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CASE REPORT

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Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids triggered by Hodgkin's lymphoma: A case report and brief literature review

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Key Clinical Message

We presented a patient, diagnosed with lymphoma-associated CLIPPERS, 11 years after lymphoma treatment. Therefore, CLIPPERS may be paraneoplastic neurological syndrome of lymphoma, which needs to be considered in the follow-up of lymphoma cases.

Abstract

Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) is a rare central nervous system disorder with a recent increase in incidence. There are few reports of lymphoma-associated CLIPPERS, although the relationship between these two diseases and the pathophysiology of CLIPPERS in general need further investigation. Here, we present a patient with a history of Hodgkin's lymphoma (HL) more than 10 years before the onset of CLIPPERS, in contrast to the majority of previously reported lymphoma-associated cases, and discuss the possibility that CLIPPERS is the paraneoplastic neurological syndrome of HL. This highlights the need to consider CLIPPERS as a differential diagnosis during follow-up of patients with a history of lymphoma.

KEYWORDS

case report, CLIPPERS, Hodgkin disease, lymphoma, paraneoplastic syndromes

1 | INTRODUCTION

Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) is a rare neurologic disorder that usually has a favorable outcome if diagnosed in time and treated appropriately.[1] Based on the initial report by Pittock et al.,[2] CLIPPERS is characterized by deep gray and white matter (mainly pontocerebellar) lymphocytic infiltrations that cause clinical features and characteristic magnetic resonance imaging (MRI) enhancements that respond to corticosteroids.[3] The etiology of the primary infiltration of lymphocytes into the perivascular space of the brain stem remains unclear, and at the time of writing this report, there have been no

controlled trials of potential treatments or recommendations for treatment. Although the disease is usually a primary relapsing disorder without typical progressive demyelination, in a few cases various malignancies, especially hematologic, have been reported before or after the diagnosis or relapse of CLIPPERS, leading to a chronic disease with a higher mortality rate.[4] In this article, in addition to presenting a patient who had Hodgkin's lymphoma (HL) years before the onset of CLIPPERS, we briefly review the previous lymphoma-associated cases and discuss the possible relationship between these conditions.

1.1 | Case

A 47-year-old Iranian man with a past medical history of type 2 diabetes and mediastinal HL at the age of 36 years, which was completely cured after chemotherapy (adriamycin, bleomycin, vinblastine, and dacarbazine), was admitted complaining of dizziness, ataxia, diplopia, and left-sided numbness. The symptoms had begun 4 months earlier and had progressed in severity and frequency. On admission, he was hemodynamically stable without fever. Neurological examination revealed diplopia, end-gaze nystagmus, dysarthria, impaired tandem gait, and left-sided ataxia and paresthesia. Other pathologies such as abnormal reflexes, sensory, or motor deficits were absent.

Laboratory tests including cell blood count, erythrocyte sedimentation rate, electrolytes, liver enzymes, serum creatinine, and thyroid hormones were within normal limits. According to the serologic evaluation, the following antibodies were all negative: collapsing response mediator protein 5, antinuclear, titin, Zic4, glutamic acid decarboxylase (GAD65), anti-SOX1, recoverin, anti-aquaporin-4, and

