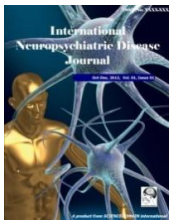

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Lymphoma with Bilateral Contrast Enhancement and Restricted Diffusion of Multiple Cranial Nerves in MRI

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Authors' contributions

This case report study was carried out in collaboration among all authors. Authors SR and KA designed the article and all authors managed the literature search and author KA wrote the first draft of the manuscript. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

We present a 50-year-old female with paresthesia on the right side of her face, facial asymmetry, hearing loss, and difficulty in walking. After clinical and radiological evaluations, non-Hodgkin lymphoma type B has been diagnosed. Cranial MRI revealed contrast enhancement of 3rd, 5th, 7th, and 8th cranial nerves bilaterally. Diffusion-weighted imaging (DWI) revealed prominent hyperintense symmetric cranial nerves involvement with corresponding signal reduction on Apparent Diffusion Coefficient (ADC) maps. In lymphoma cases, any of the 12 cranial nerves may be affected. Although such clinical phenomena have been described previously, this is the first patient to demonstrate restricted diffusion related to multiple cranial nerves.

Keywords: lymphoma; cranial nerves; diffusion / DWI; magnetic resonance imaging / MRI.

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1. INTRODUCTİON

Non-Hodgkin Lymphoma (NHL) is a hematologic malignancy with many histiologic subtypes and clinical presentations. The clinical presentation of NHL is highly variable because the lymphatic system is related to almost any organ system. In central nervous system (CNS) involvement, systemic NHL is generally thought to breach the blood–brain barrier via arachnoid vessels entering the subarachnoid space and neoplastic lymphocytes infiltrate the entire neuroaxis [1]. Neurologic signs and symptoms of patients with lymphoma are highly variable, too. The patients with leptomeningeal involvement can be asymptomatic or may present with multiple cranial neuropathies, headache, spinal cord signs (i.e., focal limb numbness or weakness), mental status changes, or spinal root symptoms (i.e., radiculopathy) [1,2]. Primary CNS lymphoma (PCNSL) can cause neuropathies in any of the 12 cranial nerves, but the 3rd, 4th, 5th, 6th, 7th, 8th, and 12th cranial nerves are most often affected [1,3,4].

The enhancement of cranial nerves which may be secondary to myelin breakdown and the low degree of inflammation seen in nerve biopsies on MRI has often been reported in inflammatory neuropathies, such as chronic inflammatory demyelization neuropathy [5]. Although we could not exclude the possibility of direct infiltration of lymphoma cells into the cranial nerves, homogenous enhancement patterns looked more like neuropathies mediated by the immunological disturbances rather than neuropathies influenced by direct tumor infiltration in our patient. In this article, we report a rare case of malignant lymphoma showing bilateral homogenous and symmetric enhancement of multiple cranial nerves and restriction in diffusion MRI. Although such clinical phenomena have been described previously [6], this is the first patient to demonstrate restricted diffusion in lymphoma case with multiple cranial nerve involvements showing restricted diffusion.

2. CASE REPORT

A healthy 50-year-old female presented to the emergency room of our Faculty with paresthesia on the right side of her face, facial asymmetry, and difficulty in walking. There was no deterioration in consciousness. Her neurological examination revealed peripheral typed facial paralysis, hypoesthesia on the right side of face, and gait ataxia. Paraparesis especially in distal parts of lower extremities was found. Deep tendon reflexes in lower extremities were decreased, and the pathological reflex was not found. There was a history of gastroenteritis 1 month ago. Ear-Nose-Throat exam showed bilateral loss of hearing. There was horizontal nystagmus on the right side during caloric test. According to the history and neurological findings, the first considered diagnosis was polyneuropathy and electromyography was performed.

Routine nerve conduction studies with F wave evaluations were normal. Lumber punction (LP) examination showed elevated protein level (473.8 mg/dL), normal glucose level (42 mg/dL), and 2-3 lymphocytes. Because of clinical findings related to cranial nerves, cranial MRI was performed. MRI was performed on a 1.5-T system (Philips, Gyroscan Intera Master, Best, The Netherlands). T1-weighted images (TR = 560 msec, TE = 15 msec) were obtained in the axial and sagittal planes. T2-weighted images (TR = 4530 msec, TE = 100 msec) were obtained in the axial and coronal planes. Cranial MRI revealed contrast enhancement of 3rd, 5th, 7th and 8th cranial nerves, and mastoiditis bilaterally (Fig. 1). For DWI a single-shot echo-planar pulse sequence (TR = 4832 msec, TE = 81 msec, field of view (FOV) = 230 mm, matrix size = 128 × 256, slice thickness = 7 mm, interslice gap = 1 mm) was used. Diffusion-weighted MRI (DWI) revealed prominent hyperintense symmetric cranial nerves involvement with corresponding signal reduction on ADC (Apparent Diffusion Coefficient) maps (Fig. 2). Lumbo-sacral MRI revealed simple central protrusions and compression fracture on L5 and S1 vertebrae corpuses. There was no contrast enhancement of roots. In differential diagnosis, Wegener granulamotosis, tumor metastasis, sarcoidosis, and leukemia were thought. Laboratory findings included: white blood-cell count 7.400/mul with 19.1% lymphocytes and 0.0% atypical lymphocytes, red blood-cell count 431 x 10^4/mul, platelet count 27.7x 10^4/mul, erythrocyte sedimentation rate 29 mm/1 h, aspartate aminotransferase 227 U/L, alanine aminotransferase 14.3U/L, serum creatine kinase 93.5 U/L, and lactate-dehydrogenase 409U/L. Rheumatoid factor was 21.8 ıu/ml. Anti-nuclear antibody titre was <1/100, anti-dsDNA was negative. Results of serum IgG and IgM antibody against borrelia, salmonella and brucella were negative. The remainder of the work-up, including serum test for hepatitis and HIV were normal or negative.

