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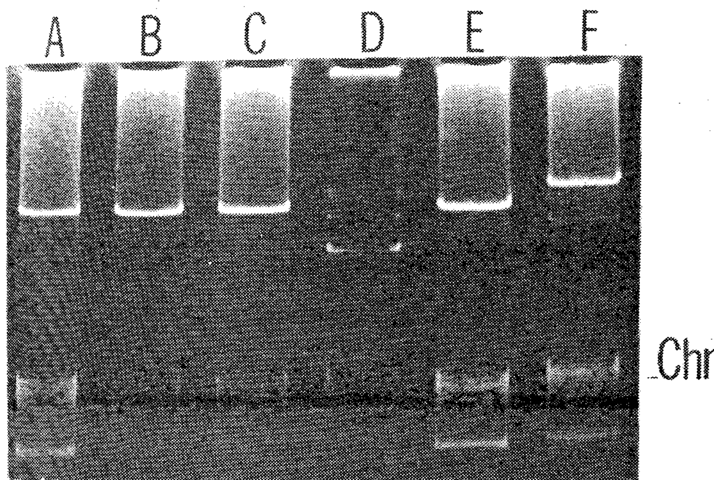

Agarose gel electrophoresis of plasmid DNA extracted from *Pseudomonas aeruginosa* strains from various sources:

A = unopened gallon iodophor container from ward 2;

B and E = peritoneal fluid from two separate patients;

C = iodophor bottle from room 2 of dialysis area;

D = molecular weight control plasmids: 2457 O s(r) (140 and 105 megadalton), R1(62 megadalton), RP4(34 megadalton); and

F = culture from infection at catheter insertion site.

these products, and our finding of two of eight containers of the same lot number positive in widely varying concentrations echoes this problem. We need to determine factors that permit survival of bacteria in iodophors, and whether the containers used for the solution can affect antimicrobial efficacy. We need to determine a measure of iodophor potency that correlates well with bactericidal activity. Most important is the need to reassess the guidelines for use of these agents as disinfectants and antiseptics.[11] Iodophors are widely used and recommended for this purpose by hospitals today, and guidelines for these uses should take into account the possibility of microbial contamination. This may be an especially important issue when these agents are considered as an antiseptic for use before surgery; before they are thus used it may be necessary to define a standard of sterility for the product.

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CHRONIC ENCEPHALOMYELITIS WITH SPECIFIC INCREASE IN INTRATHECAL MUMPS ANTIBODIES

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Summary Symptoms of severe encephalomyelitis developed in a 31-year-old man in 1967. He had a high serum antibody titre to mumps virus associated with a polymorphic cell reaction and an increased protein concentration in cerebrospinal fluid (CSF). He recovered considerably within a year and was able to resume work. In 1975 his condition deteriorated again; it improved during the following few years, but a further deterioration then occurred. In March, 1981, the complement-fixing antibody titre to mumps virus was 1/32 in the serum and 1/4 in the CSF. In November, 1981, the CSF IgG index was increased and the altered serum/CSF antibody ratio persisted. The specificity of the altered antibody ratio was confirmed by the single radial haemolysis test and an immunoassay specific for mumps virus. Antibodies against the mumps virus envelope glycoprotein, M-protein, and nucleoprotein could be demonstrated by immunoprecipitation and the antibody patterns in serum and CSF were similar. Antibodies against other microorganisms were not detected in the patient's CSF, and mumps antibodies were not found in the CSF specimens of 57 control patients. This case may be an example of a new disease—chronic mumps virus infection in the central nervous system.

Introduction

MEASLES and rubella viruses can cause chronic disease in the central nervous system (CNS).[1–5] Intrathecal CNS antibody production has been demonstrated.[5–8] Mumps virus is a major cause of meningoencephalitis,[9] but has not been associated with any form of chronic infection in man. We report a patient with chronic encephalomyelitis with a specific increase in intrathecal antibodies to mumps virus.

