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CASE REPORT

Absence of enhancement in a lesion does not preclude primary central nervous system T-cell lymphoma: A case report

Chan-Seop Kim, Chi-Hoon Choi, Kyung Sik Yi, Yook Kim, Jisun Lee, Chang Gok Woo, Young Hun Jeon

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Abstract

BACKGROUND

Primary central nervous system lymphoma (PCNSL) is a non-Hodgkin lymphoma that originates in the central nervous system (CNS) and is exclusively limited to the CNS. Although most PCNSLs are diffuse large B-cell lymphomas, primary CNS T-cell lymphomas (PCNSTLs) are rare. PCNSTLs typically demonstrate some degree of enhancement on contrast-enhanced magnetic resonance imaging (MRI). To the best of our knowledge, non-enhancing PCNSTL has not been reported previously.

CASE SUMMARY

A 69-year-old male presented to the neurology department with complaints of mild cognitive impairment and gradual onset of left lower leg weakness over a span of two weeks. Initial MRI showed asymmetric T2-hyperintense lesions within the brain. No enhancement was observed on the contrast-enhanced T1 image. The initial diagnosis was neuro-Behçet's disease. Despite high-dose steroid therapy, no alterations in the lesions were identified on initial MRI. The patient's symptoms deteriorated further. An MRI performed one month after the initial scan revealed an increased lesion extent. Subsequently, brain biopsy confirmed the diagnosis of PCNSTL. The patient underwent definitive combined chemo-radiotherapy. However, the patient developed bacteremia and died of septic shock approximately three months after diagnosis.

CONCLUSION
The absence of enhancement in the lesion did not rule out PCNSTL. A biopsy approach is advisable for pathological confirmation.

Key Words: Central nervous system neoplasms; Non-Hodgkin Lymphoma; T-cell Lymphoma; Primary central nervous system lymphoma; Primary central nervous system T-cell lymphoma; Case report

Core Tip: The characteristic features of primary central nervous system T-cell lymphoma (PCNSTL) are not widely recognized owing to its low incidence rate. However, most malignant tumors demonstrate enhancement on gadolinium-enhanced magnetic resonance imaging (MRI). Consequently, a lesion without enhancement on gadolinium-enhanced MRI can easily be misinterpreted, potentially underestimating its malignant potential. Given the significant differences in the management of malignant and benign lesions, an accurate diagnosis is imperative for appropriate intervention. We present a case of PCNSTL without MRI enhancement. In instances of non-enhancing lesions with a clinical suspicion of malignancy, a more aggressive diagnostic approach should be adopted.

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INTRODUCTION

Primary central nervous system lymphoma (PCNSL) is a non-Hodgkin lymphoma that originates within the central nervous system (CNS) and remains largely confined to the CNS. Approximately 85% of PCNSL cases are restricted within the CNS[1,2]. It is an aggressive tumor with a poorer prognosis than lymphomas outside the CNS, with a median survival of less than two years[3,4].

PCNSL affects both immunocompromised and immunocompetent patients. It is rare among immunocompetent patients, accounting for 2%-4% of all newly diagnosed intracranial tumors[5]. While most PCNSL cases are diffuse large B-cell lymphomas, primary CNS T-cell lymphoma (PCNSTL) is notably rare[2,3]. This rarity contributes to a limited understanding of the radiologic characteristics.

PCNSTLs typically exhibit some degree of enhancement on T1-weighted MRI after contrast administration, and most lesions show diffusion restriction on diffusion-weighted imaging[6]. Here, we present a case of a 69-year-old male with PCNSTL in which the diagnosis was delayed because of the absence of the expected enhancement.

CASE PRESENTATION

Chief complaints

A 69-year-old male presented to the neurology department complaining of cognitive impairment and gradual onset of left lower leg weakness over the past two weeks.

History of present illness

Two years earlier, the patient had been treated for early-stage gastric cancer using endoscopic mucosal dissection. One month before the neurology consultation, he visited the ophthalmology department with right visual discomfort and was diagnosed with anterior uveitis. This diagnosis was confirmed by positive results for the Epstein-Barr virus (EBV) polymerase chain reaction (PCR) and *Toxocara canis* antibody IgG obtained from anterior chamber aspiration. He also reported a history of recurrent oral ulcers; however, no other significant illnesses were noted.

History of past illness

Two years earlier, the patient had been treated for early-stage gastric cancer using endoscopic mucosal dissection. One month before the neurology consultation, he visited the ophthalmology department with right visual discomfort and was diagnosed with anterior uveitis. This diagnosis was confirmed by positive results for the EBV PCR and *Toxocara canis* antibody IgG obtained from anterior chamber aspiration. He also reported a history of recurrent oral ulcers; however, no other significant illnesses were noted.

