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have been established in the WHO American, European, Southeast Asian, African, and Western Pacific regions. The Eastern Mediterra nean region has not yet set such goals. Unfortunately, the COVID-19 pandemic, which began in late 2019, led to extensive disruptions of routine vaccination services in many countries. Thus, it is essential that all children are up to date with rubella vaccination, especially those who missed vaccination during the pandemic. To protect against rubella vaccine throughout the life course, additional strategies to immunize adolescents and adults will be needed to ensure adults of childbearing age are protected from the risk of having an infant with CRS.

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.who.int/teams/immunization-vaccines-and-biologicals/strategies/ ia2030. Accessed March 5, 2024.

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Mumps Mumps is an acute, self-limited, systemic viral illness typically charac terized by parotitis or other salivary gland swelling. Although mumps was once considered a universal childhood disease in the United States, routine mumps vaccination—with a one-dose policy implemented in 1977 and a two-dose policy in 1989—led to a >99% reduction in cases by the early 2000s. However, since 2006, there has been an increase in mumps cases in the United States, the majority among fully vaccinated persons. Mumps should be suspected in all patients with parotitis or mumps complications (see “Clinical Manifestations”), regardless of age, vaccination status, or travel history. ■ ■ETIOLOGIC AGENT Mumps is an acute viral illness caused by a paramyxovirus from the Rubulavirus genus in the Paramyxoviridae family. This single-stranded, negative-sense, enveloped RNA virus is ~15.3 kb in size and encodes several minor proteins and seven major proteins. There is only one mumps virus serotype. One of the seven major encoded proteins, the small hydrophobic (SH) protein exhibits hypervariability among strains; thus, the SH gene nucleotide sequence (316 nucleotides) is used to genotype the virus for molecular epidemiologic purposes. Mumps virus is rapidly inactivated by formalin, ether, chloroform, heat, and ultraviolet light. The 12 known genotypes of mumps virus are designated by the let ters A to N (except E and M). In the United States, >98% of mumps virus specimens genotyped from 2015 through 2017 were genotype G. Most mumps vaccines licensed globally are composed of virus strains from genotype A, B, or N. The mumps virus strain (Jeryl Lynn) used in vaccines in the United States is genotype A. ■ ■EPIDEMIOLOGY Mumps occurs worldwide and is endemic in many countries. In the absence of routine vaccination, the annual incidence of mumps is 100–1000 cases per 100,000 population, with epidemic peaks every 2–5 years. From 1999 to 2018, on average, >500,000 mumps cases were reported to the World Health Organization annually, with fewer reported cases from 2019 to 2021 (between 160,000 and 270,000). However, the global mumps incidence is challenging to estimate, as few countries routinely collect the pertinent data. As of 2021, mumps vaccine was introduced in 123 WHO member states. Mumps incidence has been reduced by 97–99% in countries with a routine two-dose mea sles, mumps, and rubella (MMR) vaccination schedule and by 87–88% in those with a one-dose vaccination program. However, since the mid2000s, large mumps outbreaks have been reported among populations with high two-dose MMR coverage in countries with routine mumps immunization programs. Most outbreaks have occurred in settings with intense or frequent close contact, such as universities, close-knit communities, and correctional facilities, and most of these cases have occurred in fully vaccinated persons. Despite these outbreaks, mumps incidence is still much higher in countries that do not have routine mumps vaccination. In the United States, prior to licensure of a vaccine for mumps in 1967, >100,000

mumps cases occurred annually. After the implementa tion of a one-dose mumps vaccination policy in 1977 and a subsequent two-dose policy in 1989, reported mumps cases declined to an annual average of ~300 by the early 2000s. However, since 2006, there has been an increase in mumps cases reported in the United States, with several peak years (Fig. 213-1). During the highest peak in recent cases, from January 2016 through June 2017, 150 mumps outbreaks and 9200 outbreak-associated cases were reported in a range of settings and groups, including schools, universities, athletic teams and facili ties, church groups, workplaces, and large parties and events. While a majority of cases occurred in fully vaccinated young adults in asso ciation with large university outbreaks, about one-third of cases have

