A New Look at Infectious Diseases

Mumps

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The exact incidence of mumps in Great Britain is unknown, as the disease is not notifiable. It recurs in outbreaks every seven or eight years, with sporadic infections in between. During an epidemic familiarity with the common modes of presentation makes diagnosis easy. At other times mumps is less readily recognized, particularly if it presents with involvement of the central nervous system, gonads, or pancreas rather than with classical parotitis. Patients with suspected mumps need be admitted to an infectious diseases unit only if there is doubt about the diagnosis, if complications have developed, or if isolation is advised to prevent the disease spreading in a residential establishment or the ward of another hospital. With these exceptions, patients with mumps are rightly looked after at home. As the general practitioner diagnoses and manages more patients with mumps than does any other doctor, it is with him in mind that this article is written.

Laboratory Diagnosis

The diagnosis of mumps in general practice is usually a clinical one and it is rarely necessary to call in laboratory help. When difficulties do arise, the virus laboratory can provide the answer to a diagnostic problem surprisingly quickly. A telephone call to the virologist will determine what specimens to send, in what containers, and when. Although mumps virus can be isolated from blood and urine, the most convenient specimens to send from a patient with acute parotitis are saliva or throat washings. If the patient has had a lumbar puncture in hospital, virus can usually be grown from cerebrospinal fluid—in which there is a raised cell count. If suitable specimens are sent early in the illness, the clinical diagnosis can be confirmed, sometimes within a week, by finding cytopathic changes typical of mumps in one of a variety of tissue cultures.

Mumps can also be diagnosed by demonstrating a four-fold rise in antibody titre between acute and convalescent sera sampled a fortnight apart. As the soluble antigen and virus body antigen cause an asynchronous rise and fall in serum antibody levels, it is often possible to deduce from paired sera whether the patient has an acute attack, is already protected from previous infection, or is nonimmune and still susceptible. Since up to 40% of mumps attacks are clinically inapparent,¹ serological tests may be valuable in selected cases.

The Infection

Mumps virus spreads only among human beings, and there is no known animal reservoir. Saliva and droplet spray readily transmit the infection, though fairly close contact is required, and entry is

probably through the upper respiratory passages or conjunctivae. The incubation period is usually between 14 and 21 days but may occasionally be a month or longer. The patient is infectious from one week before the onset of symptoms and up to a fortnight after. For practical purposes, he is regarded as non-infectious when the parotid swelling has subsided.

SUSCEPTIBLE GROUPS AND IMMUNITY

Asymptomatic mumps is very common so that surprisingly few contacts become clinically ill, including those who give no past history. This partly explains the apparently low infectivity of mumps compared with other droplet spread virus diseases. Nevertheless, as there are fewer young adults with significant antibody titres to mumps than there are with antibody to measles and chickenpox, mumps is probably less contagious.

The fetus may be infected transplacentally by mumps virus. Fortunately nothing as severe as the rubella syndrome occurs in mumps, though a possible connexion has been suggested between maternal mumps and primary endocardial fibroelastosis.² Until proved one way or the other, it would seem prudent for nonimmune pregnant women to avoid close contact with mumps. Therapeutic abortion is not normally advised if mumps does develop in pregnancy. Antibodies are transferred across the placenta so that the baby of an immune mother remains passively protected for the first 9 to 12 months of life. Under this age mumps is rare. Children between 5 and 15 years are notoriously prone to mumps; this probably reflects crowding together in schools, buses, and trains, just as much as an increased

susceptibility of this age group. In older people attack rates are low but there is a higher incidence of extraparotid manifestations such as orchitis. Second attacks of mumps have been recorded but a single infection usually confers life-long immunity.

Clinical Features

Typically mumps begins with fever, malaise, and pain over the angle of the jaw. The temperature often reaches 40°C (104°F). Delirium and convulsions may occur in children due to the fever or to the onset of the involvement of the central nervous system, or both. Within 24 hours there is enlargement of one or both parotid glands; usually one side is affected first, with swelling of the opposite gland a few days later, though sometimes the disease remains unilateral. Parotid enlargement may be barely noticeable or else sufficiently severe to distort the facial features and cause much discomfort and earache. Trismus is common because of the close application of the swollen parotid gland and its duct to the masseter and buccinator muscles.

