

Herpes Zoster Of The Trigeminal Nerve-Two Cases Reports.

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Citation

C Makos, G Noussios, M Peios, G Balabanis, C Evangelinou. *Herpes Zoster Of The Trigeminal Nerve-Two Cases Reports.* The Internet Journal of Neurology. 2010 Volume 13 Number 2.

Abstract

Background: The herpes zoster of the trigeminal nerve branches caused by Varicella Zoster Virus (VZV) is a clinical entity consisting of erythematous macules, papules, vesicles, bullae, small ulcers and erythematous plaques and characteristic short acute or pre-eruptive phases and long methherpetic periods with pain, burning or tingling in the involved dermatomes like in other peripheral nerves. The diagnosis of herpes zoster is primarily based upon history and clinical examination. The painful periods can induce misdiagnosis with dental signs like trigeminal neuralgia, odontalgia and acute pulpitis and the complications referred in the literature like tooth resorption, periapical lesions, periodontal destruction, osteomyelitis, jaw osteonecrosis and tooth exfoliation promote the dentist's role in diagnosis and management of this disease. Cases Reports:Two cases related to herpes zoster of the trigeminal nerve branches diagnosed at first stages as trigeminal neuralgia are presented in this report. Besides pathogenesis, clinical picture, jaw complications, diagnosis and therapeutic aspects are discussed.

INTRODUCTION

Varicella Zoster Virus (VZV) is an enveloped, spherical, 150-200 nm in diameter virus, with single, linear, double-stranded DNA molecule, 125.000 nt long. It belongs to the genus Varicellovirus, family Herpesviridae, subfamily Alphaherpesvirinae. It is also known as Human Herpes Virus type 3 (HHV 3) and is related to the Herpes Simple Viruses type 1 and 2 (HSV 1, HSV 2) sharing much genome homology. The known envelope glycoproteins (gB, gC, gE, gH, gI, gK, gL) correspond ,with those in HSV, however there is no equivalent of HSV gD [1].

The virus is ubiquitous in most populations. Primary infection with VZV causes varicella, commonly known as chickenpox. It is spread through direct person-to-person contact with viral lesions and/or airborne droplets. Maternal viremia leads to spread through placenta causing neonatal varicella. Primary infection usually occurs through the bulbar conjunctiva or upper respiratory tract mucosa, following direct contact with skin lesions or inhalation of virus-infected respiratory secretions [2]. Over the next 2-4 days a viral replication takes place in regional lymph nodes and 4-6 days later the virus spreads through circulation causing primary viremia with inoculation mainly in the liver and spleen, where virus is replicated. 14-16 days after initial infection there is a secondary viremia and spreading causing

the typical vesicular rash to the skin. At this time hepatitis, encephalitis and or pneumonia may also occur [3]. VZV is neurotropic remaining in a latent patent in sensory neurons and especially in the gasserian, geniculate and dorsal root ganglia. Reactivation related to periods of cellular immunity impairment leads to herpes zoster [4].

Herpes zoster of the trigeminal (V cranial) nerve with involvement of the ophthalmic (V1), maxillary (V2) or mandibular (V3) branch is an interesting clinical entity for the clinicians concerning oral and maxillofacial region. The painful periods can induce misdiagnosis with trigeminal neuralgia, odontalgia and acute pulpitis and the complications mentioned in the literature like tooth resorption, periapical lesions, periodontal destruction, tooth exfoliation, osteomyelitis and jaw osteonecrosis promote the dentist's role in diagnosis and management of this disease.

Cases related to herpes zoster of the trigeminal nerve branches with primary clinical picture like a trigeminal neuralgia are presented in this study. Besides dental complications, laboratory findings, diagnosis and therapeutical methods are also mentioned indicatively.