Number of Mumps Cases

FIGURE 213-1 Reported mumps cases: United States, 2000–2022. (Source: National Notifiable Diseases Surveillance System (NNDSS), Notifiable Infectious Disease Data Tables. Atlanta, GA, CDC Division of Health Informatics and Surveillance, 2022. Available at https://www.cdc.gov/nndss/data-statistics/infectious-tables/index.html.) affected children or adolescents, most of whom were vaccinated. Since 2020, there has been a large reduction in reported mumps cases likely due to social distancing and other COVID-19 prevention measures, with 150–700 reported annually. As of 2023, mumps is endemic in the United States, and there are no elimination goals for the disease. Multiple factors are likely involved in being at risk for mumps infec tion among vaccinated persons. Following vaccination, these factors include (1) failure to develop an immune response, (2) the develop ment of a low-level immune response that is insufficient for protection, (3) a decrease in immunity over time (waning immunity) after initial development of a vaccine-induced immune response, (4) lower levels of vaccine-induced antibodies to the circulating wild-type virus strains than to the vaccine virus strain, and (5) a lower frequency of subclini cal immunologic boosting due to lack of exposure to wild-type virus during periods of low disease incidence. ■ ■PATHOGENESIS Humans are the only known natural reservoir for mumps virus, which is transmitted through direct contact with respiratory droplets or saliva of an infected person. The average incubation period is 16–18 days, with a range of 12–25 days. A person is most infectious from 2 days before until 5 days after onset of parotitis or other salivary gland swelling. However, mumps virus has been detected in saliva as early as 7 days before onset and as late as 9 days after onset of these manifestations. Mumps virus has been isolated from urine and seminal fluid up to 14 days after onset of parotitis, although no studies have assessed transmissibil ity of the virus through these fluids. Primary mumps virus replication likely occurs in the nasal mucosa or upper respiratory mucosal epithelium. Given the range of symp toms, it is assumed that, after infection of the respiratory mucosa, the virus spreads to regional lymph nodes. Mononuclear cells and cells within regional lymph nodes can become infected; such infection facilitates the development of viremia, which usually lasts 3–5 days. Viremia can result in a range of acute inflammatory reactions, most commonly in the salivary glands (resulting in parotitis) and the testes (resulting in orchitis). Other sites of virus dissemination include the kidneys (reflected in the frequency of viruria), the central nervous sys tem (CNS), the pancreas, the heart, the ovaries, the mammary glands, the perilymphatic fluid within the cochlea, and (during pregnancy) the fetus. Little is known about the pathology of mumps since the disease is rarely fatal. Affected salivary glands contain perivascular and intersti tial mononuclear-cell infiltrates and exhibit hemorrhage with promi nent edema. Serum and urine amylase levels may be elevated as a result

of inflammation and tissue damage in the parotid gland. Necrosis of acinar and epithelial duct cells is evident in the salivary glands and in the germinal epithelium of the seminiferous tubules of the testes. The virus probably enters cerebrospinal fluid (CSF) through the choroid plexus or via transiting mononuclear cells during plasma viremia. Although relevant data are limited, in many cases, mumps encephalitis appears to be a para- or postinfectious process (as suggested by perive nous demyelination and perivascular mononuclear-cell inflammation) rather than the result of a direct cytotoxic effect caused by viral inva sion of the CNS. However, although rare, primary mumps encephalitis does occur, as shown by mumps virus isolation from brain tissue. Infection of the perilymphatic fluid likely develops via retrograde pen etration by the virus from the cervical lymph nodes following viremia, but infection could also occur via the CSF in cases of mumps CNS infection, given that the perilymph communicates with the CSF. Virus in the perilymph can result in infection of the cochlea and damage to the organ of Corti and the tectorial membrane, leading to transient or permanent deafness. Evidence of placental and intrauterine spread has been found in both early and late gestation. Virus frequently dis seminates to the kidneys, but kidney involvement in mumps is almost always benign. CHAPTER 213 Mumps ■ ■CLINICAL MANIFESTATIONS While typically presenting with parotitis or other salivary gland swell ing, mumps infection can be asymptomatic or present as nonspecific respiratory symptoms, though serious complications such as sensori neural hearing loss can occur. Fully vaccinated persons can contract mumps, but vaccinated persons are at a lower risk for mumps and mumps complications. Mumps infection is asymptomatic in ~20% of unvaccinated patients; the proportion asymptomatic among vaccinated persons is unknown. Parotitis can be preceded by several days by a prodrome of low-grade fever, malaise, myalgia, headache, and anorexia. Parotitis typically lasts for 5 days (range, 3–7 days); most cases resolve within 10 days. Parotitis is generally bilateral and may not occur synchronously on both sides; unilateral involvement occurs in about one-third of cases. Swelling of the parotid gland is accompanied by tenderness and obliteration of the space between the earlobe and the angle of the mandible (Figs. 213-2 and 213-3). The patient frequently reports an earache and jaw pain and finds it difficult to eat, swallow, or talk. The orifice of the parotid duct is commonly red and swollen. The submaxillary and sublingual glands are involved less often than the parotid gland and are rarely involved alone. In ~6% of mumps cases, obstruction of lymphatic drainage secondary to bilateral salivary gland swelling may lead to presternal