Methods

Antibody Determinations

The complement fixation (CF) test was used to assay antibodies to adenovirus, coronavirus, Coxsackie B5, cytomegalovirus, hepatitis B, herpes simplex, influenza A and B, measles, mumps, parainfluenza 1 and 3, polio, respiratory syncytial, rotavirus, and varicella viruses and to *Chlamydia* group antigen, *Mycoplasma pneumoniae*, and *Toxoplasma gondii*. Single radial haemolysis (SRH) tests ('Orivir', Orion Diagnostica, Helsinki, Finland) were used to detect mumps, rubella, and influenza A (Victoria strain) viruses[10–11] and solid-phase enzyme-immunoassays (EIA) for mumps and cytomegalovirus antibodies.[12–14] In addition, patient's CSF (100 μl) was absorbed with 50 μg of mumps virus for 4 h at 4°C and the virus was pelleted (100 000 *g*, 30 min), and mumps antibodies were sought by an SRH test of the supernatant.

Protein Determinations

Serum and CSF concentrations of IgG and albumin were simultaneously measured by automated fluoronephelometry (Technicon 'AutoAnalyzer'). The IgG index (CSF IgG/serum IgG × serum albumin/CSF albumin) was calculated according to Delpech and Lichtblau[15] and de novo IgG synthesis in the CNS according to Tourtellotte et al.[16] Oligoclonal bands were determined as described.[12]

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Determination of Antibody Specificity to Mumps Virus Structural Proteins by Immunoprecipitation

Purified mumps virus (Enders strain; 1 mg) grown in chickens' eggs was radiolabelled with ^{125}I (0·5 mCi) by the chloramine T method of Krohn et al.[18] Radioactive mumps virus (250 000 cpm/sample) was solubilised in 400 μl of 1% 'Triton' X-100 in 0·5 mol/l sodium chloride solution, and 20 μl of serum or 100 μl of CSF was added into the mixture. After 2 h of end-over-end mixing at 37°C, 100 μl of 10% protein A 'Sepharose' (Pharmacia, Upsala, Sweden) in phosphate buffered saline was added for 1 h at 37°C. Sepharose was washed twice with the triton/saline liquid and once with sodium dodecyl sulphate (SDS) depleted electrophoretic buffer, always pelleting with an Eppendorf microcentrifuge between washings. Sepharose-bound proteins were solubilised in 50 μl of electrophoretic buffer in the presence of 10% mercaptoethanol and boiled for 3 min. The supernatant was run in polyacrylamide (10%) gel electrophoresis in the presence of SDS (SDS-PAGE), dried, and the precipitated virus proteins were detected as bands by autoradiography.

Case-report

In February, 1967, a 31-year-old, previously healthy man developed fever, headache, and vomiting followed by paraesthesia, muscular weakness, disturbances of micturition, and diplopia. Three weeks after the onset of symptoms spastic paresis of the legs, distal weakness of the arms, and vague sensory disturbances in the legs were noted. A positive Babinski sign appeared bilaterally, tendon stretch reflexes were brisk, and abdominal reflexes were absent. In addition nystagmus, intention tremor and dysarthria were present and the patient seemed euphoric. In April his legs were paralysed. An electroencephalogram (EEG), first obtained in April, 1967, was normal, brain scan was also normal. An electroneuromyogram (ENMG) revealed marked denervation in the legs suggesting an anterior-horn lesion similar to that in poliomyelitis. The patient was treated in hospital until October, 1967, and diagnosed as having acute encephalomyelitis.

He was rehabilitated, was able to walk in a year, received professional training as a technician, and worked until 1975. His condition deteriorated in 1975 and he had difficulty in walking and some spasticity. After a period of improvement lasting some years his symptoms increased again early in 1980, with positive Babinski sign, ankle clonus, and neuropsychological deficiences. His condition has remained poor and he is unable to work. There has been a further deterioration since 1980. Both EEG and computed tomography were normal in 1981. An ENMG showed a chronic neurogenic lesion without active denervation.

Results

Laboratory Findings

Hospital records showed that in 1967 serum mumps CF titre was high (1:128) without evidence of clinical parotitis or previous mumps vaccination. At that time the CSF was not tested for antibody and no virus was detected. CSF contained 321 leucocytes per μl, total protein was raised (1500 mg/l [normal range 150–450 mg/l), and glucose concentration was normal. Serum mumps CF titre remained high and titres of 1:128, 1:64, and 1:64 were later recorded in 1967.

In March, 1981, mumps CF titre was 1:32 in serum and 1:4 in CSF and no other antibodies were detected in CSF. Thus the serum/CSF antibody ratio was 8, while the serum/CSF ratio for total IgG was normal at 252 (normal range 200–300). At this time total protein concentration in CSF was normal (353 mg/l) and there were no cells. This was also the situation in November, 1981. However, CSF IgG was slightly raised as was the IgG/albumin ratio (table I). The IgG index was raised (0·80 [normal range 0·34–0·58]), indicating intrathecal IgG production. De novo IgG synthesis in the CNS was 53·4 mg/day (normal range <3·3 mg/day).