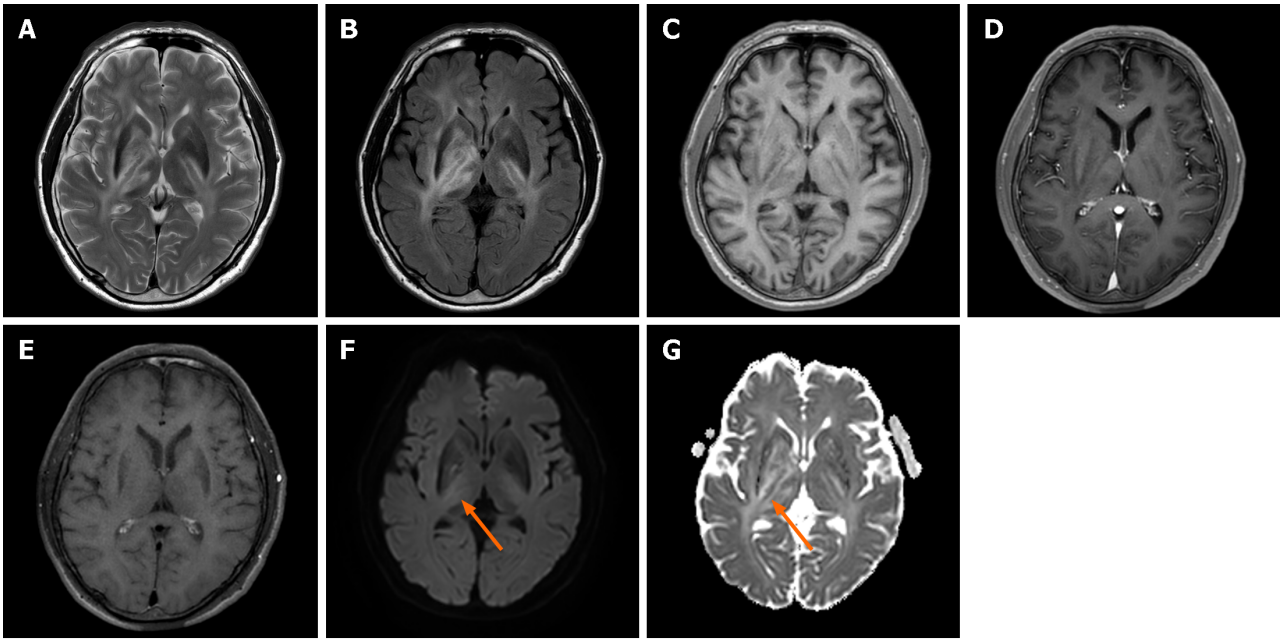

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Figure 1 Initial magnetic resonance imaging. A: T2 weighted image; B: T2 fluid attenuated inversion recovery image shows asymmetric T2 hyperintensity at bilateral basal ganglia and thalamus; C: T1 weighted image; D: Post-contrast T1 weighted image; E: Post-contrast T1 black blood image shows no enhancement at T2 hyperintensity lesions; F: B1000 diffusion weighted image; G: Apparent diffusion coefficient map showing mild diffusion restriction is noted at the right posterior limb of the internal capsule (arrows).

Personal and family history

The patient denied any family history of malignant neoplasms.

Physical examination

Vital signs on presentation were body temperature, 36.1 °C; blood pressure, 127/67 mmHg; heart rate, 65 beats per minute; and respiratory rate, 20 breaths per minute. Neurological examination revealed Medical Research Council (MRC) grade 3 weakness in the left leg, with no other notable neurological abnormalities.

Laboratory examinations

Serum white blood cell (WBC) levels were within the normal range (7680/10^3 μL), but the WBC count in the cerebrospinal fluid (CSF) showed a slight elevation (16/μL). Serum tests for human immunodeficiency virus and CSF assays for both EBV and John Cunningham virus were also negative.

Imaging examinations

The initial MRI scan showed asymmetric T2-hyperintense lesions in the bilateral periventricular white matter, basal ganglia, thalamus, right cerebral peduncle, and right middle cerebellar peduncle. Mild diffusion restriction was observed in the right posterior limb of the internal capsule. Contrast-enhanced T1 imaging showed no notable enhancement, and the magnetic resonance angiography results were unremarkable (Figure 1).