A B FIGURE 213-2 The same person before mumps acquisition (A) and on day 3 of acute bilateral parotitis (B). (Courtesy of patient C.M. From JD Shanley: The resurgence of mumps in young adults and adolescents. Cleve Clin J Med 74:42, 2007. Reprinted with permission. Copyright © 2007 Cleveland Clinic Foundation. All rights reserved.) pitting edema, associated often with submandibular adenitis and rarely with the more life-threatening supraglottic edema. The most frequent complications of mumps include orchitis, oopho ritis, mastitis, pancreatitis, hearing loss, meningitis, and encephalitis. Complications can occur in the absence of parotitis and are more com mon among adults than among children and among males than among females, likely due to rates of orchitis. Orchitis (testicular inflammation), usually accompanied by fever, is the most common complication, developing in up to 30% of unvac cinated and 6% of vaccinated postpubertal males. This complication is rare in children. Orchitis typically occurs during the first week of parotitis but can develop up to 6 weeks after parotitis. Both testes are involved in ~10–30% of cases. The testis is painful and tender and can be enlarged to several times its normal size. Pain and swelling may last for 1 week, while tenderness may last for several weeks. Testicular atrophy develops in ~30–50% of affected testicles. The development of anti-sperm antibodies,

reduced testosterone production, and impaired sperm mobility through oligospermia, azoospermia, or asthenosper mia may lead to temporary sterility or subfertility. However, no studies have assessed the risk of permanent infertility in men with mumps orchitis. PART 5 Infectious Diseases Approximately 7% of unvaccinated and ≤1% of vaccinated postpu bertal women develop oophoritis, which may be associated with lower abdominal pain and vomiting. The rate of mastitis in mumps has been estimated to be as high as 30% among unvaccinated postpubertal women and as low as ≤1% among vaccinated postpubertal women. Pancreatitis occurs in ~4% of unvaccinated and <1% of vaccinated mumps patients. Mumps pancreatitis, which may present as abdominal Parotid gland (enlarged) Parotid gland Sternocleidomastoid muscle Ear-gland axis Ear-gland axis FIGURE 213-3 Schematic drawings of a normal parotid gland (left) and a parotid gland infected with mumps virus (right). An enlarged cervical lymph node is usually posterior to the imaginary line. (Reproduced with permission from A Gershon et al: Krugman’s Infectious Diseases of Children, 11th ed. Philadelphia, Elsevier, 2004.)

pain, is difficult to diagnose because an elevated serum amylase level can be associated with either parotitis or pancreatitis. However, serum lipase is elevated in pancreatitis and the presence of both elevated serum amylase and lipase can help determine if pancreatitis is present in addition to parotitis. Hearing loss associated with mumps infection can occur in up to 4% of unvaccinated and <1% of vaccinated mumps patients. Mumps-related hearing loss is usually sudden in onset, uni lateral, and transient and may be associated with vestibular symptoms. Bilateral and permanent hearing loss are rare. Mumps virus is highly neurotropic, with subclinical CNS involve ment occurring in up to 55% of patients as manifested by CSF pleo cytosis. However, symptomatic CNS infection is less common. Aseptic meningitis occurs in ≤1% of vaccinated patients and up to 10% of unvaccinated patients and is a self-limited manifestation without sig nificant risk of death or long-term sequelae. Symptoms of aseptic men ingitis, including stiff neck, headache, and drowsiness, typically appear ~5 days after parotitis. Encephalitis develops in ≤1% of patients, who present with high fever, marked changes in the level of consciousness, seizures, and focal neurologic symptoms. Electroencephalographic abnormalities may be seen. Permanent sequelae are sometimes iden tified in survivors, and adult infections more commonly have poor outcomes than pediatric infections. The mortality rate associated with mumps encephalitis is ~1.5%. Other CNS problems occasionally asso ciated with mumps include cerebellar ataxia, facial palsy, transverse myelitis, hydrocephalus, Guillain-Barré syndrome, flaccid paralysis, and behavioral changes. Although rare and self-limited, myocarditis and endocardial fibro elastosis may represent severe complications of mumps infection; however, mumps-associated electrocardiographic abnormalities have been reported in up to 15% of cases. Other unusual complications include thyroiditis, nephritis, arthritis, hepatic disease, keratouveitis, and thrombocytopenic purpura. Abnormal renal function is common, but severe, life-threatening nephritis is rare. Mumps infection in pregnant women is generally benign and is not more severe than in women who are not pregnant. Evidence suggesting an association between maternal mumps infection and an increased rate of spontaneous abortion or intrauterine fetal death is inconclusive. Both mumps reinfection after natural infection and recurrent infection (in which parotid gland swelling resolves and then, weeks to months later, develops on the same or the other side) can occur. In the past, mumps reinfection was thought to be rare, but more recent data have suggested that it may be more common than previously thought. Death due to mumps is exceedingly rare. ■

