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Subarachnoid Hemorrhage Associated with Varicella Meningoencephalitis: A Case Report

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Abstract

Introduction: Varicella zoster virus (VZV) is an alpha herpes virus that can cause vasculopathy by production of viral load in the cerebral arteries. CNS vasculopathy is rare but can be fatal.

Case presentation: A 63 year old male with multiple medical conditions presented with a two week history of herpes zoster rash and developed waxing and waning neurological symptoms. Pt was treated with broad-spectrum antibiotics and acyclovir for meningoencephalitis. He subsequently developed extensive subarachnoid hemorrhage leading to death.

Conclusion: This case illustrates that VZV meningoencephalitis leading to VZV vasculopathy is uncommon and often associated with fatal complications. Early diagnosis and intervention are necessary to prevent serious outcomes

Keywords

subarachnoid hemorrhage; varicella cnsvasculopathy; meningoencephalitis

Introduction

Central nervous system complications caused by the reactivation of the varicella zoster virus (VZV), though reportedly rare, is often severe and can be fatal. The exact incidence of neurological sequelae is difficult to estimate; however, one study has reported 1.8 per 100,000 cases annually. VZV is understood to be the only virus that replicates in the arteries of human beings leading to vasculopathy [1]. VZV vasculopathy can cause ischemic infarction, aneurysm, subarachnoid and cerebral hemorrhage, carotid dissection, and encephalitis. Because VZV vasculopathy is rare and does not always present with typical zoster rash, it can be missed [2]. The overall mortality is low but can be as high as 9% in encephalitis patients [1]. We are reporting this adverse event to illustrate a rare CNS complication associated with VZV, which is subarachnoid hemorrhage after presenting with a zoster rash.

Case Presentation

A 63 year old male with a history of coronary artery disease, end-stage renal disease on peritoneal dialysis, diabetes, chronic obstructive pulmonary disease and prior splenectomy presented with a two day history of worsening generalized weakness and altered mental status. In addition, patient had a two week history of left facial pain and rash that possibly involved the left eye. Erythematous vesicular lesions in various sizes were found on left forehead and around the eye. Patient was alert and oriented to person,

place and time at the time of admission.

Initially, he was treated with broad-spectrum antibiotics and acyclovir for presumptive meningoencephalitis. Ophthalmologist was consulted and confirmed herpes zoster in V1 distribution. No ophthalmologic complications identified. Head computed tomography (CT) without contrast showed no evidence of an acute intracranial process. Cerebrospinal fluid (CSF) was obtained within 24 hours of admission and showed evidence of VZV (table 1). His mental status then began to wax and wane.

A repeat head CT without contrast was performed to investigate his delirium and episodes of disorientation and confusion. It showed an extensive subarachnoid hemorrhage. Neurosurgery and neurology were consulted and believed that his subarachnoid hemorrhage was due to varicella meningoencephalitis. The brain magnetic resonance imaging (MRI) revealed a significant subarachnoid hemorrhage and parenchymal hematomas over a large area of the right and left temporal lobes. Brain MRA was obtained to evaluate AVM and aneurysms, but it showed normal intracranial arteries. Due to extensive subarachnoid hemorrhage, the patient's condition deteriorated rapidly leading to coma and death, despite care provided by multidisciplinary services.

Discussion

VZV vasculopathy is caused by the production of viral infection in the cerebral arteries. Complications include cerebral aneurysm, subarachnoid and intracerebral hemorrhage, ectasia, and dissection [2]. Without prompt intervention, the outcome can be fatal. The common clinical features include headache, changes in mental status, aphasia, ataxia, hemisenory loss, hemianopia and monocular visual loss [2]. In addition, the neurological symptoms may wax and wane. Zoster rash sometimes precedes the neurological symptoms but not always. Nagel and his colleagues reported at least 40% of vasculopathy patients do not present with typical zoster rash [3].

When patients present with zoster rash and neurological symptoms, further testing is warranted. Brain imaging often reveals abnormalities in the cortical and deep matter, particularly in the grey-white junction. MRI of the brain often shows large and small ischemic or hemorrhagic infarcts in the cortical and subcortical grey and white matter [5]. With regards to CSF analysis, mild pleocytosis (less than < 100 cells, dominantly mononuclear) with a normal or elevated CSF protein and normal glucose level are seen in the majority of patients with VZV vasculopathy [2]. The characteristic findings in CSF are similar to aseptic meningitis. One study has shown that at least 33% of vasculopathy patients do not have pleocytosis in the CSF [3]. Therefore, absence of pleocytosis does not exclude the diagnosis of VZV vasculopathy.

Virological confirmation is necessary to confirm the diagnosis. Several studies have shown that VZV antibody in CSF is much more sensitive in diagnosing vasculopathy compared to PCR VZV DNA [3]. This can be explained by its protracted clinical course, averaging 4.2 months from the onset of neurologic symptoms to virologic analysis [3]. VZV DNA was undetectable 14 days after the onset of neurologic symptoms while anti-VZV IgG antibody wasstill found in the CSF. The sensitivity of anti-VZV IgG antibody is 93.3% compared to 30% in PCR DNA. Study done by Person and her colleagues demonstrated that quantitative PCR is more sensitive in detecting VZV than compared to qualitative PCR [1]. However, a gold standard to diagnose VZV vasculopathy has not yet been established.

