VZV Vasculopathy: A Case Report

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Abstract: Varicella Zoster Virus (VZV) infection can lead to a variety of neurological and systemic complications. Here we report a 58-year-old man who presented with neurological complications involving central and peripheral nervous system. The diagnosis of VZV Vasculopathy in this case was made with CSF examination and treated with anti-viral medication. [Patel A Natl J Integr Res Med, 2020; 11(5):79-80]

Key Words: Vasculopathy, Varicella Zoster Virus, complications, stroke

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Introduction: Stroke is one of the leading causes of mortality and morbidity in the world. Apart from traditional atherosclerotic vascular various risk factors like hypertension, diabetes, hyperlipidemia, atrial fibrillation etc; viral infection is a rare but an important cause of stroke. VZV is one such virus which on reactivation could present with diverse and varying neurological complications. Cerebral vasculopathy is one such complication associated with reactivation of latent virus to Zoster (Shingles)[1].

Case Report: 58-year-old man initially presented with severe right hemiparesis, headache, nausea and vomiting, while being on therapeutic anticoagulation for left arm deep vein thrombosis (DVT) developed as a complication of left brachial plexopathy. CT brain revealed a non-traumatic left frontal lobar intracerebral hemorrhage (ICH) (Fig. A) with subfalcine herniation complicated by bilateral anterior cerebral artery (ACA) distribution stroke. CTA head and neck did not show underlying vascular malformation or vasculopathy.

Few months following his discharge to inpatient rehabilitation, he represented to the hospital with worsening right hemiparesis and development of new left hemiplegia associated with change in the mental status. Neurological examination showed severe abulia, akinetic mutism, severe dysarthria and severe quadriparesis. MRI brain showed acute infarcts involving bilateral thalamocapsular regions (Fig. B), right lentiform nuclei and corona radiata. He denied any history of rash and fever. CSF examination revealed high protein of 72 mg/dl (15-45 mg/dl) and polymerase chain reaction (PCR) was positive for VZV. He was then treated with a combination of IV acyclovir and prednisone with minimal improvement and was subsequently discharged again to rehabilitation center.

Discussion: VZV is a human neurotropic alpha-herpesvirus. Following the primary infection (chicken pox); the virus remains dormant in the ganglionic neurons of central and peripheral nervous systems. Reactivation of the virus results in herpes zoster and could have varying presentations like post-herpetic neuralgia, cranial neuropathy, meningitis, encephalitis, retinal necrosis, cerebrovascular complications like ischemic stroke, intracranial hemorrhages, spinal cord infarct, intracranial cerebral aneurysm, cervical arterial dissections etc.

VZV vasculopathy results from transaxonal spread of the virus to the cerebral blood vessels – small and/or large vessels leading to inflammation and subsequent vascular remodeling; while rash occurs due to transdermal spread of the virus. Cerebral infarction due to VZV vasculoapthy could be unifocal or multifocal, superficial or deep as well as with or without preceding rash[1,2]. Diagnosis is established by demonstration of VZV DNA and/or anti-VZV antibodies in the CSF and is supported by angiographic evidence of vasculopathy.

To summarize, VZV Vasculopathy is an uncommon yet treatable cause of stroke and should be considered among the differential diagnosis despite absence of preceding Zoster-type rash as 37% of patients may never have a rash[3]. Our case reflects the broad spectrum of central nervous system (ischemic stroke and intracranial hemorrhage due to vasculopathy), peripheral nervous system (brachial plexopathy) as well as systemic complications (DVT) secondary to VZV infection without preceding rash or constitutional symptoms[4].

