Mumps Infection in Children and Hearing Loss

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50.1 Introduction

Mumps is an acute, contagious viral illness. Parotid gland swelling is the typical clinical manifestation, but many organ systems may be affected. Hearing loss (HL) is rare but a significant complication of the disease. Since it is vaccine-preventable, childhood mumps has become an uncommon disease in countries where measles-mumps-rubella (MMR) vaccination is implemented widely [1, 2]. However, recent outbreaks have been reported in vaccinated communities [1–3]. Because of populations that remain unvaccinated and breakthrough infections in vaccinated individuals, mumps remains an important epidemic problem worldwide.

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50.2 Etiology and Epidemiology

Mumps virus belongs to the *Rubulavirus* genus of the family Paramyxoviridae. It has a single-stranded, nonsegmented, negative-sense ribonucleic acid (RNA) genome with a length of 15,384 nucleotides. The viral genome encodes two nonstructural proteins and seven structural proteins: nucleocapsid-associated protein (NP), phosphoprotein (P), membrane or matrix protein (M), fusion protein (F), hydrophobic membrane-associated protein (SH), hemagglutinin-neuraminidase (HN), and polymerase protein (L). Mumps RNA is surrounded by a helical capsid in a high-lipid structured envelope [4]. The envelope contains the M protein and two surface glycoproteins, which have hemagglutinin and neuraminidase (HN protein) and cell fusion (F protein) activities [2, 4]. These two glycoproteins and the nucleocapsid protein, also referred to as S antigen, constitute the significant antigenic components of the virus. Host antibodies protective against mumps are specific to surface HN and F glycoproteins [2]. The hydrophobic protein (SH) gene is the most variable part of the mumps genome; 12 genotypes of mumps strains were described based on the nucleotide sequence of this gene [5]. Geographic distribution and outbreak relationships vary among mumps strains [5–7]. Besides, in vitro and in vivo findings suggest that cross-neutralization between strains from different genotypes might be reduced compared with strains from the same genotype. Although there is one serotype of the virus, the significance of these genomic types on outbreaks and the effect of vaccination is still a matter of investigation [8, 9].

Various human and monkey tissues and embryonated eggs are used to isolate or propagate the mumps virus. Cytopathic effects caused by the mumps virus in cell cultures include rounding and fusion of cells, causing multinucleated syncytia and intracytoplasmic inclusion formation [1, 2]. The addition of erythrocytes onto cell culture indirectly identifies viral growth through hemadsorption. Viral particles can cause hemagglutination, and partial hemolysis may occur when the virus is attached to cell surface receptors [2]. The mumps virus can be isolated from samples kept at 4 °C for a few days. Keeping viruses viable at −70 °C for an unlimited time is possible when placed in a buffered salt solution with 1–2% inactivated fetal calf serum [2, 9]. Heat (20 min at 56 °C) can destroy the infectivity of mumps. In addition, mumps infectivity is reduced by formalin, ether, Tween 80, and ultraviolet light [2].

Mumps is seen worldwide, and humans are the only hosts. Mumps' incidence is highest in the winter and spring months, but infections by this virus are present throughout the year in warm climates [8]. In the prevaccine era, mumps was a disease affecting predominantly children younger than 10 years old and causing yearly epidemics with high incidence rates [2]. In the postvaccine era, reported cases occur mainly in persons older than 10 years. After implementing routine mumps vaccination in the United States of America, a 99% decline in mumps cases has been observed [10]. However, recent outbreaks have occurred, even in vaccinated populations worldwide [11–13]. Hence, early detection and timely reporting of cases are of great importance. The possible causes of these outbreaks, such as waning immunity or strain change, are under investigation [14, 15].

The mumps virus is moderate to highly contagious. Person-to-person transmission occurs via respiratory droplets through direct contact with saliva or respiratory secretions, although transmission through fomites can also occur [8, 9]. The virus can be isolated from patients' saliva as early as 7 days before and up to 9 days after parotitis onset [9]. Besides, it has been isolated from urine and seminal fluids up to 14 days after the onset of parotitis [16]. Transmission is highest 2 days before and 5 days after the onset of symptoms, and the risk of acquiring the infection is highest among persons in close contact with infected patients, such as those living in the same household or dormitory [1, 8]. People who have an asymptomatic infection can also transmit the virus [1]. The average incubation period for mumps is 16–18 days, but it can range from 12 to 25 days [2].

