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AN ATTEMPT TO PROPOSE THE VIRAL CLASSIFICATION ALONG WITH CLINICAL PRESENTATION OF HERPES ZOSTER INVOLVING TWO DERMATOMES AND ITS OVERVIEW

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ABSTRACT

This article gives an appropriate insight into the importance of Herpes zoster infection in relation to Oro-facial region, epidemiology, etiopathogenesis, a clinical presentation of the case, diagnosis and its management in our day to day practice.

KEYWORDS: 1) Infection 2) Oral 3) Virus 4) Herpes-zoster.

INTRODUCTION

It is when, disease causing organisms for instance bacteria, virus, fungi, etc enter our body through various routes and simultaneously multiply within our body producing various toxins. Humans are susceptible for this and consequently show signs and symptoms. Occasionally, some infections do not show signs and symptoms, but most of these are appreciated clinically. Contemporarily, most of the oral lesions which are caused due to viral infections are encountered in our day to day dental practice. A whole lot of lesions associated with virus are encountered in periodontitis.[4] Recent researches suggest that Papiloma viral infections are turning up into malignancy which is of significance for study.[4] Some of the diseases do not have permanent treatment, yet they are controlled symptomatically and pathologically. Eg: HIV. Many diseases are the differential diagnosis of some viral infections; hence it is critical for the accurate diagnosis.

PROPOSED CLASSIFICATION OF VIRUS

Viral Infections

Viruses are classified into 2 main divisions depending on the type of nucleic acid they posses:

DNA CONTAINING VIRUSES

Dna viruses

Doubled stranded

HERPESVIRIDAE

1. Herpes Simplex Group I
2. Herpes Simplex Group II
3. Varicella Zoster
4. Cytomegalovirus
5. Eb Virus
6. Human Herpes Virus 6,
7. Human Herpes Virus 7
8. Human Herpes Virus 8 (Kaposi sarcoma causing)

SINGLE STRANDED

Parvoviridae

Eg: parvovirus b19

RNA CONTAINING VIRUS

Single Stranded

Rabies Virus

Paramyxo Virus

Pneumo Virus

Morbilli Virus

Orthoyxo Virus Group (Influenza Virus)

Arenoviridae

Flavoviridae

Coronoviridae

Retroviridae.

Only Double Stranded Rna Virus Is

Reoviridae Family

Reovirus

Herpes Zoster

HERPES ZOSTER (SHINGLES OR ZONA)

It is also called as Shingles or Zona.[11] Specifically, activation of this latent virus that had caused chicken pox i.e. Varicella Zoster virus. It is an acute infection in which the virus manifests mostly in the dorsal root ganglia of the trigeminal nerve. It causes painful vesicles which subsequently erupt causing secondary infections. People who are immunocompromised and facing severe illness are more prone to this infection. It affects morbidity and mortality of the human population if it is not treated.

EPIDEMOLOGY

There is no seasonal appearance of shingles infection unlike chicken-pox.[9] Higher rate of incidence is seen in women than in men. Incidence among young people is low. Incidence of this disease is increasing in the recent years globally. All studies show that there is great increase in zoster with age, particularly after 50 years.[10] The average age of onset is 22 years for adults and 59 years for older people, with 85% cases attributes in those 50 years.[10] VZV effects one in five people globally, and are suffering with dreadful complication PHN (post herpetic neuralgia).[13]

PREDISPOSING FACTORS

Immunocompromised Patients

Compromised immunity is the most common trigger for the latent varicella to become active. For example: patients with lymphoma and myeloma are not eligible for zoster vaccination.

Decreased Cell-Mediated Immunity

As there is natural decrease in cell - mediated immunity in the individuals with increasing in age, hence people with age above 50 and over are at risk.

Immunosuppressive Drugs

Anti-cancer drugs when used for the long duration, leads to immune suppression which is risk for the disease.

Debilitating Diseases

Debilitating diseases like measles, mumps in young children and diabetes mellitus in adults are facing the risk.

Idopathic

Though several studies states the above are the risk factors, but the reactivation of virus is not entirely understood.

Etiopathogenesis

Route of infection is through inhalation of infected nasal mucosal secretions as air droplets.

↓

The virus multiplicates in the nasopharyngeal tissues of the host.

↓

The virion enters the regional lymph nodes and infects cd4+ T- cells.

↓

Primary viremia.[4,5]

↓

Then replicates in the internal organs like spleen and liver.

↓

Secondary viremia and viral invasion into capillaries, endothelial cells and epithelial cells causing macules or vesicles or pustules.[4,5]

↓

Chiken pox (Primary infection)

the virus migrates from mucosal cells to dorsal rootganglia of cranial spinalnerves and exhibits latency or in a dormant stage.

↓

reactivation of virus due to above predisposing factors, VZV transported back to sensory nerves causing severe pain and initial rash.

↓

It turns into vesicles, pustules and clinically evident herpes zoster is seen.

