Mumps meningo-encephalitis

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Summary

Between July 1981 and June 1985, 49 cases (36 boys (73%) and 13 girls (27%)) of mumps meningo-encephalitis confirmed by culture of the virus from the cerebrospinal fluid (CSF) were seen. Patients presented particularly in the late spring and early summer. A CSF cell count > 500 x 10^6/l was obtained in 14 cases (28%), a total CSF protein > 0,8 g/l in 6 cases (12%) and a CSF glucose of < 2,2 mmol/l in 2 cases (4%). Two cases are reported to illustrate the diagnostic problems which the infection may cause, particularly when the CSF changes resemble those of tuberculous meningitis. In 1 case neurogenic pulmonary oedema developed after a convulsion; this caused further diagnostic uncertainty.
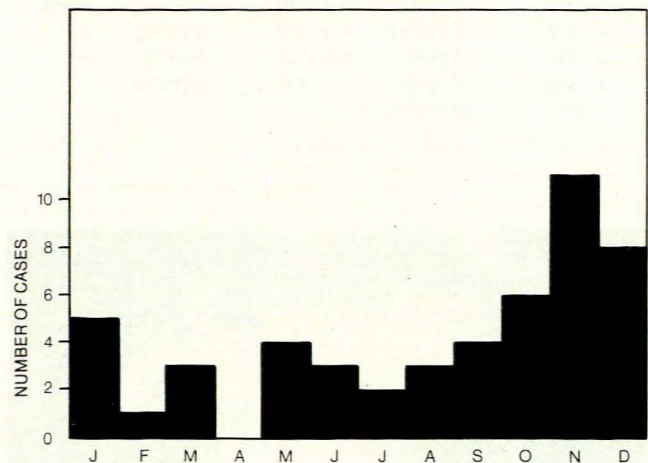
Fig. 1. Monthly incidence of confirmed mumps meningo-encephalitis cases at the Department of Paediatrics, Tygerberg Hospital, July 1981 to June 1985.

Involvement of the central nervous system (CNS) in the form of aseptic meningitis or encephalitis is one of the commonest complications of mumps and some authors have described the mumps virus as neurotropic.[1] While not often fatal, mumps meningo-encephalitis may cause considerable diagnostic difficulties at times, particularly in communities where tuberculosis is prevalent.

Experience in the Department of Paediatrics, Tygerberg Hospital, with mumps virus involvement of the CNS over a 4-year period is briefly described and some of the diagnostic problems which may be encountered are illustrated by the description of 2 cases.

Patients and methods

Since July 1981 a prospective study of the causes of meningitis in children at Tygerberg Hospital has been in progress. All cerebrospinal fluid (CSF) specimens from children with meningitis not clearly identified as being due to bacterial or tuberculous infection were submitted to the Department of Medical Virology for culture. The results of all initial diagnostic CSF investigations were recorded and the findings of the survey up to June 1984 have been published.[2] In this review findings in respect of confirmed mumps meningo-encephalitis cases seen up to June 1985 are described.

Results

During the period July 1981 - June 1985 49 cases of viral meningitis due to mumps were confirmed by culture of the virus from the

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CSF. Fig. 1 shows the month of presentation of these cases, with a prominent late spring and early summer peak. The median age of these children was 64 months and there were 36 boys (73%) and 13 girls (27%).

The CSF findings at initial diagnostic lumbar puncture are summarised in Table I. The total cell count was 500 x 10^6/l in 20% of cases and the results of conventional chemical analysis of the CSF fell within the limits usually accepted for bacterial or tuberculous meningitis in 10 - 20% of cases.

The diagnostic difficulties which may be encountered in mumps meningo-encephalitis are illustrated by the following two case reports.

Case 1

A 38-month-old boy was seen because he had been vomiting for 2 days. He was febrile (38°C), not dehydrated, and was admitted for observation overnight. Shortly afterwards he had a generalised convulsion lasting 1 minute, after which he was unconscious with marked hypertonicity of legs and arms. The fundi were normal with no signs of papilloedema. Lumbar puncture revealed a pressure of 14 cm H_2O and clear CSF containing 206 lymphocytes x 10^6/l and no polymorphs on microscopy. Globulin was absent on Pandy's test, the total serum protein level was 0,32 g/l, the CSF glucose level was 5 mmol/l and the blood glucose level 11,8 mmol/l (ratio 42%). His condition was unchanged 8 hours later and computed tomography of the brain revealed small ventricles with signs of cerebral oedema. An electro-encephalogram (EEG) showed widespread nonspecific disturbances, possibly post-ictal. A chest radiograph also taken approximately 8 hours after the convulsion surprisingly showed widespread opacification in both upper lobes and the right middle lobe area (Fig. 2). In view of the possibility of tuberculous meningitis treatment with antituberculosis drugs was started.