CASES REPORTS

CASE 1

In 2000, a 61-years-old male patient was referred for acute, periodic and long standing pain like electricity or knife cut for the last week in the left aspect of the head extending in side of the nose, upper eyelid, frontal, parietal and temporal area. Washing his face with cold water or wiping it out with towel was a characteristic trigger action. Trigeminal neuralgia was suspected and carbamazepine was administered. The re-examination 5 days later disclosed erythematous macules, papules, vesicular rash, bullae, fluid excretion, desiccation and crusts with unilateral dermatomal distribution (Fig. 1 and 2) and no improvement with the administered drug was accessed. A collaboration with Dermatological and Neurological Department concluded about herpes zoster of the ophthalmic (V1) and maxillary (V2) branch of the trigeminal (V) nerve and the administration of acyclovir and non steroidal anti-inflammatory drugs for one week was effective.

Figure 1

Figure 1. The erythematous macules, papules, vesicular rash, bullae, fluid excretion, desiccation and crusts with unilateral dermatomal distribution on the nose, upper eyelid, frontal, parietal and temporal area.



Figure 2

Figure 2. A parietal aspect of the skin lesions with unilateral dermatomal distribution.



CASE 2

In 1997, a 73-years-old female patient was referred for acute and periodic pain like electric discharge for two weeks in the left aspect of the face including preauricular area, lower labia, lower lip, chin and posterior one third of the tongue. Washing her face, wiping it with towel and eating were characteristic trigger actions. The diagnosis in an external medical office before two weeks was trigeminal neuralgia and diphenylhydantoin had been administered with no relief. The painful area was covering in a unilateral dermatomal distribution by erythematous macules, papules, vesicular rash, bullae, fluid excretion, desiccation and crusts in dermis and erosions or ulcerations with erythematous circumscription in oral and tongue mucous (Fig. 3 and 4). A collaboration with Dermatological and Neurological Department concluded about herpes zoster of the mandibular (V3) branch of the trigeminal (V) nerve and the glossopharyngeal (IX) nerve. The administration of acyclovir and non steroidal anti-inflammatory drugs for 10 days was effective.

Figure 3

Figure 3. The erythematous macules, papules, vesicular rash, bullae, fluid excretion, desiccation and crusts in a dermatomal distribution on the left aspect of the face including preauricular area, lower labia, lower lip and chin.



Figure 4

Figure 4. The erosions and/or ulcerations with erythematous circumscription in oral mucous and latero- posterior one third of the tongue.



DISCUSSION

In most patients with herpes zoster a two-three day primary

acute or pre-eruptive phase with pain, burning or tingling in the involved dermatome is recorded. Fatigue, headache, low-grade fever and myalgia may be present [2,5,6]. The eruptive phase is characterized by erythematous macules and papules diverting into vesicular rash with unilateral dermatomal distribution. Over the next 3-5 days new lesions tend to rise forming bullae [2]. The vesicular rash is followed by rupture of the vesicles, fluid excretion, crusts and desiccation leaving small ulcers² and erythematous plaques with deliberate improvement without typically visible scars. Metherpetic pain in the affected dermatomes is not rare [6].

Herpes zoster of the trigeminal (V) nerve is divided into herpes zoster of the ophthalmic (V1), maxillary (V2) and mandibular (V3) nerve respectively. Herpes Zoster Ophthalmicus (HZO) occurs when VZV invades the Gasserian Ganglion with a frequency 10-15% of all zoster cases and it is five times commonest than the maxillary (V2) and mandibular (V3) type. The eruptive phase of HZO includes ipsilateral erythema and vesicular rash in V1 dermatome of the forehead and upper eyelid. The ipsilateral involvement of the preauricular and occasionally submaxillary lymphnodes, along with headache, pain, nausea and vomiting are common prodrome symptoms. The recurrent meningeal branch (Arnold's nerve) to the tentorium cerebelli may account for the meningeal irritation signs and therefore, meningitis must be excluded. The involvement of the nasociliary nerves correlates with vesicles on the tip and/or the lateral nose (Hutchinson sign) [7,8].