■DIFFERENTIAL DIAGNOSIS

Mumps is the only cause of epidemic parotitis, although an increase in parotitis cases may also result from increased influenza activity— specifically, infection with

influenza A virus subtype H3N2. Other infectious causes of parotitis include parainfluenza virus types 1–3, Epstein-Barr virus, human herpesviruses 6A and 6B, herpes simplex viruses types 1 and 2, coxsackievirus A, adenovirus, parvovirus B19, echovirus, lymphocytic choriomeningitis virus, and HIV. Laboratory testing for sporadic parotitis cases caused by these infectious pathogens can help rule out mumps. Parotitis can also develop in the setting of sarcoidosis, Sjögren's syndrome, Mikulicz's syndrome, Parinaud's oculoglandular syndrome, uremia, diabetes mellitus, laundry starch ingestion, malnutrition, cir rhosis, and some drug treatments. Unilateral parotitis can be caused by ductal obstruction, cysts, and tumors. In the absence of parotitis or other salivary gland enlargement, symptoms of other visceral-organ and/or CNS involvement may predominate, and a laboratory diagnosis is required. Other entities should be considered when manifestations consistent with mumps appear in organs other than the parotid. For example, testicular torsion may produce a painful scrotal mass resem bling that seen in mumps orchitis. Orchitis can also be caused by bac terial infections in the prostate and urinary tract, sexually transmitted diseases such as chlamydia and gonorrhea, and other viral infections such as those with coxsackievirus, varicella, echovirus, and cytomega lovirus. Oophoritis can also be caused by sexually transmitted diseases

such as chlamydia and gonorrhea. A number of viruses (e.g., enterovi ruses) can cause aseptic meningitis that is clinically indistinguishable from that due to mumps virus. ■ ■LABORATORY DIAGNOSIS If mumps is suspected, infection is confirmed by virologic methods, but serologic testing can aid in diagnosis. Especially in vaccinated patients, a negative virologic or serologic result in a person with clinical signs of mumps does not rule out mumps infection. Virologic methods for confirming mumps include reverse transcrip tion polymerase chain reaction (RT-PCR) and viral culture. RT-PCR is preferred because of its sensitivity, specificity, and timeliness. Mumps virus and viral RNA can be detected in blood, saliva, urine, and CSF. Buccal swabs provide the best specimens for virus detection. The parotid gland should be massaged for 30 s prior to collection of the buccal swab sample. As maximal viral shedding occurs within 5 days after symptom onset, specimens for mumps virologic testing ideally should be collected as close to parotitis onset as possible. The diagnos tic yield of urine specimens increases over time up to 10 days after par otitis onset, but buccal specimens are more likely than urine specimens to result in virus detection at any time point. Serologic methods that can aid in the diagnosis of mumps include detection of mumps-specific IgM antibodies or a fourfold rise between acute- and convalescent-phase IgG antibodies. In unvaccinated per sons, IgM antibody is usually detectable within 5 days after onset, reaches a maximal level a week after onset, and remains elevated for weeks or months. Failure to detect mumps IgM in vaccinated patients is very common, as the IgM response is often undetectable, transient, or delayed in these individuals. Collection of specimens >3 days after onset may improve IgM detection. Additionally, IgM can yield falsepositive results due to serologic cross-reactions. Use of IgG testing is generally not recommended, as IgG titers in vaccinated or previously infected patients may already be elevated at the time of acute-phase specimen collection, such that a fourfold rise is not detected in the convalescent-phase specimen. TREATMENT Mumps Mumps is generally a benign, self-resolving illness. Therapy for parotitis and other clinical manifestations is symptom based and supportive. The administration of analgesics and the application of warm or cold compresses to the parotid area may be helpful. Testicular pain may be minimized by the local application of cold compresses and gentle support for the scrotum. Anesthetic blocks also may be used. Neither the administration of glucocorticoids nor incision of the tunica albuginea is of proven value in severe orchitis. Mumps immune globulin is not recommended for postexposure prophylaxis or treatment. ■ ■PREVENTION Vaccination is the best

