

## A STUDY OF THE DERMATOMERS IN HERPES ZOSTER

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**Abstract:** *We have performed a clinical study on a number of 53 herpes zoster patients who were hospitalized in the Infectious Diseases Hospital of Brasov in 2008. In the studied group we focused on the affected body areas, on the number of dermatomes, distribution on sexes and age groups, as well as on the possible immunodepressing factors triggered by associated chronic diseases.*

**Key words:** *dermatome, herpes zoster.*

### 1. Introduction

Herpes Zoster is a type of neurodermitis caused by the reactivation of the varicella-zoster virus (VZV) as a result of immunodepression, after natural immunization caused by chickenpox the patients suffered from in their childhood. The virus remained latent in the nervous ganglia and was reactivated by a series of factors which harm the body. This virus harms the sensitive, dorsal roots of the spinal or cranial nerves, the disease manifesting itself through skin rash with blisters, forming a stripe on the dermatomes corresponding to the affected sensitive nerves. The varicella-zoster virus belongs to the Herpes Viridae family. It is made up of a nucleocapsida that contains a double-stranded DNA and a lipid layer, whose surface presents a large number of spicules glycoproteins. The varicella-zoster virus can be cultivated on continuous or discontinuous human or simian cell lines. The reproduction of the varicella-zoster virus is obtained by viral replication. Each infected cell is capable of providing the material necessary for the production of over 100 new viral particles.

As a result of the accumulation of viral particles, the cell growth and disintegrate releasing new virions. For the replication, the virus uses the proteic device of the host cell, which instead of producing cell proteins will produce viral proteins. By means of electronic microscopy it has been noticed that immature viral cells start appearing 12 hours after the onset of the infection. We do not know yet all the details concerning viral latency and reactivation, but with the aid of PCR techniques the VZV genome has been shown to exist at the level of the neurons, each infected neuron having 2 or 3 copies of viral DNA. Copies of the infecting DNA have been reported at the level of the non-neuronal satellite cells in the sensitive ganglia. By means of in-situ hybridization it has been estimated that the VZV remains dormant in 0.01-0.03% of the neuronal population. The reactivation consists in the resumption of the viral replication, the result of which is the inflammation of the ganglion, and thus an intense neuralgia along the affected nerve and the centrifugal propagation of the VZV along the nerve ramifications towards the dermatomes.

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Once in the skin, the VZV will multiply intensely in the deep layers of the epidermis, triggering specific cytopathic effects: giant cells, with multiple nuclei and with eosinophilic intranuclear inclusions. These cell lesions together with the local inflammation and the immune response of the host will bring about the formation of blisters.

### 1.1. Clinical Presentation

The incubation period is not known due to the fact that herpes zoster consists in the reactivation of the dormant virus. The eruptive period lasts a few days and manifests itself through intense neuralgia at the level of one or two dermatomes specific of the affected nerve. The burning pain may be accompanied by fever and skin hypersensitivity, which may lead to confusions concerning the diagnosis: pleuritis, pneumonia, myocardial arrest, appendicitis, kidney or biliary colics. The eruptive period is marked by the emergence of vesicles and lasts 2-3 weeks. The eruption in the case of herpes zoster is characterised by radicular distribution which affects only the dermatomes specific of the infected nerve; the distribution is unilateral and stops at the anterior and dorsal midline, having a 'belt' character. The eruptive elements follow the same stages as chickenpox: macula, papula, blisters and crust. The vesicles form within 12-14 hours, grouped in hives, then in confluent polycyclic blisters. The vesicles become cloudy in the 5<sup>th</sup> day, they crust over between the 7<sup>th</sup> and the 10<sup>th</sup> days; after 2-3 weeks the crusts fall off, leaving pinkish-whitish scars. The chronic period or the post-zoster neuralgia is represented by the pre-existent pain which persists 4 to 12 weeks after the onset of the eruption or by the re-occurring pain that lasts for about 30 days. If not treated properly, post-zoster neuralgia may last for years, having even a higher intensity.

## 2. Aims

The present paper aims at highlighting the possible dermatomeric areas, the incriminated factors in reactivating the varicella-zoster virus and the incidence of the disease with respect to sexes and age groups.

## 3. Material and Method Employed

The study has been performed on a group of 53 patients with ages between 15 and 82, who were hospitalized in the Hospital of Infectious Diseases of Brasov in the year 2008. The 53 patients diagnosed with herpes zoster and treated for it were divided into 3 age groups: teenagers (15-19), adults (20-59) and elderly people (60-82).