50.3 Pathogenesis and Immune Response

After contact with infected secretions, the mumps virus replicates in the upper respiratory mucosa of the host and spreads to regional lymph nodes. Subsequently, primary viremia occurs, and the infection spreads to multiple organs [4]. The most prominent infection site is the salivary glands (mainly the parotids); however, the nervous system (meninges and brain), inner ear (cochlea), gonads, pancreas, heart, joints, thyroid, liver, and kidneys may also become secondary infection sites [2].

The virus replicates in the ductal epithelium in the salivary glands, leading to a local inflammation with lymphocytes and macrophages and periductal interstitial edema [4, 9]. Subsequently, tissue damage with necrosis occurs due to the accumulation of lymphocytes and debris in the ductal epithelium [2]. Orchitis can be a direct or indirect consequence of mumps virus propagation [4, 9]. The mumps virus was isolated from semen in a patient with orchitis [17]. While the mumps virus is known to have tropism for testicular tissue, the specific receptors responsible for viral affinity for the testis have not been clearly defined [18]. The infection results in interstitial edema and lymphocytic infiltration. Due to these pathological changes, necrosis and hyalinization of the seminiferous tubules can occur, with subsequent fibrosis and atrophy within the testes [1, 2, 9].

Infected mononuclear cells provide entry of the mumps virus into the central nervous system through the choroid plexus [4]. As the virus propagates in the choroidal epithelium and ependymal cells lining the ventricles, infected cells' desquamation and accumulation into the cerebrospinal fluid (CSF) lead to meningitis and hydrocephalus [2, 4]. Encephalitis may occur with perivascular infiltration by mononuclear cells, scattered foci of neuronophagia, and microglial rod-cell proliferation. Demyelination of the periventricular area also can be observed [2].

In response to mumps infection, specific immunoglobulin M (IgM), immunoglobulin A (IgA), and immunoglobulin G (IgG) antibodies are produced [1]. IgM may appear on the second day, peak within the first week, and become undetectable after 3 months; however, in some instances, it may persist for 5–6 months. Mumps-specific IgG becomes detectable at the end of the first week, peaks after 3 weeks, and lasts throughout life. Salivary IgA appears simultaneously with the cessation of

viral shedding in saliva [2]. A specific cell-mediated immune response to the mumps virus also develops [19]. In addition, increased levels of interleukin (IL)–6, IL-8, IL-10, IL-12, IL-13, and interferon-gamma (IFN-γ) were found in the sera of the patients with mumps meningitis and encephalitis [20, 21]. In general, lifelong immunity against mumps persists after the infection's resolution; however, studies show possible reinfection [22].

50.4 Clinical Manifestations

Mumps causes subclinical or mild respiratory tract illness in one third of patients. In symptomatic patients, the initial clinical manifestations include fever, headache, anorexia, vomiting, and generalized aches and pains, constituting the prodromal phase of the illness, which lasts 1 or 2 days [8, 9]. After the prodromal period, painful parotid swelling occurs; this is usually unilateral in the beginning and later becomes bilateral in 70% of cases. Parotitis is the most common manifestation, with an incidence rate of 95% in symptomatic patients [9]. It may be accompanied by earache and discomfort while eating and drinking sour liquids. Pain and difficulty while opening the jaw may be seen [2]. Parotid swelling does not have discrete borders; it shifts the earlobe out and upward and obscures the angle of the mandible [9]. Other salivary glands may be affected separately or in combination with parotids in up to 10% of cases [9]. Salivary production and flow can change during the illness, leading to dry mouth or extreme salivation [2]. Redness of the orifices of the Stensen or Wharton ducts can be seen at the onset of the illness and may be a helpful finding to guide the diagnosis [2, 9]. Presternal and supraglottic edema have been described in a few cases due to probable obstruction of lymphatic drainage by bilateral glandular swelling [9, 23]. Although systemic symptoms resolve in 3–5 days, parotitis may last up to 10 days. Adults and adolescents are more prone to severe disease and complications [1]. Laboratory tests usually show low white blood cell (WBC) count, slight relative lymphocytosis, and elevated serum amylase in uncomplicated mumps [2].