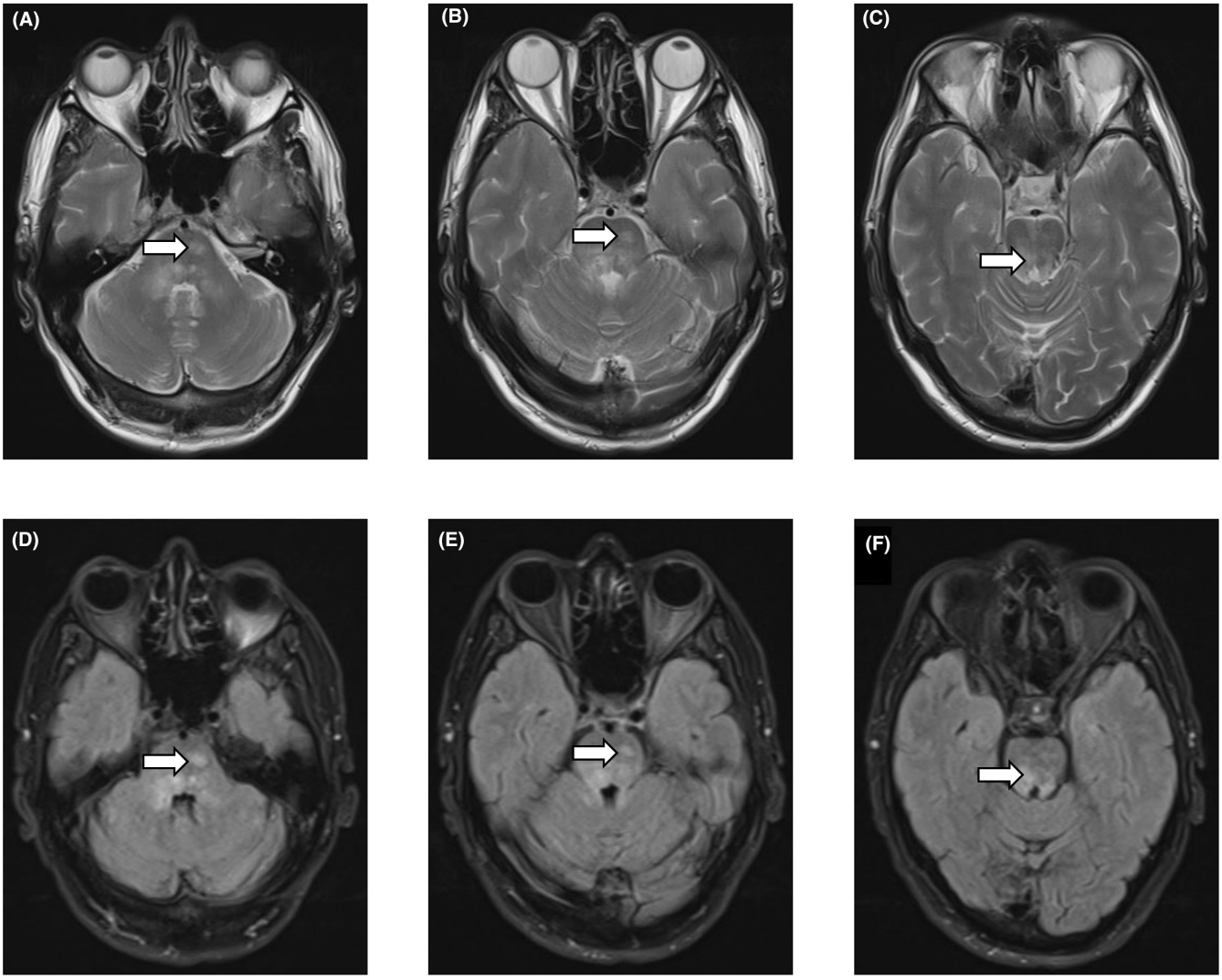

FIGURE 1 Axial plane T2 (A, B, C) and fluid-attenuated inversion recovery (FLAIR) (D, E, F); multiple T2 high peppering curvilinear enhancing lesions (marked by arrows) in the brain stem and cerebellum suggesting chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS). 236 × 190 mm (300 × 300 DPI).

anti-MOG, anti-Hu, anti-Ri, anti-Yo, anti-amphiphysin, and anti-PNMA2. Human T-cell leukemia virus I, syphilis, Epstein–Barr virus (EBV), and human immunodeficiency virus were not detected. Cerebrospinal fluid (CSF) analysis showed pleocytosis (23/mm^3 white blood cells) and a high protein level (0.998 g/L), while no malignant cells or oligoclonal bands were seen. The bone marrow specimen also showed no evidence of lymphoma. Neck, chest, and abdominopelvic computerized tomography scans of the case had no pathology except for kidney stones. However, MRI of the brain demonstrated multiple peppering curvilinear enhancing lesions in the brain stem and cerebellum (Figures 1 and 2a,e). In magnetic resonance spectroscopy of the mentioned lesions, the ratio of choline to N-acetyl aspartate was about 0.88 and the ratio of choline to creatine was 1.42, which were against the diagnosis of lymphoma.

Clinical and paraclinical findings ruled out the recurrence of HL and raised the suspicion of CLIPPERS. Therefore, the patient was treated with corticosteroids (1 g intravenous methylprednisolone for 4 days followed by oral dose of 25 mg of prednisolone per day), which confirmed the diagnosis by resolution of symptoms and reduction of MRI enhancements (Figure 2b,f). The patient returned approximately 2 months later complaining of dysphagia, ataxia, and numbness of the left limbs. Neurological examination revealed recurrence of diplopia and gait ataxia. He was admitted to the hospital and underwent plasmapheresis with a total plasma exchange volume of 10 liters, followed by oral prednisolone 25 mg per day. Gait ataxia improved significantly, other neurological deficits resolved completely, and MRI enhancements were reduced compared with the first hospitalization (Figure 2c,g). After discontinuing oral prednisolone for 2 months, no relapse of CLIPPERS was observed. At the 6-month follow-up after the second hospitalization, the brain MRI lesions were completely resolved (Figure 2d,h).

