ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 10 | October - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE REPORT ON VASCULOPATHY SECONDARY TO VARICELLA ZOSTER VIRUS INFECTION IN A NEWLY DIAGNOSED CASE OF RETROVIRAL DISEASE

Internal Medicine

Dr Jagruti Palav* Junior Resident II. *Corresponding Author
Dr Sachin Bangar Neurologist And Unit Incharge.
Dr Neeti Battalwar Junior Resident I.
Dr Vishal Jagdale Junior Resident I.

ABSTRACT

Varicella zoster virus (VZV) is a ubiquitous, exclusively human alpha herpes virus that produces varicella then becomes latent in ganglionic neurons. VZV is commonly known for causing chicken pox, a disease characterized by fever with maculopapulovesicular rash. In elderly and immunocompromised individuals, VZV gets reactivated and typically produces herpes zoster commonly known as shingles. Varicella zoster virus (VZV) infection of intra- and extracranial arteries (VZV vasculopathy) may be associated with a granulomatous vasculitis characterized by vessel wall damage and transmural inflammation, with multinucleated giant cells and/or epithelioid macrophages. VZV vasculopathy has previously been called granulomatous angiitis, VZV vasculitis, or post-varicella arteriopathy. Here we present a case of a 25 years old female who presented with paraparesis with newly diagnosed retroviral disease.

KEYWORDS

Varicella zoster virus, chicken pox, herpes zoster, VZV vasculitis, post-varicella arteriopathy, immunocompromised individuals, retroviral disease.

INTRODUCTION

Varicella zoster virus is one of the nine human herpes viruses, HH3 to be specific which causes a infection characterized by fever with rash which is pruritic and evolves into dried crusts over a period of 3-7 days period. This infection is often self-limiting; however, the virus remains dormant in the cranial nerve root ganglia. The elderly and the immunocompromised individuals are prone to the reactivation of this dormant virus due to a decline in VZV specific cell mediated immunity, resulting in a painful, limited to a single dermatome, papulo-vesicular eruption (often multidermatomal in the immunocompromised) which is serpentine in pattern. Another life-threatening complication of VZV infection is VZV vasculopathy. The clinical spectrum of VZV vasculopathy ranges from TIAs, ischemic and hemorrhagic stroke to multifocal VZV vasculopathy with temporal arteritis, extracranial vasculopathy, aneurysm with or without SAH, arterial dissection, ischemic cranial neuropathies, CVST, spinal cord infarction and peripheral thrombotic disease.

CASE REPORT

We present a case of a 25 years old female brought to our tertiary health care center by her relatives with complaints of bilateral lower limb weakness since 8 days and diminution of vision in the right eye since 3 days before presenting to the hospital.

She had no history of altered sensorium, loss of consciousness, head injury, seizure disorder, any known cardiac illness. She was not a known case of diabetes mellitus, hypertension.

The patient's past history was significant for varicella zoster infection 2 months prior to the presentation with the aforementioned symptoms.

The parents of the patient had succumbed due to the complications of human immunodeficiency virus (HIV), the details of which could not be elicited from the accompanying relatives.

The relatives gave an ambiguous history about the screening of the patient for HIV. No prior screening or registration had been done.

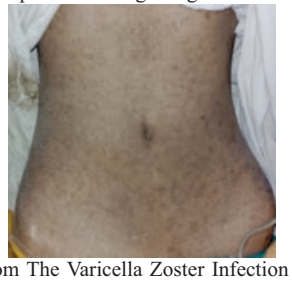

Fig 1: Scars From The Varicella Zoster Infection Rash The Patient Suffered From 2 Months Prior To The Presentation.

On clinical examination, the patient was laying in a supine position, conscious, oriented to time, place and person, obeying given commands, afebrile with a lean built. Vitally the patient had a pulse rate of 120/min, regularly regular, moderate volume, no radio radial or radio femoral delay, no pulse apex deficit with all peripheral pulses palpable. The blood pressure of the patient was 100/70 mm Hg and the SpO2 was 99% on room air. The patient had no pallor, icterus, clubbing, cyanosis, oedema or lymphadenopathy. The patient showed small, numerous scars generalized over her body, more so on her abdomen from the varicella zoster infection.

Systemic Examination

Cardiovascular System -

Both heart sounds were heard with no audible murmurs.

Respiratory System –

Breath sounds were equally heard on bilateral lung fields with no abnormal breath sounds.

Per Abdominal Examination –

On inspection, the abdomen was scaphoid with scar marks of prior varicella zoster infection, Soft on palpation with no organomegaly. No tenderness or guarding or rigidity was present. Bowel sounds were present.