50.5 Complications

50.5.1 Central Nervous System Infection

Central nervous system infection is the most frequent complication of mumps in children [2, 9]. Meningeal inflammation accompanies parotitis in about 50% of patients (evidenced by CSF pleocytosis), but less than 10% show symptoms of meningitis [1, 9]. Mumps meningitis is usually a benign entity, and severe cases or deaths are unusual. Headache, fever, vomiting, neck stiffness, and lethargy are typical symptoms that resolve within 7–10 days [9]. Seizures can be seen in 20% of hospitalized patients [1]. Meningeal signs are more prominent in older children and adolescents [2]. Physicochemical analysis of the CSF shows normal or slightly

decreased glucose and normal or elevated protein concentration; the WBC count is usually lower than 1000 cells/mm^3, with lymphocytes predominating over polymorphonuclear leukocytes [1]. Mumps encephalitis is rare (0.1%) and may manifest with changes in the level of consciousness, seizures, focal neurological signs, ataxia, behavioral changes, and abnormal electroencephalography [9]. Mumps encephalitis's outcome is generally good; however, complications, long-term morbidities, and deaths may rarely occur (1.4%) [1, 2]. Ependymitis, acquired aqueductal stenosis, hydrocephalus, cerebellitis, transverse myelitis, progressive encephalitis, paralysis, neuroretinitis, and sensorineural HL (SNHL) due to mumps have been described [1]. Adults are more prone to unfavorable outcomes than children [9].

50.5.2 Epididymo-Orchitis and Oophoritis

Epididymo-orchitis and oophoritis with mumps infection are rarely seen before puberty [9]. However, the orchitis rate is between 14% and 35% in postpubertal males with mumps [24]. The highest risk is observed in males aged 15–29 years [1, 2]. Symptoms usually manifest 4–8 days after parotitis, but may appear up to 6 weeks later [9]. Generally, unilateral testicular involvement occurs, and epididymitis accompanies orchitis [1]. Malaise, fever, lower abdominal pain, testicular pain, and vomiting are the most common symptoms in the clinical onset. Physical examination usually reveals swelling, warmth, tenderness of the affected testicle, and inflammation of the scrotum. Symptoms typically progress for 2–3 days and resolve within 1–2 weeks, but testicular tenderness may persist longer [9]. The C-reactive protein level usually is elevated [1]. Mumps virus can be isolated in seminal fluid, and viral RNA may remain detectable for several weeks [17]. Half of the patients recover completely, but testicular atrophy occurs in the other half at varying degrees [16]. Infertility in patients with bilateral orchitis is rare. However, sperm count and motility decrease can be seen in up to 25% of patients [9]. In addition, there are reports about cancer development in affected testes [2]. Oophoritis occurs in 5–7% of postpubertal women with mumps infection. Symptoms include fever, lower abdominal pain, and vomiting [9]. Pelvic pain and tenderness are noted in physical examination [2]. Rarely, premature menopause and infertility have been reported following oophoritis due to mumps [9].

50.5.3 Other Manifestations

Pancreatitis develops in about 4% of mumps infections. It generally occurs subclinically or follows a mild course with epigastric pain. However, severe hemorrhagic pancreatitis was reported in some patients [1]. Although insulin-dependent diabetes mellitus cases were identified after mumps outbreaks, the relationship between the mumps virus and this disorder remains controversial [1]. Hematuria, proteinuria, and abnormal renal function can frequently occur in children during mumps, but severe glomerulonephritis is rare [9]. Mumps can cause arthralgia and arthritis,

especially in young males. The clinical course mainly includes single-joint arthritis or migratory polyarthritis of large joints [1, 9]. Joint symptoms usually appear 1–3 weeks after the onset of parotitis, and they can last 2 days to 6 months. Full recovery is usually observed without recurrence or joint damage [9]. Electrocardiographic abnormalities indicating myocarditis are observed in up to 15% of patients with mumps infection. Although clinically apparent myocarditis is rare, sequelae and mortality have been reported [1, 9]. Mastitis, thyroiditis, thrombocytopenic purpura, hepatitis, acalculous cholecystitis, hemophagocytic syndrome, and kerato-uveitis are other rare manifestations of mumps disease [1, 9]. Hearing loss is a well-known and important complication of mumps virus infection [9].

