VIROLOGY CHAPTER FOURTEEN

MEASLES (RUBEOLA) AND MUMPS VIRUSES

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TEACHING OBJECTIVES

Brief review of structure and properties of measles and mumps viruses. Discussion of viral pathogenesis and disease, epidemiology, prevention and treatment.

INTRODUCTION

Infections with measles, mumps and rubella viruses are confined to man and occur worldwide. They are all spread primarily via the aerosol route. Each of these viruses exists as a single serotype. MMR (mumps, measles, rubella) vaccine contains live, attenuated forms of all three of these viruses.

Measles and mumps viruses belong to the Paramyxovirus Family and are enveloped, non-segmented, negative-sense RNA viruses with helical symmetry (figure 1A).

PARAMYXOVIRUS FAMILY

The name, paramyxovirus, comes from beyond (para) and slime (myxo). There are there are two subfamilies of Paromyxoviridae (table 1).

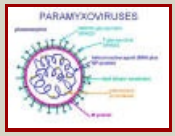


Figure 1A
Paramyxovirus structure

TABLE 1 Members of the Paramyxovirus Family

SUB-FAMILY	GENUS	MEMBERS	GLYCOPROTEINS
Paramyxovirinae	Respirovirus	Human parainfluenza virus1 (HPIV 1) Human parainfluenza virus3 (HPIV 3)	HN, F
	Rubulavirus	Human parainfluenza virus2 (HPIV 2) Human parainfluenza virus4 (HPIV 4) Mumps virus	HN, F
	Morbillivirus	Measles	H, F
	Henipavirus	Hendravirus Nipahvirus	G, F
Pneumovirinae	Pneumovirus	Respiratory syncytial virus	G, F
		Metapneumovirus	

MEASLES (RUBEOLA)

Measles (the name is derived from the German word for blister) is a highly contagious, serious, mainly childhood disease. It was once widespread in developed countries and remains common in less developed countries. CDC estimates that for every 1000 cases of measles, one or two children die and one quarter of measles cases in the United States require hospitalization. The disease is so easily spread by water droplets in the air after a cough or sneeze by an infected person that it is likely that every non-vaccinated person in that room will contract the disease. Indeed, the air-borne virus may remain viable for some time.

Epidemiology - Eradication of measles in the United States by vaccination

Prior of vaccination, almost everyone got measles and developed childhood immunity. In 1941, there were about 900,000 cases of measles in the United States (almost 7000 cases per million population) but as a result of better preventative measures and better hygiene, the number of cases dropped to around 500,000 per year by 1960. The attenuated vaccine was introduced in 1962 and within a few years measles had dropped to around 20,000 cases per year, a 96% reduction (figure 1B, see here for further statistics on vaccine effectiveness). In 1981, there were 13,506 measles cases leading to a recommendation in 1989 for a second booster vaccination. Cases dropped further to below one per million population (figure 1C) and by 2000, the United States declared the elimination of endemic measles because almost all cases were shown to be either imported or associated with imported cases. No endemic measles genotype has been detected in laboratory analyses in the United States since 1994.

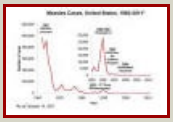


Figure 1B
Reported cases of measles in the United States 1960-2010
CDC

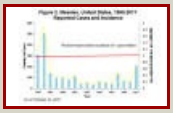


Figure 1C
Reported cases and incidence of measles in the United States 1995-2011
CDC

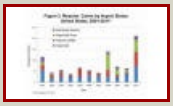


Figure 1D
Measles cases by import status in the United States 2001-2011
CDC

Figure 1E
Measles cases January 1 to 31, 2015
CDC


Figure 1F
Reported measles cases by WHO region, 2005 to 2010.
AFR=Africa
AMR=Americas
EMR=Eastern Mediterranean
EUR=Europe
SEAR=South east Asia
WP=Western Pacific
WHO