Further diagnostic work-up

Based on the patient's history of anterior uveitis, an initial diagnosis of neuro-Behçet's disease was considered, although the imaging findings were inconsistent. Given the absence of enhancement and significant diffusion restriction, a tumor lesion was initially considered less probable. Considering the patient's age, a preliminary diagnosis of neuro-Behçet's disease was made without further investigation. The patient was administered a high-dose steroid regimen, specifically prednisolone at 1 mg/d for five days. The patient reported an improvement in his subjective symptoms. However, on neurological examination, left leg weakness remained at MRC grade 3, and a follow-up MRI performed five days post-steroid initiation revealed no change in the previously identified lesions, an unanticipated result (Figure 2). The patient was discharged at his request, against medical advice.

Over the subsequent weeks, the patient's mental status became stuporous, and he was soon comatose. The patient was hospitalized in the emergency room. MRI conducted one month after the initial imaging illustrated a reduction in the extent of the lesion in the right basal ganglia. However, the other lesions had expanded, causing a mass effect on the left lateral ventricle. New subarachnoid space multifocal hemorrhages were identified, and arterial spin labeling-perfusion MRI revealed increased cerebral blood flow (Figure 3). The patient's symptoms, MRI findings, and diagnoses are summarized in Table 1. Based on these findings, conditions such as glioblastoma, lymphoma, and metastasis were considered. Chest and abdominal computed tomography (CT) and positron emission tomography-CT were performed to

Table 1 The summary of patient's clinical symptoms, exam results, considered diagnosis, and treatment

Time	Initial	5 d after	1 month after
Neurologic symptom	Mild cognitive impairments MRC grade 3 left lower leg weakness	No significant change in MRC grade 3 left lower leg weakness	Changed mental status to comatose
MRI	Asymmetric hyperintensities at the bilateral periventricular white matters, basal ganglia, thalamus, cerebral and cerebellar peduncles	No significant change in extent of T2 hyperintense lesion	Reduced extent of T2 hyperintense lesion in right basal ganglia, but increased extent in left hemisphere compressing left lateral ventricle Newly appeared SAH
Laboratory exams	Slight elevation of WBC in CSF	Nonspecific	Nonspecific
Considered diagnoses	Neuro-Behçet's disease	Neuro-Behçet's disease	Glioblastoma Lymphoma Metastasis
Treatment	Prednisolone at 1 mg/d for 5 d	None	Supportive care for bacteremia and sepsis

MRC: Medical Research Council; WBC: White blood cell; CSF: Cerebrospinal fluid; SAH: Subarachnoid hemorrhage; MRI: Magnetic resonance imaging.