Conclusion

There have been rare reports of subarachnoid hemorrhage complicating varicella-zoster viral encephalitis. One report in 2008 by M paka et al in Greece described such complication in an immunocompetent adult with no significant medical history. In this report, the patient has no zoster rash and had diffuse multifocal hemorrhagic lesions. These were atypical features in an immunocompetent adult as they usually present with rash and focal large vessel arterial disease resulting in focal neurological deficits similar to stroke [6]. In contrast, our patient is clearly immunocompromised due to his end stage renal disease. Because of his immunodeficiency, his encephalitis affected mainly the diffuse small vessels of his brain. The brain imaging showed multiple bilateral hemorrhagic lesions which were consistent with the small vessel vasculopathy seen in immunocompromised patients [6].

VZV vasculopathy is rare and can be chronic in nature. Thus, clinicians should have a high index of suspicion in adults who present with neurological symptoms whether or not the zoster rash is present. Brain MRI and anti-VZV IgG antibody in the CSF should be obtained to evaluate for VZV vasculopathy. Early suspicion and diagnosis can lead to valuable intervention and help prevent fatal outcomes. Additional studies are needed in the area of diagnosis VZV vasculopathy and its antiviral treatments.

Table

Table 1: Cerebrospinal fluid analysis

Hematology	***Chemistry***	***Serology***	***Microbiology***
WBC 195 ref range 0 - 5	Glucose 90 (ref range 40 – 70)	Varicella zoster PCR - detected	AFB - NEG
RBC 260 ref range 0 - 1	Protein 72 (ref range 15 – 45)	Crytococcus antigen - NEG	Mycology culture - NEG
Neut 12%		HSV1/HSV2 DNA – not detected	Bacterial Culture - NEG
Lymph 30% Monos/Macro 58% Ref range: Lymphocytes 60 -70% Monocytes/mac 28-30% Others 1 to 2%		Bacterial AG: Group B strep - NEG H. Influenza - NEG N. Meningitidis – NEG S. Pneumonia - NEG	
		Bartonella Henselae AB, IgM < 1:20 Bartonella Henselae AB, IgG < 1:64 Bartonella Quintana AB, IgM < 1:20 Bartonella Quintana AB, IgG < 1:64	

HSV – herpes simplex virus, NEG – negative result

Figures

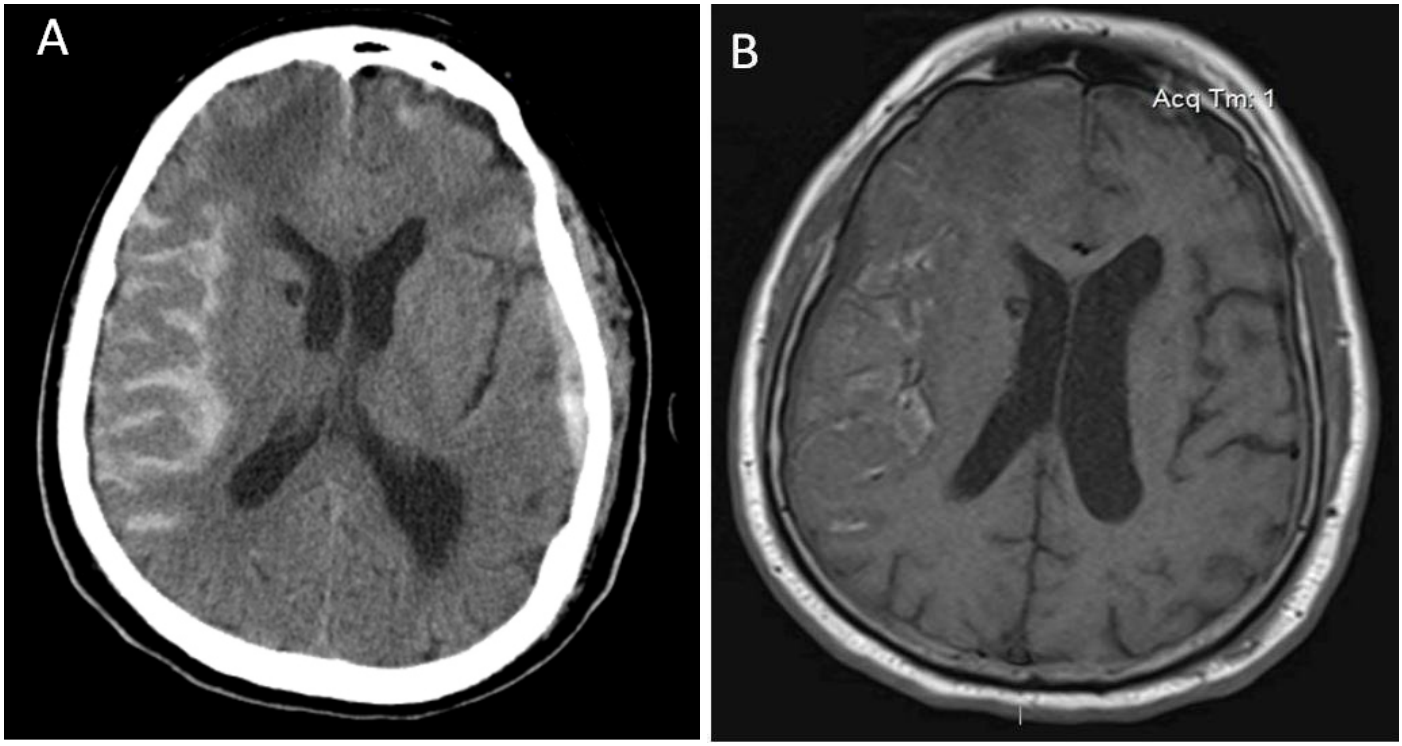

Figure 1: A: Head CT w/o contrast reveals subarachnoid hemorrhage in the parietotemporal region. **B**: Brain MRI shows a large subarachnoid hemorrhage and parenchymal hematomas over a large area of the right and left temporal lobes.

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