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Acute bilateral total deafness complicating mumps

by
S. BITNUN, M.D., Y. RAKOVER, M.D., and G. ROSEN, M.D., F.A.C.S. (Afula, Israel)

Abstract
While it is well known that mumps is a potential cause of deafness, it is not generally appreciated that it can produce total deafness. The damage is usually unilateral but bilateral disease has been recorded. A case is presented of severe bilateral hearing loss during a mumps epidemic in which, in spite of intensive conventional as well as lesser known therapy, no improvement occurred. In the absence of a successful therapeutic regime it is clearly advisable to intensify immunization in non-developed countries.

Introduction

Mumps is a very common childhood illness, particularly in those parts of the world where specific immunization has not been introduced. Although mild-to-moderate unilateral hearing loss is a well-known complication, it is probably rare. Amongst a group of more than 40,000 students enrolled in special education programmes for the hearing impaired, in only 0.6 per cent of cases was mumps responsible for the hearing loss (Jensema, 1975). Severe bilateral deafness is very rare (Everberg, 1957). We would like to present a case of sudden total bilateral hearing loss occurring during active mumps, and our attempts at conventional and less well-known therapy.

Case report

A boy aged seven years, a non-immunized new immigrant from South America, fell ill with nausea, vomiting, high fever and bilateral tender parotid swellings. His illness occurred during the middle of an epidemic of mumps in a closed rural community. On the third day of the illness, when the vomiting had abated and the fever and parotid swelling were improving, sudden deafness was noted by the parents. At this stage no headache, dizziness, nausea or meningeal signs were present. Complete blood count and urinalysis were unremarkable. Serum and urinary diastase were normal. Complement fixation test for mumps was positive at 1/80.

On audiometry a bilateral sensorineural deafness of 100 decibels was found. Brainstem evoked response of the auditory nerve at 120 sound pressure level was found to be completly absent bilaterally. With the diagnosis of severe bilateral deafness immediate therapy was instituted with intravenous 2 per cent novocaine injected daily, for a week, commencing with 2.5 ml and gradually increasing the dose to 10 ml daily. In addition 250 mgs rhaeomacrodex was given intravenously daily over a 3-hour period, for a week. One hour prior to the novocaine, intramuscular phenobarb 30 mgs was given and oral meticorten 10 mgs every six hours was instituted. Alternate breathing for 15 minutes into a closed circuit was instituted, in order to elevate the blood CO_2 level. After a week on this regime no improvement was noted on audiometry. It was then felt worthwhile to institute hyperbaric oxygen therapy. This was continued for 10 days, during which period he was in the hyperbaric chamber on six occasions. Repeat examination after seven days revealed no change in hearing.

The child returned home and a comprehensive rehabilitation regime instituted.

Discussion

Mumps is a common disease, especially prevalent between the ages of five and 15 years (Philips,

*From the Departments of Family Medicine and Otolaryngology, Central Emek Hospital (Afula, Israel)

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1983). The frequency has been reduced considerably since the institution of mumps vaccination (Gunby, 1983) but is still common, particularly in situations where children are concentrated such as boarding schools, kindergartens or hospitals. It is not a benign illness, the most common complications being meningoencephalitis and orchitis (Bjorvatu and Skoldenberg, 1978).

Deafness following mumps is probably uncommon but there is considerable variance in the literature on the subject. The reported incidence varies from a low of 1/2000 (Goodhill and Gussen, 1979) to a high of four per cent in an epidemic involving Finnish soldiers (Vuori *et al.*, 1962). The average appears to be around 1/30,000 (Everberg, 1957). The hearing loss may occur before (Dawes, 1963), during or after the parotitis (Everberg, 1957; Vuori *et al.*, 1962), but also in subclinical non-diagnosed cases, particularly during epidemics (Dawes, 1963). Typically the deafness is insidious in onset, occurring days or weeks after the parotitis has ceased. Since it affects young children, the diagnosis is often delayed or missed entirely. The hearing loss may only be detected years later at routine audiometry at school. The deafness is usually severe, especially in the high and middle frequencies, and generally permanent (Robin, 1971). The presentation may be in one of three fashions (Jaffe, 1978); the commonest is insidious in onset, unilateral with complete hearing loss. The next common variety is unilateral and partial deafness, and although not often reported may well be frequently undiagnosed, especially in young children (Jaffe, 1978; Rowson *et al.*, 1975). Bilateral complete deafness is very rare, and had in fact been reported in only 22 patients until 1957 (Everberg, 1957). Very few additional cases have since been recorded (Vuori *et al.*, 1962; Lindsay *et al.*, 1960).

The hearing loss is perceptive (sensorineural) (Vuori *et al.*, 1962; Lindsay *et al.*, 1960) and may thus involve the cochlea directly, affecting the organ of Corti and tectorial membrane (Jaffe, 1978; Lindsay *et al.*, 1960; Sawada, 1979) or the myelin sheath of the eighth nerve (Sawada, 1979; Westmore *et al.*, 1979).

Microscopic evidence of the damage has only been recorded in two patients (Lindsay *et al.*, 1960; Westmore *et al.*, 1979). Both (one adult, one child) showed atrophy and degeneration of the stria vascularis, tectorial membrane and organ of Corti and collapse of Reissner's membrane (Westmore *et al.*, 1979).

The most striking direct proof that mumps is the cause of deafness was the successful isolation of the virus, in a single unique instance, from the perilymphatic fluid of the inner ear in a patient during the acute phase of mumps (Westmore *et al.*, 1979). It was postulated that the virus penetrates the perilymphatic fluid in retrograde fashion, *via* the cervical lymph nodes, into the cerebrospinal fluid and hence into the perilymphatic zone (Westmore *et al.*, 1979). It is clear that deafness is generally not associated with mumps meningitis since, amongst more than 50 children suffering from proved mumps meningitis, none were found to have hearing loss (Azimi *et al.*, 1969). The chances of recovery from viral damage to the inner ear, whether uni- or bi-lateral, is generally regarded as remote (Robin, 1971; Jaffe, 1978), although in one epidemic where only unilateral damage occurred, in only eight per cent was the deafness complete (Vuori *et al.*, 1962).

A therapeutic regime has been outlined (Meyerhoff, 1979). 500 ml of 10 per cent low molecular weight dextran should be given intravenously over four hours, every 12 hours, in an attempt to reduce blood viscosity and sludging, and thereby improve vascular perfusion. Steroids, heparin and papaverine have been recommended to improve oxygenation to the injured area. Inhalation of five per cent CO_2 with 95 per cent O_2 has also been employed for the same purpose (Bahgat and Shenoi, 1982) and in more desperate cases stellate blocks and hyperbaric oxygen have been tried (Goto *et al.*, 1979). In our patient we instituted the conventional methods for intensive management of acute deafness to no avail. In addition we attempted therapy in the hyperbaric chamber, in an effort to improve oxygenation to the organ of Corti (Goto *et al.*, 1979). This, too, was ineffective and the child has since been fitted with a hearing aid enabling him at least to perceive very loud noises. An intensive regime of speech and family therapy as recommended by Towne (1983) has also been instituted. The boy, his family, classmates and teachers, as well as the medical and paramedical pesonnel have undergone a very traumatic period of rehabilitation and re-education. This very severe hearing loss in a young child with all the difficulties associated with it emphasizes the need and value of immunization on as large a scale as possible (Koplan and Preblud, 1982).

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Address for Correspondence:
Dr. G. Rosen,
Department of Otolaryngology,
Central Emek Hospital,
Afula 18101,
Israel.

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