FACIAL PALSY AS FIRST PRESENTATION OF ACUTE LYMPHOBLASTIC LEUKEMIA: A CASE REPORT

S. Inaloo MD [1],
M. Karimi MD [2]

Abstract:

Objective

Facial paralysis in children is very often idiopathic and isolated facial nerve palsy, resulting from leukemic infiltration is a rare occurrence.

Here we present the case of a 14 year-old boy with acute lymphobastic leukemia, who first presented with isolated right side peripheral facial nerve paralysis and was initially diagnosed with Bell's palsy.

Conclusion

The presence of Bell's palsy in young children requires a complete evaluation, keeping in mind the possibility of leptomeningeal disease.

Key words: Lymphoblastic Leukemia, Facial nerve palsy, Children.

Introduction

Idiopathic peripheral facial paralysis (Bell's palsy) is the most cause of common facial palsy in adults and older children, whereas other causes of acquired peripheral facial weakness are much less common. Associated conditions include diabetes mellitus, hypertension, HIV infection, Lyme disease, the Ramsay Hunt syndrome (facial palsy with zoster oticus caused by varicella–zoster virus), sarcoidosis, Sjogren's syndrome, parotid nerve tumors and amyloidosis.

The incidence of Bell's palsy in adult is 20-35 case per 100/000 people per year; it accounts for 60-75 percent of all cases of unilateral facial paralysis.

Although the precise incidence in children and infants is not available, it is believed to be lowest in children under 10 years (1,2).

Facial paralysis is a known complication of leukemia of the central nervous system; however facial palsy as the presenting symptom of childhood leukemia is not well recognized. Indeed standard textbooks in neurology, pediatrics, and oncology do not cite facial palsy as one of the presenting features of leukemia (3).

In this report, we present a boy with acute lymphoblastic leukemia who referred with isolated unilateral facial palsy, a rare presenting feature of leukemia.

Case report

A 14-year old boy presented with isolated acute onset of right peripheral facial palsy without any other symptoms and signs. The patient was treated with prednisolone, 1 mg/kg/day for 2 weeks. After 2 weeks of the drug, the patient showed relative improvement in facial paralysis but had complaints of bone pain, backache and fever. No abnormalities were seen in the general and neurologic exams, except for slight right peripheral facial palsy. Hematogramed revealed hemoglobin, 11.5g/dl, total

1. Asssistant Professor of Pediatric, Pediatric Neurologist, Shiraz University of Medical Sciences
2. Professor of Pediatric Hematology/ Oncology,Hematology Research center, Shiraz University of Medical Sciences
Corresponding Author:
S.Inaloo MD
Tel :+ 98 711 62625024
Fax: + 98 711 62625024
Email: Sinaloo@Sums.ac.ir

leukocyte count: 6200/µL (57% poly, 41% lymphocyte), platelet count: 238000/µL and sedimentation rate was 23. Brain MRI with and without contrast was recommended. After 2 weeks the patient referred with normal MRI results, but the fever and bone pain were still there. A physical exam of the patient showed no facial palsy, but he still looked pale and ill; he had mild hepatosplenomegaly with slight right sided parotid enlargement. Cell blood count showed hemoglobin: 8.8 g/dl, total leukocyte count: 31700/µL (71% blast) platelet count of 47000/µl. Bone marrow aspiration showed 90% blast cells indicating acute lympholastic leukemia. The flow cytometry revealed T cell acute lympholsastc leukemia.

Cerebrospinal fluid analysis showed protein 75mg/dl, sugar 65mg/dl, total cell 150/mm^2, WBC of 130 /mm^2 , segmmented 10%, lymphoblast 30%, lymphocyte 60%. He underwent chemotherapy for high-risk leukemia. The child ultimately recovered, and was discharged after 6 weeks of hospitalization. During a 7-month follow up, the patient was found to be in remission and was receiving maintenance therapy.

Discussion

Leukemia is the most common childhood malignancy and can present with varying clinical features; cranial neuropathy, especially of the seventh nerve, is well known to occur in children with central nervous system leukemia caused by leukemic infiltration but isolated facial palsy is a rare presenting feature of leukemia. There have been few reports of facial palsy as the presenting symptom of leukemia in adults and children (1, 4, 5).

Most of the case reports available in present day literature are of adults and mostly about cases with acute myelogenous leukemia as granulocytic sarcoma that involved the parotid and facial nerve (6, 7). Our patient however had lymphocytic leukemia.

Facial paralysis in children with leukemia is caused by infiltration of the nerve with leukemic cells (8). Magnetic resonance imaging (MRI) with contrast especially of the facial nerve canal facilitates facial nerve enhancement.

Meningeal infiltration in the early stages, however, may not reveal an abnormality, either on MRI or CSF analysis (9). The absence of positive MRI in this case may reflect the fact that thin slicing of the facial canal was not performed or may be due to partial remission of leukemia with prednisolone therapy. Current MRI techniques are well equipped to demonstrate facial nerve enhancement more readily (10). Existing case reports about acute lymphoblastic leukemia suggest a preponderance of T cell leukemia for this complication (1,4) ; our patient was also diagnosed with T. cell acute lymphoblastic leukemia. Evidence of active leukemia in other organs may be lacking or absent at the onset of facial palsy, as it was in our patient.

The routine use of steroids in the treatment of patients suspected of having idiopathic facial palsy may be to provide partial remission, which could cause a delay in the actual diagnosis. In our patient too, the delay in diagnosis was due to steroid therapy.

In any patient with facial palsy, physical examinations including complete neurologic examination, ostoscopy and blood pressure evaluation are mandatory; further investigations are not indicated in the absence of any abnormal symptoms or signs (7). We suggest complete physical examination, including in particular, lymph node, liver, and spleen examination; complete blood count and a peripheral smear should also be done as a routine test for all children presenting with a pheripheral type of facial palsy. All such patients must be followed up and revaluated for the development of any new sign or symptoms.

Acknowledgment

The author would thank Dr Ashkan Mola for his editorial assistance and also the Center for Development of Clinical Research of Shiraz University of Medical Sciences, Shiraz, Iran for their support of this research.

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