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Case Report

CASE REPORT

Multiple Cranial Neuropathies Involvement in Varicella Zoster Virus Infections

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ABSTRACT

Unlike the children, Varicella Zoster Virus (VZV) rarely cause disseminated infection of the CNS except those who have immunocompromised state in the form of HIV infection or using immunosuppressents. However, it may cause focal root infection in the form of shingles and rarely as an involvement of the cranial nerves. Infection involving the multiple cranial nerves has been observed only in some case reports. It is usually a self-limiting disease however early diagnosis and timely treatment may help in quick recovery. We are reporting an interesting case of VZV infection having typical rash and multiple cranial neuropathies.

Key Words: Immunocompromised, Meningoencephalitis, Postherpetic neuralgia, Radiculopathy, Rash,

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Article info.
Received: November 9, 2018
Accepted: November 22, 2018

Cite this case Report: *Zaman Q, Malik WT, Ehsan-ul-Haq, Khan MF. Multiple cranial neuropathies involvement in Varicella zoster virus(VZV) infections: A Casre Report. JIMDC. 2018; 7(4):304--306*

Funding Source: *Nil*
Conflict of Interest: *Nil*

Introduction

Varicella zoster virus (VZV) infection commonly affects the central and peripheral nervous system, the commonest presentation being meningoencephalitis causing vasculitic arteriopathy and acute cerebellitis in children[1]. However, it may affect the peripheral nervous system in the form of acute radiculopathy or neuropathy. Shingles refers to the local VZV infection resulting from reactivation of dormant virus commonly after lapses in immunity of the individual. It may affect various dermatomes and myotomes, most commonly the thoracic and lumbar regions but also rarely the cervical roots[2]. Cranial nerves commonly affected are trigeminal and facial nerves referred as (Ramsay Hunt syndrome), other cranial nerve involvement is rare[2]. We are reporting a patient with multiple cranial neuropathies having contralateral superior oblique weakness resulting from left trochlear nerve palsy and associated lesions of right trigeminal and vestibulocochlear nerves. She responded to treatment with antivirals and recovered significantly on follow up

Case Report

A 24-year-old lady who had no previous co-morbids or any history of immunosuppressive illness (like HIV or diabetes) and was not taking any medication causing immunosuppression. She presented with 5 days' history of rash in the right auricular canal and around the ear followed by fever, sore throat and right ear pain. It was followed shortly by double vision maximum on looking to the right side and downward with nausea, vomiting, vertigo and imbalance on walking. Her symptoms gradually worsened over 2 days and she had to visit hospital. There was no history of headache or visual blurring but had pain involving right half of the face.There was no speech or swallowing difficulty, no weakness of arms or legs, no paraesthesias or numbness involving the

limbs. On examination she had normal higher mental functions and speech, normal vision and fundi. Pupils were bilaterally equal and reactive. She had left superior oblique palsy andthe rest of the ocular movements were normal. She had vesicular rash involving the right ear and decrease sensation on the right half of the face. Both sides of the face were symmetrical; the patient had normal hearingand tongue and pharyngeal movements. Rest of the neurological examination including sensory,motor andcerebellar examination was normal, but had some ataxia on tandem walking.MRI brain showed contrast enhancement of the right 8th nerve (Figure 1). Her CSF showed 10 cells, with 70 % neutrophils,30% lymphocytes,proteins were 59 mg/dl and CSF glucose was 61 mg/dl with serum glucose level of 75 mg/dl. MTB PCR was done to rule out tuberculosis as it is another common cause of such presentation. It was found to be negative, and ESR was 14.Other causes were ruled out. She was treated with acyclovir orally and her symptoms improved in a week. On followup, diplopia had markedly improved as well as ataxia, facial pain and rash.

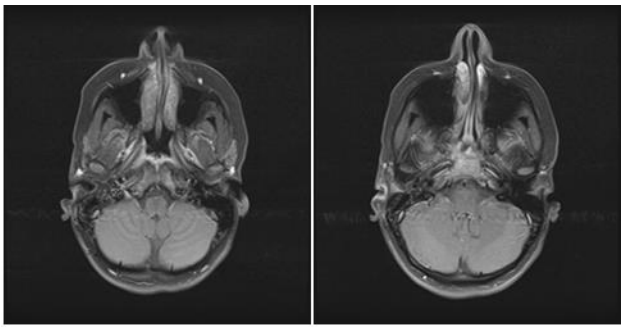

Figure 1: T1weighted image with contrast showing contrast enhancement of the 8th nerve

Discussion

Herpes zoster virus is a DNA virus, commonly transmitted through direct spread and hematogenous route. It commonly affects the children and cause meningoencephalitis resulting in vasculitic arteriopathy and as acute cerebellitis. Besides CNS it also affects various systems of the body and may cause disseminated systemic infection. Sometimes infection may be subclinical and virus may become dormant in the dermatomes of cranial and spinal nerves. Reactivation of the virus commonly occurs in the immune deficiency states like HIV,hematological malignancies and use of immunosuppressant drugs[3]. It commonly affects the thoracic dermatomes followed by lumbar and cervical dermatomes causing pain and rash. It can affect the myotomes resulting in atrophy and weakness and can cause urine and bowl incontinence by affecting the sacral dermatomes. Cranial nerves most commonly affected are trigeminal nerve and facial nerve where it causes auricular rash and lower motor neuron facial palsy. Other cranial nerves affected include 3rd,4th,6th,8thand lower cranial nerves have been reported rarely.The reported incidence of extraocular muscle palsies has ranged between 7% and 31%[4]. In some reported cases there were multiple cranial neuropathies without the signs of meningoencephalitis and various mechanism have been proposed which include cytopathic/allergic, occlusive vasculitis and myositic processes affecting eye muscles. CSF usually shows lymphocytic pleocytosis with mildly raised proteins, however monocytic or neutrophilic picture can also be seen. According to a case series of VZV patients, Lymphocytic predominance was observed in 52% of patient cases, while monocytes predominated in 26% and neutrophils in 22%[5]. Brain imaging may show contrast enhancement of the affected nerves[6]. Investigations are needed to rule out immune deficiency states especially HIV infections and immune malignancies[7]. It is mostly self-limiting condition and improves significantly within 2 months in many cases[8]. However, it has been reported that the duration of diplopia can vary from 2 to 23 months[8]. Long term squeal include post herpetic neuralgias and atrophy involving the distribution of affected nerves, systemic involvement may occur with wide spread dissemination. Early treatment with antivirals may hasten the recovery and prevent complications[7].

Conclusion

Herpes zoster virus infection should be suspected in patients presenting with multiple cranial neuropathies, especially in immunocompromised patients. Early treatment with antiviral medications help to treat and prevent complications of herpes zoster viral infection

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