The next day the child was still febrile (38 - 39°C), but his level of consciousness had improved and he now responded to commands. Tonus in the limbs was normal. A follow-up lumbar puncture produced a slightly cloudy CSF with 440 x 10^6/l lymphocytes, and no polymorphs were seen on microscopy. CSF total protein level was 0,71 g/l, no globulin was present on Pandy's test and the CSF glucose level was 3 mmol/l (blood glucose not determined).

TABLE I. CSF FINDINGS ON INITIAL LUMBAR PUNCTURE IN MUMPS MENINGO-ENCEPHALITIS*

Cell count (x 10^6/l)	No.	Total protein (g/l)	No.	Pandy's test	No.	Glucose (mmol/l)	No.	Blood glucose ratio	No.
0 - 100	16 (33%)	<0,4	28 (58%)	Absent	32 (65%)	<2,2	2 (4%)	<0,4	9 (22%)
1 - 200	4 (8%)	0,4 - 0,6	8 (17%)	Trace	12 (25%)	>2,2	47 (96%)	0,4 - 0,6	22 (54%)
2 - 300	12 (25%)	0,6 - 0,8	6 (13%)	+	3 (6%)			>0,6	10 (24%)
3 - 400	3 (6%)	0,8 - 1,0	1 (2%)	++	2 (4%)				
4 - 500	4 (8%)	>1,0	5 (10%)						
> 500	10 (20%)								

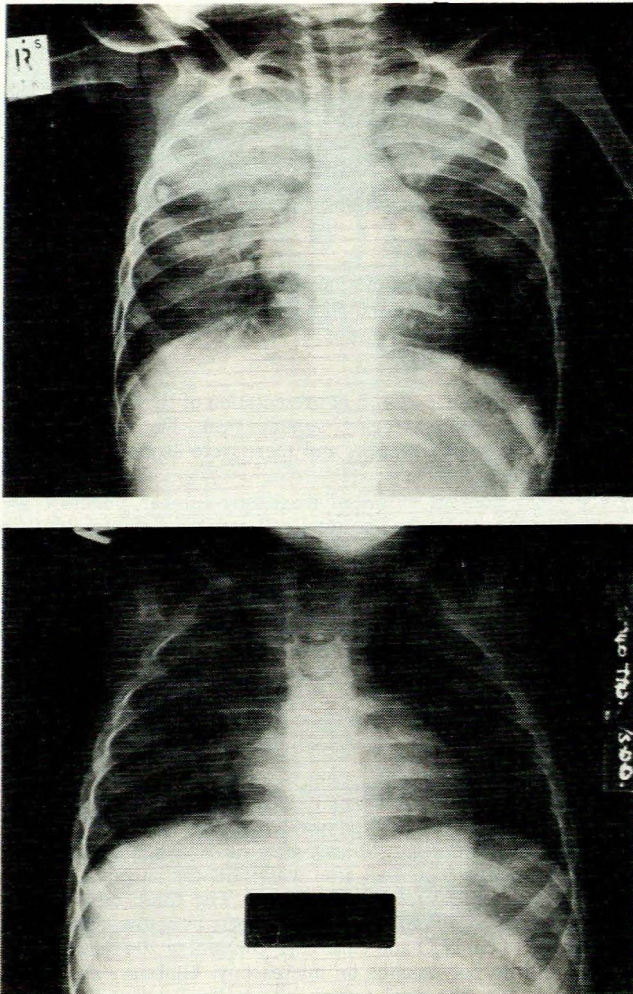*Investigation not carried out on all patients.

Fig. 2. Case 1: chest radiograph (top) on admission showing bilateral pulmonary opacities with air bronchogram visible in the right upper zone; and (bottom) 2 days later showing resolution.

By the 3rd day after admission the child was afebrile and neurologically normal. His serum amylase level determined on admission was reported to be 691 U/l (normal 16 - 180 U/l). A diagnosis of mumps meningo-encephalitis was made and all treatment stopped. Follow-up chest radiograph at this point was normal and the diagnosis of mumps was confirmed later by culture of the virus from the CSF specimen obtained on admission.

Case 2

A 46-month-old boy was seen with a 4-day history of loss of appetite and weakness. On examination he was irritable and had marked neck rigidity. His CSF was slightly cloudy and contained 667 x 10^6/l lymphocytes but no polymorphs on microscopy, there was a trace of globulin on Pandy's test and the CSF glucose level was 1,7 mmol/l (blood glucose and total CSF protein levels not determined). A strong family history of tuberculosis was obtained — the patient was in fact the only person in the home not on antituberculosis therapy — and consequently treatment for a possible tuberculous meningitis was started. Two days later the child was very much better and a follow-up lumbar puncture contained 550 x 10^6/l lymphocytes, no polymorphs, globulin + on Pandy's test (total protein level not determined), a CSF glucose level of 2,1 mmol/l and a blood glucose level of 5,6 mmol/l (ratio 38%). The child's serum amylase level determined on admission was reported as 199 U/l and mumps virus was subsequently grown from the initial CSF specimen enabling the antituberculosis therapy to be stopped.