Epidemiology - Resurgence

After the introduction of the second booster shot, endemic measles was essentially gone from the United States but there was a small resurgence in 1989-1991. From 2001 to 2011, there were 904 cases of measles in the United States (37 - 212 cases per year; median 60). The highest number of cases were in infants who were not old enough to be vaccinated and about two thirds of the cases were in persons who were known not to be vaccinated. In 2008, there were 140 cases and almost all of these were in people who were either unvaccinated or whose vaccination status was unknown. In 2011, there were 212 cases mostly in people known to be unvaccinated. Almost all of these 2011cases resulted from infection in Western Europe which was experiencing a measles epidemic. Indeed, most cases in the 21st century were import-linked. For example, of the 140 cases in 2008 :

- 25 cases (18%) were directly imported from abroad
- 129 cases (92%) could be shown to be import-associated

For further statistics on import cases, go here

Epidemiology - 2014 - 2015
The Disneyland-associated cases

There was a record number, 644, of measles cases in the United States in 2014 and these occurred across 27 states in 23 outbreaks. Many of these cases were associated with cases imported from the Philippines. In January 2015, there were 102 cases across 14 states (figure 1E). Almost all of these cases consist of a single major outbreak linked to Disneyland in California. Among the cases were five Disneyland employees. Again, the majority of people who contracted measles were unvaccinated.

Epidemiology - Worldwide

Estimated number of measles deaths worldwide (in thousands) 2000 to 2010	
2000	535.3
2001	528.8
2002	373.6
2003	484.3
2004	331.4
2005	384.8
2006	227.7
2007	130.1
2008	137.5

2009	177.9
2010	139.3
2013	145.7

Measles is still one of the leading causes of childhood deaths around the world. Before widespread vaccination outside developed counties, there were 2.6 million deaths per year (1980 figures). By 2000, this had been reduced to about half a million (out of over 30 million measles illnesses). Of these, over 50% were in Arica (figure 1F).

Between 2000 and 2013, global measles cases fell by 75%, according to WHO. This resulted from more widespread vaccination and in 2013, 84% of the children in the world had received the vaccine by their first birthday. WHO estimates that vaccination prevented 15.6 million deaths between 2000 and 2013. Nevertheless, in 2013, there were 145,700 deaths worldwide, mostly in children under 5.

WEB RESOURCES

Mumps, measles and rubella vaccine
CDC

Measles, Mumps, and Rubella -- Vaccine Use and Strategies for Elimination of Measles, Rubella, and Congenital Rubella Syndrome and Control of Mumps: Recommendations of the Advisory Committee on Immunization Practices (ACIP)
CDC

Pathogenesis and disease (figure 2)

Infection is via an aerosol route (coughs, sneezes) and the virus is very contagious. Ninety per cent of unvaccinated people who are exposed will become infected. The virus replicates initially in the upper/lower respiratory tract, followed by replication in lymphoid tissues leading to viremia and growth in a variety of epithelial sites. The disease develops 1 - 2 weeks after infection. Thus, an infected person may not know that he/she is infected.

Uncomplicated disease is characterized by the following:

- Fever of 101 degrees Fahrenheit (38.3 C) or above
- Respiratory tract symptoms: running nose (coryza) and cough
- Conjunctivitis (table 2)
- Koplik's spots on mucosal membranes (table 2) - small (1 - 3mm), irregular, bright red spots, with bluish-white speck at center. The patient may get an enormous number and red areas may become confluent.
- Maculopapular rash which extends from face to the extremities. This seems to be associated with T-cells targeting infected endothelial cells in small blood vessels (table 2).

The infection is prostrating but recovery is usually rapid. The peak of infectiousness is during the prodromal phase, that is before the onset of obvious symptoms (Koplik's spots, rash). Some virus shedding continues to occur during the overt disease phase; thus spread of the virus to other individuals can be somewhat reduced by minimizing contact with others.

The cell-mediated response is important since patients with agamma-globulinemia recover normally. Measles tends to be more severe in adults and the very young (under 5 years of age) and is less severe in older children and teenagers.