An altered serum/CSF ratio for mumps antibodies was also detected by both EIA for IgG and SRH (table I). No mumps-specific IgM or IgA antibodies were found by EIA. The patient also had detectable amounts of CF antibodies to rubella, influenza A, cytomegalovirus, adenovirus, and Coxsackie B viruses in his serum but we found no antibodies to these viruses or to 12 other microbes tested in the CSF (table I). After absorption of CSF with mumps virus, the SRH value for mumps fell from 5 to <3.

TABLE I—DETERMINATION OF PROTEINS AND ANTIBODIES IN SERUM AND CSF SAMPLES

—	Serum	CSF	Serum/CSF ratio
*Proteins:**			
IgG (g/l) (serum 8·0–19·9; CSF 0·014–0·38)	10·6	0·042	252
Albumin (g/l) (serum 35–55; CSF 0·082–0·290)	38·0	0·187	203
IgG/albumin ratio (serum 0·15–0·57; CSF 0·08–0·20)	0·28	0·22	1·27
Mumps antibodies:			
CF (titre)	1:32	1:4	8
SRH (mm)	11	5	10†
IgG EIA 1:50 (A_{405})	1·082	0·372	..
IgG EIA 1:500 (A_{405})	0·555	0·178	11‡
IgG EIA 1:5000 (A_{405})	0·201	0·025	..
IgM EIA 1:50 (A_{405})	0·127	0·016	..
IgA EIA 1:50 (A_{405})	0·014	0·002	..
Other antibodies:			
Rubella SRH (mm)	8	<3	>20†
Influenza A CF (titre)	1:16	1:<2	>8
Influenza SRH (mm)	8	<3	>20†
Cytomegalo CF (titre)	1:16	1:<2	>8
Cytomegalo EIA 1:5 (A_{405})	ND	0·026	..
Cytomegalo 1:50 (A_{405})	0·540	0·011	>50
Cytomegalo 1:500 (A_{405})	0·117	0·005	..
Adenovirus CF (titre)	1:8	1:<2	>4
Coxsackie B CF (titre)	1:8	1:<2	>4
Other CF§ (titre)	1:<8	1:<2	..

*Reference ranges are given in parentheses.

†Ratio is calculated from standard curve with serial dilutions of positive sera diluted with a negative (SRH<3 mm) serum.

‡Ratio is calculated from interpolated dilutions corresponding to $A_{405}=0·200$.

§Herpes simplex, varicella zoster, influenza B, parainfluenza 1 and 3, respiratory syncytial, measles, rota and hepatitis A viruses, *Chlamydia* group antigen, *Mycoplasma pneumoniae*, and *Toxoplasma gondii*.

Symbols: CF = complement fixing; EIA = enzyme immunoassay; A_{405} = absorbance values in EIA; SRH = single radial haemolysis; ND = not done. In EIA absorbance values of $A_{405}<0·200$ are regarded as not significant.

The specificity of the CSF mumps antibody finding was further assessed by determination of mumps antibodies from 22 control patients with serum mumps antibodies and 35 seronegative control patients with various neurological symptoms. None of the 57 controls had mumps antibodies in the CSF specimen tested at a 1:50 dilution (table II).

Since oligoclonal CSF IgG bands have been described in

TABLE II—MUMPS ANTIBODIES IN SERUM AND CSF SAMPLES OF CONTROL PATIENTS WITH POSITIVE AND NEGATIVE SERUM COMPLEMENT-FIXING ANTIBODIES

—	CF titre		EIA 1:50 (A_{405})		SRH (mm)	
	Serum	CSF	Serum	CSF	Serum	CSF
Present patient	1:32	1:4	1·082	0·372	11	5
Mumps-positive controls (n = 22)	1:16–1:256	1:<2	0·208–0·741	0·003–0·068	3–17	<3
Mumps-negative controls (n = 35)	1:<16	1:<2	ND	0·000–0·039	<3	<3

CF = complement fixing; EIA = enzyme immunoassay; SRH = single radial haemolysis; ND = not done.

