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Case report

Infectious mononucleosis with cranial nerve palsies

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The neurological complications of infectious mononucleosis may rarely occur in the absence of the classical symptoms and signs of infectious mononucleosis. Widespread involvement of the cranial nerves is very rare. It is important to consider infectious mononucleosis in the differential diagnosis of any young adult presenting with a neurological illness.

CASE HISTORY

A 19-year-old female student presented with a one-week history of headaches in the left temple, each lasting two hours and preceding a sudden onset of diplopia and left ptosis, which came on while she was photocopying at work. The diplopia deteriorated over the next few days and she developed an aching pain in the jaw. There was a three-day history of difficulty in swallowing and of slurring of speech. In particular, she had difficulty moving food around her mouth. There was no history of weakness in the limbs. She had had a mild irritation of her throat two or three weeks prior to her admission. There was no history of fever. She was well nourished, alert and well orientated. There was no significant enlargement or tenderness of the lymph nodes. She was not icteric. There were no rashes. She was normotensive, in sinus rhythm and had no cardiac murmurs. The chest was clinically clear. Liver and spleen were not palpable.

The pupils were normal size and reacted normally to light and accommodation. Visual acuity was 6/6 on both sides. Fields of vision were full. Optic fundi were normal. There was no bilateral ptosis. External ocular movements were restricted on both sides. Depression and adduction were full in both eyes. In the right eye, adduction was reduced to 50% of the full range and elevation to 25%. In the left eye, elevation was reduced to 50% and adduction to 75%. There was no nystagmus. Corneal reflexes and facial sensation were normal. Jaw jerk was not present. There was no wasting or weakness of the jaw muscles. There was an almost complete bilateral facial palsy. Hearing was not impaired and she could hear a whisper at two feet. Rinnie's and Weber's tests were normal. The soft palate moved normally, and gag reflexes were normal. The sternomastoids and trapezii had full power. The tongue was weak and could not be protruded fully. It

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could not be placed in either cheek. In the limbs, the power tone and co-ordination were normal. The tendon reflexes were all present, equal and normal. The plantar responses were flexor. There was no limb ataxia. There was no sensory loss. Tensilon test was negative.

The monospot test was positive, and the Paul-Bunnell reaction positive to a titre of 512, with absorption by ox cells but not by guinea pig cells. Epstein-Barr virus specific IgM was present in the serum, and about three months after the onset of her illness antibody to Epstein-Barr nuclear antigen was detected. Haemoglobin concentration was 12.5 g/l, with normal red cell indices. White cell count was 6,500/ml, with an excess of abnormal mononuclear cells. Chest X-ray was normal, CT scan of skull normal, EEG normal. Cerebrospinal fluid protein 0.30 g/l red cells <1 per µl lymphocytes 5 per µl. Serum sodium was 141 mmol/l, potassium 4.0 mmol/l, urea 3.6 mmol/l, total protein 82 g/l, albumin 45 g/l. Liver function tests showed total bilirubin 4 µmol/l, alkaline phosphatase 138 U/l, lactic dehydrogenase 303 U/l (normal range 130-270), aspartate transaminase 72 U/l, alanine transaminase 198 U/l, gamma glutamyl transpeptidase 43 U/l (normal 5-34), pseudocholinesterase 103 U/l. Prothrombin time was 62%. HBs Ag was negative, antinuclear factor negative, antistriated muscle antibody negative. Nerve conduction studies were normal in the right median and lateral popliteal nerves. (The usual range is quoted in brackets only where the value obtained was abnormal).

Following admission there was no deterioration in her condition. She was started on prednisolone 60 mg and this was tailed off over 10 days. It made no impression on her palsies, which slowly improved over a period of three months. At recent review she was symptom-free and had no sequelae.

COMMENT

The neurological complications of infectious mononucleosis were first reported in 1931.[1,2] The range of complications includes meningitis, encephalitis, cranial nerve lesions, mononeuritis, polyneuritis and spinal cord lesions; transient psychotic episodes have also been reported.[3,4] The incidence of neurological complications is difficult to assess, because many series are based on hospital patients and thus tend to include the more severe cases of infectious mononucleosis; however, it has been reported as varying from 0.37% to 26.5%.[5]

The neurological involvement can precede or follow the common manifestations of fever, pharyngitis, lymphadenopathy and splenomegaly, and in some cases the only clinical signs of infectious mononucleosis have been related to the nervous system.[5] All the cranial nerves have been involved in the disease,[4] although multiple involvement of cranial nerves, as in this case, is rare.[6] We have found only one case reported in the literature where the range of cranial nerve involvement was so widespread.[3] The Miller Fisher syndrome (ophthalmoplegia, ataxia and areflexia) has been reported in association with infectious mononucleosis[8] but does not satisfactorily describe this case. The pathological changes in the brain in patients dying with signs of cerebral damage consist of inflammatory lesions with dense perivascular cuffing and diffuse infiltration of the parenchyema mainly with atypical mononuclear cells.[7]

The prognosis is good for a complete recovery of the neurological complications.[5] In the cases involving the cranial nerves, the mean time to complete recovery was 70 days with a range of two to 240 days.[6] The use of steroids is controversial. Some authors recommend that steroids should be used in cases where infectious

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mononucleosis is complicated by neurological involvement,[9, 10] but there does not seem to be good evidence that their use is beneficial.[6] There is also the consideration that steroids may enhance replication of herpes viruses.

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