Figure 2 The pathogenesis of measles. The virus invades the body via blood vessels and reaches surface epithelium first in the respiratory tract where there are only 1-2 layers of epithelial cells then in mucosae (Koplik's spots) and finally in the skin (rash). Adapted from Mims et al. Medical Microbiology, 1993, Mosby

Complications of measles

About 30% of people who are infected by the measles virus develop some complications such as pneumonia, ear infection or diarrhea. However, a small number develop much more serious complications.

If a patient has an impaired cell-mediated immune response, there is continued growth of the virus in the lungs leading to giant cell pneumonia (such patients may not have a rash). This is rare, but often fatal. The reason for the giant cells is that, since F protein can function at physiological pH, it can facilitate cell-cell (syncytial) fusion.

Since virus grows in epithelia of the nasopharynx, middle ear and lung, all of these sites may then be susceptible to secondary bacterial infection. Otitis media and bacterial pneumonia are quite common.

The outcome of the disease is affected by the nourishment of the patient and access to medical care. Measles is still a major killer in underdeveloped countries and several studies in areas with severe vitamin A deficiency problems have found that vitamin A treatment of children with measles has resulted in reduction in

morbidity and mortality. Pneumonia accounts for 60% of deaths from measles.

One in 1000 patients may get encephalitis a few days after the rash disappears. Most patients (90%) survive encephalitis but there may be complications such as deafness, seizures and mental disorders.

Sub-acute sclerosing pan encephalitis

Very rarely (7 in 1,000,000 cases) the patient may get **s**ubacute **s**clerosing **p**an**e**ncephalitis (SSPE). This develops 1 to 10 years after the initial infection. It is a progressive, usually fatal, disease and those who survive are severely impaired mentally and physically. First signs are behavioral, followed by loss of motor control and coordination. There are jerky movements known as myoclonic seizures. As the disease progresses speech and swallowing are affected and vision may be impaired. The course of the disease may be a few weeks although it may also last for years.

Risk factors include acquiring primary measles at an early age (usually under two years). The incidence of SSPE has decreased since vaccination against measles was initiated. SSPE is associated with defective forms of the virus in the brain and so it is difficult to isolate infectious virus from such patients. Certain viral proteins are often not expressed, the M protein being frequently absent.

TABLE 2

CLINICAL ASPECTS OF MEASLES

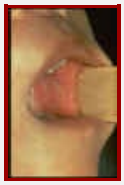
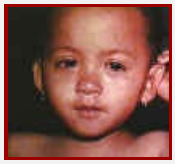
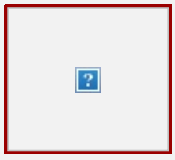
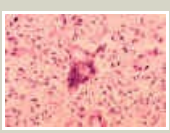
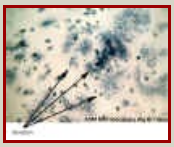
Site of replication of virus	Symptoms in a well nourished child with good medical care	Symptoms in a malnourished child with poor medical care
Lung	Temporary respiratory illness	Pneumonia (life threatening)
Ear	Otitis media is quite common	Otitis media is experienced more often and is more severe
Oral mucosa	 *Koplik's spots* WHO/Immunization Action Coalition.  Patient who presented with Koplik's spots on palate due to pre-eruptive measles on day 3 of the illness CDC/Dr. Heinz F. Eichenwald	Severe ulcerating lesions
Conjunctiva	 Conjunctivitis: Eyes of child with measles. CDC/Barbara Rice ber2@cdc.gov	Severe corneal lesions. There may be secondary bacterial infections of the eyes and blindness may occur
		

Figure 3

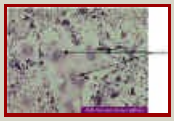
A 

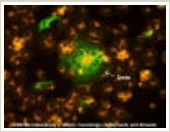
B 
Histopathology of measles pneumonia. Giant cells. CDC/Dr. Edwin P. Ewing, Jr. epe1@cdc.gov

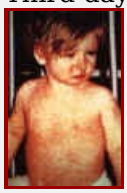
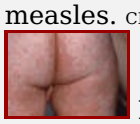
C 

