Mumps and Guillain-Barré syndrome

A variety of neurological sequelae to mumps have been reported including meningitis, encephalitis, myelitis and rarely polyneuropathy.1 By 1981 a total of ten cases fulfilling accepted diagnostic criteria for Guillain-Barré syndrome had been reported1 and half of these cases were associated with orchitis. We can find no subsequent reports of this association and would like to report a further case of Guillain-Barré syndrome following mumps parotitis and orchitis.

A 48 year old male was admitted on 9 December 1988 with progressive generalised muscle weakness. Three weeks earlier he had developed bilateral painful parotid swelling and within days this was followed by pain and swelling of the left testis. There was no previous history of mumps infection but in 1963 the patient had suffered a traumatic upper lumbar fracture dislocation at T12/L1 with moderate residual flaccid paraparesis.

On admission the patient gave a five day history of distal paraesthesia and progressive weakness of all limbs and a 24 hour history of increasing bilateral facial weakness. He normally practised intermittent self-catheterisation and had not noticed any change in sphincter function. Examination revealed severe bilateral facial weakness, moderate weakness of all limbs and marked weakness of the trunk. In the limbs there was generalised weakness graded MRC 3 proximally and MRC 2 distally and all deep tendon reflexes were absent. Pinprick sensation was impaired bilaterally to the wrist and there was widespread abnormality of superficial sensation in the legs related to the previous injury.

The clinical diagnosis of Guillain-Barré syndrome was investigated. The cerebrospinal fluid (CSF) was acellular with an elevated protein content of 1.2 G/L and nerve conduction studies revealed normal peripheral conduction and delayed F waves. Other routine investigations were normal, including urinary porphyrin estimation.

Complement fixation tests for mumps antigen were performed on admission and again two weeks later. The S antigen in mumps infection peaks within the first two weeks of infection and declines thereafter. The V antigen appears at the end of the first week after infection and persists, sometimes for years, as a marker of previous infection.1 In the current case the S antigen on admission was 128, declining to 64 two weeks later. The V antigen was 64 on admission, and had not changed at the second examination. These titres are compatible with recent mumps infection. Viral cultures were negative, but mumps virus is only detectable in the CSF for four days after infection,1 and our failure to culture the virus is in keeping with the third week of mumps infection.

In the days following admission the limb weakness progressed and the patient became bed-bound with no useful upper limb function. Respiratory function was carefully monitored throughout admission and assisted ventilation was not required. Plasmapheresis was started on the second day and the patient had five exchanges. An improvement in muscle power was noted on the second day and he subsequently made a rapid recovery. By the time of discharge two weeks after admission there had been a complete functional recovery.

The clinical and investigative features in this patient agree with accepted diagnostic criteria for Guillain-Barré syndrome.1 The temporal relationship to serologically proven mumps infection suggests a causative relationship as in other viral infections including mononucleosis, acute encephalitis and cytomegalovirus. Muscle weakness associated with a lymphocytic pleocytosis, thus mimicking poliomyelitis, may occur in mumps but this usually accompanies the acute illness.1 Polyneuropathy develops one to three weeks following the viral infection and in the majority of cases is preceded by mumps orchitis. This is not likely to reflect the severity of infection and is unexplained.

Nonetheless, it is important to recognise Guillain-Barré syndrome as a potentially serious complication of mumps infection and this could be of particular relevance in view of the recent widespread introduction of mumps vaccination.


The role of combined valve prolapses in the prognosis of cerebro-vascular ischaemic attacks associated with mitral valve prolapse

The role of Mitral Valve Prolapse (MVP) as a cause of ischaemic stroke (IS) remains controversial. MVP has been associated with cerebral ischaemic events, especially in young adults, though the incidence of ischaemic stroke in subjects with MVP is quite rare. This paradox may be explained by the existence of a subgroup of patients with MVP with specific characteristics that may lead them to carry a high risk for suffering IS.

In a recent paper Barletta et al.2 suggest that ischaemic stroke associated with MVP could be related to the presence of myxomatous degeneration of several valves. These authors found that MVP patients were more frequent in patients with IS and MVP than in a control group with MVP and suggested that the group of patients with such characteristics would have a higher risk of cerebral embolism.2

In a prospective study carried out from January 1982 to December 1986 in the Hospital "Valle de Hebron" in Barcelona, 386 patients under 50 years old had presented with a cerebral ischaemic event. The mean (SD) age was 42±7 (6) years old (range: 22-86 years). Baseline studies included ECG, laboratory determinations, chest and skull radiology, continuous wave Doppler ultrasoundography, M-mode and bidimensional echocardiography, and computerised tomography scan. Angiography studies were performed in 166 patients. The criteria for MVP were defined as those proposed by Alpert et al.4 Aortic valve prolapse was identified using the criteria proposed by Morganroth et al.4 and tricuspid valve prolapse was diagnosed using analogous criteria as used for MVP.

Out of all 386 patients studied, 17 had MVP. One had atrial fibrillation and was excluded from the study. The mean (SD) age of the 16 remaining patients was 43±7 (3) years old. Seven were male and nine female. Seven patients presented with TIA and four with RIND. The other five had established stroke. Only one had had previous ischaemic events and none of them was hypertensive or diabetic. ECG was normal in 13 patients, but one had an ischaemic pattern and two showed branch block. The non-invasive study of extracranial arteries was performed in two patients. Two had aortic valve prolapse and another had tricuspid valve prolapse in the echocardiographic study.

During a mean (SD) follow up period of 36 (16) months on treatment with aspirin and platelet drugs, only three patients had new ischaemic events: one had TIA, another RIND, and the third six TIAs. Such patients coincided with those who presented with multiple valvular prolapse.

The evaluation of our series suggest that the incidence of MVP is not so great in the group of young adults with ischaemic stroke as has been stated, especially not in the youngest patients. However, our study reinforces the force of high risk groups in those patients with multiple valvular prolapse.

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