Varicella-zoster virus is neurotropic and epitheliotropic.[8] Primary infection causes chicken-pox where as reactivation of latent virus causes herpes-zoster infection. In the primary infection, immune response develops and mucosal infections subside. During this course, virus is transported back from the mucosal cells to neuronal cell bodies i.e. mainly dorsal root ganglia of spinal nerves where it remains as a latent virus for a lifetime. In this period of latent infection there are no symptoms, virus does not replicate and patient is not infectious. Reactivation of the virus leads to replication, subsequently transportation of virus to sensory

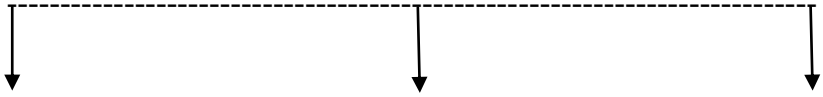
nerves, and then the clinically evident herpes-zoster is developed. It affects the dermatome of the effected sensory nerve. Reactivation of the latent virus depends on the host cell, not the virus, triggers and risk factors. Unlike HSV, reactivation is rare phenomenon in herpes-zoster infection although two in three individuals effected. It is mostly unilateral in distribution affecting one or two dermatomes. Prodromal pain appears before the rash appears.[12]

Ramsay-Hunt Syndrome

Zoster infection of geniculate ganglion which is clinically evident as skin lesions of external auditory meatus, oral mucosa, and involvement of facial and auditory nerve. Order of effecting dermatomes is thoracic (50-60%), cervical (10-20%), trigeminal (10-20%), lumbar (5-10%) and sacral(5%).[5]

Clinical Features

Clinical featres are divided into three catogories namely;-



1. Prodromal phase

It is characterised by severe pain in the dermatome of the effeced sensory nerve and the pain is described as burnig, tingling, knife-like. It is due to virus travels down the infected nerve. Initial symptoms are fever, malaise and headache. Prodromal pain is false perceived as otitis media, myocardial infarction, appendicitis, headache, migraine.

2. Acute phase

Prodromal phase is succeded by the acute phase rash subsequently, group of vesicles are formed (some clear fluid is seen), then vesicles are transformed to pustules (filled with more or less pus) which often rupture to form crusts these lesions are surrounded by the erythmatous base Lesions usually culminate at the midface (unilateral-which is the characteristic of the disease) Ocassionally, there is pain in the respective affected dermatome. However, the rash is not devoloped. This is termed as Zoster Sine Herpete. Ocular involvement- (10-25%) cases, may also lead to permanant blindness. **Hutchinson's sign**-devolopment of vesicles on the tip of the nose.

3. Chronic Phase

Postherpetic neuralgia is the most threatened complication of herpes zoster infection. It is characterised by severe, throbbing, burning, persistant pain after the resolution of the rash.Chronic phase is was observed in only 15% of patients amd may subside by 2months or 1 year or rarely 20 years. Secondary infection with bacteria may also cause encephalitis.[18]

Oral Manifestation

Vesicles or blisters seen on oral mucosa especially buccal mucosa, tongue, pharynx.

Case Presentation

A patient named Mrs. Sarojini devi, 63 years old female, came to our OPD at EHS/JHS wellness center, khairthabad with a cheif complaint of intensive burning pain on the left region of the face predominantly of cheek, malar region, lips, nose, chin and fever for a past days. Pain was excrutiating and radiating to ear, upper temporal region. Vesicular eruptions on cheek and lips. The patient had a history of chiken pox during her childhood. There is a medical history of diabetes and patient is on anti-diabetic medication since last 5 years and no dental history. On extra-oral examination, patient had vesicles and some of them were erupting in the distribution of maxillary and mandibular divisions of Trigeminal nerve (V2 and V3). Skin lesions at different stages were clinically seen such as vesicles pustules, scabs. Erythma surrounding the vesicles were noticed (Fig: 1).

Pictorial Representation of The Case

Fig 1: Clinical presentation of Herpes zoster.

Fig: 2: Clinical picture of Herpes Zoster showing some of the erupted vesicles.

Fig 3: Post treatment of Herpes zoster and clinical scarring of lesions were seen in the picture.

DIAGNOSIS

Diagnosis is the major criteria for detection of this disease.

1. Classic Clinical Pattern

Severe painful rashes that are like vesicles can be seen mostly unilaterally in a dermatomal distribution.

LAB DIAGNOSIS

2. Polymerase Chain Reaction

PCR of VZV-DNA, which is derived from vesicle of host, is an accurate method.[15]

3. Tzank Smear

It is the standard diagnostic test for herpes zoster .vesicular scrapings are collected from edge and base. These are isolated, smeared on to a glass-slide and stained with Diff-Quick stain. Then the slides were observed under microscopy which reveals, multinucleate gaint cells that are characteristic of the disease.[14]

4. Direct Flourescense Antibody Test

In this test VZV antigen is detected using a tagged antibody. it is most widely used test than Tzanck test.[15]

Treatment and its Management

Antiviral drugs such as acyclovir, famciclovir or valacyclovir reduces the vesicular rashes. Along with the antiviral medication, opioid analgesics, corticosteroids or tricyclic antidepressants reduces the pain caused by herpes zoster infection. Increasing age is the primary risk factor for herpes zoster infection and there are studies which say antiviral therapy during the acute phase can prevent the posttherapetic neuralgia. Immunization is available to prevent herpes zoster and posttherapeutic neuralgia for individuals 60 yrs and above.[19]

CONCLUSION

Hence it is very crucial to diagnose viral diseases as it is concerned with morbidity and mortality of humans. Herpes zoster is significant as we can see the rise of this disease globally. This infection can be manageable effectively and patient needs supportive care after the treatment as in cases of the older people.

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