MUMPS ENCEPHALOMYELITIS

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ABSTRACT
A 7-year-old Indian girl developed complete paralysis of her lower limbs and acute urinary retention 10 days after suffering from mumps. Encephalomyelitis due to mumps was not suspected initially since it is a rare complication of mumps, although relatively well-documented. However, the preceding history of parotitis and the presence of mumps-specific IgM in both blood and cerebrospinal fluid led to the diagnosis. The initially severe acute neurological deficits resolved completely three months after onset of her illness. Serological investigations were helpful in diagnosing neurological complications of mumps in this case, and especially where there is no preceding parotitis.

Keywords: Mumps, encephalomyelitis, infection.

INTRODUCTION
Mumps is a common and usually benign viral infection in children. Estimates of the incidence of central nervous system (CNS) complications of mumps vary from 0.5 to 30% and meningitis is undoubtedly the commonest complication. Brain and/or spinal cord involvement are rare manifestations of mumps, occurring only once in each 6000 cases. Mumps encephalitis is a serious condition with high mortality and less favourable prognosis in contrast to mumps meningitis which is a relatively trivial complication.

It is likely to be a more protracted illness and can be followed by lasting sequelae. We report a case of mumps encephalomyelitis with a good outcome in a young girl.

CASE REPORT
A 7-year-old Indian girl presented with abdominal pain and weakness in both legs for three days, associated with difficulty in passing urine. History from the mother revealed that 10 days prior to admission she had developed bilateral painful parotid swellings that subsided over 6 days. The weakness of her legs was progressive and she was completely paralysed in her limbs by the third day of admission. She had also become increasingly drowsy. She was previously well and there was no significant family history.

On examination she was drowsy but responsive to verbal commands. Axillary temperature was 38.2°C. There was bilateral ptosis with normal extra ocular movements. The pupils were equal and reactive and there was no papilloedema. The nasolabial groove was less prominent on the left side. There was severe neck stiffness. The lower limbs were hypotonic and areflexic with grade 0 power while the upper limbs were normal. The plantar responses were equivocal. The anus was patulous and the anal reflex was absent. The sensation was intact. Abdominal examination revealed a grossly distended bladder. There were no cutaneous lesions overlying the lumbar spine. The cardiovascular and respiratory systems were normal.

Investigation showed a haemoglobin of 10 g/dL with a total white cell count of 7.2 x 10⁹/L, polymorphs 48% and lymphocytes 52%. The cerebrospinal fluid (CSF) was clear and there were no cells. The CSF protein was 0.85 g/dL, chloride 126 mmol/L and glucose 3.2 mmol/L. The plasma glucose was 5.0 mmol/L. Virological examination of both blood and CSF confirmed presence of IgG (titre > 1:400) and IgM antibodies to mumps virus.

The patient required bladder catheterisation for 21 days, after which she was able to micturate on her own. The power in her legs began to improve steadily with physiotherapy. She could walk a few steps 33 days later. She was finally discharged about 5 weeks after admission, with mild weakness in her lower limbs. She was found to be completely normal on neurological examination during her last follow-up visit, 3 months after onset of her illness. She had not complained of any hearing loss on enquiry.

DISCUSSION
Mumps virus can affect many organs, with or without involvement of the salivary glands. The diagnosis of CNS complications is relatively easy if there is salivary-gland involvement, but when this is absent an accurate diagnosis can be made only by serologic tests. In 50% or more of cases with neurologic involvement by the mumps virus, serologic tests provide the only indications of the etiologic diagnosis. On the basis of clinical and pathologic observations, it has been suggested that CNS injury may be caused by direct viral invasion alone (primary encephalomyelitis) or through an immune response of the host to breakdown products of cells and myelin (postinfectious encephalomyelitis).

The pathological picture at autopsy is of an acute perivascular demyelination which is similar to other forms of postinfectious encephalomyelitis. Mental symptoms are prominent in encephalitis, and clinical features include drowsiness, convulsions, headache, psychoses, ataxia and hemiplegia. Neck stiffness, involuntary movements, increased reflexes, and extensor plantar responses may be found. Cranial nerve palsies involving the optic, oculomotor, trigeminal or facial nerves may occur. Myelitis can present in 2 forms either as a segmental involvement of the spinal cord involving the lower
thoracic or lumbar regions or a paralytic poliomyelitis-like syndrome with muscular weakness and loss of tendon reflexes [10].

The prognosis of mumps encephalomyelitis varies from complete recovery without neurological deficit, through a range of cranial nerve and limb palsies, to death from respiratory paralysis [11]. A case of chronic mumps encephalomyelitis has also been recently documented in the literature [12].

The mortality in mumps encephalitis is about 20% and fatalities have also been reported in cases of mumps myelitis [13]. Initial convulsions, psychic symptoms, affected consciousness and lack of salivary gland swelling are associated with poor outcome [14]. This case report helps to create an awareness of unusual complications of mumps, particularly in areas where mumps vaccination is not routinely offered. It also highlights the difficulty one may encounter in making a clinical diagnosis of CNS involvement following mumps in cases with no history of parotitis.

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REFERENCES