D 
Fusion of Measles Virus Infected Cells. Cell fusion occurring 2 hours (C)) and 5 hours (D) after infection of a human cell line. Cell nuclei become pyknotic, and by 5 hours, small syncytia fuse into giant ones which show beginning evidence of vacuolation. These syncytia will eventually die and detach from the cell monolayer, leaving behind a visible plaque. © Linda E. Fisher
University of Michigan - Dearborn
Dearborn, Michigan and The Microbe Library

E 

F 
CPE typical of measles virus infection of HeLa cells. The large syncytia, or multinucleated giant cells, result from fusion of cell membranes bearing viral glycoproteins. Also visible are inclusion bodies, eosinophilic areas of altered staining in the cytoplasm. Cells stained with hematoxylin, a basic dye, and eosin, an acidic dye. © Maria-Lucia Rácz, Institute of Biomedical Sciences, University of São Paulo, São Paulo, Brazil and The Microbe Library

G 
Indirect fluorescent antibody serological test for measles virus immunoglobulin G (IgG) antibody. All cells are counterstained with Evans blue which fluoresces orange

Skin	Maculopapular rash: Face of boy with measles. Third day of rash. CDC  Maculopapular rash: This child shows a classic day-4 rash with measles. CDC/NIP/Barbara Rice  Maculopapular rash: This child with measles is displaying the characteristic red blotchy pattern on his buttocks during 3rd day of the rash CDC	Possibility of hemorrhagic rashes (black measles)
Intestinal tract	No lesion	Diarrhea which increases malnutrition, halts growth and impairs recovery
Urinary tract	Virus in urine	No further effect
Overall impact	Serious disease in a small proportion of patients	Major cause of infant death (estimates of 1.5 million deaths per year)

Adapted from Mims et al. Medical Microbiology

Other consequences of measles infection

Measles can cause temporary defects in the immune response; for example, tuberculin-positive individuals may temporarily give a negative response. There may be reactivation of herpes or exacerbation of tuberculosis with natural measles, but this does not seem to happen with the vaccine strain.

Measles virus replicates in the cytoplasm, but inclusions containing nucleocapsid protein can also accumulate in the nucleus. It is not known if this has any effect on the host cell, but histologically typically giant cells with cytoplasmic and nuclear inclusion bodies are seen (figure 3). There may also be nucleocapsid protein in the nucleus but the significance of this is unknown.

Diagnosis

The clinical picture is the first part of diagnosis; that is exposure plus upper respiratory tract symptoms, Koplik's spots (table 2) and rash (which is usually quite characteristic for physicians familiar with measles). This diagnosis is confirmed by serodiagnosis, RT-PCR or isolation. Serodiagnosis by IgG levels is simpler than isolation but two samples are needed, one 10 to 21days post rash, and so takes longer. There is now also an IgM test. It is recommended that all suspect cases in the United States be confirmed by laboratory testing

Almost all infected individuals show signs of disease. There is only one serotype of measles and a single natural infection gives life-long protection. The main route of infection is via inhalation. Measles virus is highly contagious and the period of maximum contagiousness is the 2 to 3 day period before onset of the rash.

Prevention

Vaccine

There is an attenuated virus vaccine that is grown in chicken embryo fibroblast culture. Current recommendations are to give a first dose of the vaccine at 12 to 15 months. If given earlier, the recipient does not mount a strong immune response to the vaccine. A second dose is administered at 4 to 6 years of age, before the recipient enters kindergarten or first grade. This reduces the

red. Measles virus induces the fusion of infected cells resulting in a large multinucleated cell (syncytia).
Kristina M. Obom, Patrick J. Cummings, Maria A. DeBernardi, Gary Brooker , Johns Hopkins University and The Microbe Library

proportion of persons who remain susceptible due to primary vaccine failure. Students in post-high school education who do not show evidence of immunity should be given two vaccine doses 28 days apart.

The vaccine gives long term immunity and the vaccine virus does not spread from the vaccinee.