The anatomical relations of the parotid are important in the accurate diagnosis of facial and cervical swellings. The gland nestles between the upper anterior border of the sternomastoid posteriorly and the zygoma anteriorly and above. It lies below the auricle as well as in front of it so that when the gland is very swollen the ear lobe is characteristically displaced upwards and outwards. The inflamed parotid is firm and tender with tense, flushed overlying skin and in severe cases a jelly-like oedema extends down the neck. The parotid duct orifice, which usually lies opposite the crown of the second upper molar tooth, is sometimes pouting and inflamed. Submandibular and sublingual glands may also be involved with or without parotitis. The salivary gland swelling usually reaches its peak in 48 hours and then slowly subsides over the next week together with deferescence of fever.

EXTRAPAROTID MUMPS

As mumps viraemia inevitably leads to invasion of the central nervous system and glands throughout the body, dysfunction of these tissues should be accepted as part of the natural disease. Nevertheless, salivary gland disease alone is so common that when signs and symptoms do develop in other organs clinical usage refers to them as "complications" rather than as the pathologically more acceptable term "extraparotid mumps."

Central Nervous System Involvement

Routine lumbar punctures and electro-encephalograms in patients with clinical parotitis alone show that involvement of the central nervous system is common³⁻⁵ and this suggests that mumps virus is neurotropic. Stiffness or soreness on flexing the head may result from painful parotitis, meningism in a febrile child, or frank mumps meningitis. Kernig's sign may be negative. If there is doubt about the cause of neck stiffness, especially when parotitis is absent or slight, hospital admission for lumbar puncture is advisable.

In mumps meningitis, the cerebrospinal fluid is not usually under pressure, but it is opalescent owing to the presence of several hundred lymphocytes per cubic millimetre. Early in the illness mumps virus can be isolated from the cerebrospinal fluid. The protein and sugar content is usually normal. The patient with mumps meningitis nursed in bed for 10 to 14 days and then slowly mobilized almost always makes an uneventful recovery without sequelae.⁶ Very occasionally deafness results and a simple assessment of hearing is always advisable after mumps.

When signs and symptoms develop in the central nervous system some days after the onset of parotitis the outcome may be quite different. Postinfectious encephalomyelitis with demyelination is likely and the patient must be sent to hospital. The cerebrospinal fluid need not show a pleocytosis, and protein and sugar levels are often normal. Mumps virus cannot usually be isolated. This illness is probably due to an antigen-antibody reaction in the central nervous system rather than to virus invasion as such. The prognosis varies from complete recovery without neurological deficit, through a range of cranial nerve and limb palsies, to death from respiratory paralysis. A guarded but happier prognosis usually accompanies the polyneuritis sometimes found two or three weeks after the onset of mumps. The cerebrospinal fluid is sterile, the cell count normal, but the protein concentration is often raised in this condition. Fortunately both post-mumps meningoencephalitis and polyneuritis are very rare compared with the relatively common but less serious aseptic meningitis.

Orchitis

About one-fifth of men with mumps develop testicular pain, swelling, and tenderness—which often comes on when the parotid swelling is waning. From 16 to 30% of them have both testes affected.¹ The fever, headache, and systemic upset accompanying orchitis is often sufficiently severe to warrant hospital admission. Though testicular atrophy occurs in about one-third of these men, this must not be equated with sterility even when the disease is bilateral: probably only about one patient in ten with orchitis becomes sub-fertile or infertile.¹ Fortunately orchitis is rare before puberty in the age groups most likely to catch mumps.

Abdominal Pain

Early on in an attack of mumps abdominal discomfort, nausea, and vomiting may occur. Pelvic or epigastric tenderness should suggest, respectively, oophoritis in women, or pancreatitis in patients of either sex. The clinical diagnosis is often difficult. As the serum amylase level is usually raised in acute mumps, this test contributes little in the differential diagnosis of suspected pancreatitis. Although both oophoritis and pancreatitis occasionally mimic an acute surgical abdomen, most patients recover spontaneously and permanent sequelae are unusual. Mumps oophoritis is not thought to cause subfertility and mumps pancreatitis is an excessively rare cause of diabetes mellitus.

Involvement of Other Organs

Myocarditis, arthritis, mastitis, thyroiditis, nephritis, prostatitis, and labyrinthitis, have all been recorded in mumps but usually no permanent damage results. Myocarditis is commonest but in the absence of pericarditis it is rarely detected clinically without electrocardiographic examination.