50.6 Mumps and Hearing Loss

Permanent HL is an important long-term sequela of mumps. The precise frequency of HL as a result of mumps virus infection has not been established due to the limited number of studies in which this complication has been systematically analyzed. The estimated incidence of HL has been reported to be 0.5–5.0 per 100,000 cases of mumps [2, 9]. However, the incidence of HL may be higher when non-severe cases are included [2]. The patients or their guardians may not recognize unilateral HL when it is mild or even profound [25, 26]. There was transient high-frequency HL in 4.1% of adult males (military) with mumps infection [27]. In a prospective study of children with the clinical diagnosis of mumps, the incidence of SNHL was approximately 1 in 1000 cases [28].

The precise mechanism leading to HL associated with mumps infection has not been defined; alterations in the stria vascularis, organ of Corti, and vestibulocochlear nerve have been considered [29]. Mumps virus may enter the inner ear through viremia or the CSF, reaching the perilymphatic space through the cochlear aqueduct. Mumps virus has been isolated from perilymphatic fluid in a patient with sudden deafness occurring within 2 days of mumps [30]. The vestibular system is also affected due to labyrinthine lesions with possible eighth cranial nerve involvement following mumps infection [31–33].

Hearing loss may be the sole clinical manifestation in patients with mumps virus infection or might develop in patients with parotitis with or without associated meningoencephalitis [34, 35]; however, mumps patients with clinical meningoencephalitis can suffer from HL more frequently than those without central nervous system involvement [35]. Mumps-associated deafness usually is unilateral and often permanent; bilateral, severe HL is rare [9]. Hearing loss may occur in an acute onset or gradually and is frequently accompanied by vertigo [2, 9]. Vestibular symptoms may go unnoticed in pediatric patients, as they can be confused with the malaise associated with the disease, and infants may have difficulty expressing the presence of this symptom [32].

Hearing loss due to mumps is a diagnostic and therapeutic emergency since it may result in permanent HL [36]. Thus, the suspicion threshold for HL should be

kept low in patients with mumps, and audiological evaluation should be performed readily when HL is suspected [25]. Different treatments, including steroids, vasodilators, vitamin B12, and hyperbaric oxygen therapy, are used; however, there is no definite treatment protocol for HL due to mumps infection. Among these options, systemic steroids have become the standard therapy for children and adults [37]. Steroids show therapeutic properties via their anti-inflammatory effects by reducing cytotoxic immune responses, activating ion transport in the stria vascularis and spiral ligament in the cochlear canal, controlling endolymph homeostasis, and increasing blood circulation in the cochlea [37]. However, the effectiveness of steroid therapy in reducing the degree of HL or attaining full recovery remains unclear [38]. While the time of treatment initiation from the onset of HL may affect the prognosis, this might depend on other factors, including the etiology of HL [39].

Profound and bilateral HL due to mumps generally has a poor prognosis [25, 36]. Accompanying vestibular symptoms are also considered poor prognostic factors [38]. In patients with profound HL without improvement after medical therapy, cochlear implant (CI) installation and audiologic rehabilitation should be considered [33, 36]. The optimal time of CI implantation must be determined on an individual basis. Since the need to carry out this intervention is not an emergency, it is reasonable to wait for some time to assess the response to medical therapy and to allow for the family's or patient's acceptance of the permanent HL and the need for CI [33, 37]. An essential factor to take into consideration regarding the optimal time for CI is the patient's age. Young children with deafness can rapidly show a delay in language development and dysarthria [33, 37]. In addition, sudden HL at school age can lead to language and understanding impairments and disruptions in education [33]. In patients with bilateral HL, CI installation showed promising results for speech perception [26, 40]. Early installation of CI may be associated with better outcomes; therefore, it should be considered after 2–3 months of the onset of HL [33, 37]. However, patients may not benefit from a CI when the HL is due to central nervous damage associated with meningitis and encephalitis [40].

50.7 Mumps in Pregnancy

Mumps has a benign course in pregnant women, similar to nonpregnant women. Although case reports suggest the potential effects of mumps infection on the developing infant, there is no evidence of an increased risk of fetal malformations related to gestational mumps disease in prospective and retrospective studies [41]. Several studies reported that spontaneous abortion could occur due to mumps infection in the first trimester, but comparative studies controlled with the normal population are needed to confirm these observations [9, 41]. The relationship between maternal mumps infection and fetal endocardial fibroelastosis was investigated, and the results were inconclusive [41]. Perinatal mumps generally results in a mild course of illness; however, parotid swelling, pneumonia, and pulmonary hypertension have been reported [41, 42].

