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Bell's Palsy as the Initial Presentation of Multiple Myeloma: A Case Report

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Abstract A 54-year-old white woman presented to the emergency department with inability to smile, difficulty in closing her right eye and drooling when attempting to drink liquids for two days prior to presentation. Neurological examination revealed classic signs of right peripheral seventh cranial nerve paresis. The patient also complained of fatigue, general malaise and weight loss of 20 pounds during the past few weeks.

Keywords: *Bell's palsy, multiple myeloma*

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1. Background

Neurologic symptoms are common in multiple myeloma and may be the chief complaint of some patients. Some manifestations are spinal or nerve root compression and peripheral neuropathy [1]. Treatment-related neurologic symptoms are more common with use of proteasome inhibitors and immune-modulators lenalidomide and thalidomide [2]. Metabolic consequences of multiple myeloma may present a broad spectrum of neurologic symptoms. Facial nerve paralysis has been described in cases of Waldenstrom's macroglobulinemia [3]. A case has been reported of a plasmacytoma affecting the facial nerve that mimicked Bell's palsy [4]. We report a case of immunoglobulin (Ig) A kappa multiple myeloma presenting with Bell's palsy that resolved with treatment. This association has not been reported previously.

2. Case Report

A 54-year-old white woman presented to the emergency department with inability to smile, difficulty in closing her right eye and drooling when attempting to drink liquids for two days prior to presentation. Neurological examination revealed classic signs of right peripheral seventh cranial nerve paresis. The patient also complained of fatigue, general malaise and weight loss of 20 pounds during the past few weeks.

At presentation, the patient was hyperuricemic, hypercalcemic, hyponatremic and hyperglycemic with relatively normal renal function (Table 1).

Table 1.

Laboratory Results	Dec 2014	Jan 2015	Feb 2015	April 2015
Total protein (g/dl)	15.2	9.2	7.1	7
Globulins (g/dl)	11.5	6.1	3.3	3.6
IgA Kappa monoclonal proteins (g/dl)	10.6	n/a	n/a	0.6
Albumin (g/dl)	2.4	3.1	3.8	3.6
Creatinine (mg/dl)	1.2	0.7	0.8	0.8
Corrected calcium (mg/dl)	14.7	9.9	10	10.1
Immunoglobulin A (mg/dl)	8,740	3,050	1,080	376
Uric acid (mg/dl)	40.5	< 0.2	7.6	n/a
Serum viscosity (cps)	12.3	n/a	4.2	n/a
Beta-2 microglobulin (mg/L)	10.6	n/a	n/a	2.5
Monoclonal plasma cells in bone marrow	82%	n/a	n/a	10%
T(4;14)	47%			8.5%
Monosomy 13	pos			neg
1q21 amplification	2.9			1.4
Hemoglobin (g/dl)	6-7	9-10	11-12	12-13

Complete blood count showed 5.5k/mcl white blood cells, hemoglobin at 7.5 gm/dl and 204k/mcl platelets. Erythrocyte sedimentation rate was >120 mm/hr. A serum viscosity sample taken after several hours of hydration was elevated at 12.2 (normal reference range 1.4-1.8 cP). A posterior iliac crest bone marrow biopsy showed 90%

cellularity with 82% monoclonal IgA kappa-restricted plasma cells. Fluorescence in situ hybridization (FISH) studies on the bone marrow showed the presence of t(4;14), monosomy 13 and 1q21 amplification. Magnetic resonance imaging (MRI) of the brain did not show any abnormalities. Positron emission tomography-computed tomography showed a single hypermetabolic lesion in the right scapula.

The patient underwent two full-volume plasma exchanges with significant improvement in facial weakness over two days. The patient received weekly cyclophosphamide, bortezomib and dexamethasone for four cycles resulting in reduction of bone marrow plasma cells to 10%. Total monoclonal protein decreased from 10.6 g/dL to 0.6 g/dL. Facial nerve paralysis resolved in two months. Subsequently the patient received two cycles of bortezomib, lenalidomide and dexamethasone prior to high-dose melphalan therapy and an autologous hematopoietic stem cell transplant.

3. Discussion

In this case of isolated seventh nerve palsy as the initial presentation of a patient with IgA kappa multiple myeloma, it is likely that the nerve palsy was due to hyperviscosity with evidence of the negative MRI, markedly elevated serum viscosity and prompt response to plasmapheresis.

Hyperviscosity is more commonly associated with Waldenstrom's macroglobulinemia than multiple myeloma [5] and symptoms usually occur when monoclonal protein levels are >15 g/dl for IgG, >4g/dl for polymerized IgG3, >10 g/dl for IgA and >3 g/dl for IgM. The incidence of symptomatic hyperviscosity in Waldenstrom's macroglobulinemia is 10-30% and 2-6% in IgG myeloma. FISH studies of our patient's bone marrow showed three unfavorable cytogenetic changes. The severity of presentation and prognosis is likely a function of the cytogenetic/molecular abnormalities [6].

It is also possible that the nerve paralysis was incidental Bell's palsy. The onset of Bell's palsy is sudden and not generally associated with systemic symptoms other than those of isolated seventh nerve paralysis. The natural history of Bell's palsy ranges from complete recovery in a few weeks in the majority of patients to permanent nerve damage in the minority [7]. The presence of systemic symptoms in patients with isolated seventh nerve palsy should alert the clinician to explore alternative diagnoses.

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