### 3.1. Pathologic Anatomy

In order to understand the evolution of the disease, we thought it appropriate to take into account the neuro-anatomy of the affected areas. Thus, the tegument of each area is divided into dermatomes, specific innervated areas with sensitive nervous fibres that belong to one spinal nerve. There are 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal. The sensory information at the level of each dermatome is taken over by the sensitive nervous fibres and directed to the nervous roots corresponding to the respective dermatome. In the herpes zoster on the chest, the hives of blisters appear in the area where the sensitive nervous fibres of the inter-costal nerves emerge. Sometimes, due to the fact that the neurons in the anterior medullary horns are harmed, it may come to paralysis or palsies, vegetative or trophic disfunctions (vasomotric, secretion).

The herpes zoster ophthalmicus appears as a result of the reactivation of the dormant

VZV in the Gasser ganglion situated on the trajectory of the sensitive fibres of the trigeminal nerve. The ophthalmic branch of the trigeminal nerve has 3 sub-branches: frontal, lachrymal and nasal. The VZV can affect only one sub-branch. The diagnosis depends on the topographic localisation of

the lesions. Ocular complications are the result of the inflammations (stromal keratitis, uveitis), of viral replication (lesions of the cornea), trophic disfunctions (neuro-paralytic keratitis) and ischemia.

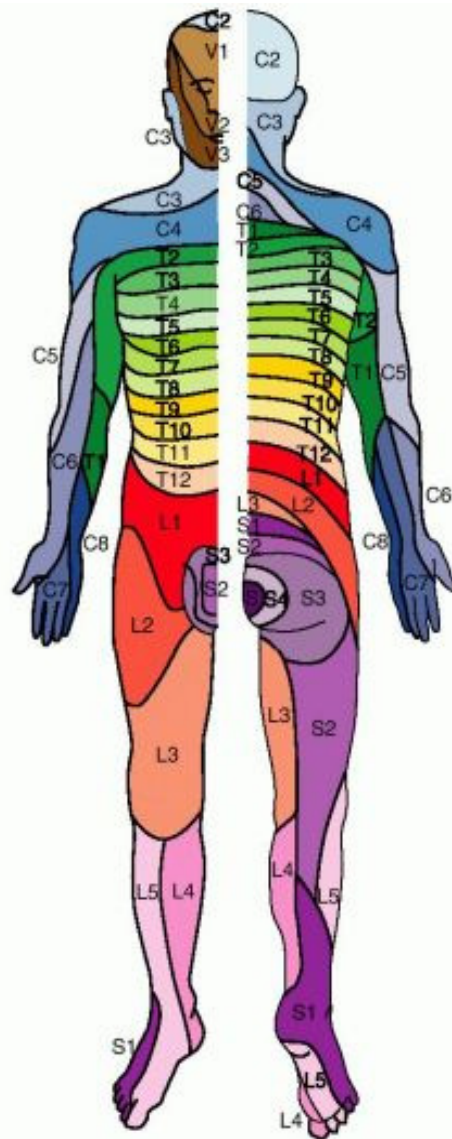


Fig. 1. *Dermatomers- antero-posterioara*

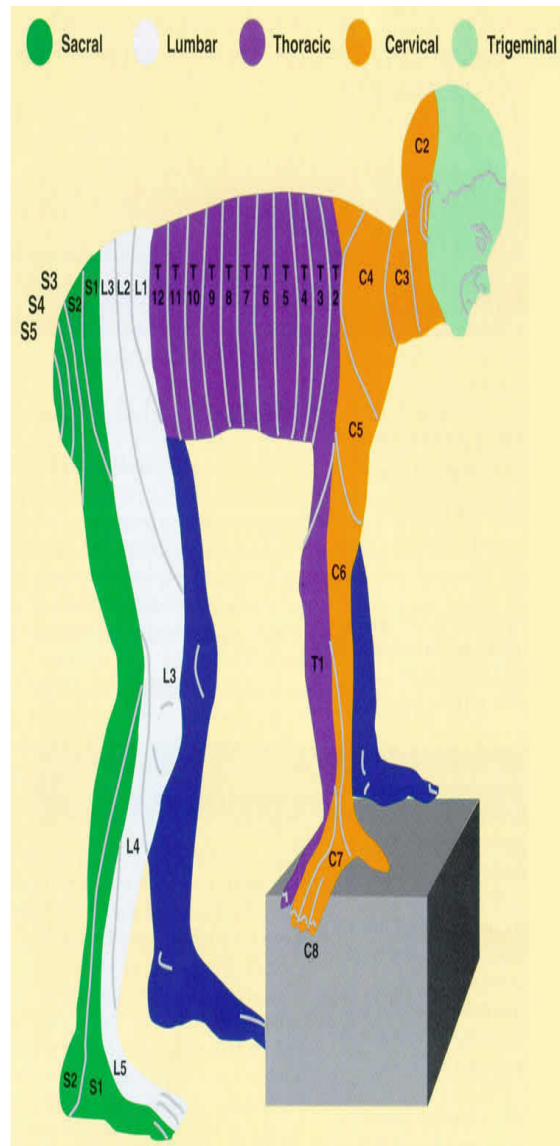


Fig. 2. *Dermatomers- vedere laterala*

#### 4. Findings and Comments

The distribution on age groups and sexes shows a higher incidence of the disease in the case of females (36 persons, representing 68% of the total number of subjects) as opposed to men (17 persons representing 32% of the total number of