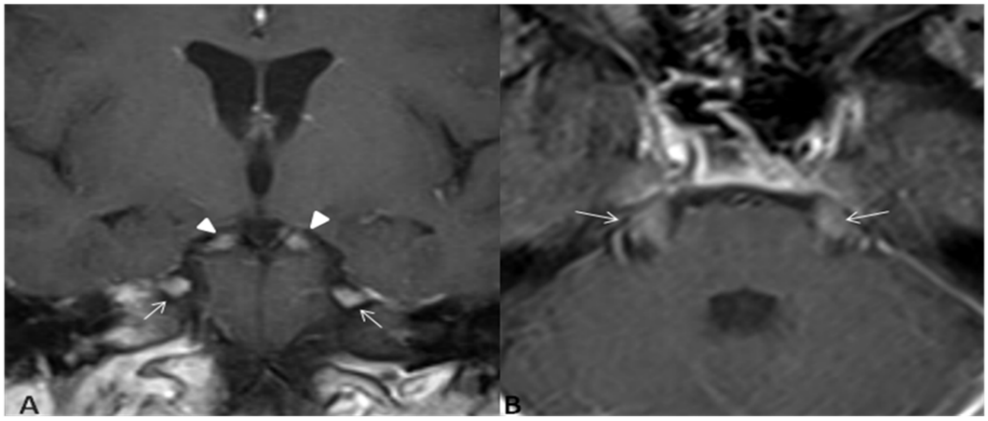

Fig. 1. Coronal (A) and axial (B) gadolinium-enhanced T1-weighted MRI views shows enlargement and enhancement of the trigeminal (arrowhead) and oculomotor nerves bilaterally (arrows)