50.8 Differential Diagnosis

Parotitis or parotid swelling can also be caused by viruses other than mumps virus, such as influenza, echovirus, coxsackievirus A, parainfluenza virus types 1 and 3, cytomegalovirus, Epstein-Barr virus, human herpesvirus-6, lymphocytic choriomeningitis virus, adenovirus, parvovirus B19, and human immunodeficiency virus (HIV) [43, 44]. These viral agents can be distinguished by epidemiological and clinical characteristics, serologic studies, and culture [2]. Purulent parotitis may occur due to bacterial agents; the clinical course is characterized by parotid tenderness, increased WBC count, and pus coming out of the Stensen duct [2, 9]. Drugs (e.g., iodides, phenylbutazone, phenothiazines, and thiouracil), tumors, malnutrition, salivary gland stones, cysts, metabolic disorders, and miscellaneous syndromes (e.g., Parinaud's, Mikulicz's, and Sjogren's syndromes) may be other causes of parotid swelling [9]. Since mumps can occur without parotitis, it should be kept in mind in all children with aseptic meningitis, meningoencephalitis, and encephalitis [2].

50.9 Diagnosis

All children with acute parotitis, orchitis, and oophoritis lasting for 2 or more days should be tested for the mumps virus when no other cause is detected [1, 9, 16]. The main laboratory finding is elevated serum amylase levels, and low or normal WBC count with a relative lymphocytosis can be observed [1, 9]. Isolation of mumps virus in cell culture, detection of the viral genome by reverse transcriptase-polymerase chain reaction (RT-PCR), positive serum mumps-specific IgM antibody test result for a single serum sample, or demonstration of a fourfold or greater rise in mumps-specific IgG antibody titers between acute and convalescent serum samples can confirm mumps infection [8]. Throat swabs, Stensen duct exudates, saliva, CSF, and urine specimens are suitable for mumps virus isolation [1, 8]. Since vaccinated individuals may shed the virus for a shorter time or in a lower concentration, samples should be obtained as soon as possible after the onset of symptoms to increase the likelihood of detecting the virus [1]. In cell culture, the typical cytopathic effect of cellular degeneration and syncytium formation can be seen, and the virus is confirmed by hemagglutination inhibition assays or immunofluorescent antigen detection [1]. Recently, RT-PCR has replaced viral culture as it is more rapid and sensitive [1, 16]. However, a negative RT-PCR test result does not exclude infection with this virus when clinically compatible symptoms are observed [16].

Quantitative and semiquantitative serologic tests can measure mumps-specific IgM and IgG antibodies [1]. Although these serologic assays can help to diagnose mumps, there may be false-positive results due to cross-reaction between mumps and parainfluenza viruses [2]. Usually, enzyme-linked immunoassay (ELISA) is used to determine mumps IgM; positivity shows a current or recent infection [1, 2]. The optimal serum collection time for IgM testing is 7–10 days after the beginning of symptoms [1]. Mumps-specific IgM antibodies can also be determined by ELISA

in oral fluid [45] and detected in CSF samples of patients with mumps meningitis [1]. A positive IgG result indicates recent or past wild-type mumps virus or vaccine exposure. A second serum sample should be obtained 2–3 weeks after symptom onset to assess if there is a significant increase in IgG titers, indicating a recent infection [46].

Serologic confirmation of mumps can be difficult in vaccinated people. In previously vaccinated patients, mumps IgM response is less likely to be positive and may be transient or delayed [1, 16]. Besides, detection of a fourfold increase in mumps IgG titer may not be possible in vaccinated individuals since IgG titers may already have been elevated in the acute phase of the illness [1, 16]. Serologic tests cannot differentiate wild-type mumps virus exposure from vaccine effect, but RT-PCR can detect viral RNA in previously vaccinated children who develop mumps [1, 16]. A buccal or oral swab sample is required for genotyping to distinguish wild-type mumps virus from vaccine virus [16].

50.10 Treatment and Prognosis

There is no specific antiviral drug for the mumps virus. According to the symptoms, treatment is supportive since the clinical course is usually benign and self-resolving [9, 16]. Analgesics may be used for the pain occurring due to parotitis or orchitis. A lumbar puncture may relieve the headache related to meningitis. Adequate hydration and alimentation should be provided [1, 9].

The prognosis of uncomplicated mumps is good. The outcome of mumps meningoencephalitis is primarily good, but neurologic damage and even death may occur. Sterility and HL are rare complications [2].

