TUBERCULAR MENINGITIS MASQUERADING AS UNILATERAL FACIAL PALSY

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ABSTRACT

We report an unusual case of sudden onset lower motor neuron facial palsy in 60 year old male. The diagnosis was based on cerebrospinal fluid examination and MR imaging. The bells palsy resolved after starting antitubercular therapy and steroids. our case is an uncommon case of acute onset Lower motor neuron bells palsy due to tuberculosis, in absence of any symptoms like fever headache vomiting and meningeal signs.

KEYWORDS: Cerebrospinal, MR imaging, antitubercular therapy.

INTRODUCTION

TB of central nervous system accounts for 5% of extrapulmonary cases in US. The early manifestations are usually low grade fever, malaise, headache (more than 50% of cases) lethargy, confusion and stiff neck (75% of cases). Because of inherent chronicity of the disease, signs of cranial nerve involvement (usually ocular palsies, less often facial palsies or deafness) and papilledema may be present at the time that the infection is recognized(20% of cases).in approximately two thirds of patients with tubercular meningitis there is evidence of of active tuberculosis elsewhere. CSF shows 50 to 500 white cells per cubic millimeter. Lymphocytes predominate. protein content being 100 to 200mg/dl. gucose is reduced to below 40 mg/dl.

CASE REPORT

A 60 year old male presented with sudden onset facial asymmetry, deviation of angle of mouth to left and inability to close right eye since five days. There was no history of headache, vomiting, fever, upper respiratory tract infection, any discharge from ears. There was right lower motor neuron complete facial palsy. Blood investigations and pure tone audiometry was normal. Patient was diagnosed as bells palsy.patient was treated with steroids, physiotherapy and eye care.however there was no improvement. Patient came to tertiary hospital where after thorough history taking and examination there was a doubt of chronic meningitis in view of terminal neck stiffness and long standing decreased appetite and poor response to treatment. His evaluation revealed normal baseline investigations but CSF examination revealed lymphocytic pleocytosis and high ADA.and CEMRI showed meningeal enhancement with focal lesion in pons right side accounting for his nuclear seventh cranial nerve palsy. Patient was started on ATT and steriods and after few days patient started showing signs of improvement.

DISCUSSION

Bell’s palsy is an acute peripheral facial nerve affection, usually affecting only one side of the face. The clinical picture varies, depending on the location of the lesion of the facial nerve along its course to the muscles. Symptoms and signs result from the fact that the facial nerve not only carries motor fibers including fibers to the stapedius muscle but also supplies autonomic innervation of the lacrimal gland, submandibular gland, sensation to part of the ear, and taste to the anterior two thirds of the tongue via the chorda tympani. Thus, Bell’s palsy is diagnosed upon abrupt onset of impaired facial expression due to unilateral facial weakness of all facial nerve branches, dry eye, if saliva runs out of the mouth, the inability to close or wink the eye or close the mouth, to droop the brow or the corner of the mouth, numbness or pain around the ear, temple, mastoid, or angle of the mandible, an altered sense of taste, hypersensitivity to sounds, or decreased tearing. Unilateral peripheral facial nerve palsy may have a detectable cause (secondary facial nerve palsy) or may be idiopathic (primary) without an obvious cause (Bell’s palsy). Secondary facial nerve palsy is due to various causes like metabolic, stroke, vascular, trauma tumour, infections (otitis media, mastoiditis, herpes simplex infection, varicella zoster...
infection tubercular meningitis), surgery drugs and immune system disorder.

CONCLUSION

Patients developing Bell’s palsy should be seen by a neurologist, oto-rhino-laryngologist, and ophthalmologist with the least possible latency after onset of the palsy. All patients in whom secondary facial nerve palsy is suspected a diagnostic work-up for the presence or absence of possible causes should be promptly initiated. If any of these causes is detected, it should be assessed if there is a causal relation between the palsy and the detected cause or not. Though a final decision on the optimal therapy of acutely developing Bell’s palsy cannot be actually proposed, patients should be provided with all measures to avoid secondary infection of the eyes if the lid closure is insufficient or in case of impaired tearing. According to a recent double-blind, placebo-controlled trial on 496 patients early administration of corticosteroids resulted in a significantly better outcome than placebo. In case steroids are used in diabetic patients, serum glucose should be frequently followed.

REFERENCES