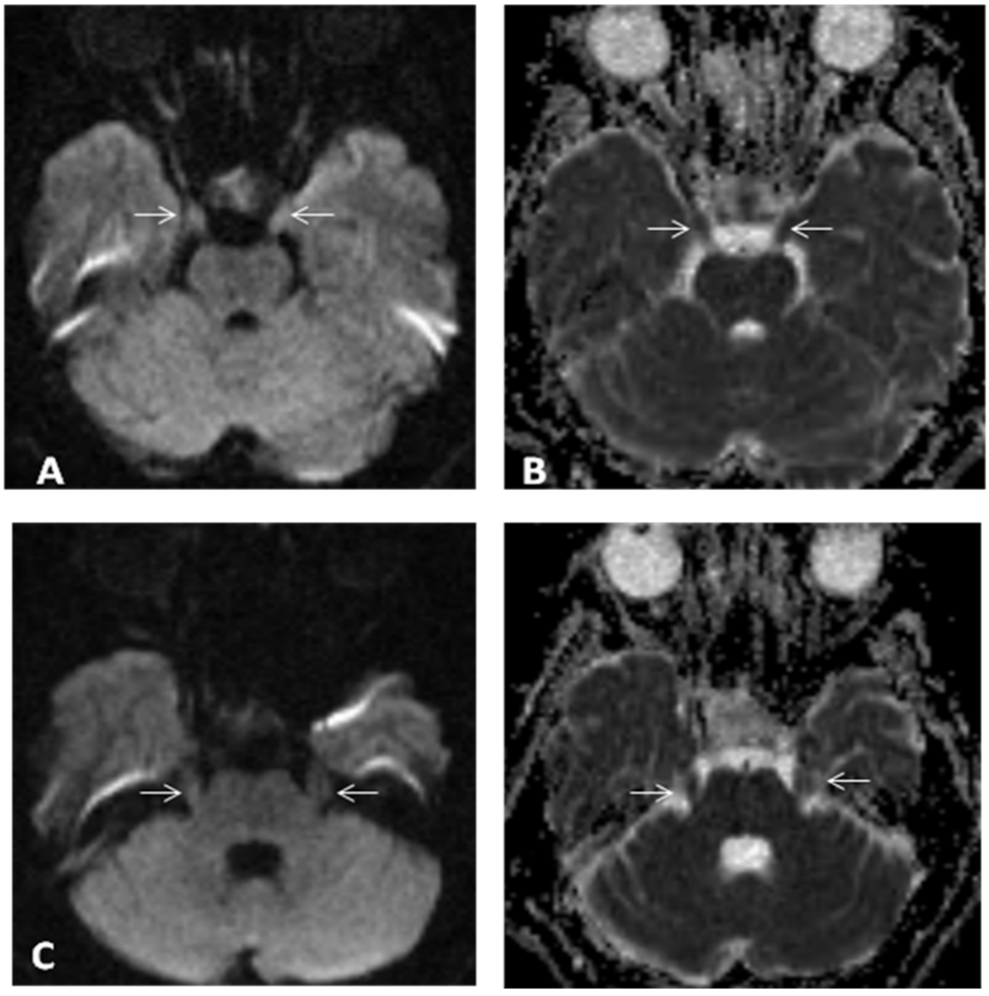

Fig. 2. DWI (A, C) with corresponding ADC map (B, D) shows diffusion restriction along the oculomotor (arrows on A and B) and trigeminal nerves (arrows on C and D) bilaterally

Abdominal ultrasound exam showed grade 2 hepatosteatosis and hemangioma in the liver. Thorax Computed Tomography (CT) and abdominal CT were normal. Angiotensin-converting enzyme level were (11.6 ıu/L) normal. Tumor markers were negative. Diagnosis of multiple cranial neuropathy related to nervous system lymphoma was made. Steroid therapy (1 gram i.v. pulse/day) was started. After 10 day pulse steroid therapy, oral prednisolone therapy (64 mg/day) was started. Repeated LP exam showed very high level of protein (465 mg/dL), too. Atypical lymphoid cells were found in cytological examination of LP material. Supraclavicular region lymphadenopathy was found, but ultarsonographic neck exam and oral exam were normal. After oncology consultation, Positron Emission Tomography (PET) was evaluated and PET exam showed F-18 fluorodeoxyglucose (FDG) uptake within medulla spinalis especially in thoraco-lumber part (Standardized uptake value was 6). There was not uptake through cranial nerves. Our patient was conciliated by hematology clinics of another university. After examination of LP material specimen, cytological examinations showed increased cells and malignant lymphoma cells with positive CD-20 which was the most popular protein marker in lymphoma, and the patient has taken a diagnosis of non-hodgin lymphoma type B.

3. DISCUSSION

Almost all primary PCNSL shows brain parenchymal lesions, and meningeal involvement is quite rare. But two thirds of them demonstrate meningeal involvement and the others have parenchymal lesions [7]. Non-hodgin lymphoma type B in our patient infiltrated 3rd, 5th, 7th, and 8th cranial nerves bilaterally and produced persistent restricted diffusion on DWI in our patient. To our knowledge, this is the first reported case of PCNSL with multiple cranial nerves showing restricted diffusion.

We know that CT, FDG uptake and the hybrid FDG-PET/CT are the most commonly used diagnostic tools for the initial staging and assessment of malignant lymphomas [8]. Whole-body MRI and diffusion-weighted imagings are radiation-free alternative techniques [9,10]. DWI is an MRI technique that measures the diffusion rate of unbound extracellular water molecules. Early investigations into DWI demonstrated that densely packed tumor cells with a high nuclear-to-cytoplasmic ratio could reduce water molecule motion [9]. DWI is characterized by high sensitivity for the detection of lesions in the evaluation of malignant lymphomas [8-10]. Formerly this technique was mainly used in stroke diagnostics but the spectrum of applications is becoming increasingly larger. It is now a useful tool for assessment of the depth of invasiveness of tumors and for differential diagnosis of brain tumors and tumor-like conditions [8-10]. Additionally, it has been previously proposed that primary CNSL can be histologically subclassified on the basis of cellular growth patterns into high and low cellular density tumors, which may have prognostic implications [10,11]. A further advantage in addition to the diagnostic capabilities is the speed of the sequence because DWI is little influenced by motion [8].

Recently, it has been reported that DWI-derived ADC measurements inversely correlate with histopathologic assessment of PCNSL tumor cellular density [10]. Akter et al. [9] reported on DWI in 16 PCNSL, approximately 80% lesions were hyperintense or partially hyperintense and the other lesions were isointense. The lesions with ADC value less than 0.7×10^{-3} mm2/s were all hyperintense on DWI [10]. Markedly reduced ADC values have been reported for PCNSL, especially in areas of dense cellularity [10,11]. Similar to Matano et al. [11] we have found that cranial MRI did not detect any intraparenchymal lesions, but thickening of multiple cranial nerves was detected. Swelling and contrast enhancement of bilateral cranial nerves on MRI was found.

4. CONCLUSION

To our knowledge, the use of DWI as findings of the cranial nerve involvement in PCNSL has not been previously reported. In our case, cranial nerves were shown as hyperintense relative to the normal-appearing cerebral cortex on T2 and FLAIR images. Contrast-enhanced T1-weighted axial and coronal images showed significant enhancement on cranial nerves bilaterally. DWI revealed prominent hyperintense symmetric cranial nerves involvement with corresponding hypointensity on ADC maps.

CONSENT

Not applicable.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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