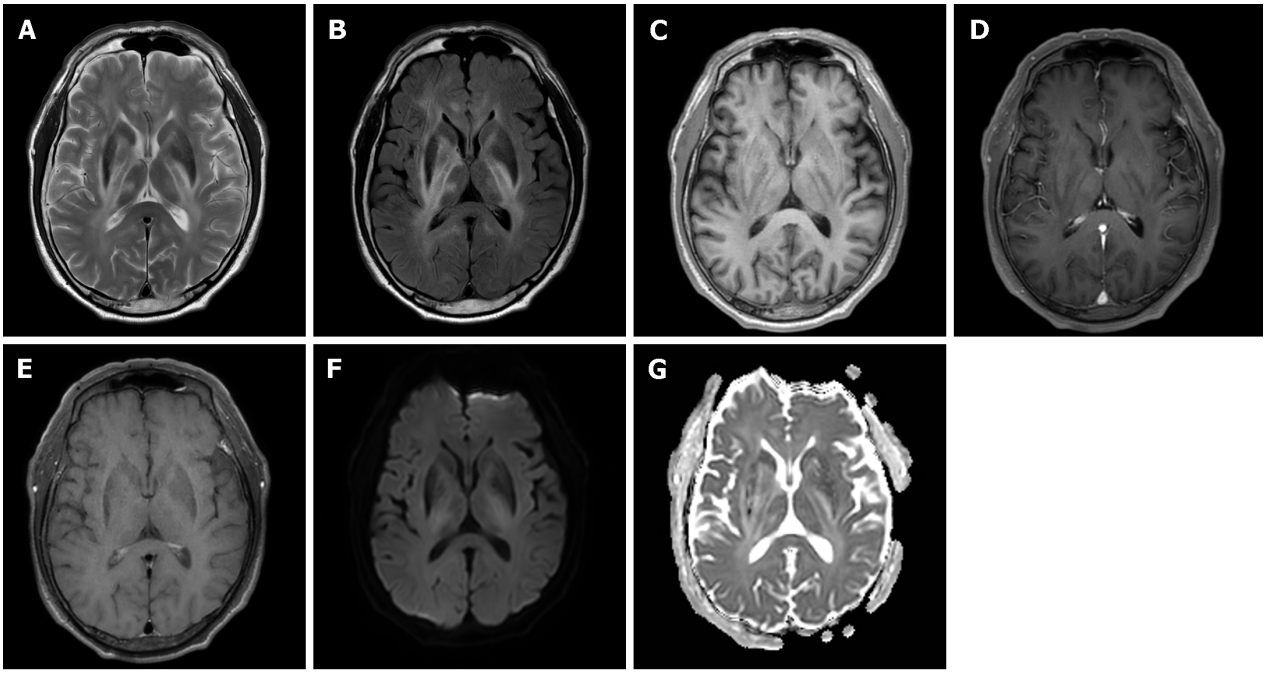

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Figure 2 Magnetic resonance imaging scan five days after high-dose steroid therapy is initiated. A: T2 weighted image; B: T2 fluid attenuated inversion recovery image; C: T1 weighted image, D: Post-contrast T1 weighted image; E: Post-contrast T1 weighted black blood image; F: B1000 diffusion weighted image; G: Apparent diffusion coefficient map showing minimal change of previously noted T2 hyperintense lesions.

rule out metastases. Brain biopsy was performed.

A stereotactic biopsy of the left putamen was performed. Immunohistochemical analysis revealed cluster of differentiation (CD)3 and CD8 positivity, indicating T-cell infiltration. CD68-positive microglia were also observed, suggesting a response to brain injury or pathology. Positive P53 staining was observed, which might indicate disrupted cell cycle regulation, often associated with malignant tumors. Moreover, Antigen Kiel-67 positivity in approximately 40% of the cells indicated a high proliferation rate, which is typical of aggressive neoplasms (Table 2, Figure 4). Electron microscopy corroborated a lymphoid neoplasm diagnosis, describing neoplastic lymphoid cells and virus particles indicative of a possible associated viral infection, like EBV, which has been linked to certain lymphomas. This evidence pointed towards a possible diagnosis of PCNSTL (Figure 5).

Table 2 The pathologic report

Immunohistochemical	P53: Positive
	CD68: Positive in the microglia
	Glial Fibrillary Acidic Protein: Positive in the reactive astrocytes
	Myeloperoxidase: Negative
	K27M: Negative
	Vimentin: Focal positive in the lymphocytes and reactive astrocytes
	CD56: Nonspecifically positive
	Phospho-Histone H3: 23/10 HPFs
	LFB: Positive in the myelin sheath (no loss of myelin sheath)
	Isocitrate Dehydrogenase 1: Negative (no mutation)
	EZHIP: Negative
	Olig2: Focal positive
	TMHH3 (Lys27): Positive (no mutation)
	CD3: Some CD3-positive T-cells infiltration
	CD20: A few positive cells in a perivascular area
	IBA-1: Positive in the microglia
	Ki-67: Positive in 40.0%
	CD8: A few positive cells
Electronic microscopy	Ultrathin sections show sheets of round to oval-shaped neoplastic lymphoid cells. The nuclei of tumor cells are round to oval with heterochromatin and prominent nucleoli. The cytoplasm is scanty and contains eccentrically located a few mitochondria and electron-dense granules
Diagnosis	Brain, putamen, left, stereotactic biopsy: Scattered proliferative T-cell infiltration, consistent with T-cell lymphoma

CD: Cluster of differentiation; LFB: Luxol fast blue; EZHIP: Enhancer of Zest homologs inhibitory protein; Olig2: Oligodendrocyte transcription factor 2; TMHH3 (lys27): Trimethylation at lysine 27 of histone H3; IBA-1: Ionized calcium-binding adaptor molecule 1; Ki-67: Antigen Kiel-67.

FINAL DIAGNOSIS

Considering both the pathological findings and confinement of the lesion within the brain, the definitive diagnosis was PCNSTL.

TREATMENT

The patient was slated for combined chemoradiotherapy. However, this was postponed due to the onset of bacteremia during hospitalization.

OUTCOME AND FOLLOW-UP

The patient died of septic shock approximately three months post-diagnosis.

DISCUSSION

PCNSL in immunocompetent patients is predominantly a diffuse large B-cell lymphoma, comprising up to 90% of all cases. Other types, including PCNSTL, constitute the remainder. The prevalence of PCNSTL among all cases of PCNSL is 2% in Western countries, 8.5%-14.3% in Japan, and 16.7% in Korea[4,7]. However, these elevated rates in East Asian countries may be biased due to small sample sizes. Despite its rarity, the clinical features and prognosis of PCNSTL resemble those of other types of PCNSL[8].

