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Case Report

Multiple myeloma presenting as isolated sixth cranial nerve palsy

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ABSTRACT

Multiple myeloma with isolated third nerve palsy as the presenting complaint is a rare entity. We report a case of a 30-year-old man who developed sudden onset left-sided complete abducens nerve palsy without pain. Cranial and orbital magnetic resonance imaging and cerebrospinal fluid examination demonstrated no abnormalities. He was diagnosed as having IgG type multiple myeloma, based on the immunological and pathological investigation. After instituting chemotherapy, palsy resolved within 3 weeks. Etiology is not established beyond doubt but probably palsy was caused by nerve ischemia due to hyperviscosity of the serum.

Keywords: Multiple myeloma, Abducens nerve

INTRODUCTION

Multiple myeloma (MM) is a plasma cell malignancy characterized by monoclonal expansion of lymphoplasmacytic cells in the bone marrow. Neurological manifestations of myeloma may include diffuse cerebral dysfunction, cranial nerve palsies, and spinal radiculopathies, chronic inflammatory demyelinating polyradiculoneuropathies. In MM, abducens nerve palsy (6NP) may be caused by its invasion to the clivus or intracranial plasmacytoma or by speculative mechanisms like nerve ischemia due to hyperviscosity. Here, we describe a case of MM presenting 6NP without intracranial lesion, which might be associated with hyperviscosity of the serum (HV).

CASE REPORT

A 30 year-old non-diabetic, non-hypertensive male presented with binocular horizontal diplopia of two weeks duration. No associated pain, blurring of vision was present. Diplopia increased on right gaze and corrected after closing right eye. He reported that the diplopia did not have a fixed pattern and was not aggravated by fatigue or any diurnal variation. . Patient gave a history of vague low back pain and two episodes of transient blurring of vision in last 6 months. On examination; corrected visual acuity was 6/6 in both eyes. Pupillary light reactions were normal. Fundoscopic examination demonstrated no papilledema. The extraocular motility examination revealed restricted abduction of the left eye. Remaining neurological examination was reasonably within normal limits. Systemic examination was unremarkable. Serum acetylcholine receptor binding antibody and tensilon test were negative. Cranial and orbital MR imaging demonstrated no abnormalities. Cerebrospinal fluid examination demonstrated that cell count was 3/µL (all lymphocyte), protein concentration at 32 mg/dL, and glucose at 62 mg/dL with no malignant cell. c-ANCA, p-ANCA,ANA were all negative. Serum ACE, Thyroid function were within normal ranges. Complete blood count showed presence of normocytic, normochromic

anemia .ESR was significantly raised (88mm/1st hour). Rouleaux formation was reported. Albumin-2.9g%; Globulin-5.6g%; X-ray skull showed presence of lytic lesions. Serum protein electrophoresis revealed monoclonal M spike. Bence-Jones protein was also detected in urinary examination. Markedly elevated IgG level (N--768-1632) mg/dl at 6,024 mg/dL. Bone marrow biopsy revealed a hypercellular marrow with diffuse infiltration of atypical plasma cells, which accounted for about 60% of the total marrow cellularity. On the basis of these findings, a diagnosis of IgG multiple myeloma was made.Upon institution of chemotherapy, nerve palsy resolved within 6 weeks without residual deficits almostin parallel with normalisation of Hb and Ig levels.

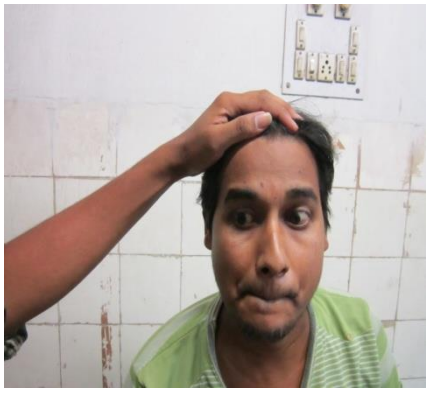

Figure 1: Patient with right 6th nerve palsy.

