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Ear, nose and throat manifestation of viral systemic infections in pediatric patients

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ABSTRACT

Objective/Methods: An exhaustive review of literature was performed to investigate available data and evidences regarding pediatric otolaryngologic manifestations of viral systemic infections.
Results/Conclusions: Modern otolaryngologists should be familiar with viral systemic infections since many have head and neck manifestations. Cooperation between otolaryngologist, paediatrician and virologist can be considered and excellent tool in diagnosis and treatment of these diseases in particular when complications occur.

Contents

1. Introduction

There are multiple systematic viral infections that can manifest themselves in ORL related organs. Their actions can work directly or indirectly causing an alteration in the human immune system and a consequent secondary bacterial invasion. Notable advances in the diagnosis and treatment of viral infections have been mitigated by the appearance of new pathological processes, for example AIDS, which often has its initial manifestations in ORL regions. Table 1 is a list of illnesses affecting different anatomical sites and the viral etiologies that commonly strike each particular location.

Considering the vast nature of the subject, we subdivided our treatment into three parts corresponding to the same groups of interrelated viral illnesses:

- Viruses that can cause deafness.
- Viruses that can cause inflammation in the upper respiratory tract.
- Viruses particularly relevant to ENT (infectious mononucleosis, papillomatosis, herpes infections).

Ascertaining specific viral causes of most infections is neither necessary nor cost-effective, and should be reserved only for specific cases. Clinical and epidemiological acumen remain the basis for a presumptive diagnosis. When a specific diagnosis is necessary, diagnostic procedures based on biochemical and molecular biological processes provide sensitive, specific and rapid results [1].

In most viral infections, immunity to re-infection generally lasts a short period of time due to the host's limited immunological response or, rather for an antigenic change in the virus.

2. Viral illnesses which cause deafness

Viral pathologies that can cause deafness can be congenital, appear in either the pre-natal or postnatal period and can also be acquired upon contact with the pathogen [2–4] (Table 2).

In particular, the hearing damage caused by congenital infections can be part of a severe syndrome (such as "congenital rubella syndrome") but more frequently it is the first and only manifestation of intrauterine infection. Common childhood viral infections, such as measles and mumps are probably an unrecognized cause of acute or progressive damage to hearing [5].

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Table 1
Viruses and syndromes in childhood

Pathology	Virus
RHINITIS	Adenovirus – Coronavirus – Parainfluenza viruses – Rhinovirus
STOMATITIS	EBV – Herpes simplex
PHARYNGITIS	Adenovirus- Coxsackie A – Parainfluenza viruses – Enterovirus-echo – Herpes simplex – EBV –VRS – Influenza viruses – Cytomegalovirus
TONSILLITIS	Adenovirus – EBV – Parainfluenza viruses – Other viruses (42%)
ACUTE OTITIS MEDIA (miringite bullosa)	Parainfluenza and influenza viruses
LARYNGOTRACHEITIS	Parainfluenza virus type 1 and 2 – Influenza viruses
RHINOSINUSITIS (predisposing to bacterial infection)	Parainfluenza and influenza viruses, – Adenovirus – Rhinovirus
LABYRINTH DISEASE	Herpes simplex – Varicella zoster – Rubella virus – Citomegalovirus
DEAFNESS	Parotitis (mumps) virus – Measles virus – VRS – Cytomegalovirus – Rubella virus – Herpes zoster – Parainfluenza and influenza viruses
LARYNGEAL PAPILLOMATOSIS	Papovavirus
FACIAL PARALYSIS	Herpes zoster

Table 2
The viral etiology of childhood deafness

Prenatal or congenital	Post-natal or acquired
Rubella virus	Measles virus
Citomegalovirus	Mumps (parotitis)
Herpes varicella zoster	Varicella zoster virus
HIV	HIV

2.1. Prenatal deafness

In prenatal deafness, a pathogen introduced during pregnancy can provoke an arrest or alteration of the normal development of the ear, even causing lesions on the already-formed hearing mechanism [6]. The most serious lesions manifest themselves in the first three months of pregnancy, especially between the seventh and tenth week, when the cochlea is developing; this would be considered a case of embriopathy. Fetopathy refers instead to lesions that form between the fourth month of pregnancy and birth. Since the hearing organ has already formed in these cases, patients do not generally suffer serious alterations although the inner ear is certainly sensitive. The viruses that most frequently cause prenatal deafness are rubella and citomegalovirus (CMV).