Measles vaccine can cause problems (e.g. fatal giant cell pneumonia) in those with severely compromised cell-mediated immunity. No inactivated vaccine is available, due to past problems in which subsequent infection with naturally acquired measles was sometimes associated with an atypical, severe form of measles.

The attenuated vaccine has few side effects:

- Mild fever (1 in 6 vacinees)
- Mild rash and mild swelling of glands in cheek and neck (1 in 20)
- More severe fever that may cause a seizure (1 in 3000)
- Temporary joint pain and stiffness (1 in 4)
- Temporary low platelet count (1 in 30,000)
- There may be a severe allergic reaction in one in a million vaccinees

Other problems have been reported after administration of the vaccine but they are so rare that it is not known if they were the result of the vaccine or coincidental.

The vaccine is usually given by injection in combination with vaccines for mumps and rubella (MMR vaccine) and sometimes also with a varicella vaccine (MMRV vaccine).

Large studies in the United States and Europe have found no association between the MMR vaccine and autism.

Why do we need to vaccinate when measles has been declared eliminated from the United States?

As noted already, all or almost all cases of measles in the United States are imported. Even in major developed countries in Europe, there are outbreaks of measles from time to time in unvaccinated communities. Thus, if we stopped vaccination, unvaccinated children would still be likely to be susceptible to infection. Until the disease is eliminated worldwide, vaccination must be maintained. By having a large fraction of the population immune to measles infection, we get what is known as "herd immunity" and this protects people in whom the vaccine has not elicited a sufficient level of immunity and people who cannot take the vaccine such as very young children and immunocompromized people.

Herd immunity

If no one were vaccinated (as was the case before 1963), an infected person would infect anyone with whom he came in contact who had not already had measles (infection gives life-long immunity). The newly infected people would infect more and the initial infection in the community would rapidly become an epidemic. A successfully vaccinated person will not be infected and will not spread the virus, thus the disease will spread more slowly. This is the "herd effect". There will simply be fewer people to pass the virus on from the original patient.. If a very large proportion of the population cannot be infected (as a result of having already had the disease or because of vaccination), it is less likely that an infected person will meet a non-immune person to whom to pass the virus. Thus if the infected person runs the course of the disease and becomes non-infectious before he or she can infect another susceptible person, the disease will simply die out. This is "herd immunity".

Immune globulin

Immune serum globulin can be used for at risk patients during an outbreak; that is those less than 1 year old or with impaired cellular immunity.

Treatment

No antiviral therapy available for primary disease. Dehydration should be countered with rehydration solution and the patient needs good nutrition and fluid intake. Complications should be treated appropriately such as antibiotics for pneumonia and eye and ear infections. All children in developing countries who contract measles should be given two doses of vitamin A, 24 hors apart. This restores low vitamin A levels seen in infected children and can prevent eye damage. Vitamin A has also been shown to reduce the number of deaths by 50%.

More on the measles virus receptor

MUMPS

The name comes from the British word "to mump", that is grimace or grin. This results from the appearance of the patient as a result of parotid gland swelling although other agents can also cause parotitis. Clinically, mumps is usually defined as acute unilateral or bilateral parotid gland swelling that lasts for more than two days with no other apparent cause.

Mumps is caused by a paramyxovirus. There is one serotype of the virus and in an affected patient it can be found in most body fluids including cerebro-spinal fluid, saliva, urine and blood. The virus can be grown in cell cultures and in eggs.

WEB RESOURCES

CDC (requires Acrobat)
Association of State and Territorial Directors of Health Promotion and Public Health Education

Pathogenesis and disease

Mumps is very contagious and is probably usually acquired from respiratory secretions and saliva via aerosols or fomites. The virus is secreted in urine and so urine is a possible source of infection. It is found equally in males and females. Before 1967, most mumps patients were under 10 years of age but since the advent of the attenuated vaccine, the remaining cases occur in older people with almost half being 15 years of age or older.

