HERPES ZOSTER INDUCED CEREBELLAR ATAXIA- A CASE REPORT 
AND POSSIBLE RELATIONSHIP BETWEEN KELOIDS AND 
VARICELLA VIRUS

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ABSTRACT
Varicella Zoster virus (VZV) has 2 expressions of the same clinical spectrum- Varicella (Chickenpox) and Herpes Zoster (Shingles). Here two clinically separate manifestations of VZV are reported, one having a Cerebellar ataxia post Herpes Zoster and the other is a Keloid post Varicella. Both were treated with Aciclovir and responded favorably with complete remission.

Key Words: Varicella Zoster Virus, Varicella, Herpes Zoster, Cerebellar Ataxia, Keloid, Aciclovir

INTRODUCTION
VZV has two clinically distinct diseases. Primary infection results in Varicella (Chickenpox), a fairly ubiquitous and contagious acute infection, usually as epidemics in Pre School and school children, characterized by generalized vesicular rash (John, 2002). Post primary infection, VZV establishes latency in the dorsal and spinal root ganglia. In conditions of stress and Immunosuppression, there is reactivation of this latent virus in the form of Herpes Zoster (HZ) characterized by neuralgic pain with or without cutaneous manifestations (figure 1 gives an outline of the complications and progress of VZV). Most typical manifestations can be easily diagnosed, but, few atypical cases can be clinically challenging and taxing on the patient. Two such less common case reports are being discussed, that occur both in immunocompetent and immunocompromised individuals.

CASES
Case Report 1
A 70 year old male with severe pain in the right upper back of shoulder and neck was admitted in the Medicine ward for further investigations. Three weeks later, he developed difficulty balancing while walking, illegible speech and clumsy movements of the right hand.

Examination
Patient was afebrile but in pain. Tender erythematous vesicular rash was noted along the right C4-C5 dermatomal distribution of his back.

CNS
Conscious and oriented. Fine motor showed intention tremors. Right sided knee jerk was pendular. He had Staccato (Scanning) speech and Drunken gait. Gross incoordination was noticed on performing Finger- nose test and Rapid Alternating Movements of right hand.

A provisional diagnosis of HZ Cerebellar ataxia was made to investigate further.

Investigations
CSF studies were normal without any increase in IgG or lymphocytosis. However, MRI angiography of the brain showed evidence of Granulomatous angiitis.

Treatment
Patient recovered completely with 800 mg of Aciclovir 5 times daily for 10 days. MRI 2 months later showed no trace of Granulomatous angiitis.
Case Report

Discussion
HZ Cerebellar ataxia is an extremely rare condition. Ataxia occurs in 1 in 4000 individuals with varicella (Underwood, 1935; Choo et al., 1995). The pathogenesis of this condition is poorly understood because of the low pathological studies and low mortalities (Boughton, 1966). A similar case was reported by Keswani et al., (1993) investigations (MRI brain) in both these cases were consistent with Granulomatous angiitis which may have resulted in focal neurologic deficit. Though the role of anti viral therapy is not fully understood in these cases, the fact that Cerebellar ataxia remitted completely with treatment suggests that HZ may have a direct role in the vasculitic reaction. While documented evidence is still small, a vasculitic reaction is a known complication of HZ and hopefully with the advent of widespread VZV vaccination, the incidence of such cases would remain restricted.

Case Report 2
A 14 year old male with excrutiating pain in his left arm, forearm and wrist progressing over a week visited the Medicine OPD. The pain and swelling aggravated when his mother applied hot fomentation for symptomatic relief 2 days ago. The pain went from 4/10 to 7-8/10 in intensity. The patient also noticed reddish itchy scars on both knees after a previous episode of chickenpox 5 years ago.

Examination
Patient was afebrile with hand held in supine and flexed at the elbow. Any active or passive movements were painful. The medial aspect of left arm, forearm and wrist was erythematous and warm to touch to. Pain was present along the C8 dermatomal distribution of the left arm, forearm and wrist. A keloid had formed over both knees and no progression was noted over time. Interestingly NO vesicular lesions were found on the left arm, forearm or wrist. A provisional diagnosis of HZ reactivation was made.

Investigation
X-ray of the left hand was WNL, CBC and ESR was WNL.

Treatment
A therapeutic trial of Aciclovir orally and topical application was initiated with concurrent Prednisone 60 mg tapered weekly to 15 mg in 3 weeks. The pain and erythema subsided, and interestingly even the keloid started regressing with the above treatment. Eventually over a few weeks, the keloids became flat and the red color faded.

Figure 1: Complications of Varicella Zoster virus
Case Report

Discussion

HZ reactivation is known to occur along the dermatomal distribution, but commonly presents with vesicular eruptions. Lesions without vesicular rash with neuralgia are called Zoster Sine Herpete (Dan Longo et al., 2011). This is an uncommon occurrence, the incidence of which in the general population remains to be determined. While the mechanism of recurrence is not understood clearly, the use of VZV vaccines can significantly reduce this complication.

Keloid is a benign proliferative type of scar tissue. The pathogenesis for keloid formation is not well understood. Though anti-fibroblast antibodies are theorized to be responsible for it, there is no outstanding evidence as yet (Ozge Keseroglu and Meltem Onder, 2008). Such a Keloid formation over an unrelated, already healed lesion is called “Wolf’s Isotopic Response (Wolf et al., 1995)”. HZ is considered the most common preceding condition for this Isotopic response. The pathogenesis for this response is not clearly understood, but VZV DNA was found only in early (less than a month) Isotopic reactions. Hence, VZV may not be related directly to keloid formation (Ozge Keseroglu and Meltem Onder, 2008). Though the mechanism is poorly understood, there are instances where Keloids are formed over HZ lesions. The duration between HZ and keloid formation, degree and intervention required to regress vary largely. It may also be possible that the coincidence of regression of keloids and anti viral therapy in this case were merely coincidence, but the duration and the promptness with which the keloids regressed after the anti viral therapy is impressive. More research is probably required which may also help us theorize the effect of VZV on tissues and get a clearer picture on the pathogenesis and mechanism of action of the virus.

REFERENCES