2 | DISCUSSION

In this patient, CLIPPERS was diagnosed 11 years after complete remission of HL. Brain biopsy was not performed because the radiologic and clinical findings

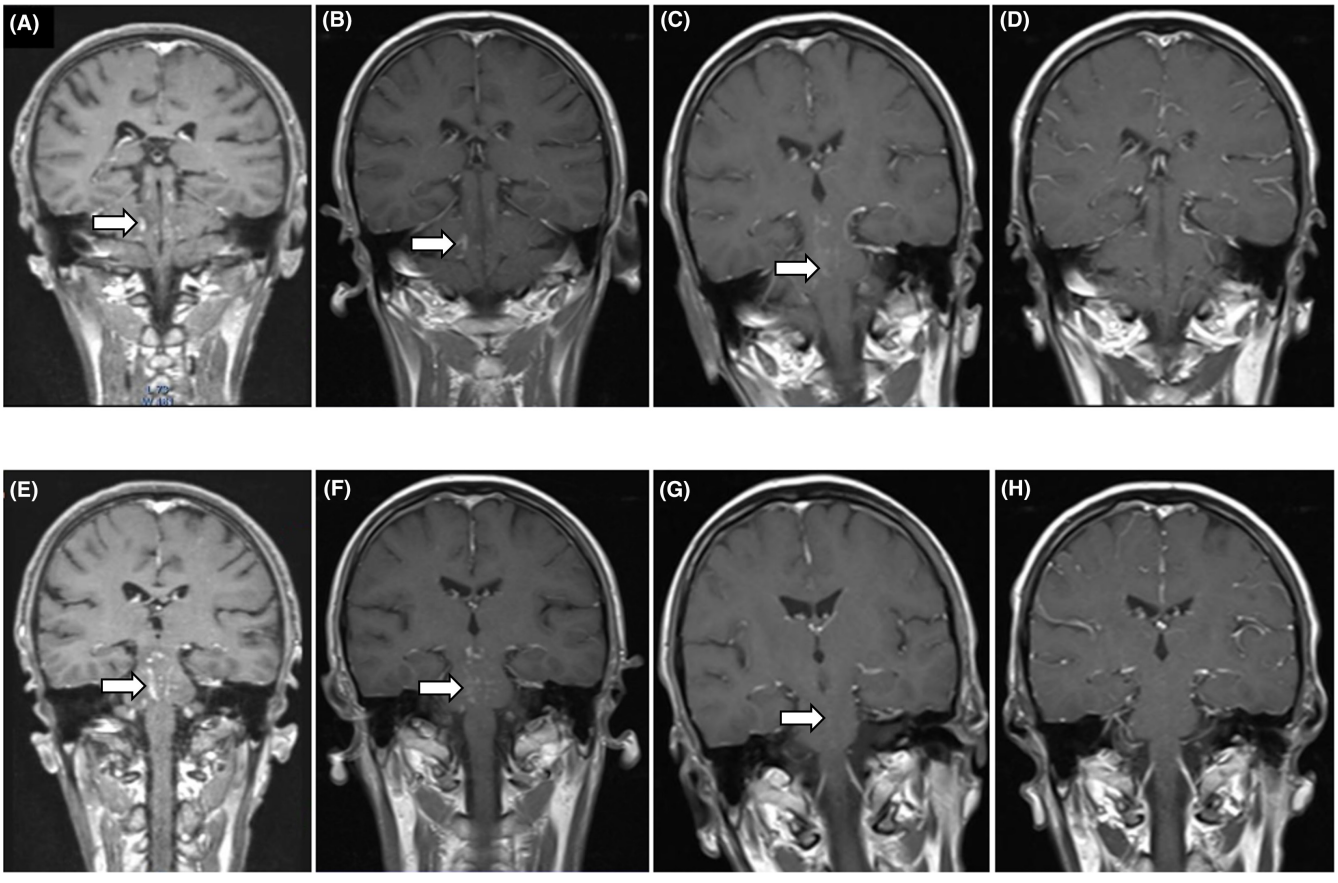

FIGURE 2 Coronal plane T1; multiple T1 low peppering curvilinear enhancing lesions (marked by arrows) in the brain stem and cerebellum suggesting chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) before treatment (A, E); reduction in enhancing lesions after initial treatment with corticosteroids (B, F) and plasmapheresis at the next hospitalization (C, G); disappearance of enhancing lesions at the 6-month follow-up visit (D, H). 264 × 181 mm (300 × 300 DPI).

