Mumps

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Mumps is an acute self-limited infection. It is characterized by fever, bilateral or unilateral parotid swelling and tenderness, and the frequent occurrence of meningoencephalitis and orchitis.

Etiology;

Mumps virus is in the family Paramyxoviridae and the genus Rubulavirus.. Mumps virus exists as a single immunotype, and **humans are the only natural host**.

Epidemiology;

In the prevaccine era, mumps occurred primarily in young children between the ages of 5 and 9 and in epidemics about every 4 years. Mumps infection occurred more often in the winter and spring months. The outbreak subsequently spread to all age groups. Mumps is spread from person to person by respiratory droplets. Virus appears in the saliva from up to 7 days before to as long as 7 days after onset of parotid swelling. The period of maximum infectiousness is 1–2 days before to 5 days after parotid swelling.

Pathology and Pathogenesis;

Mumps virus targets the salivary glands, central nervous system (CNS), pancreas, testes, and, to a lesser extent, thyroid, ovaries, heart, kidneys, liver, and joint synovia. Following infection, initial viral replication occurs in the epithelium of the upper respiratory tract. Infection spreads to the adjacent lymph nodes by the lymphatic drainage, and viremia ensues, spreading the virus to targeted tissues. Mumps virus causes necrosis of infected cells and is associated with a lymphocytic inflammatory infiltrate. Salivary gland ducts are lined with necrotic epithelium, and the interstitium is infiltrated with lymphocytes. Swelling of tissue within the testes may result in focal ischemic infarcts. The cerebrospinal fluid (CSF) frequently contains mononuclear pleocytosis, even in individuals without clinical signs of meningitis.

Clinical features;

The incubation period for mumps ranges from 12 to 25 days, but is usually 16 to 18 days. Mumps virus infection may result in clinical presentation ranging from **asymptomatic** or **non specific symptom** to **typical illness** associated with parotitis with or without complications involving several body systems. The typical case presents with a prodrome lasting 1–2 days consisting of fever, headache, vomiting, and achiness. Parotitis then appears and may be unilateral initially but becomes bilateral in about 70% of cases. The parotid gland is tender, and parotitis may be preceded or accompanied by ear pain on the ipsilateral side. Ingestion of sour or acidic foods or liquids may enhance pain in the parotid area. **As swelling progresses, the angle of the jaw is obscured and the ear lobe smay be lifted upward and outward.** The opening of the Stensen duct may be red and edematous. The parotid swelling peaks in approximately 3 days then gradually subsides over 7 days. Fever resolves in 3 to 5 days along with the other systemic symptoms. Submandibular salivary glands may also be involved or may be enlarged without parotid swelling. Edema over the sternum due to lymphatic obstruction may also occur.

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Diagnosis;

When mumps was highly prevalent, the diagnosis could be made based on history of exposure to mumps infection, an appropriate incubation period, and development of typical clinical findings. Confirmation of the presence of parotiditis could be made with demonstration of an elevated amylase level. Leukopenia with a relative lymphocytosis was a common finding. Today, in patients with parotiditis of >2 days of unknown cause, a specific diagnosis of mumps should be confirmed or ruled out by virologic or serologic means. This may be accomplished by isolation of the virus in cell culture, detection of viral antigen by direct immunofluorescence, or identification of nucleic acid by reverse transcriptase polymerase chain reaction. Virus can be isolated from upper respiratory tract secretions, CSF, or urine during the acute illness. Serologic testing is usually a more convenient and available mode of diagnosis.

Differential diagnosis;

Parotid swelling may be caused by many other infections and noninfectious conditions. **Viruses** that have been shown to cause parotitis include parainfluenza 1 and 3, influenza A, cytomegalovirus, Epstein-Barr virus, enteroviruses, lymphocytic choriomeningitis virus, and HIV. **Purulent parotitis**, usually caused by *Staphylococcus aureus*, is unilateral, extremely tender, and associated with an elevated white blood cell count, and may have purulent drainage from the Stensen duct. Submandibular or anterior cervical adenitis due to a variety of pathogens may also be confused with parotitis. Other **noninfectious causes of parotid swelling** include obstruction of the Stensen duct, collagen vascular diseases such as Sjögren syndrome, systemic lupus erythematosis, and tumor.

