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Editorial

Neurological complications in Varicella

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Editorial

Varicella is a high infectious disease with a worldwide distribution. According to a WHO position paper, global annual disease burden of Varicella is estimated to be 140 million cases [1]. Even if it is often considered a harmless disease, it may have a serious course and may require hospitalization. Among complications, neurological ones have been reported in previous studies. They may be caused by a primary infection or may be associated with a virus reactivation. Generally, children present with neurological complications associated to a primary infection. Those associated with recurrences are often due to the direct effect of viral replication in the central nervous system. Varicella zoster virus reaches the nervous system either through the bloodstream or by direct spread from sensory ganglia where it remains latent. The neurological consequences of Varicella reactivation are more frequent detached among elderly individuals and immunodeficient patients. Neurological involvement in immunocompetent children following Varicella reactivation is exceptionally rare. Varicella-zoster virus can infect a wide variety of cells in the central and peripheral nervous system, including neurons, oligodendrocytes, meningeal cells, ependymal cells, and cells of the blood vessel wall. The wide range of susceptible cells explains the diversity of the clinical and pathologic nervous system manifestations of VZV. The spectrum of VZV infection can range from mild symptomatic infection to meningoencephalitis.

In fact, neurological complications are numerous and include acute cerebella ataxia, stroke or stroke-like episodes, meningitis, encephalitis, myelitis, and vasculopathy.

The pooled prevalence of neurological complications resulting from a systematic review of the literature from January 1990 till January 2012 identifies the likelihood of neurological complications in the pediatric age in the range 13.9-20.4% [2].

Among children, acute cerebella ataxia is one of the most frequent, occurring in about 4-8% of those hospitalized for Varicella [3]. At admission, ataxia is the most frequent symptom, with wide-based gait. Clinical presentation may be also characterized by dysmetry, difficult speech, vomiting and cephalea. It has a limited time course and is generally followed by complete recovery.

In recent years, Varicella has been reported to be the second most common infectious an etiology of encephalitis after herpes simplex virus. Diffuse encephalitis most often occurs in adults, especially in immune suppressed hosts, such as patients with AIDS and transplant recipients. The clinical manifestations include delirium, seizures, and focal neurologic signs. Reported mortality rates approach 10 percent and neuropsychological sequelae reported in up to 15 percent of survivors [4]. In immunocompetent patients, myelitis may complicate acute Varicella or zoster, usually one to two weeks after the development of rash. Its clinical features are paraphrases with sensory-level and sphincter impairment. The cerebrospinal fluid either is normal or shows mild pleocytosis with a normal protein level or a mild elevation. The condition of most patients improves substantially, but some patients have persistent stiffness and weakness of the legs. In immunocompromised patients, myelopathy is often more insidious and progressive, and it is sometimes fatal [5].

Cranial nerve palsies may complicate Varicella. Among the cranial nerves, the trigeminal one is the most commonly involved. The distribution of symptoms varies, depending on which of the three branches of this nerve is affected.

Meningitis and meningoencephalitis have been more frequently detached in the elderly and are associated with a prolonged hospitalization [6] .The outcome is generally good, as adults are reported to have few, mild neurological sequelae and children no reported neurological deficit [4].

Vasculopathy caused by Varicella may occur after both primary and reactivated infection. It may interest both children and adults.

In children, even if the incidence rate is very low, Varicella is the most common cause of acute is chaemicstroke. One or several large or small cerebral arteries may be involved. Visual loss has been reported as a sequela in patients affected by Varicella with small artery involvement, such as the central retinal artery and the posterior ciliary artery. When the middle cerebral artery is affected, contra lateral hemiplegic is often detached [6,7].

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