Neurological Examination –

The patient was conscious, cooperative and oriented to time, place and person. Higher mental functions were normal. Speech was normal. The patient had bowel and bladder incontinence since a day before. Power in bilateral upper limbs was 5/5 but bilateral lower limbs was 2/5 – the patient was unable to move her lower limbs against gravity. The tone in both upper limbs was normal but spastic hypertonia was observed in bilateral lower limbs. The deep tendon reflexes of upper limbs were normal and of the lower limbs were brisk. The plantar reflex bilaterally was extensor. The patient could experience the touch and pain sensations in the bilateral upper limbs and lower limbs. Bilateral pupils were reactive to light. Neck rigidity was found to be present with presence of Kernig's and Brudzinski's sign.

INVESTIGATIONS

On ECG the heart rate was 120/min with normal axis and no significant ST-T segment changes with poor R wave progression. Chest Xray of the patient revealed no significant findings. Fundus examination of the patient showed no significant findings. The patient had blurred vision with finger counting upto 1 metre distance in both eyes. On urine routine and microscopy examination +1 albuminuria was present along with 8-10 pus cells/hpf and bacteria were present. Hemoglobin – 12.2 g/dL; TLC – 6,600/cumm; RBC – 4.45 x 10^6/cumm; Platelet count – 2.17 x 10^6/cumm; hematocrit – 32.2%; mean corpuscular volume – 80.6 fL; urea – 63 mg/dL; serum creatinine – 1.2 mg/dL; Na –

148 mEq/L; K – 3.7 mEq/L; total bilirubin – 0.5 mg/dL of which direct – 0.4 and indirect 0.1; total protein – 7.6 g/dL of which serum albumin – 3.3 and serum globulin – 4.3.

CSF cytology – 1 ml clear fluid with 40 nucleated cells of which 75% were neutrophils and 25% were lymphocytes.

CSF protein – 115mg/dL and sugar – 57 mg/dL.

MRI brain plain was suggestive of multiple T2/FLAIR hyperintensities involving left corona radiata, bilateral thalami, right gangliocapsular region, midbrain, left caudate nucleus and putamen, cerebral peduncle, pons, bilateral hippocampus, left ventrolateral aspect of medulla, superior aspect of left cerebellar hemisphere and left medial temporal lobe showing diffusion restriction on DWI. On **MRI Brain Contrast study** there is thin ring enhancement seen in left putamen, bilateral thalami, right ganglio-capsular region suggestive of infective etiology more than neoplastic etiology. Focal leptomeningeal enhancement seen in left parietal lobe. **MR Angiography** was within normal limits.

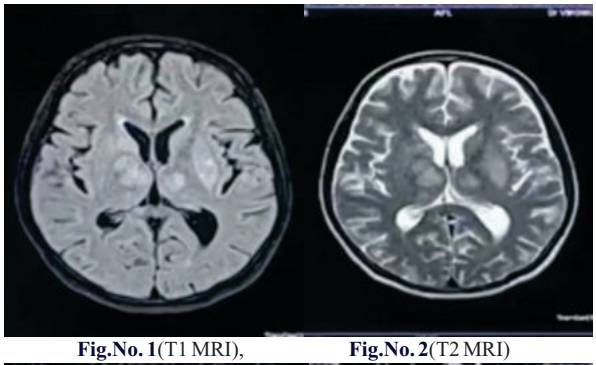

Fig.No. 1(T1 MRI), **Fig.No. 2**(T2 MRI)

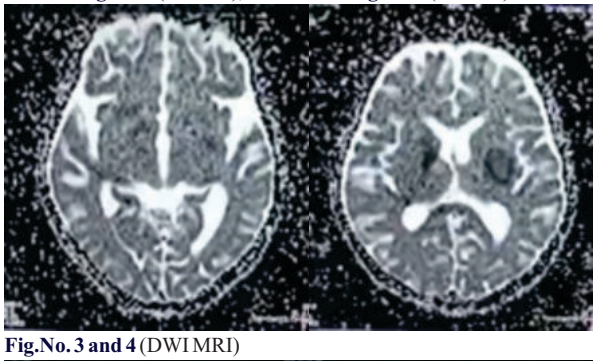

Fig.No. 3 and 4 (DWI MRI)

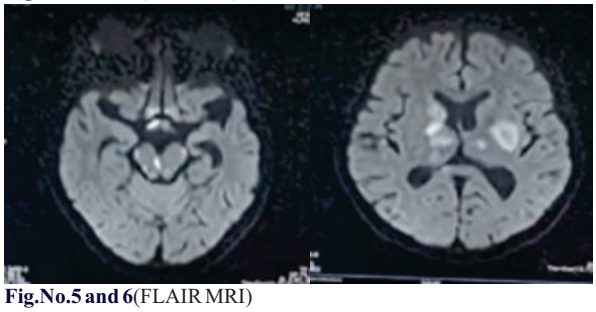

Fig.No.5 and 6(FLAIR MRI)