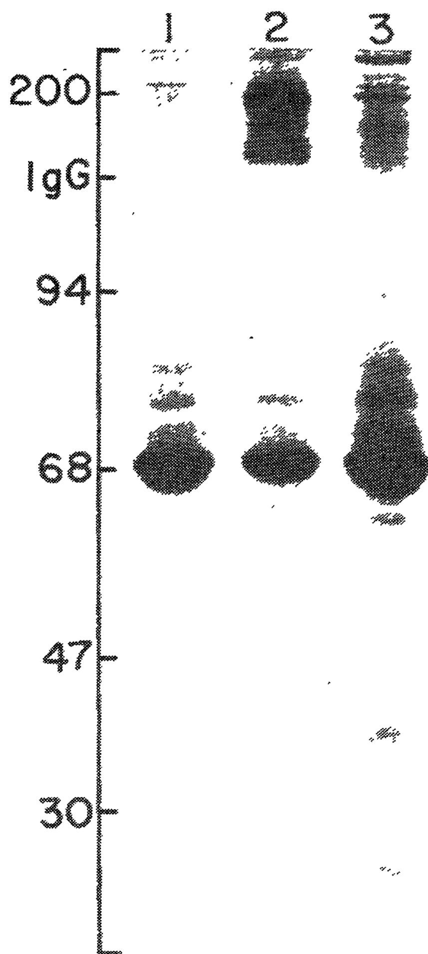

Fig. 1—Analysis of oligoclonal IgG bands by polyacrylamide gel electrophoresis in CSF specimens.

Lane 1 shows analysis of a specimen from a myasthenia gravis patient (negative control), lane 2 of a multiple sclerosis patient (positive control), and lane 3 of the present patient in November, 1981. Apparent molecular weights in kilodaltons and positions of IgG bands are indicated on left.

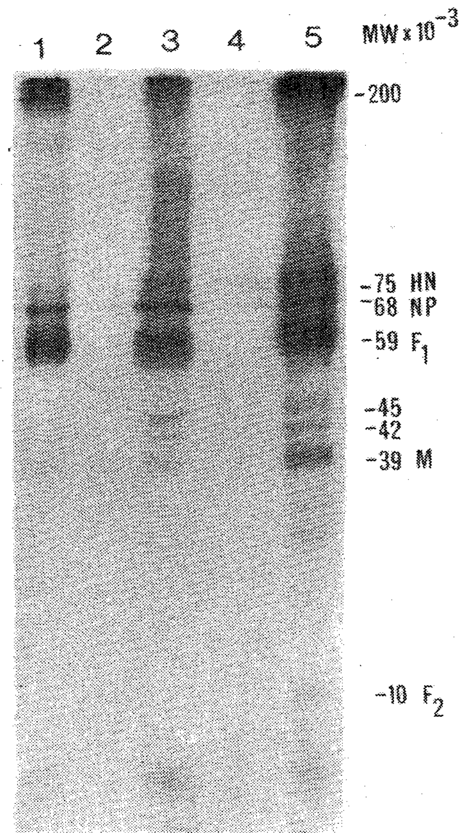

Fig. 2—SDS-PAGE and autoradiography of immunoprecipitated mumps virus polypeptides.

Patient's CSF (lane 1) and sera (3), control patient's CSF (2) and serum (4). Lane 5 represents radiolabelled whole-virus preparation. HN, haemagglutinin-neuraminidase; NP, nucleoprotein: F_1 and F_2, fusion protein; M, membrane protein. Precipitated proteins correspond to the antibody response.

acute as well as in chronic infections of the central nervous system,[7,16,19] the patient's CSF specimen was further analysed by the SDS-PAGE procedure.[17] We found a moderate increase in the IgG region (fig. 1).

Specificity of Antibody Response to Mumps Virus Structural Proteins

The patient's antibody response to mumps virion proteins was investigated by immunoprecipitation. The precipitated protein bands indicate the presence of antibodies. We found antibodies to envelope glycoproteins haemagglutinin-neuraminidase (HN, 75K), fusion protein (F_1, 58K), viral membrane protein (M, 39K), and various nucleocapsid proteins (200K, 68K, 45K and 42K) (fig. 2).[20] Our lysis buffer leaves the nucleocapsid intact and possibly different nucleocapsid proteins are precipitated together. The antibody patterns in serum and CSF were similar and resemble the antibody response in serum of patients with acute mumps infection (Julkunen I, unpublished observations). Control sera and CSF with no mumps antibodies did not precipitate any virion proteins (fig. 2).

