphetamine tracer. Contrary to this, immunocompromised patients' PCNSL lesions may be differentiated from infectious intracranial lesions by the increased thallium-201 tracer uptake on SPECT and the increased FDG tracer uptake on ^{18}F-FDG PET/CT [10].

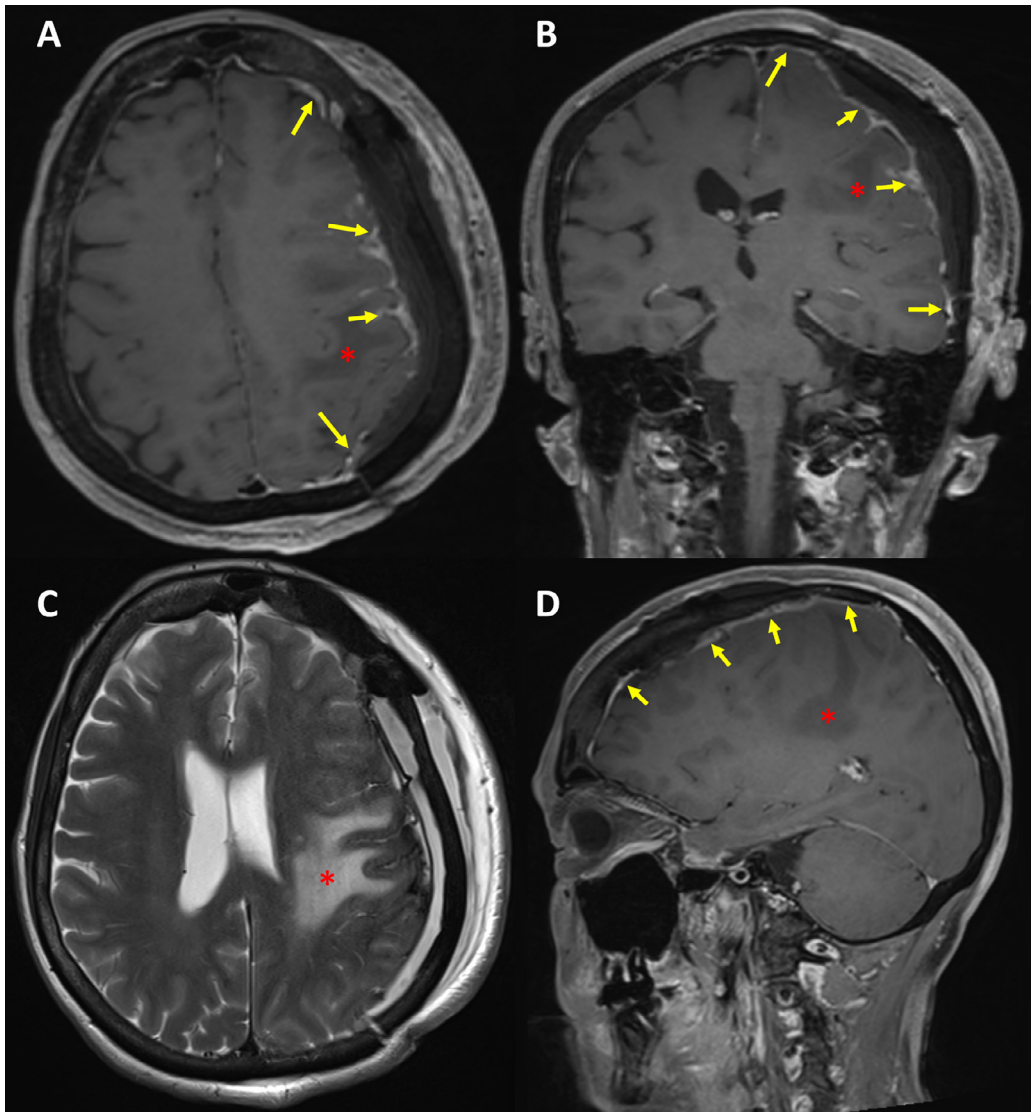

Fig. 3 – MR imaging of the brain with contrast in the axial (A, C), coronal (B), and sagittal (D) planes demonstrating interval resection of the dural-based mass with small residual enhancing tissue at the margins of the surgical cavity and underlying left cortical sulci (yellow arrows) on contrast-enhanced T1-weighted sequence (A, B, D). On T2-weighted sequence imaging (C), vasogenic edema is seen within corona radiata (red asterisk).

The absence of head trauma and patient history aids in differentiating PDL from hematomas. Though, this may be complicated if the patient either presents with head trauma or cannot report any recent falls. Furthermore, Iaccarino et al reported a case that clinically mimicked a chronic epidural hematoma in that the patient had a right biconvex parietal mass in setting of a known history of partial seizures and mild head injury. This patient's clinical and neuroradiological status progressively worsened and surgical excision was ordered. The histopathological diagnosis chronic lymphocytic leukemia/small lymphocytic lymphoma was given. Several other studies report an original misdiagnosis of subdural hematomas [8,9,16,25,26]. At times, PDL may coexist with the presence of hematoma or an actively hemorrhagic lesion [26–28]. Given that MR imaging is not routinely ordered for subdural hematomas and also non-contrast-enhanced CT lacks the resolution to discriminate between subdural hematomas and PDL, it is important to use the patient's clinical history and symptoms to guide management. PDL is more favorable on the differential compared to a subdural hematoma if patients have an incongruency between the size of the "hematoma" and their symptomatology, absence of head trauma or other factors that may lead to frequent falls, or there is evolution or progression of the lesion.

Conclusion

PDL is a rare subtype of PCNSL and may mimic several intracranial pathologies. Characteristics that aid in its differentiation include the patient's history, symptomatology, and

specific, multimodality radiologic findings. The classic PDL lesion is a hyperdense, vividly enhancing, dural-based mass with long dural tails on CT. On MR imaging, PDL is isointense to grey matter on T1-weighted imaging, isointense to hypointense on T2-weighted imaging, and restricts diffusion on DWI.

Patient consent

The authors obtained written informed consent for publication.

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