Complications;

The most common complications of mumps are **meningitis**, with or without **encephalitis**, and gonadal involvement. Uncommon complications include conjunctivitis, optic neuritis, pneumonia, nephritis, pancreatitis, and thrombocytopenia. Maternal infection with mumps during the 1st trimester of pregnancy results in increased fetal wastage. No fetal malformations have been associated with intrauterine mumps infection. However, perinatal mumps disease has been reported in infants born to mothers who acquired mumps late in gestation.

Meningitis and Meningoencephalitis.

Mumps virus is neurotropic and is thought to enter the CNS via the choroid plexus and infect the choroidal epithelium and ependymal cells, both of which can be found in CSF along with mononuclear leukocytes. Symptomatic CNS involvement occurs in 10–30% of infected individuals, but CSF pleocytosis has been found in 40–60% of patients with mumps parotitis. The meningoencephalitis may occur before, along with, or following the parotitis. It most commonly will present 5 days after the parotitis. Clinical findings vary with age. Infants and young children will have fever, malaise, and lethargy, while older children, adolescents, and adults will complain of headache and demonstrate meningeal signs. In typical cases, symptoms resolve in 7–10 days. CSF in mumps meningitis has a white blood cell pleocytosis of 200–600/mm³ with a predominance of lymphocytes. The glucose is normal in most patients, but a moderate hypoglycorrhachia (20–40 mg/dL) may be seen in 10–20% of patients. Protein is normal or mildly elevated.

Less common CNS complications of mumps include transverse myelitis, aqueductal

stenosis, and facial palsy. Sensorineural hearing loss is rare but has been estimated to occur in 0.5-5.0/100,000 cases of mumps. There is some evidence that it is more likely in patients with meningoencephalitis.

Orchitis and Oophoritis.

In adolescent and adult males, epidymo-orchitis is 2nd only to parotitis as a common finding in mumps. Involvement in prepubescent male children is extremely rare, but following puberty it occurs in 30–40% of males. It begins within days following onset of parotitis in the majority of cases and is associated with moderate to high fever, chills, and exquisite pain and swelling of the testes. In $\leq \frac{1}{3}$ of cases the orchitis is bilateral. Atrophy of the testes may occur, but sterility is rare even with bilateral involvement.

Oophoritis is uncommon in postpubertal females but may cause severe pain and when on the right side it may be confused with appendicitis.

Pancreatitis.

Pancreatitis may occur in mumps with or without parotid involvement. Severe disease is rare, but fever, epigastric pain, and vomiting are suggestive. Epidemiologic studies have suggested that mumps may be associated with the subsequent development of diabetes mellitus, but a causal link has not been established.

Cardiac Involvement.

Myocarditis has been reported in mumps, and molecular studies have identified mumps virus in heart tissue taken from patients with endocardial fibroelastosis.

Arthritis.

Arthralgia, monoarthritis, and migratory polyarthritis have been reported in mumps. It is seen with or without parotitis and usually occurs within 3 weeks of onset of parotid swelling. It is generally mild and self-limited.

Thyroiditis.

Thyroiditis is rare following mumps. It has not been reported without parotitis and may occur weeks following the acute infection. Most cases resolve, but some become relapsing and result in hypothyroidism.

Treatment;.

No specific antiviral therapy is available for mumps. Management should be aimed at reducing the pain associated with meningitis or orchitis and maintenance of adequate hydration. Antipyretics may be given for fever.

Prognosis;.

The outcome of mumps is nearly always excellent, even when complicated by encephalitis, although fatal cases due to CNS involvement or myocarditis have been reported.

Prevention:.

Immunization with the live mumps vaccine is the primary mode of prevention used in the United States. It is given as part of the MMR 2 dose vaccine schedule, at 12–15 mo of age for the 1st dose and 4–6 yr of age for the 2nd dose. If not given at 4–6 yr, the 2nd dose should be given before children enter puberty. Antibody develops in 95% of vaccinees after 1 dose. One study showed vaccine effectiveness of 88% for 2 doses of MMR vaccine compared with 64% for a single dose. Immunity appears to be long lasting, with existing serologic and epidemiologic evidence indicating protection for >25 yr.