50.11 Prevention

50.11.1 Vaccination

There are numerous mumps vaccines in use worldwide. All mumps vaccines are live-attenuated virus vaccines [47, 48]. Mumps vaccines may be administered as monovalent or combined vaccines. The most common combination vaccines include measles and rubella in addition to mumps (MMR vaccine); recently, formulations that include varicella in addition to measles, mumps, and rubella are also in use (MMRV vaccine) [9]. More than 80–85% of vaccine recipients develop neutralizing antibodies after a single dose [49]. According to post-licensure data, the protective efficacy of one dose is about 78%, ranging from 49% to 92%, and after two doses, it increases to approximately 88%, ranging from 66% to 95% [50, 51]. It is estimated that a 90–92% rate of population immunity is needed for herd immunity [2]. The mumps vaccine should be applied to all susceptible children, adolescents, and adults [52]. Individuals who document previous mumps diagnoses, two doses of live mumps virus vaccine, or laboratory evidence of immunity should be considered immune [2].

50.11.1.1 Vaccine Recommendation

The mumps vaccine is administered in two doses, separated by at least 1 month by subcutaneous injection of MMR or MMRV vaccine at any age on or after the first birthday. Both vaccines can be administered during the same visit with other vaccines [48, 49]. If the MMR vaccine is received before 12 months of age, the vaccine dose is not taken into account to assess the completeness of the immunization schedule, and two additional doses are recommended beginning at 12–15 months of age, separated for at least 28 days [48, 49].

During an outbreak, people with evidence of immunity to mumps or documentation of two doses of mumps vaccination should receive a third dose if they are in a high-risk group for acquiring mumps infection. No additional mumps vaccination is recommended for individuals previously vaccinated with three doses [16]. Routine administration of the mumps vaccine is not recommended for people born before 1957; this recommendation excludes healthcare personnel with no laboratory evidence of immunity or history of having had the disease [16]. Before mumps vaccination, testing for susceptibility is unnecessary, especially in adolescents and young adults [2]. There is no evidence of a harmful effect of MMR or MMRV on people who already have immunity from previous infection or immunization [48].

50.11.1.2 Adverse Reactions

Adverse reactions associated with the mumps component of the vaccines, including parotitis, fever, febrile seizures, encephalitis, aseptic meningitis, and orchitis, are rare [1, 2, 16]. Allergic reactions such as pruritus, rash, and purpura associated temporally with mumps vaccination are uncommon, usually mild, and of short duration. Severe allergic reactions, including anaphylaxis, are rare [1]. No causality has been identified for temporally related reactions such as pruritus, rash, purpura, SNHL, encephalitis, and aseptic meningitis [2, 16]. Other reactions following MMR or MMRV vaccine administration may be related to other vaccine components [49].

50.11.1.3 Precautions and Contraindications

Fever is not a contraindication of mumps vaccination. Children with minor febrile illnesses should be immunized. However, in patients with severe diseases, with or without fever, vaccination should be deferred until recovery [16, 48].

Hypersensitivity reactions are rare and usually minor, presenting mainly as wheal-and-flare reactions or urticaria at the injection site [16]. Anaphylaxis rarely occurs. Since mumps vaccines produced in chicken embryo cell culture have minimal amounts of ovalbumin cross-reacting proteins, individuals with egg allergy but no anaphylaxis history are at low risk for anaphylactic reactions and can be vaccinated routinely. However, according to published protocols, persons with egg allergy and anaphylactic reactions should only be vaccinated with caution [2]. No proven increased risk exists for allergic reactions to the mumps vaccine in people allergic to chickens or feathers [16]. People with a history of anaphylactic reactions to gelatin or neomycin should be assessed by an allergist or immunologist and receive mumps vaccine in hospital settings where adverse reactions, if present, can

be managed [16]. Contact dermatitis is the most common finding of neomycin allergy and is not a contraindication to the mumps vaccine [2, 16].

Although mumps vaccination during pregnancy has not been shown to be related to congenital malformations, pregnant women should not be vaccinated due to the theoretical risk of fetal damage. Conception should be avoided for 4 weeks after receiving the mumps vaccine. In the case of vaccination during pregnancy, the termination decision should be made individually since MMR vaccination is not a definite indication [49].