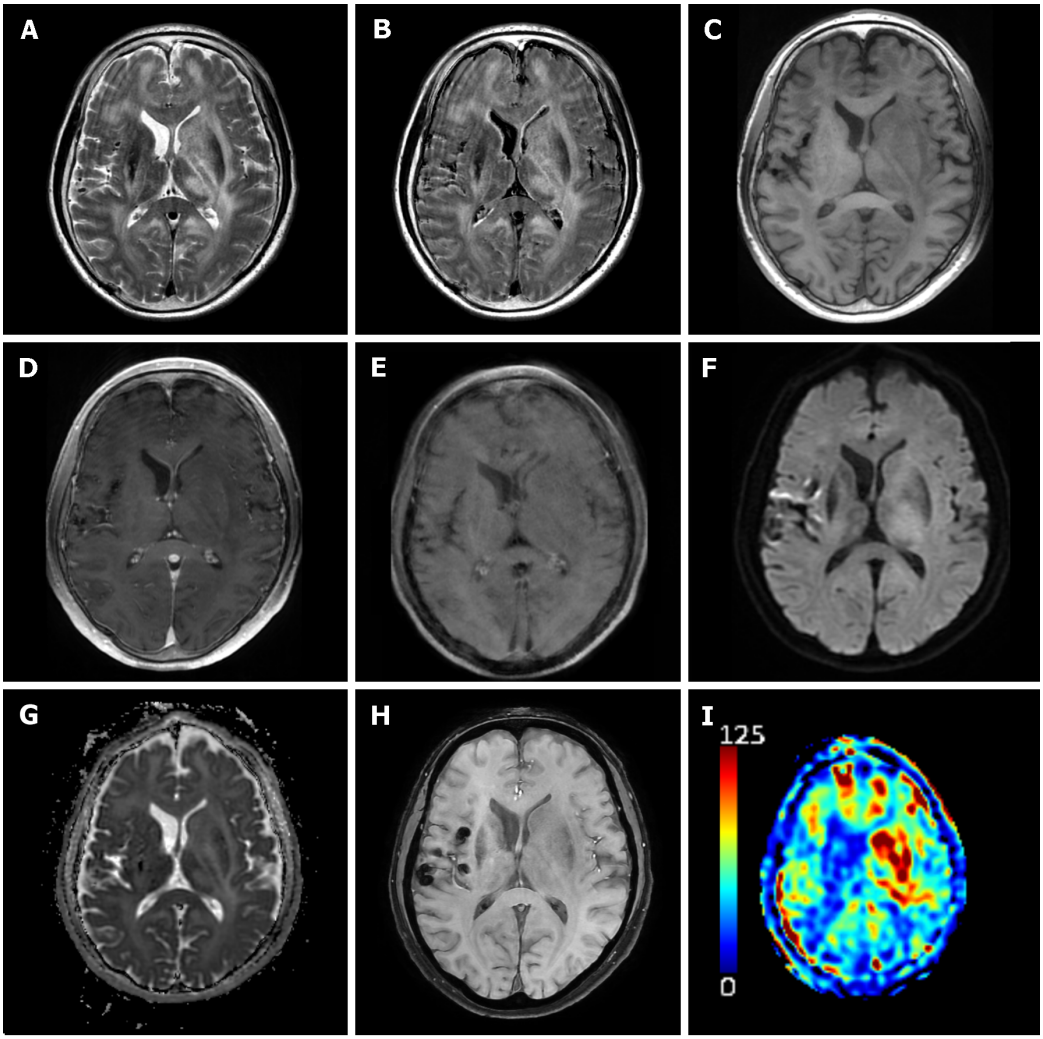

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Figure 3 Follow up magnetic resonance imaging one month after initial scan. A: T2 weighted image; B: T2 fluid attenuated inversion recovery image shows decreased extent of hyperintense lesion at right basal ganglia, but increased extents of remaining hyperintense lesions; C: T1 weighted image; D: Post-contrast T1 weighted image; E: Post-contrast T1 black blood image shows no enhancement at T2 hyperintense lesions; F: B1000 diffusion weighted image; G: Apparent diffusion coefficient map shows mild diffusion restriction at both basal ganglia; H: Susceptibility weighted image shows newly appeared multifocal hemorrhages at subarachnoid space; I: Arterial spin labeling perfusion magnetic resonance imaging shows increased cerebral blood flow of left basal ganglia lesion.

The present case posed a diagnostic challenge owing to the atypical imaging characteristics, which, to our knowledge, have not been previously reported. Given the low incidence of PCNSTL, limited imaging findings have been reported, and its diagnosis is histopathologically challenging because of the frequently absent atypical cytological features of most T-cell lymphomas[7,9]. Additionally, no specific immunohistochemical markers for neoplastic T-cells exist[10].