Discussion

The epidemiological characteristics of mumps meningo-encephalitis seen in this study are in accordance with descriptions in the literature. Thus the male predominance and the occurrence of the disease especially in the late spring is not unexpected.[3]

The CSF findings in the children and in the two case reports illustrate the possible diagnostic problems of mumps meningo-encephalitis.[4] A cloudy CSF with a total CSF cell count > 500 x 10^6/l is not infrequent (20% of our cases), while conventional CSF chemistry gave results falling within a 'bacterial' or 'tuberculous' range in 10 - 20% of cases. Furthermore, some of the investigations used to differentiate viral and tuberculous meningitis, such as the bromide partition test, may also give false-positive results in mumps.[5]

The first child described presented a particularly difficult diagnostic problem. In retrospect, it seems likely that the pulmonary opacities seen on the initial chest radiograph and which cleared within 2 days were due to neurogenic-based pulmonary oedema. Neurogenic pulmonary oedema may occur after cerebral trauma or as a complication of other neurosurgical procedures, but may also develop post-ictally.[6,7] Raised intracranial pressure from an increase in central sympathetic nerve activity may lead to peripheral α- or β-adrenergic discharge and an increase in cardiac pre- and afterload.[8] Our patient's CSF pressure was normal at the time of lumbar puncture but may have been raised immediately after the seizure.

In considering mumps as a possible cause of meningitis it must be remembered that a large proportion of mumps patients may not have clinically detectable parotitis at the time of presentation, while others, as in the case of the 2 patients discussed, may never develop detectable parotitis.[1] In common with other authors, we have found the elevated levels of serum amylase present in many cases of mumps to be of considerable diagnostic help even in the absence of parotitis.[9]

In conclusion, mumps meningo-encephalitis in the Western Cape follows the expected epidemiological pattern of spring exacerbation with a male predominance. The CSF results of 49 cases are summarised and indicate that these may be confusing and at times lead to the consideration of tuberculous meningitis as a possible diagnosis. Two cases of mumps meningo-encephalitis are described which caused diagnostic difficulties, one of which presented with neurogenic pulmonary oedema.

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Pulmonary aspergilloma — indications for surgical intervention

An analysis of 22 cases

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Summary

Surgical resection of aspergillomas has generally been associated with excess mortality and morbidity; 22 patients who had a resection of complicated mycetomas were studied retrospectively. Indications for surgery were serious haemoptysis (14), massive haemoptysis (6), and recurrent infection (2). Extrapleural pneumonectomy was required in 9 patients and extrapleural lobectomy in 12; thoracoplasty alone was done in 1 patient. There was 1 hospital death (4,5%); 4 patients developed postoperative empyemas (18%), 2 with associated bronchopleural fistulas. Two further patients (9%) had stable postresectional spaces. Surgery for complicated aspergilloma was associated with significant postoperative morbidity.

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Aspergillus fungi are found world-wide in decaying vegetation and soil. The spores are thus widespread in rural and urban environments, and cause disease in man when they are inhaled into the nose and lungs. By far the majority of human *Asper-*

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gillus infections affect the lungs, and the major pulmonary disease states so caused are grouped into four syndromes.[1-3] These are: (*i*) aspergillomas (fungal balls or mycetomas) which develop in pre-existing lung cavities; the fungus remains confined to the cavity and induces a strong immune response; (*ii*) allergic bronchopulmonary aspergillosis in patients with chronic respiratory disease, usually asthma or mucoviscidosis, the fungal growth is limited to the airways but the vigorous immune response causes chronic progressive asthma; (*iii*) extrinsic allergic alveolitis due to inhalation of aspergillus spores in patients with no previous lung disease and immunologically normal; a diffuse self-limiting alveolitis is produced by inhalation; and (*iv*) invasive aspergillosis, when the fungus invades the lung and disseminates systemically; this infection is acute, progressive and life-threatening, and occurs in patients whose immune competence, especially T-cell function, is impaired.

While the disease states caused by *Aspergillus* have been presented above as distinct syndromes, degrees of overlap occur.[1-3]

The intracavitary aspergilloma or mycetoma is due to saprophytic colonisation of lung cavitated by previous disease. Most often this is tuberculosis, but the cavities of bronchiectasis, sarcoid, lung abscess, pulmonary infarction, cysts and bullae, even chronic cavitating neoplasm and pre-existent mycosis may harbour mycetomas. Aspergillomas may also occur in lung cavities due to ankylosing spondylitis and rheumatoid disease. Proliferation of the fungus within a cavity in the lung is recognised by the formation of an intracavitary mass, consisting of mycelia and debris which forms the typical fungal ball or mycetoma. The characteristic radiographic appearance is provided by the circular cresent of air visible between the intracavitary mass and the wall of the cavity (Fig. 1). Radiographic views of the lung apices show the