Figure 4 Pathogenesis of mumps
Adapted from Mims et al Medical Microbiology 1993. Mosby, 1993

TABLE 3

CLINICAL ASPECTS OF MUMPS

Site of replication of virus	Symptoms		Notes
Salivary glands	 Inflammation, parotitis, in a child with mumps. CDC/NIP/Barbara Rice Virus is shed in saliva from 3 days before to 6 days after symptoms		Salivary gland symptoms are often absent or may be unilateral
Meninges Brain	Meningitis Encephalitis	Up to 7 days after parotitis	Meningitis is found in about 10% of cases. Encephalitis is less common. Usually there is complete recovery; nerve deafness is a rare complication
Kidney	Virus in urine		No clinical consequences
	epididymo-orchitis; rigid tunica albuginea around		Common in adults (20% in adult males), often

Testis, ovary	testis makes orchitis more painful, more damaging in male	unilateral; not a significant cause of sterility
Pancreas	Pancreatitis	Rare complication (There is a possible role in juvenile diabetes)
Mammary gland	Virus detectable in milk; mastitis in 10% post-pubertal females	
Thyroid	Thyroiditis	Rare
Myocardium	Myocarditis	Rare
Joints	Arthritis	Rare

Adapted from Mims et al. Medical Microbiology

Virus infects upper/lower respiratory tract leading to local replication. The virus spreads to lymphoid tissue which, in turn, leads to viremia. The virus thus spreads to a variety of sites, including salivary, other glands and other body sites (including the meninges).

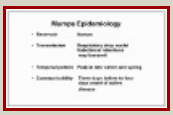
The average time to full manifestation of disease is 2 - 3 weeks but there may be fever, anorexia, malaise, myalgia during prodromal phase. Many mumps infections (up to 20%) result in no symptoms at all and about half of infections result only in the primary respiratory symptoms.

The symptoms of mumps (figure 4 and table 3) include:

- Fever
- Parotitis. Pain from parotitis swelling persists for 7 - 10 days. This is the most common feature of mumps and is seen in about 40% of patients. It may be unilateral or bilateral depending on which salivary glands are infected by the virus.
- Meningitis. Aseptic meningitis is usually mild. About three times as many males than females get this . In about half of patients the meningitis is asymptomatic. In symptomatic meningitis, which occurs in about 15% of patients, there is stiff neck and headache which usually resolves in up to 10 days with no further problems. Mumps-related meningitis is more severe in adults. In very rare cases mumps can result in encephalitis.
- Deafness. Mumps was a leading cause of acquired deafness before the advent of mumps vaccines but nevertheless hearing loss is rare (one in every 20,000 mumps cases). It is usually unilateral. The patient may not, in fact, have overt mumps. Deafness may improve with time but is usually permanent.
- Orchitis (testicular inflammation). This is especially severe in adolescent and adult males and occurs in about 50% of cases. Sometimes, it occurs along with parotitis. The painful swelling diminishes after about seven days but tenderness can last for weeks. In 70% of cases, orchitis is unilateral and results in some degree of testicular atrophy. Damage tends to be patchy and rarely causes infertility.
- Pancreatitis. This is an infrequent side effect of mumps. There is transient hyperglycemia that resolves. However, there is very little evidence from controlled studies that mumps plays any role in diabetes mellitus although outbreaks of diabetes have been reported after mumps outbreaks.
- Myocarditis. Myocarditis is observed from electrocardiograms in a minority of patients but is usually otherwise asymptomatic.
- Rare complications. These include nephritis, arthralgia (joint pain) and arthritis (joint inflammation)

Mumps is more severe in adults and it seems that cell-mediated immunity is important in recovery. On average, one person dies per year in the United States now that most people are vaccinated.