2.1.1. Rubella

Rubella is caused by an RNA virus of the Togaviradae family of the Rubivirus genus. Congenital rubella is typically passed on to the fetus from a primary infection in the mother. The virus invades the upper airways of the mother causing viremia and spreading into different sites including the placenta. It has been hypothesized that in the first gestational phases, the rubella virus provokes a chronic intrauterine infection. Fetal infection in the first trimester, particularly in the first 8–10 weeks, has an extremely high risk of malformations such as hypoacusia, cardiac and ocular defects (Gregg Triad); however, if the rubellum infection is contracted in the second or third trimester, it results in hypoacusia and pigmented retinas. Thus, the more precocious the maternal rubellum, the greater the risk of fetal infections and the more serious the fetal malformations (100% in the first month, 80% in the first trimester, 70% in the second trimester and 30% in the third). This reduction is likely due to either a maturation of the placenta after the first trimester which limits the transfer of the virus, or the greater resistance of the differentiated cells [5]. The deriving hypoacusia is generally sensorineural and bilateral and at birth can already be progressive or it can manifest itself later. The hearing damage seems to be caused by a "teratogenic" effect of interference with the normal development of the organ at the cochlear level [6,7]. Unlike other congenital infections, rubella is easily prevented. Between 12 and 15 months of age, a live-virus rubellum vaccine is administered along with a measles and mumps vaccination, giving the patient immunity to rubellum for about 15 years (MMR); a booster vaccination is administered before elementary or middle school. Women of child-bearing age who are not immune to rubella must undergo vaccination and not get pregnant in the following three months. Vaccination immediately after giving birth is advisable for mothers at risk of being infected.

2.1.2. Citomegalovirus

Citomegalovirus (CMV) is a DNA virus that belongs to the Herpesviridae family. It can go into latency and then reactivate and has been isolated in various sites including saliva, urine, breast milk, sperm, brain fluid, and amniotic fluid. *Congential CMV infection* is thought to be derived from transplacental infection from a primary or recurring maternal infection occurring in the first half of pregnancy. *Prenatal CMV infection* is contracted by contact with infected cervical secretions, breast milk, or blood derivatives. It is believed that maternal antibodies have a protective function and that most of these newborns are either born asymptomatic or are not infected by the virus in the case of contact. Many women who are infected by CMV during pregnancy are asymptomatic, but occasionally develop an illness similar to mononucleosis. It is still unclear if more serious lesions are a consequence of a precocious maternal infection or of a later one during the course of gestation. Nearly 10% of children with congenital CMV infection are symptomatic at birth. Manifestations include delayed intrauterine growth, premature birth, microcephalus, jaundice, petechia, hepatosplenomegalia, periventricular calcification, corioretinitis e pneumonia. The virus causes deafness by infecting the inner ear and altering the Organ of Corti. Moreover, it can cause malformations of the labyrinth of ethmoid and at the same time also lesions on the auditory tract due to secondary toxicity. The hypoacusia that establishes itself is sensorineural, almost always bilateral, and profound, generally regarding acute tones. Symptomatic newborns have a mortality rate of up to 30% and 70–90% of those who survive have neurological deficits such as hearing loss, mental retardations and visual disturbances [8–10]. A vaccine for CMV is still under research. Exposure to the disease in non-immunized pregnant women must be controlled, despite the fact that CMV is ubiquitous everywhere. Since it is frequent in children who attend preschools, pregnant women must observe all the common norms of good hygiene after contact with or being exposed to the urine or expectorate of such children [11,12].

2.2. Post-natal deafness

In post-natal deafness, numerous infective illnesses can be responsible for serious damage to the VIII nerve and the cochlear apparatus. A large part of hearing defects arising in childhood can be traced back to the intrauterine period. The most frequent forms are: viral meningoencephalitis (arbovirus, herpesvirus, mixovirus, poxvirus, etc.), mumps, chickenpox and measles.

2.2.1. Meningoencephalitis

Meningoencephalitis can be primitive or constitute the secondary complication of a viral infection. The forms of primitive