Previous imaging studies of PCNSTL in immunocompetent individuals have reported features such as heterogeneous or rim enhancement, which are uncharacteristic of PCNSL, given that typical primary CNS B-cell lymphoma (PCNSBL) usually presents with homogenous enhancement[11]. Moreover, PCNSTL exhibits hemorrhage or cystic changes more frequently than PCNSBL. In East Asian countries, PCNSTL typically manifests in the supratentorial or subcortical areas without leptomeningeal involvement[11]. Other studies have described nodular, patchy, homogenously enhancing lesions[12] and multifocal mass-like lesions with heterogeneous enhancement and mass effects[13]. In contrast, our patient had asymmetric T2 high signal intense lesions in the pons and basal ganglia with minimal diffusion restriction and no enhancement. These features were not previously associated with PCNSTL.

Histologically, PCNSTL predominantly presents with small cells, occasionally interspersed with medium-sized cells. These cells do not typically form a solid mass or aggregate but demonstrate notable perivascular infiltration, a significantly different histopathologic growth pattern from that of PCNSBL[7]. Our patient exhibited scattered proliferative T cells, leading to a diagnosis of PCNSTL. This histopathologic pattern might explain the observed lack of enhancement, suggesting that there was no solid mass-like lesion to enhance, but rather relatively sparse proliferative T-cells.

The lesion distribution was also distinctive and localized in deeper brain regions, such as the basal ganglia and pons, which are uncommon sites for PCNSL. Previous studies have indicated that low-grade PCNSLs have different imaging features from their high-grade counterparts[9]. Given that PCNSTL more often presents with low-grade histology than B-cell-origin lymphomas, this case might represent a low-grade PCNSTL with a deep brain location and no enhancement[2,