subjects). When brought to the hospital, the patients showed the following symptoms: fever (56%), head aches (29%), pain (94%), skin rash (98%), itching (88%). Of the total number of subjects, 24 persons (45%) suffered from post-zoster neuralgia. The elderly persons also showed associated secondary pathologies

which could have caused immunodepression, such as lung problems (9%), liver problems (13%), kidney microlithiasis (9%), renal insufficiency (6%), liver problems (6%), diabetes (8%), ischemic cardiopathy (23%), anaemia (9%), HTA (25%), obesity (13%), AIDS (4%), leukaemia (2%). All these have diminished the immunity of the patients, creating a medium favourable for the reactivation of the varicella-zoster virus.

The topographic localisation of the lesions differed among the 53 patients: in 48 cases, the lesions appeared along the spinal nerves, whereas in the case of 5 patients, the lesions followed the trajectory of the cranial nerves, as follows (see table no. 1):

The most frequently affected nerves were the intercostals (42%), followed by the lumbar nerves (17%), then the brachial (15%), the trigeminal (9%), the sacral (9%) and the cervical nerves. In all the 5 cases in which the trigeminal nerve was affected, the ophthalmic sub-branch was infected. In 30 (57%) cases we identified the infection of a single dermatome, whereas multiple dermatomeric localisations were identified in 23 cases (43%). The areas affected in the latter are presented in table 2 below.

At the same time, we also detected a number of non-pathogenic factors which could have contributed to the herpes zoster infection, such as old age, stress or increased emotional tension, immunosuppressant medication, irradiation or inappropriate eating habits.

*Dermatome distribution on cranial and spinal nerves.*

Table 1

Trigeminal nerve	Cervical nerves C1-C4	Brachial nerves C5-T1	Intercostal nerves T2-T12	Lumbar nerves L1-L4	Sacral nerves L5-S4
5	48		22	9	5

*Multiple dermatomeric localizations*

Table 2

Cervico-brachial	Thoracic-brachial	Thoracic -lumbar	Lumbar-sacral	Disseminated
3 (13%)	5 (22%)	3 (13%)	3 (13%)	9 (39%)

## 5. Conclusions

1. We noticed that the elderly people and the adults are equally affected by shingles (45% vs. 42%), while the young people and the teenagers are less affected by this disease (13%).
2. We also noticed that females (68%) are more affected by the disease than men (32%).
3. Our study shows that the most frequent localisation occurs at the level of the intercostal nerves (42%), followed by the lumbar nerves (17%), then the brachial (15%), the trigeminal (9%), the sacral (9%) and the cervical nerves (8%).
4. The affection of a single dermatome has been recorded in (57%) of the cases as opposed to multiple dermatomeric localisations (43%).

5. The multiple dermatomeric localisations are more frequent in the case of female immunocompromised patients.
6. We noticed that the most frequent risk factors that favour the herpes zoster infection are: severe immunodeficiency (in the case of HIV positive persons), cytostatic treatments, liver problems, kidney problems, heart problems), a weak immune system due to pre-existent problems (chronic liver diseases, chronic lung diseases), immunodepressant medication, old age, stress or increased emotional tension, pregnancy, inappropriate eating habits or/and irradiation.

## References

1. Arama, V. and Streinu, C. *Infectii cu Herpes Virusuri*. Bucureşti: Editura Info Medica, 2002.
2. Chiotan, M. *Boli Infectioase*. Bucuresti: Editura National, 2002.
3. Netter, F. *Atlas de Anatomie Umană*. Bucuresti: Editura Info Medica, 2005.
4. Niculescu, C. Th. and Onisai, L. *Anatomia Functionala a Nervilor Cranieni*. Brasov: Editura Lux Libris, 1996.
5. Rebedea, I.; Dumitru, C.; Căruntu, F. Al. *Boli Infectioase*. Bucureşti: Editura Medicala, 2002.
6. Voiculescu, M. *Tratat de Boli Infectioase*. Bucureşti: Editura Medicală, 1990.