Possible serious complications are ocular inflammation and corneal denervation[6]. In herpes zoster of the maxillary branch (V2) the evidences are in relation to V2 dermatome i.e. cheek, lateral nose, nasal mucosa, lower eyelid, upper eyelid, cheek mucosa, maxillary gums, palate, tonsil and nasopharynx. Sometimes, except oral mucosa, there are no skin manifestations[6]. Corneal involvement in maxillary zoster is rare [9]. In herpes zoster of the mandibular branch (V3) the evidences are localized to V3 dermatome i.e. lateral head, external ear, external auditory meatus, lower lip, lip mucosa, mandibular gums and lateral neck[6].

In herpes zoster of the glossopharyngeal (IX cranial) and vagal (X cranial) nerve the jugular and petrosal ganglia are involved respectively and the painful vesicular rash is found on palate, posterior tongue, epiglottis, tonsillar pillars and, occasionally, external ear. The affection of Arnold's recurrent nerve may be accompanied by a typically mild encephalomyelitis and rarely by a significant zoster

meningoencephalitis[6]. In Ramsay-Hunt syndrome the involvement of the geniculate ganglion of the facial (VII cranial) nerve is indicated with ipsilateral deep otalgia, tinnitus, loss of hearing, vertigo, facial paresis and characteristic herpetiform vesicles on the external ear meatus [10,11]. Ramsay Hunt syndrome is also frequently associated with VIII cranial nerve and more scarce with V, VI, IX, and X cranial nerves involvement [12]. A case of Frankl-Hochwart syndrome (polyneuritis cranialis menieriformis) as a variety of Ramsay-Hunt syndrome[13] and a case of Fisher's syndrome associated with trigeminal herpes zoster explained via propagation of the virus to the brainstem through the trigeminal root [14] is reported. Immunocompromised patients may develop a disseminated or a multidermatomes form of herpes zoster with a high risk in developing visceral affections, like pneumonia or hepatitis [6]. Concerning herpes zoster of the occipitocollaris ganglion (C2 and C3 spinal nerves) the lesions are estimated in the posterior scalp, neck and part of the external ear, lower mandible and anterior neck.

The diagnosis of herpes zoster is primarily based upon history and clinical examination. Sometimes herpes zoster may be present in an atypical form, especially in immunocompromised patients, requiring further laboratory examination like direct immunofluorescence with fluorescein-tagged antibody (DFA) or polymerase chain reaction (PCR). The Tzanck smear in vesicular lesions is a classical method, but there is no differentiation between herpes zoster and herpes simplex. Biopsy is applied in difficult cases and VZV can be cultured successfully [2,5,6].

Because the manifestations of a trigeminal herpes zoster resemble to other oral entities the oral practitioners must be aware about the differential diagnosis and definitive treatment modalities before any "dental therapy" is applied and intraoral examination is necessary when skin facial lesions are observed by professionals [15-19].

The therapy in herpes zoster infection aims to shorten the clinical course, provide analgesia and to prevent complications. Antiviral agents (acyclovir, famcyclovir, pencyclovir, valacyclovir) are nucleotide-like substances with per os administration for 7-10 days. Corticosteroids (prednisone) have anti-inflammatory action used mainly in acute but not in long-term pain. Analgesics (acetaminophene, non steroidal anti-inflammatory drugs) and tricyclic antidepressants (amitriptylene) can be used [2,5]. Active immunity is provided by vaccines with a 99% effectiveness against varicella infection [6].

CONCLUSION

The herpes zoster of the trigeminal nerve and /or other cranial nerves involving the oral and maxillofacial area may be faulty attributed to other neuralgic conditions mainly in the primary painful stages lacking any other clinically obvious lesions. The history, a detail clinical and radiographical examination, the pain distribution and the short re-examinations may offer in differential diagnosis before any “pulpitis”. “Tooth extraction” or “trigeminal neuralgia” is the administered solution.

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