TABLE 1 Diagnosis, management, and prognosis of CLIPPERS cases associated with Hodgkin's lymphoma.

First author	Mashima K[17]	Ma Y[18]	Deng Y[15]	Our report
Publication year	2015	2016	2021	2023
Country	Japan	China	China	Iran
Age	31	37	54	47
Sex	Male	Male	Male	Male
Initial presentations	Ataxia, dysarthria, nystagmus, dizziness numbness of hands, increased deep tendon reflexes, and diminished position and vibration sensations of legs	Ataxia, dysarthria, dysphagia, seizure, hoarseness, 6th cranial nerve palsy, cough, decreased muscle strength, limited eyeball movement, increased deep tendon reflexes, pyramidal signs, and decreased pharyngeal reflex	Ataxia, mild dysarthria, gait disturbance, intermittent diplopia, horizontal nystagmus, and impairment of coordinated limbs' movements	Ataxia, diplopia, left-sided numbness, and dizziness
Location of perivascular contrast enhancement in MRI	Pons, cerebellum, medulla, cerebral cortex, and cervical spinal cord	Pons, midbrain, pontine brachium, basal ganglia, and diffuse white matter	Pons, cerebellum, middle cerebellar peduncle, and midbrain	Brainstem and cerebellum
CSF abnormalities	Elevated protein (0.96 g/L), pleocytosis (5/mm^3)	Elevated protein (1.3 g/L), pleocytosis (108/mm^3)	Elevated protein (0.77 g/L) no pleocytosis	Elevated protein (0.998 g/L) pleocytosis (23/mm^3 white blood cells)
Initial treatment for CLIPPERS	Intravenous 1 g methylprednisolone daily for 5 days	Intravenous 15 mg dexamethasone daily for 28 days then tapering by 5 mg every 5 days until discontinuance	Intravenous 1 g methylprednisolone daily for 5 days	Intravenous 1 g methylprednisolone daily for 4 days
Response to initial treatment	Controlled well	Gradually improved	Significantly improved	Significantly improved
Maintenance treatment	Oral prednisolone (1 mg/kg body weight daily)	Oral prednisolone (30 mg daily tapering to 5 mg daily)	Oral prednisolone (1 mg/kg body weight tapering to 20 mg daily)	Oral prednisolone (25 mg daily tapering to 12.5 mg daily)
Total relapses	0	0	1, after 2 months	1, after 2 months
Diagnosed CLIPPERS biopsy	N/A	Lymphocytic inflammatory infiltration in the perivascular and parenchymal area	Lymphocytic inflammatory infiltration in the perivascular and parenchymal area	N/A
Diagnosed lymphoma biopsy	Stage II Hodgkin's lymphoma (mixed cellularity type)	Mediastinal Hodgkin's lymphoma (lymphocyte predominant type)	N/A	Mediastinal Hodgkin's lymphoma
Time between lymphoma treatment and the onset of neurological symptoms	16 years	11 years	9 years	11 years
Chemotherapy regimen	Cyclophosphamide, vincristine, procarbazine, and prednisone	Adriamycin, bleomycin, vinblastine, and dacarbazine	N/A	Adriamycin, bleomycin, vinblastine, and dacarbazine
Outcome	Remission	Remission	Remission after parotid carcinoma resection	Remission

Abbreviations: CLIPPERS, chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids; CSF, cerebrospinal fluid; g, gram; L, liter; mg, milligram; mm, millimeter; MRI, magnetic resonance imaging; N/A: not available.

were consistent with the current diagnostic criteria for CLIPPERS.[3,5] To find previous lymphoma-associated cases up to November 2023, PubMed and Scopus online databases were searched using the keywords "CLIPPER" and "Lymphoma". To the best of our knowledge, 20 patients with lymphoma-associated CLIPPERS have been reported[6–16] since the first introduction of CLIPPERS.[2] A review of 10 patients by Zhang et al. showed that patients with lymphoma-associated CLIPPERS had worse clinical outcomes and higher risk of mortality compared to those without lymphoma.[16] These patients also presented with different clinical and laboratory findings, such as long tract involvement and elevated CSF protein level, particularly above 0.9 g/L. However, the different courses of CLIPPERS in different underlying lymphoma pathologies (i.e., HL, T-cell, and B-cell lymphoma) require further explanation.