Discussion

This patient had chronic encephalomyelitis with a slowly progressive course and a specific increase in mumps antibodies in the CSF and intrathecal IgG production with an oligoclonal pattern. Intrathecal antibody production has been reported in chronic CNS infections caused by measles[8] and rubella,[5] and probably herpes viruses.[21] In acute mumps virus meningitis CNS antibody production has been detected.[13] Vandvik et al.[22] reported prolonged pleocytosis in CSF as late as a year after mumps meningitis, suggesting persistence of virus infection in the CNS. An earlier paper[23] reported a case of aqueductal stenosis two years after mumps encephalitis but with no serological follow-up in the CSF or other evidence of ongoing mumps infection. We know of no other reports of prolonged disease or antibody production caused by mumps virus. Mumps virus can cause spinal-cord as well as pontine and cerebellar lesions.[24,25] Our patient displayed these features but a chronic progressive course also developed. In addition, some cerebral signs such as neuropsychological deficiences were observed.

Increased titres to measles virus and to several other viruses including mumps virus have been found in multiple sclerosis without evidence of viral antigen in CNS.[19] It has been suggested that the broad-spectrum viral antibody response in the CNS in this disorder results from a polyclonal B cell activation. In the present case only antibodies to mumps virus were increased in the CSF, and we were able to absorb the antibodies with purified mumps virus.

By immunoprecipitation we demonstrated that the antibody response to mumps virus was similar in the patient's serum and CSF and was directed against nearly all structural proteins including M-protein. In chronic measles-virus encephalitis a lack of antibodies against M-protein has been

detected and the possibility of defective viral protein synthesis has been discussed.[26,27]

The present case could represent a chronic form of mumps virus infection in the CNS. This can only be confirmed by virus isolation or demonstration of antigen within brain tissue. Mumps virus has a remarkable tendency to produce chronic infections in cell cultures,[28] but has not previously been associated with chronic human infection.

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DOES PSEUDOMONAS CROSS-INFECTION OCCUR BETWEEN CYSTIC FIBROSIS PATIENTS?

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Summary Over a 12-month period respiratory *Pseudomonas aeruginosa* isolated from CF patients were typed by serology and pyocin production to determine whether cross-infection was occurring. Results of typing were interpreted in relation to the degree of contact patients had with each other. One strain appeared in 4 unrelated patients. However, since none of these patients had been in contact with each other the strains were considered to have been acquired from the environment. Each of six pairs of siblings shared the same strain, but the pairs of strains were distinct from each other. These results suggest that the environment is the most important source of *Pseudomonas* strains for CF patients and that for cross-infection to occur prolonged intimate contact is required.

Introduction

Pseudomonas aeruginosa is a common respiratory pathogen in cystic fibrosis (CF) patients. Where patients acquire their infecting strains from is unclear. Some workers believe that cross-infection contributes to the frequent isolation of *Ps. aeruginosa*.[1,2] If cross-infection did occur, clearly it would be important to attempt to separate CF patients from each other socially and during hospital attendances. We tried to determine the extent of pseudomonas cross-infection by investigating the types of respiratory isolates and relating them to the degree of contact that patients had with each other. The study was based on the premise that the greater the degree of contact that patients had with each other, the more likely it was for cross-infection to occur. It did not dismiss the possibility that infection could be acquired from the environement.

Patients and Methods

Patients

Patients were grouped according to the degree of contact they were likely to have had with each other.

Group A (low-contact) consisted of 26 unrelated adolescent CF patients (mean age 17·15 years) who attended a general respiratory outpatient clinic in St Vincent's Hospital, Elm Park, Dublin. Here, generally only 2–3 CF patients attend the same outpatient session, and only rarely are there more than 2 CF inpatients at any one time.

Group B (medium contact) consisted of 26 unrelated paediatric CF patients (mean age 6·5 years) who attended a special CF outpatient clinic at Our Lady's Hospital for Sick Children, Crumlin, Dublin. Here there are generally 12–18 CF patients at each outpatient session, and there are likely to be more than one CF inpatient at any one time.

Group C (close contact) consisted of 14 pairs of CF siblings.

Bacteriology

Patients usually attended as outpatients every 3–4 months, and sputum samples, or occasionally throat swabs, were taken at each outpatient visit over the course of a year. The samples were plated on blood agar, cystine lactose electrolyte deficient agar (CLED), and cetrimide agar ('Pseudosel' agar, BBL).