Case Report

Post-mumps acute disseminated encephalomyelitis in an adolescent

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Abstract

Mumps is an acute communicable self-limiting swelling of the parotid or other salivary glands. Various organs can be involved including the testes, central nervous system, mammary glands, ovary, pancreas, kidneys, and heart. We hereby present a rare case of an 18-year-old unvaccinated male with acute disseminated encephalomyelitis following mumps without parotitis.

Keywords: Acute disseminated encephalomyelitis, adolescent, mumps, unvaccinated

Introduction

Mumps, a paramyxovirus, commonly infects the salivary glands and the testes. Neurologic complications are documented with aseptic meningitis being the most common neurologic complication.[1] Other central nervous system (CNS) manifestations include cranial nerve palsies, cerebellar ataxia, transverse myelitis, Guillain-Barré syndrome (GBS), and behavioral changes.[2] Acute disseminated encephalomyelitis (ADEM) following mumps without parotitis is an uncommon presentation.

Case

An 18-year-old man presented with complaints of fever for 3 days, which was high grade, continuous in nature, and not associated with chills and rigor. The patient developed altered sensorium 1 day prior to admission. There was no significant medical or surgical history. There was no history of any vaccination in childhood or in recent past. On examination, he was unconscious with Glasgow Coma Scale (GCS) score of E4V1M4. His core temperature was 100°F. The blood pressure and pulse rate were 110/80 mmHg and 82 beats/minute, respectively. CNS examination revealed spastic tone along with bilateral positive Babinski sign. Meningeal signs were positive, and no cranial nerve involvement was seen. Rest of the general physical and systemic examination did not reveal any abnormality.

Laboratory investigations revealed blood count, liver function tests, and renal function tests were all within normal limits. Tests for viral hepatitis, malaria serology, and HIV were negative. Dengue serology and chikungunya serology were negative. Noncontrast computed tomography of head was normal. Cerebrospinal fluid (CSF) examination revealed total leukocyte count of two cells with protein 49 mg/dL and sugar 59 mg/dL. Mumps serology [immunoglobulin M (IgM) antibodies by MAC ELISA; NIV, Pune] was positive, but IgG antibodies were absent. Serology and CSF polymerase chain reaction for Epstein–Barr virus, herpes simplex virus 1, cytomegalovirus, herpes simplex virus 1, measles, and rubella were negative. CSF for oligoclonal bands and antiaquaporin-4 antibodies were negative. CSF polymerase chain reaction for mumps was positive.

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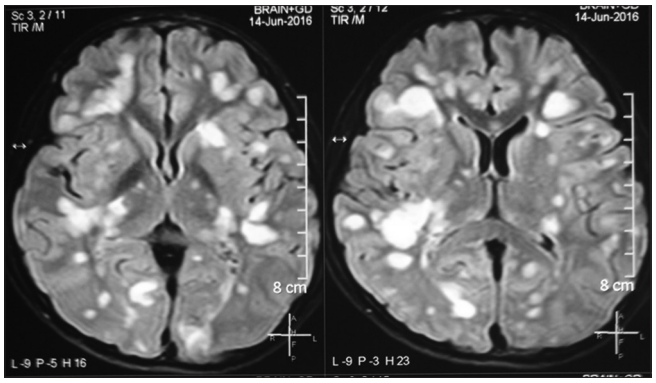

Figure 1: CEMRI/T2 FLAIR of brain showed altered signal intensity in bilateral subcortical white matter and cortex, thalamus, pons, and middle cerebellar peduncle with restricted diffusion and no significant postcontrast enhancement suggestive of ADEM

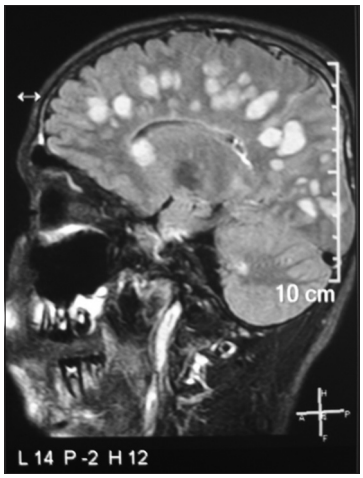

Figure 2: CEMRI/T-2 FLAIR of sagittal section of brain showing involvement of cortex with no periventricular involvement

Contrast-enhanced magnetic resonance imaging (CEMRI) brain showed altered signal intensity in bilateral subcortical white matter and cortex, thalamus, pons, and middle cerebellar peduncle with restricted diffusion and no significant postcontrast enhancement suggestive of ADEM [Figure 1]. CEMRI T-2 flair image of sagittal section showed involvement of cortex with no periventricular involvement [Figure 2].

Pulse steroid therapy was initiated, but the patient, however, developed respiratory distress and was shifted to the intensive care unit and intubated and put on SIMV mode of ventilation. The patient showed no significant improvement on pulse steroid therapy, and plasmapharesis was planned due to nonaffordability of intravenous immune globulin. After seven cycles of plasma exchange, he showed improvement with GCS of E4VTM6. The patient was discharged later after tracheostomy closure. Three months after the discharge, the patient requires assistance in daily activities. GCS has improved to E4V2M6 and no relapse or deterioration was seen.

Discussion

Mumps is characterized by an acute-onset tender, self-limited swelling of the parotid or other salivary glands, lasting at least 2 days, but may persist longer than 10 days.[3] Parotitis usually precedes manifestations of involvement of other sites of virus infection, but the latter can be clinically evident before, during, or even in the absence of parotitis. With mumps meningitis, half may not have detectable salivary gland enlargement.[4] Aseptic meningitis is the most common neurological manifestation of mumps, present in 10% of patients with mumps-related neurological complication.[5] Other reported neurological complications are encephalitis, ADEM, brain stem encephalitis, sensory neural hearing loss, GBS, and peripheral neuropathy.[5,6] ADEM is frequently preceded by an antecedent viral or bacterial infection in 75% of cases.[5,6]

Primary encephalitis is typically the response to direct viral invasion of neural cells, whereas postinfectious encephalitis is believed to be an autoimmune attack on CNS myelin sheaths. Symptoms of postinfectious encephalitis and associated demyelination appear 1–3 weeks after the onset of parotitis.[7] However, in vaccinated individuals reliance only on IgM detection is no longer advised, given the difficulty of its detection in anamnestic immune responses. Thus, in vaccines, the absence of IgM mumps antibodies is not confirmation of the absence of mumps infection. Furthermore, testing of sera too early or too late in the course of disease can also yield false-negative IgM results.[8] Up to 90% of patients with acute mumps meningitis produce virus-specific IgG within the CNS compartment and one-half show IgM antibodies.[9]

In ADEM, it is important to first consider a treatment with antibiotics and/or acyclovir until an infectious cause is ruled out. A pulse steroid therapy is the primary treatment of ADEM. Plasma exchange is recommended if there is no response to corticosteroids. Intravenous immunoglobulin is recommended if there is no response to plasma exchange. Mumps per se is treated symptomatically.[10]

Conclusion

Parotitis in mumps is usually present though it must not be assumed that it is either primary or essential. With involvement of the CNS, there is higher fatality and the apparent ineffectiveness of passive protection leaves vaccination as the only practical control measure. Thus, emphasis on vaccination coverage needs to be underlined. Encephalomyelitis following mumps is an uncommon event in young adults, and diagnosis without parotitis requires sound clinical judgment and laboratory evaluation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients

understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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