

Herpes zoster: A review

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Mankind has always been affected by some diseases which have the tendency to spread from one person to another. Amongst these, the diseases of viral origin are one of the most common diseases.

“Virus Is A Piece of Bad News Wrapped In A Protein Coat” - Peter Medawar

A virus is an intracellular parasite composed of DNA or RNA that is surrounded by a protective protein shell. Frequently, this shell is itself enclosed within an envelope that contains both protein and lipid. They multiply only in living cells. In essence, viruses are nucleic acid molecules that can invade cells and can replicate within them. Later they encode proteins capable of forming protective shells around them [1].

Viruses have been classified into various families among which Herpesviridae is the one. It is a family of DNA viruses that cause diseases in animals including the humans. The members of this family are also known as herpes viruses. The family name “Herpesviridae” is derived from the Greek word herpein which means to creep. This refers to the latent recurring infections which are typical of this group of viruses. Herpes viruses can cause latent or lytic infections. [1]

Herpes viruses can be classified as alpha, beta and gamma herpes viruses. One of the ubiquitous human alpha herpes viruses is Varicella-zoster virus (VZV) which can cause varicella (chicken pox) and herpes zoster. Diagnosis for the herpes virus, particularly varicella zoster is considered vital for the patient because of the resultant adverse effects. Symptoms that point to this infection, such as rashes go unnoticed in patients that receive antibiotics because they can be misinterpreted as a side-effect of the medicine. As a result, the medication of the patient is discontinued leading to potentially severe consequences resulting from the spread of the infection.

Herpes zoster (HZ) is the reactivated form of the varicella zoster virus (VZV). It is more commonly known as shingles, which has been derived from the Latin cingulum, “girdle.” This is so because herpes zoster commonly involves a unilateral rash that can wrap around the waist like a girdle. Similarly, the name zoster is derived from classical Greek word. Referring to a belt like binding (known as a zoster) which was used by warriors to secure armor [2].

Annually, over 500,000 people in the United States experience a shingles outbreak. Over 90 percent of the adult population in the United States has serological evidence of a prior varicella zoster virus infection and thus are at risk for developing shingles [2].

Primary infection is clinically identified as varicella or chickenpox with initial exposure typically occurring during childhood. It may transmit through inhalation, ingestion, skin or mucosal contact, blood and blood products. The virus enters the host via the respiratory system, replicates at an undefined site (presumably the nasopharynx), infiltrates the reticuloendothelial system, and eventually makes its way into the bloodstream. Evidence of viremia is manifested by the scattered nature of the telltale skin lesions on the body. The usual incubation period for varicella is 14-16 days with communicability ranging from 10- 21 days after initial exposure. An individual can no longer transmit varicella zoster virus once the final skin lesions have crusted. Indirect transmission (via an immune third person) is not thought to occur [3].

Once the initial outbreak has subsided, varicella zoster virus then retreats into the dorsal root ganglia where it can lie dormant for years until some excitatory factor triggers reactivation. The associated outbreak is then clinically identified as herpes zoster or shingles. Microscopic examination of select dorsal root ganglia tissue during active herpes zoster shows presence of hemorrhage, edema and lymphocytic infiltration [2].

Lowered cellular immunity places an individual at risk for herpes zoster. Henceforth, the immunocompromised and the elderly individuals are more susceptible. It has been proposed that because genes involved in reactivation of herpes simplex virus are missing in varicella zoster virus, varicella zoster virus may not recur as frequently as other herpes viruses [3].

Although herpes zoster occurs at all ages, but its incidence is highest among individuals in the sixth to the eighth decade of life [4].

Shingles symptoms may be vague and nonspecific at first. People with shingles may experience tingling, itching or pain before the classic rash appears. In the pre-eruption stage, diagnosis may be difficult and the pain can be so severe that it may be mistaken for pleurisy, kidney stones, gallstones, appendicitis or even a heart attack depending on the location of the affected nerve [5]. Some patients may have prodromal symptoms without developing the characteristic rash. This situation is known as “*zoster sine herpate*” and it may further complicate the eventual diagnosis [6].

The prodromal phase is followed by development of the characteristic skin lesions of herpes zoster. The skin lesions begin as a maculopapular rash that follows a dermatomal distribution commonly referred to as a “belt-like pattern.” The maculopapular rash evolves into vesicles with an erythematous base. The vesicles are generally painful and their

development is often associated with the occurrence of anxiety and flu-like symptoms. The vesicles eventually become hemorrhagic or turbid and crust over within seven to 10 days. As the crusts fall off, patients are generally left with scarring and pigmentary changes^[6].

Intraorally herpes zoster is characterized by formation of vesicles & bullae that are scattered and surrounded by an erythematous zone. These lesions soon become ulcerated & get covered by a white pseudomembrane. The vesicles ulcerate and form pustules within three to four days. A crust lesion then forms and healing takes place within seven to ten days. These lesions often heal with scarring and areas of hypo or hyperpigmentation may be seen. When the Nucleus facialis and Nucleus auditorius are involved (via the geniculate ganglion) facial paralysis, vesicles on the external ear, tinnitus, deafness and vertigo may follow. This is known as Ramsay-Hunt syndrome^[7].

In herpes zoster infection, the ophthalmic division of the trigeminal nerve is most often affected (herpes zoster ophthalmicus). The corneal involvement may also lead to blindness. Involvement of this nerve leads to lesion on upper eyelid, forehead, scalp, mid face and lower lip. Patients experienced prodrome of pain, burning and tenderness, usually on palate on one side. This is followed several days later by the appearance of painful, clustered 1 to 5 mm ulcers on the hard palate or even on buccal gingiva. Ulcers heal within 10 to 14 days. Involvement of mandibular division of trigeminal nerve results in blisters and ulcers on mandibular gingival and tongue^[8].

In immunocompetent patients infected with herpes zoster, the most distressing symptom is typically the pain while the most feared complication is post herpetic neuralgia (PHN), in which there is persistence of pain for long even after healing of the rash.⁹

Prior to the 1940's shingles was thought to be non threatening and little was done in the way of treatment. Patients were instructed not to scratch or to apply cold-water compresses to weeping blisters. By the 1950's it became evident that shingles occurring near an eye or ear could lead to serious complications including the loss of hearing or vision^[2].

Nowadays many options are available to the clinician for the treatment of herpes zoster and post herpetic neuralgia, albeit with variable degrees of success. Antiviral agents, such as acyclovir, valacyclovir and famciclovir have been shown to reduce both the pain and healing time of skin lesions associated with herpes zoster but have marginal success in preventing and treating post herpetic neuralgia. Corticosteroids may be used for pain management in herpes zoster, but do not seem to be effective in prevention of post herpetic neuralgia. Analgesics provide effective temporary pain relief for both herpes zoster and post herpetic neuralgia. Nerve block injections offer more long-term pain relief in both conditions provided they are administered early in the course of the disease.²The availability of a safe and effective vaccine for zoster offers an opportunity to decrease the burden of this disease and its complications among persons with high levels of risk.

An attempt has been made to understand the nature of herpes zoster, its etiopathogenesis, clinicopathological features and diagnostic criteria. It is emphasized that early diagnosis is must to reduce morbidity and initiate prompt early management.

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