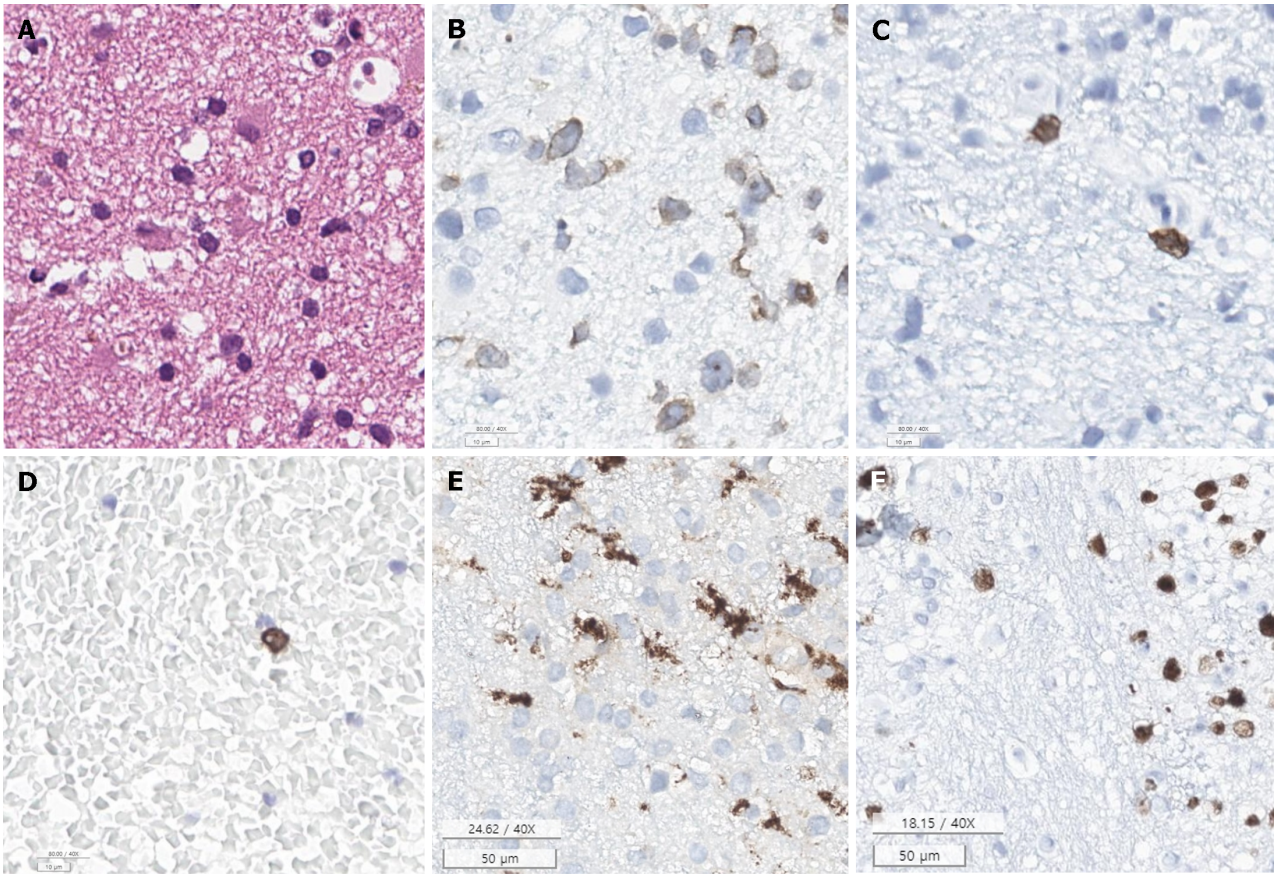

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Figure 4 The immunohistochemical staining test results. A: Hematoxylin & eosin stain (× 40) shows scattered lymphocyte infiltration in the specimen; B: Cluster of differentiation (CD)3 immunostain (× 40); C: CD8 immunostain (× 40), CD3 and CD8 positive lymphocytes suggests T-cell infiltration; D: CD20 immunostain (× 40), CD20 positive cells suggest presence of an immune response, likely towards the neoplastic cells; E: CD68 immunostain (× 40), CD68 positive microglia suggests an ongoing response to an injury or pathology in the brain; F: Antigen Kiel-67 (KI-67) immunostain (× 40), KI-67 index of 40% indicates the presence of high proliferation rate. Overall, these findings suggest a high proliferation neoplastic disorder involving lymphoid cells.

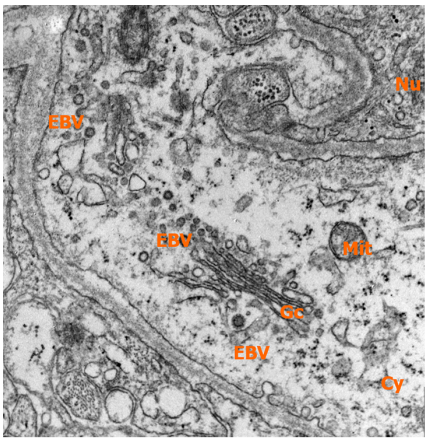

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Figure 5 Transmission Electron Microscope revealing the presence of numerous infiltrations of Epstein-Bar virus (80 kV, 40000 ×) within tumor cells. Nu: nucleus; Cy: Cytoplasm; Gc: Golgi-complex; Mit: Mitochondria; EBV: Epstein-Bar Virus.

7]. Unfortunately, further grading and subtyping were not possible.

The patient was initially considered to have potential neuro-Behçet's disease, given that the clinical history and imaging findings aligned with the neurological manifestations of Behçet's disease[14]. High-dose corticosteroids, the first-line treatment for neuro-Behçet's disease[15], were administered. However, corticosteroids induce T-cell apoptosis, which complicates the diagnosis of PCNSTL[10]. Some CNS lymphomas respond to corticosteroids, resulting in symptomatic and radiological improvements that mimic vasculitis. Over time, the growth of corticosteroid-resistant clones may render

the lymphoma resistant to corticosteroid treatment[9,10]. Consequently, corticosteroid therapy before stereotactic brain biopsy is discouraged as it may delay an accurate diagnosis. Given the significant differences in treatment strategies for vasculitis (such as neuro-Behçet's disease) and PCNSTL, adopting a more proactive biopsy approach could potentially enhance the diagnosis and prognosis of PCNSTL, especially in cases presenting with atypical radiological features, such as the absence of enhancement.

CONCLUSION

Herein, we present a unique case of PCNSTL without enhancement. Given its rarity, known imaging features of PCNSTL are limited. To the best of our knowledge, the absence of enhancement, as observed in the present case, has not been previously documented. This case underscores the importance of not ruling out PCNSTL in the differential diagnosis, even without enhancement. Considering the stark differences in the management of PCNSTL and other benign conditions, biopsy for definitive pathological confirmation is strongly recommended.

FOOTNOTES

Author contributions: Kim CS, Choi CH, and Yi KS contributed to manuscript writing and editing and data collection; Kim CS contributed to data analysis; Kim CS, Choi CH, Yi KS, Kim Y, Lee J, Jeon YH and Woo CG contributed to conceptualization and supervision; All authors have read and approved the final manuscript.