Fig. A: MRI Brain Showing Left Frontal Parasagittal Parenchymal Hemorrhage

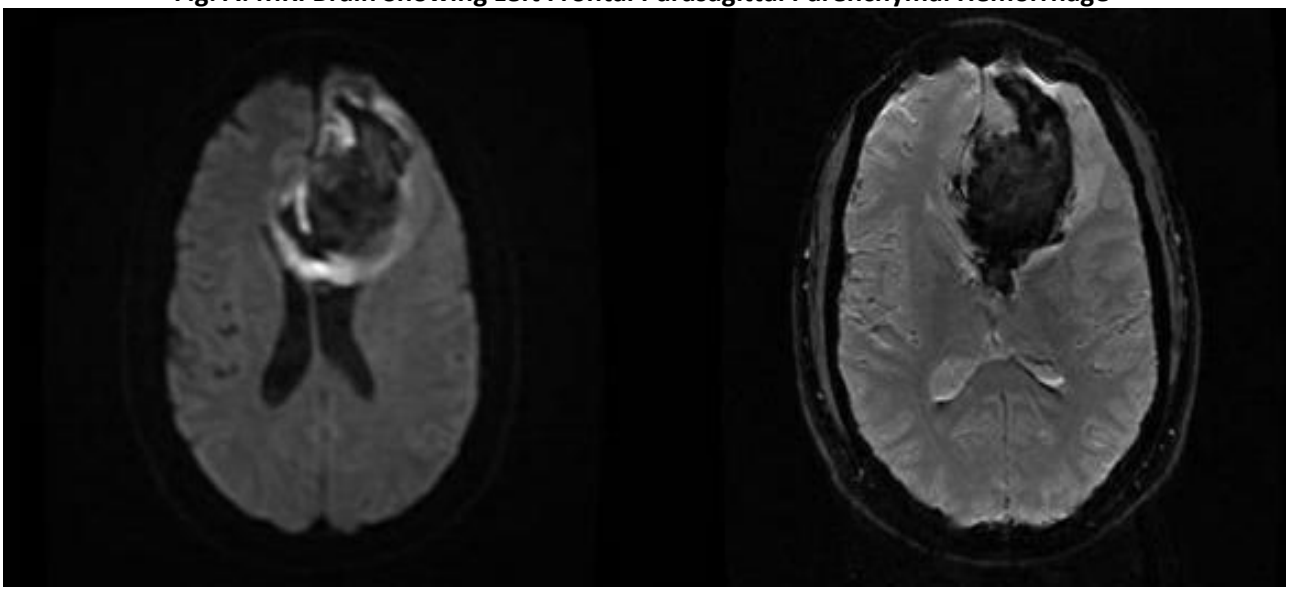
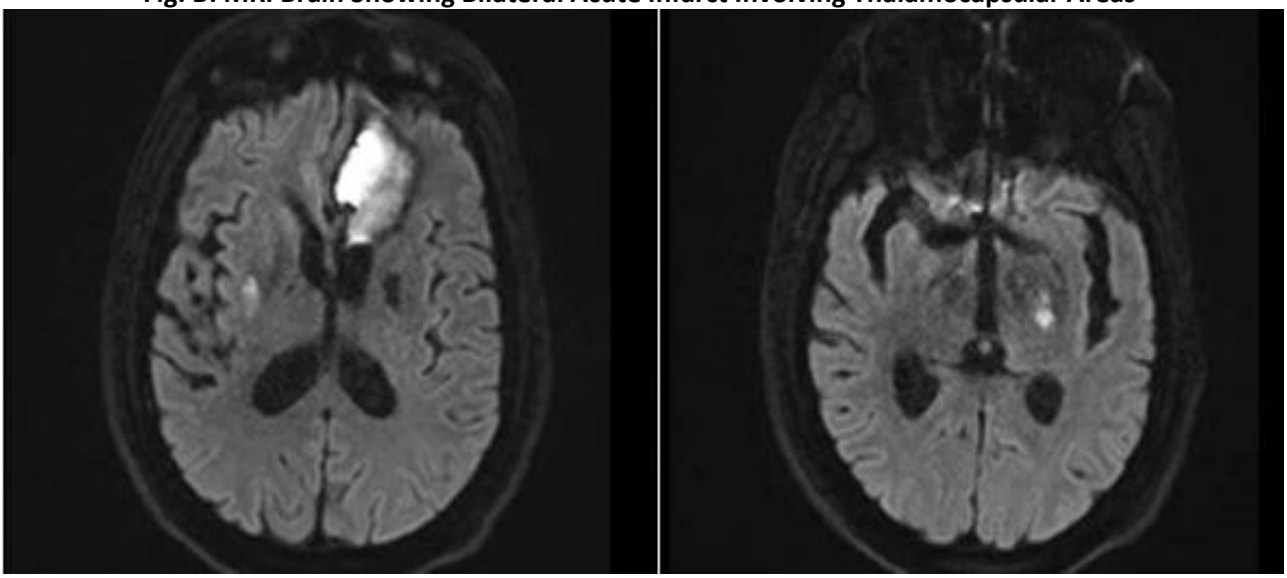


Fig. B: MRI Brain Showing Bilateral Acute Infarct Involving Thalamocapsular Areas



References:

1. Nagel MA, Gilden D. The relationship between herpes zoster and stroke. Curr Neurol Neurosci Rep. 2015;15(4):16. doi:10.1007/s11910-015-0534-4
2. Nagel MA, Bubak AN. Varicella Zoster Virus Vasculopathy. J Infect Dis. 2018;218(suppl_2):S107–S112. doi:10.1093/infdis/jiy425
3. Gilden D, Cohrs RJ, Mahalingam R, Nagel MA. Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, pathogenesis, and treatment. Lancet Neurol. 2009; 8(8):731–740.doi:10.1016/S1474-4422 (09)70134-6.
4. Mohanty B. Deep vein thrombosis: A rare complication of varicella zoster infection. Arch Gen Intern Med. 2017;1(2):1-2

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