prevention measure against mumps. Mumps vaccine is commonly included as part of the combination measles– mumps–rubella (MMR) vaccine or the combination measles–mumps–rubella–varicella (MMRV) vaccine. All mumps vaccines currently on the market are live attenuated virus vaccines. Strains used in mumps vaccines have included Jeryl Lynn, RIT 4385, Urabe Am9, Rubini, Leningrad-3 and Leningrad-Zagreb. Urabe- and Rubini-containing vaccines are no longer available. The Jeryl Lynn and RIT 4385 strains are the only strains used in mumps vaccines in the United States since 1967. In the United States, children are recommended to receive the first MMR dose at 12–15 months of age and the second dose at 4–6 years. MMR vaccine is licensed for use in persons age ≥12 months; MMRV vaccine is licensed for use in persons age 12 months through 12 years. Due to the potential increased risk of febrile seizures, it is recom mended that MMR vaccine and varicella vaccine be administered for the first dose in children 12–15 months of age. Adequate vaccination

against mumps is defined as two doses of MMR for school-aged chil dren (i.e., grades K–12) and for adults at high risk (i.e., health care workers, international travelers, and students at post–high school educational institutions) and one dose for preschool-aged children and adults not at high risk. During an outbreak, a second dose should be considered for children age 1–4 years and adults who have received one dose. In 2017, after an increase in cases among persons with two MMR doses and a study demonstrating added benefit of a third MMR vaccine dose for individual protection, a third dose was recommended for use during outbreaks, specifically for groups whom public health authorities identify as at increased risk of acquiring mumps; public health authorities will inform providers of these groups at increased risk. As the duration of protection provided by a third dose of MMR vaccine is unknown and may be short term (<1 year), there is no cur rent recommendation for a routine third dose.

The effectiveness of Jeryl Lynn containing MMR vaccine in pre venting mumps was 72% after one dose (RR 0.24, 95% CI 0.08 to 0.76; 6 cohort studies; 9915 children; moderate certainty evidence), 86% after two doses (RR 0.12, 95% CI 0.04 to 0.35; 5 cohort studies; 7792 children; moderate certainty evidence). The effectiveness of the mumps component is lower than that of the measles component (twodose effectiveness of 97%) and the rubella component (one-dose effec tiveness of 97%). Incremental vaccine effectiveness of a third MMR dose—compared with two doses—during outbreaks is estimated at 78% (range, 61–88%). In general, most recipients of mumps vaccine will seroconvert after vaccination and will have detectable antibodies to mumps virus; how ever, antibody levels start to decline soon after vaccination. Vaccineinduced neutralizing antibodies to wild-type strains may be lower in titer and may decline more rapidly than antibodies to the vaccine strain (Jeryl Lynn). However, most young adults given two vaccine doses in childhood appear to retain memory B cells. CHAPTER 213 Mumps vaccines are generally very safe. Urabe and LeningradZagreb mumps strain vaccines have been associated with a slightly increased risk of aseptic meningitis, but there is no evidence of this risk for Jeryl Lynn and RIT 4385 mumps strain vaccines. There is a twofold greater risk of febrile seizures among children 12–23 months of age after receipt of the first dose of MMRV vaccine than after the first dose of MMR vaccine, with or without simultaneous varicella vaccination; this risk has not been found among vaccinated children 4–6 years of age. Mumps There is no known immune correlate of protection for mumps; a positive IgG titer indicates only that a person has been exposed to mumps virus through either vaccination or natural infection and does not predict protection against infection. Therefore, all close contacts of a mumps patient should be advised to self-monitor for mumps symp toms for 25 days after their last exposure. Further, IgG titers should not be used to infer immunity in close contacts as it may

indicate acute infection rather than immunity. MMR vaccine has not been shown to prevent illness or alter clinical severity in persons already infected with mumps virus and is not recommended as postexposure prophylaxis for immediate close contacts of mumps patients. Acknowledgment The authors acknowledge and thank Drs. Mariel Marlow and Stephen Rubin, authors of prior editions of this chapter. ■ ■FURTHER READING Di Pietrantonj C et al: Vaccines for measles, mumps, rubella, and varicella in children. Cochrane Database Syst Rev 4:CD004407, 2020. Krow-Lucal E et al: Measles, mumps, rubella vaccine (PRIORIX): Recommendations of the Advisory Committee on Immunization Practices—United States, 2022. MMWR Morb Mortal Wkly Rep 71: 1465, 2022. Marin M et al: Recommendation of the Advisory Committee on Immunization Practices for use of a third dose of mumps virus–con taining vaccine in persons at increased risk for mumps during an outbreak. MMWR Morb Mortal Wkly Rep 67:33, 2018. Masarani M et al: Mumps orchitis. J R Soc Med 99:573, 2006.

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