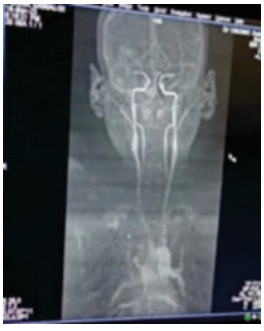

Fig.No. 7(MR angiography)

The patient presented with paraparesis and diminution of vision first started in right eye followed by left eye. She was examined thoroughly for a provisional diagnosis and localization of lesion. After the history and examination, a provisional diagnosis of VZV vasculopathy was made and injection Acyclovir 600 mg IV TDS was initiated along with injection Mannitol 100ml IV TDS and prophylactic antiepileptic cover. She was being conservatively managed when she started deteriorating where she developed upper limb weakness followed by decrease in consciousness level. CSF examination revealed normal sugar levels whereas CSF proteins were raised. To get a final diagnosis, the patient underwent MRI brain plain with contrast and MR Angiography which were suggestive of multiple hyperintensities as shown in images (Fig No. 1-7). Meanwhile, her HIV reports came to be positive for which she was registered and TLD (Tenofovir, Lamivudine, Dolutegravir) regimen was started. After taking advice from the Neurologist of our institute, final diagnosis of VZV Vasculopathy in newly diagnosed RVD made. Her GC further deteriorated with drop in saturation for which she was intubated. Despite of all the measures of stabilizing the patient, she further deteriorated and did not respond to resuscitation and succumbed.

DISCUSSION

Primary infection by VZV causes varicella, followed by establishment of virus latency in cranial nerves, dorsal roots, and autonomic ganglionic neurons. With a decline in VZV specific cell mediated immunity in elderly and immunocompromised individuals, the virus reactivation occurs leading to shingles or herpes zoster, which can be complicated by postherpetic neuralgia. The source of the virus during the reactivation may be trigeminal and autonomic ganglia of the head and the neck and the viruses can travel transaxonally to cerebral arteries where nerves terminate in the adventitia. The deposition of the virus can lead to persistent inflammation that leads to pathological remodeling of the vascular tissue which is believed to cause vasculopathy. VZV vasculopathy should be suspected in the individuals, particularly if immunocompromised, who have had a stroke or aneurysm with

- a recent history of varicella infection
- recurrence of unclear cause with or without rash
- unclear etiology and absence of common risk factors of stroke

The subject of this discussion, the patient was an immunocompromised individual with a recent history of varicella infection with absence of the common stroke risk factors, which lead to an immediate provisional diagnosis of VZV vasculopathy. The treatment of this condition consists of antiviral agents such as Acyclovir and Ganciclovir. Some physicians resort to the usage of intravenous and oral corticosteroids to curb the inflammatory changes but long-term usage of corticosteroids is harmful. Hence, the patient on corticosteroids is observed improvement following which the dose of steroids is gradually tapered off. If during the tapering of the steroid dose, the symptoms worsen, oral antivirals should be added rather than increasing the dose of steroids.

CONCLUSIONS

Though the primary infection caused by VZV is self-limiting, the secondary infection – herpes zoster is distressing and the complications can be fatal. The discovery of occurrence of vascular events such as vasculopathies, infarcts and rarely hemorrhages following VZV infection has opened a new avenue for the treatment and mainly prevention of a significant number of such events. Given our increasing aging population and recognition that VZV reactivation manifesting as zoster is a risk factor for stroke and myocardial infarction, recognition of VZV as a potential cause of vascular disease with or without associated zoster rash is essential to decrease associated morbidity and mortality because VZV vasculopathy can be treated with antiviral therapy.

REFERENCES:

[1] *Varicella zoster virus vasculopathy, The journal of infectious diseases, volume 218, 1 November 2018.*

[2] *Update on varicella zoster virus vasculopathy, Curr Infect Dis Rep 2014 June.*

[3] *Harrison's Principles of Internal Medicine, Twenty-First Edition (Vol.1 & Vol.2) 21st Edition.*

[4] *Muna Ba'Omar,a,⁎ Shabnam Chhetri,a Nenad Pandak,a Faryal Khamis,a Zakariya Al-Balushi,a Aliaa Al-Hajri,b Hend Abouelhamd,c and Zakariya Al-Fahdia Varicella Zoster Virus Vasculopathy; An HIV adult presenting with multiple strokes.*