Short report

Bell's palsy and infection with rubella virus

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SUMMARY Viral antibody-titres were measured in 28 patients with Bell's palsy seen in Baghdad. These cases were selected to include only those seen within 24 hours from onset. No association with recent viral infection other than rubella virus was demonstrated. Four cases showed immunological evidence of simultaneous rubella virus infection but without other clinical evidence of the disease.

The cause of Bell's palsy is unknown and the disease is still labelled "idiopathic".¹ Park and Watkins² analysed 500 cases of Bell's palsy. They suggested that an inflammatory process was the essential part as evidenced by the clinical course, pleocytosis and elevated protein in the CSF in some cases. Other causes such as vasospasm, secondary ischaemia, viral infection or immunological disturbances have been postulated.^{1,3} The present study was designed to detect those patients with serological evidence of simultaneous infection with a number of viruses including rubella, mumps, measles, herpes simplex, varicella-zoster, cytomegalovirus and influenza viruses.

Patients, materials and methods

The material of our study consisted of all patients with Bell's palsy seen in the first 24 hours after onset of symptoms. Patients seen after 24 hours, or those who had an obvious cause for the facial palsy (such as trauma, Guillain-Barré syndrome or middle ear disease), were excluded from the study. An 18-year-old woman with bilateral simultaneous facial palsy was included after exclusion of Guillain-Barré syndrome by repeated CSF examination over 3 weeks, and by electromyography and nerve conduction studies. Of the 28 patients, 14 were male and 14 female. Ages ranged between 11-70 years with a mean age of 39.6 years.

All patients had a full blood count, ESR and blood chemistry (including fasting blood sugar, serum creatinine, blood urea, liver function tests, serum electrolytes, serum cholesterol and serum uric acid). Skull radiology with special views of petrous bone and internal auditory meatus on the affected side, and electromyographic and nerve con-

duction studies also were performed. All patients had their serum estimated for viral antibody titres on presentation and in the third week after presentation. Antibody titres to herpes simplex, mumps, measles, rubella, varicella-zoster, cytomegalovirus and influenza A and B viruses were measured. A four-fold or more rise in the titre was regarded as evidence for recent infection with the virus concerned. Rubella virus isolation was not performed for technical reasons. All patients were monitored daily during the first week for clinical evidence of infection with these viruses. Thereafter, they were followed up at fortnightly intervals for at least 6 months and a few of them for up to one year.

Results

Among the 28 patients, only three had hyperacusis. Taste disturbance in the same side as the lesion occurred in four patients. Both these symptoms disappeared within two weeks from the onset. Three patients had history of Bell's palsy in one parent. None had other cranial nerve abnormality. Six were hypertensive and one had a borderline blood pressure which settled down two weeks later without treatment. Two of these hypertensive patients had high serum cholesterol. Two patients were known diabetics. Another two were discovered to be so for the first time. All were of late onset type. Three of the diabetic patients had raised serum cholesterol and uric acid. Two other patients had high serum cholesterol as their only abnormality.

There was no significant rise in the titres of antibodies against mumps, measles, herpes simplex, varicella-zoster, cytomegalovirus and influenza viruses. Four patients showed a more than four-fold rise in anti-rubella antibody titres, measured by a haemagglutination inhibition method, between samples taken on presentation and on the 15th day in two and the 16th and 17th day after presentation

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Received 11 February 1982 and in final revised form 19 February 1983.
Accepted 12 March 1983

in the other two. The positive sera were not tested for rubella-specific IgM antibodies. These results were regarded as good evidence for recent rubella virus infection^{4,5} and the probable association of this infection with facial palsy in these patients. None of the four patients developed any of the classic clinical features of rubella (such as a rash or lymphadenopathy). They had no previous history of this disease or MMR (mumps, measles, rubella) vaccination. None of them had abnormalities of the biochemical tests nor clinical hypertension. One of the four patients had a bilateral simultaneous Bell's palsy and in this patient careful exclusion of Guillain-Barré syndrome was made.

Discussion

The term Bell's palsy is currently restricted to isolated sudden peripheral facial paralysis of unknown aetiology.³ The cause of Bell's palsy remains controversial.³ Several hypotheses have been proposed to explain the aetiology. The most popular include the ischaemic, immunological and viral aetiologies, but none of these can be regarded as proven.¹

It is known that some viruses can cause a facial paralysis.^{1 6-11} Some authorities believe that a number of cases of Bell's palsy are due to herpes zoster without a rash,¹²⁻¹⁵ or to reactivation of herpes simplex virus.^{16 22} Others claim that mumps, herpes simplex, measles, influenza or other viruses are responsible in a few cases.^{6 7 11 12 15 17-23} However these seem to be exceptional. In reviewing the literature, there have been papers showing evidence of simultaneous infection with herpes simplex, varicella zoster, influenza, cytomegalovirus, mumps, coxsackie, Epstein-Barr and adenoviruses.^{6 7 11 13 15 17-27}

Other authors, investigating large numbers of Bell's palsy patients, failed to demonstrate any evidence of viral infection.^{16 28-31} To date there has been no report on unequivocal serological evidence of rubella virus infection coincident with Bell's palsy. This report of four patients is believed to be the first. These patients had a four-fold or more rise in their basic antirubella HAI antibody titres in the third week from the onset of the Bell's palsy. There was no clinical evidence of rubella and no history of previous rubella or antirubella vaccination. Subclinical rubella infection has been reported previously with serological evidence without clinical manifestations.^{5 32} On the basis of our results the role of rubella virus in patients of Bell's palsy warrant further study. Antiviral antibody titres to rubella should be studied in all patients with Bell's palsy.

The authors thank Professor JA Simpson and Dr JP Ballantyne for their assistance.

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