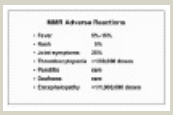
Complications of mumps CDC


Mumps epidemiology CDC


Cases of mumps in the United States 1968-2001 CDC


Cases of mumps in the United States 1980-2001 CDC


Age distribution of mumps cases in the United States 1980-2000 CDC


MMR Vaccine -adverse reactions CDC

WEB RESOURCES

Facts about autism
CDC

Facts about MMR vaccines and autism
CDC

See also:
The MMR-autism scare - our story so far

Diagnosis

Approximately 30% of infections are sub-clinical. Parotitis is suggestive as it occurs in 30 - 40% of infections but there are other causes of parotitis. The disease is confirmed by isolating the virus, RT-PCR or by serology. Hemagglutination inhibition, radial hemolysis and complement fixation assays are rather insensitive. Better is enzyme immunoassay which detects IgM or IgG. The level of IgM rise during the prodromal phase and peak at about seven days. Normally, when testing for IgG a specimen is taken during the acute disease and then during the convalescent phase. The latter should show a higher antibody titer than the former.

Complement fixing antibody to the S (soluble) antigen (nucleocapsid protein) is seen for a few months after infection and is used to diagnose a recent infection. However, one needs to be careful as there is some cross reaction with other human parainfluenza virus nucleocapsid proteins. CF antibody to the viral envelope (V antigen) persists.

Epidemiology

Man is the only known natural host and the disease is found worldwide. There is no 'carrier state'. Since many (about 30%) infections are sub-clinical, spread is usually via these persons. Mumps is contagious from about 7days before the infection becomes clinically apparent and continues until about 9 days afterwards.

Until the development of the highly effective attenuated vaccine, mumps was a very common disease.; for example, there were 212,000 reported case in the United States in 1964. Occurrence dropped to about 3,000 cases by the mid 1980's which is about one case per 100,000 population. In 2001, there were 231 United States cases. In 1986/87, there was a jump in mumps in people in the 10 - 19 years age group (12,848 cases) which was attributable to the fact that these people were born before routine immunization. Vaccine failure may also have contributed.

From time to time, outbreaks of mumps still occur, often in the winter or spring in crowded environments such as schools and colleges. In 2011 to 2013, there were outbreaks on college campuses in the United States in California, Virginia and Maryland but these were limited in extent. In 2009-2010, there were approximately 3,000 cases in a single outbreak among school children in a religious community in New York. The center of the epidemic was one child who had contracted mumps in the United Kingdom during an outbreak there.

Prevention

The attenuated vaccine virus, which is made in chick embryo fibroblasts, does not spread to contacts and gives long-term immunity (greater than 95% efficacy with immunity lasting more than 25 years). It is usually given as MMR vaccine that contains three live, attenuated viruses: **m**umps, **m**easles and **r**ubella. It is also available as a single virus preparation or combined with the rubella vaccine. Normally, two doses separated by four weeks are recommended for children more than one year of age.

Vaccine is contraindicated in immunosuppressed patients and in pregnant women, although there is no evidence that the vaccine can damage the fetus. Also people who have severe allergic reactions after a previous mumps vaccination should not receive the MMR vaccine.

The virus is rapidly inactivated by organic solvents such as chloroform and ether (as would be expected of enveloped viruses) and also by UV light and formaldehyde.

Treatment

There is no specific treatment for mumps.

MMR VACCINE AND AUTISM

There have been reports in the media linking autism to administration of the MMR vaccine. These were the result of a small study (12 children) by Dr Andrew Wakefield and his colleagues in London who suggested that the vaccine caused inflammatory bowel disease. This allowed proteins to enter the circulation that would otherwise not be there and which could then move to the brain and cause neurological damage. Larger studies have failed to establish such a link and the original paper has been retracted.

Among the findings in such studies reported by CDC (link) are:

- There is no indication that measles vaccine contributes to the development of long-term neurological damage, including educational and behavioral deficits

- There is no difference in the prevalence of autism among children born before the introduction of the MMR vaccine in Sweden and those born after the vaccine was introduced
- In a 1999 study of 498 children with autism in the UK, it was found that the age at which they were diagnosed was the same regardless of whether they received the MMR vaccine before or after 18 months of age or whether they were never vaccinated. The first signs or diagnoses of autism were not more likely to occur within time periods following MMR vaccination than during other time periods. Also, there was no sudden increase in cases of autism after the introduction of MMR vaccine in the UK. Such a jump would have been expected if MMR vaccine was causing a substantial increase in autism.

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