Blood products, such as immunoglobulin administration, may interfere with the immune response to MMR or MMRV vaccines; therefore, in individuals who have received such products, vaccination should be deferred for at least 3 months if possible [48]. Mumps vaccine should be administered at least 2 weeks before the planned immunoglobulin administration, or it should be deferred for 3–11 months according to the dose of immunoglobulin received [49]. If immunoglobulin is administered within 14 days of MMR or MMRV administration, these vaccines should be repeated after the appropriate time interval [49]. When immediate protection is required, MMR or MMRV vaccine can be applied to a person who recently received a blood product, but the dose should be repeated after 3 months [48].

50.11.1.4 Altered Immunity

Patients with immunodeficiency diseases and suppressed immune responses due to lymphoma, leukemia, generalized malignancy, or those who have received immunosuppressant treatments such as corticosteroids, antimetabolites, alkylating drugs, or radiation within the previous 4 weeks should not receive live attenuated virus vaccines such as MMR or MMRV [2, 16]. Since vaccinated people do not spread the virus, immunocompromised persons can be protected by vaccinating their close contacts [49, 53].

For HIV-infected children, mumps vaccination recommendations differ according to their immune status. All asymptomatic HIV-infected children and adolescents without severe immunosuppression should be vaccinated with the MMR vaccine [49, 53]. In contrast, severely immunocompromised HIV-infected persons should not receive measles virus-containing vaccines. The quadrivalent MMRV vaccine should not be administered to HIV-infected infants at this time because of this population's lack of safety data [49, 53].

Immunization with the MMR vaccine should be deferred for at least 3 months after finishing immunosuppressive therapy such as chemotherapy for leukemia patients [53]. However, the proper interval for a safe and effective immunization depends on the type and intensity of immunosuppressive or radiation therapies, underlying disease, and other factors, so that a definite recommendation is not possible [16]. According to recommendations of an international consensus conference, allogeneic hematopoietic stem cell transplant recipients may receive two doses of MMR vaccine starting 24 months after transplant if they are no longer on immunosuppressive therapy [54]. Data regarding the safety and efficacy of live virus vaccines in transplant recipients still receiving immunosuppressive therapy were not found to be sufficient in a review [55].

Patients receiving short-term corticosteroid therapy, including intra-articular, bursal, or tendon corticosteroid injections, as well as topical corticosteroid therapy (e.g., skin, nasal), can be vaccinated with the mumps vaccine. However, children receiving immunosuppressive levels of corticosteroids (≥2 mg/kg per day of prednisone or its equivalent, or ≥20 mg/day if they weigh 10 kg or more, for 14 days or more, as well as those with prolonged and extensive topical corticosteroid application) should wait at least 4 weeks after discontinuation of therapy before they receive live-virus vaccines [2, 16].

50.11.2 Control Measures

Mumps is a reportable disease. Recommendations to prevent transmission include standard and droplet precautions for managing patients with mumps and a 5-day isolation period after the onset of parotitis [56]. For postexposure prophylaxis, mumps vaccine and immunoglobulin are ineffective [16, 48]. However, people without evidence of immunity should receive a mumps vaccine since vaccination will provide protection against the disease after future exposures [48]. Immunization during incubation is not related to an increased risk of adverse events [16]. Therefore, vaccination programs, isolation of patients, contact tracing, and exclusion of unimmunized persons can be implemented to control a mumps outbreak [2]. During an epidemic, the mumps vaccination status of all individuals in the community should be updated appropriately according to age. According to public health recommendations, people with an increased risk for mumps and its complications might receive a third dose of the mumps vaccine [16]. In school settings where outbreaks occur, unimmunized students should be excluded from attendance until 26 days have passed since the onset of symptoms of the last person with parotitis. Excluded students can return to school as soon as they receive a dose of the MMR vaccine [16].

50.12 Conclusion

Mumps outbreaks are still occurring throughout the world. Since mumps is a vaccine-preventable disease, all susceptible individuals should be vaccinated unless contraindicated. Hearing loss due to mumps disease is rare but an important complication. It may be overlooked when HL occurs gradually or as a mild affection. No effective treatment exists for HL due to mumps infection. Although rare, bilateral or severe HL may occur, requiring CI installation. Otological evaluation should be performed to assess HL in children in whom mumps is suspected. Also, because mumps virus infection may be asymptomatic, it should be kept in mind in any patient with sudden HL.

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