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REFERENCES

1 **Plasswilm L**, Herrlinger U, Korfel A, Weller M, Küker W, Kanz L, Thiel E, Bamberg M. Primary central nervous system (CNS) lymphoma in immunocompetent patients. *Ann Hematol* 2002; **81**: 415-423 [PMID: 12223997 DOI: 10.1007/s00277-002-0498-8]
2 **Jahnke K**, Thiel E, Schilling A, Herrlinger U, Weller M, Coupland SE, Krümpelmann U, Stein H, Korfel A. Low-grade primary central nervous system lymphoma in immunocompetent patients. *Br J Haematol* 2005; **128**: 616-624 [PMID: 15725082 DOI: 10.1111/j.1365-2141.2004.05361.x]
3 **Grommes C**, DeAngelis LM. Primary CNS Lymphoma. *J Clin Oncol* 2017; **35**: 2410-2418 [PMID: 28640701 DOI: 10.1200/JCO.2017.72.7602]
4 **Menon MP**, Nicolae A, Meeker H, Raffeld M, Xi L, Jegalian AG, Miller DC, Pittaluga S, Jaffe ES. Primary CNS T-cell Lymphomas: A Clinical, Morphologic, Immunophenotypic, and Molecular Analysis. *Am J Surg Pathol* 2015; **39**: 1719-1729 [PMID: 26379152 DOI: 10.1097/PAS.0000000000000503]
5 **Song KW**, Issa S, Batchelor T. Primary central nervous system lymphoma: epidemiology and clinical presentation. *Ann Lymphoma* 2021; **5**: 16 [DOI: 10.21037/aol-20-50]
6 **Haldorsen IS**, Espeland A, Larsson EM. Central nervous system lymphoma: characteristic findings on traditional and advanced imaging. *AJNR Am J Neuroradiol* 2011; **32**: 984-992 [PMID: 20616176 DOI: 10.3174/ajnr.A2171]
7 **Choi JS**, Nam DH, Ko YH, Seo JW, Choi YL, Suh YL, Ree HJ. Primary central nervous system lymphoma in Korea: comparison of B- and T-cell lymphomas. *Am J Surg Pathol* 2003; **27**: 919-928 [PMID: 12826884 DOI: 10.1097/00000478-200307000-00007]
8 **Ferreri AJ**. How I treat primary CNS lymphoma. *Blood* 2011; **118**: 510-522 [PMID: 21613254 DOI: 10.1182/blood-2011-03-321349]
9 **Dulai MS**, Park CY, Howell WD, Smyth LT, Desai M, Carter DM, Vogel H. CNS T-cell lymphoma: an under-recognized entity? *Acta Neuropathol* 2008; **115**: 345-356 [PMID: 18196250 DOI: 10.1007/s00401-007-0338-y]

10 **Fraser E**, Gruenberg K, Rubenstein JL. New approaches in primary central nervous system lymphoma. *Chin Clin Oncol* 2015; **4**: 11 [PMID: 25841718 DOI: 10.3978/j.issn.2304-3865.2015.02.01]

11 **Kim EY**, Kim SS. Magnetic resonance findings of primary central nervous system T-cell lymphoma in immunocompetent patients. *Acta Radiol* 2005; **46**: 187-192 [PMID: 15902895 DOI: 10.1080/02841850510022714]

12 **Byun SH**, Kim DM, Lee IH, Song CJ, Kim KH, Choi SY. Primary Central Nervous System Involvement in Peripheral T-Cell Lymphoma: A Case Report. *Taehan Yongsang Uihakhoe Chi* 2021; **82**: 255-260 [PMID: 36237451 DOI: 10.3348/jksr.2020.0008]

13 **Manenti G**, Di Giuliano F, Bindi A, Liberto V, Funel V, Garaci FG, Floris R, Simonetti G. A Case of Primary T-Cell Central Nervous System Lymphoma: MR Imaging and MR Spectroscopy Assessment. *Case Rep Radiol* 2013; **2013**: 916348 [PMID: 23781374 DOI: 10.1155/2013/916348]

14 **Kim SW**, Kim TG, Oh J, Kim DY, Choi YC, Kim SM, Shin HY, Bang D. Clinical and Radiographic Characteristics of Neuro-Behçet's Disease in South Korea. *J Clin Neurol* 2019; **15**: 429-437 [PMID: 31591829 DOI: 10.3988/jcn.2019.15.4.429]

15 **Alpsoy E**, Leccese P, Emmi G, Ohno S. Treatment of Behçet's Disease: An Algorithmic Multidisciplinary Approach. *Front Med (Lausanne)* 2021; **8**: 624795 [PMID: 33996847 DOI: 10.3389/fmed.2021.624795]

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