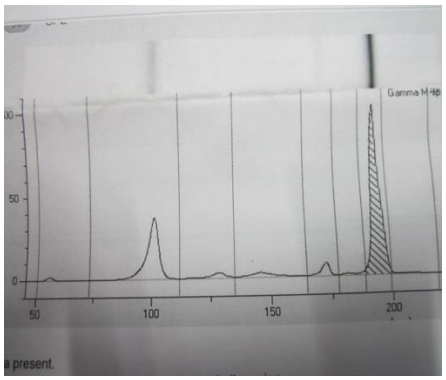

Figure 2: M spike on serum protein electrophoresis.

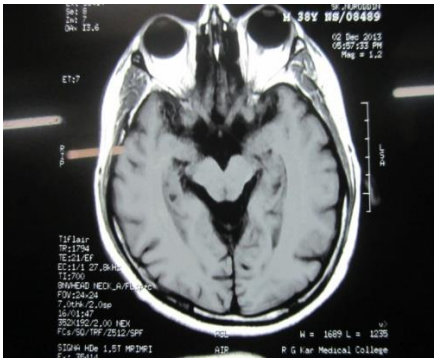

Figure 3: MRI brain (T2 flair) showing no lesion in brain.

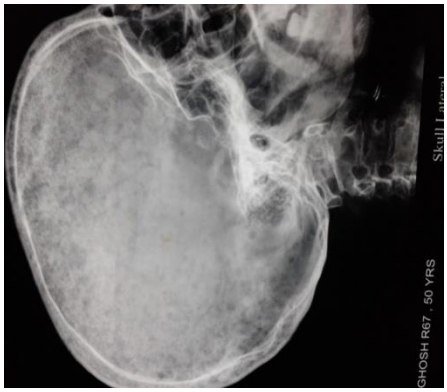

Figure 4: Skull X ray showing multiple lytic lesions suggestive of multiple myeloma.

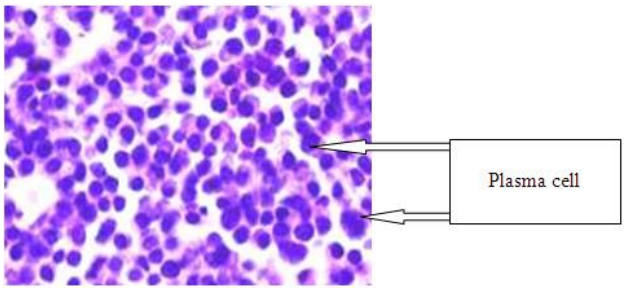

Figure 5: Bone marrow slide showing plasma cell infiltration.

DISCUSSION

The temporal relationship with multiple myeloma and its disappearance following treatment of myeloma suggests myeloma as the most probable etiology behind the sixth nerve palsy. The abducens nerve is running pieces the dura of the clivus, passes through the Dorello's canal formed by the petroclinoid ligament, and then enters the cavernous sinus. This nerve exits the cavernous sinus through the superior orbital fissure and innervates the lateral rectus muscle. In multiple myeloma, the mechanism of sixth cranial nerve palsy may include the following: hematologic effect, compression, meningeal metastasis,[1-3] direct infiltration of the nerve itself, invasion to the clivus or intracranial plasmacytosis compressive lesions, meningeal deposits, direct spread to cranium.[4-7] In our case, alternative causes of sixth nerve palsy have been excluded by relevant investigations. The remaining plausible mechanism is nerve ischemia due to hyperviscosity in myeloma. Presence of history of transient blurring of vision in this patient lends credibility to the hyperviscosity and rouleaux formation being the possible mechanism behind the abducens palsy.

Patients with a vasculopathic abducens palsy usually have complete resolution as was noticed in our case. The presumed mechanism of vasculopathic abducens nerve palsy involves thickening and hyalinization of nutrient vessels, which results in ischemic demyelination of a

portion of the nerve.[8] The area of ischemic demyelination subsequently undergoes remyelination over time, accounting for spontaneous recovery. Other risk factors for ischemic sixth nerve palsy, such as diabetes mellitus, hypertension, hyperlipidemia and smoking habit were not present. Therefore, it can be reasonably concluded that hyperviscosity due to underlying multiple myeloma might have caused ischemic abducens palsy in our case.

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Conflict of interest: None declared
Ethical approval: Not required

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