Table 1 presents pertinent information from three previously reported patients with HL-associated CLIPPERS. Although the demographic characteristics and clinical course of CLIPPERS are almost similar among the cases, differences in HL classification, time interval between lymphoma and onset of CLIPPERS, and chemotherapy regimens are noticeable. The association of CLIPPERS with types of lymphoma in different ways, and when it comes to HL, in different clinical conditions, highlights the need for further research to find a potential relationship or possible common mechanisms.

The pathophysiology of CLIPPERS as a specific disease is still a matter of debate, although lack of evidence has maintained CLIPPERS as an independent disease, albeit by exclusive diagnosis.[4] Different hypotheses have been proposed regarding the relationship between lymphoma and CLIPPERS, which need further validation: (a) Lymphoma may be misdiagnosed as CLIPPERS at the onset or recurrence of symptoms. Studies reporting these cases highlight the role of biopsy and red flags such as inadequate response to corticosteroids in differentiating CLIPPERS from lymphoma.[6–8,16]; (b) CLIPPERS may be a premalignant state that progresses to malignancies such as lymphoma or histiocytic sarcoma[19] after a variable time in response to EBV reactivation,[11,20] chronic stimulation of perivascular antigens,[12] blood brain barrier dysregulation,[21] or compromised immune system resulting from treatment of CLIPPERS[9,10,12]; (c) CLIPPERS may be a lymphocytic response through immune reconstitution or autoimmune reaction to various triggers such as HBV infection, influenza vaccination, or natalizumab withdrawal that may resolve or progress to more severe diseases such as lymphoma depending on management and other unknown prognostic factors[22,23]; (d) CLIPPERS may be a spectrum rather than a disease caused by heterogeneous mechanisms. The presence of hemophagocytic lymphohistiocytosis gene mutations in CLIPPERS cases with progression to lymphoma or CLIPPERS cases with predominant B lymphocytes in brain biopsy supports this idea[24–26]; (e) CLIPPERS may be triggered by lymphoma. Among the lymphoma-associated CLIPPERS cases, CLIPPERS occurred after lymphoma in four patients (three with HL, one with T-cell lymphoma). Nakamura et al. have suggested abnormal proliferation of $CD4^+$ T cells as the pathogenic pathway from lymphoma to CLIPPERS.[27] In the HL case reported by Deng et al, CLIPPERS was subsequently accompanied by parotid carcinoma, and the symptoms did not disappear until the parotid cancer was treated. They proposed CLIPPERS as a paraneoplastic neurological syndrome (PNS) of parotid carcinoma.[15]

The history of our patient is consistent with the latter hypothesis that CLIPPERS may be triggered by lymphoma. Whether CLIPPERS in this case could be the PNS of HL warrants future studies. PNSs are rarely seen in lymphoma, but they are more common in HL, mostly unique, and without specific onconeural antibodies.[28] There are reports of HL patients with paraneoplastic cerebellar degeneration, the most common PNS in HL, that the lymphoma preceded the PNS by months to years and even in HL remission.[29,30] In our case, CLIPPERS was diagnosed more than 10 years after HL remission, which could be an immune-mediated PNS of HL through antibody or T-cell related mechanisms.[31] Considering the rarity of CLIPPERS and the lack of original studies on its pathophysiology, further histopathologic and serologic investigations and long-term follow-up of patients with different types of lymphoma would shed light on clarifying of the relationship between lymphoma and CLIPPERS and its pathogenesis.

3 | CONCLUSION

CLIPPERS may be triggered by HL years after its remission. In patients with a history of lymphoma, regardless of CNS involvement, PNS, and autoimmune diseases such as CLIPPERS should be considered.

AUTHOR CONTRIBUTIONS

Atiyeh Karimi Shervedani: Conceptualization; investigation; methodology; writing – original draft. **Farinaz Tabibian:** Investigation; methodology; supervision; writing – review and editing. **Mahdiyeh Gholipour Khotbesara:** Investigation; writing – review and editing. **Iman Adibi:** Project administration; supervision; writing – review and editing.

FUNDING INFORMATION

No funding was received for this study.

CONFLICT OF INTEREST STATEMENT

We declare that all authors have read and approved the submission. The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The approval was received from Research Ethics Committees of Research Ethics Committee of the "Alzahra Research Centers." Approval ID: IR.ARI.MUI.REC.1402.100. This study was performed in accordance with the Helsinki Declaration of 1964, and its later amendments.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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