Differential Diagnosis

Consideration of the regional anatomy of the salivary glands should distinguish mumps parotitis from cervical lymphadenitis. Examination of the throat is essential. It may show a streptococcal pharyngitis, anginos glandular fever, or even diphtheria. Septic and tuberculous cervical lymphadenitis are less common today. Non-infectious cervical lymphadenopathy includes the whole range of malignant disease from leukaemia and reticulosis to cancer metastasis. In difficult cases a throat

swab, white blood cell count, Paul Bunnell test, or an outpatient chest x-ray examination, may prove invaluable.

If genuine salivary gland swelling is diagnosed, a history of mumps contact should be sought. In the elderly with poor oral hygiene, septic parotitis is likely and pus may be seen exuding from the inflamed salivary gland ducts. Recurrent "parotitis" accompanied by painful salivation suggests a duct calculus, which may be palpated with the finger or shown by x-ray examination. Carcinomas and mixed salivary gland tumours are usually not tender, are of longer standing than mumps, and are unaccompanied by a generalised upset. In Miculicz's syndrome disease of the lachrymal glands usually precedes the painless salivary gland enlargement. Persistent non-tender parotid swelling may suggest sarcoidosis and demands a search for other manifestations of this disease. Salivary gland enlargement is also one of the less well known side effects of phenylbutazone.

In the absence of parotitis, the differential diagnosis of symptoms in the central nervous system includes a wide range of serious conditions such as bacterial meningitis, brain abscess, tumours, and vascular lesions. Urgent investigation in hospital is required.

When testicular enlargement occurs some days after salivary gland swelling mumps orchitis is easily recognized. When orchitis is the only clinical finding the diagnosis may be difficult without laboratory help, for the enteroviruses and mycoplasmas may also cause testicular disease. Acute gonococcal epididymo-orchitis can usually be diagnosed by examining the urethral discharge. All these conditions, including mumps orchitis, are acutely painful. Tuberculous orchitis is less tender and of slower onset. In seminomas and teratomas painless enlargement of the organ is often the only presenting symptom. Mumps orchitis without parotitis is difficult to distinguish from torsion of a maldescended testicle and expert surgical advice should be obtained at once. The natural history and clinical examination should quickly differentiate mumps orchitis from the common scrotal swellings of hydrocoele, varicocele, and hernia.

Mumps pancreatitis and oophoritis can simulate a surgical abdomen and if doubt exists, observation in hospital is advisable. A white blood cell count may be helpful but the serum amylase level is of limited value as it is often raised in parotid mumps without pancreatitis.

Management

The patient with salivary gland mumps requires no treatment other than bed rest during the acute illness. Pain may be relieved by paracetamol or aspirin. Even though chewing and swallowing are painful, an adequate fluid intake should be encouraged. For the first few days the youthful appetite may be pandered to with sweet drinks, ice cream, and jelly. Secondary

salivary gland sepsis does not occur in mumps so there is no justification whatsoever for giving antibiotics.

Involvement of the central nervous system may cause severe headache and vomiting requiring a combination of analgesics and antiemetics. Dihydrocodeine bitartrate (D.F. 118) 50 mg and cyclizine lactate (Valoid) 50 mg in a single injection are often effective when the patient is vomiting. The management of post-mumps encephalomyelitis in hospital with high doses of corticosteroids remains controversial but these drugs certainly have no place in uncomplicated mumps meningitis.

The traditional treatment of mumps orchitis with a scrotal support so often worsens the discomfort that it should be abandoned. Prednisolone, 15 mg six-hourly for four days, often accelerates pain relief and brings about a quicker defervescence of fever. After this the dose should be dropped by 15 mg daily and then the drug stopped. Corticosteroids do not, however, reduce the risk of testicular atrophy nor do they prevent unilateral orchitis from becoming bilateral. A sympathetic explanation will do much to boost the injured male ego. It should be made absolutely clear that sterility is a very unlikely outcome and that mumps orchitis does not cause impotence.

Abdominal pain and vomiting from suspected mumps pancreatitis or oophoritis may be severe enough to merit hospital admission for closer observation of the patient or for intravenous fluid therapy.

Prevention

Isolation of an index case occurring in a residential school at the beginning of term may curtail the spread of mumps. It should be remembered, however, that the patient is infectious before the onset of symptoms and at the same time there may be other children with clinically inapparent infections. A quarantine period is no longer advised. Prevention of orchitis is possible with convalescent immunoglobulin, when available, but pooled human normal immunoglobulin is ineffective.⁷ Neither is routinely recommended. The desirability of live attenuated mumps vaccine is in doubt.⁸ It has not been widely enough used in Britain to assess its efficacy.

References

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