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Behavioural Shift as a Subtle Mental Change Occurring in a Case of Rheumatoid Arthritis Experiencing Zoster Encephalitis

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Abstract

Primary varicella (chickenpox) and herpes zoster (shingles) are caused by the highly contagious varicella zoster virus (VZV). Due to underlying immunological dysfunction, people with rheumatoid arthritis (RA) may be more vulnerable to serious infection. A 52-year-old woman with rheumatoid arthritis, who was receiving hydroxychloroquine and sulfasalazine, is the subject of our case study. She had a broad vesicular rash, subtle cognitive difficulties, and VZV hepatitis, a known complication of disseminated infection. Through positive immunoglobulin M (IgM), immunoglobulin G (IgG), and polymerase chain reaction (PCR) detection of VZV deoxyribonucleic acid (DNA) in blood and cerebrospinal fluid (CSF), laboratory studies verified the spread of VZV. Our case highlights behavioral shift as a unique presentation of VZV encephalopathy. An interdisciplinary approach continues to be important in the management of such complicated cases, with timely interventions and better outcomes.

Keywords

Cognitive Change, Behavioural Shift, Encephalitis, Varicella Zoster, Emergency Department

1. Introduction

Varicella-zoster virus (VZV) is an extremely contagious, neurotropic human herpes virus that causes primary varicella (chickenpox) and herpes zoster (shingles). Although varicella is in most cases a mild, self-limiting illness in immune-competent humans, reactivation of VZV in the immune-compromised patient is associ-

ated with possible severe complications like disseminated infection, encephalitis, and hepatitis. Disseminated VZV is defined as extensive cutaneous lesions outside of the normal dermatome pattern plus visceral organ participation, necessitating prompt medical consultation [1].

It has been postulated that defects in innate and adaptive immunity due to RA could increase susceptibility to viral reactivation [2]. VZV encephalitis is a severe, but rare complication of disseminated VZV infection. Although mental changes in VZV encephalopathy are usually a given (eg., confusion, seizures, altered consciousness, memory loss, speech problems, and focal neurologic deficits), it is still relatively rare for such patients to manifest subtle behavioral and cognitive shifts (like emotional labiality, personality shifts, and disorganized thinking) as the only presentation to the Emergency Department [3] [4].

This case report underscores the need to have a high index of suspicion for disseminated VZV in immune-compromised hosts with unusual symptoms. Early diagnosis, aggressive antiviral therapy, and an interdisciplinary treatment approach are critical in treating this potentially lethal disease.

2. Case Presentation

A 52-year-old woman came to the Emergency Department (ED) complaining of fever, malaise, and a generalized vesicular rash that began on her face, and quickly progressed to her trunk and extremities within a few days. She complained of severe headaches; subtle abnormal behaviour with involuntary movement of hands and lips, with intermittent agitation were noted by the husband.

Her past medical history was notable for rheumatoid arthritis (RA), for which she had been treated with hydroxychloroquine and sulfasalazine for many years. She had no history of herpes zoster, or varicella vaccination. There was no history of recent exposure to people with active varicella infection, or herpes zoster.

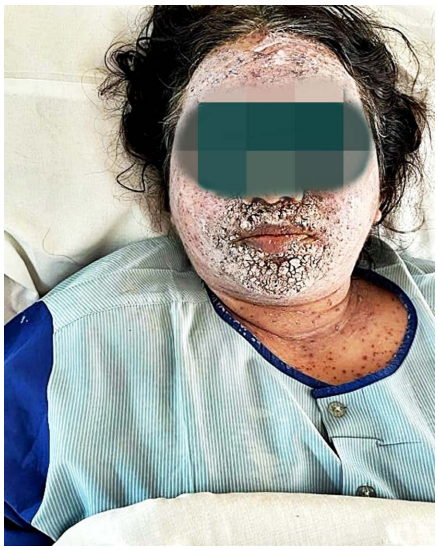

Figure 1. Vesicular lesions in different stages of healing over the face.

On physical exam, she was febrile (38.7˚C), disoriented to place and time, and had scattered vesicular lesions at different stages of healing on her body; including the face, trunk, and limbs (Figure 1, Figure 2). Neurological exam showed mild neck stiffness, but no focal deficit. She was conscious and interacting with the physicians, however, she was unable to answer beyond simple questions, or perform complex tasks. Her Glasgow Coma Scale was E4V4M6. The Mini Mental State Exam score was 22. The abdomen was tender in the right upper quadrant, and scleral icterus was present.

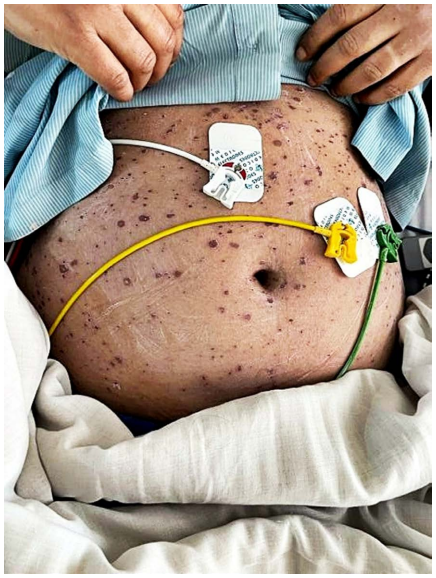

Figure 2. Healed scabs over the trunk.

She was admitted for further evaluation, and initial laboratory (Table 1) and imaging studies were ordered to assess the extent of systemic involvement, and confirm the diagnosis of disseminated VZV infection. Given her altered mental status and immune-compromised state, a lumbar puncture, and neuroimaging were performed.

Table 1. Relevant laboratory results including blood and cerebrospinal fluid analysis.

No.	Laboratory Test	Result	Reference Range
1.	Varicella Zoster (VZV) IgM	> 2.30 ISR	< 0.90 - 1.1 ISR
2.	Varicella Zoster (VZV) IgG	695 mIU/ml	< 50 - 100 mIU/ml
3.	VZV DNA (in blood)	Positive	Qualitative
4.	VZV DNA (in spinal fluid)	Positive	Qualitative
5.	Alanine aminotransferase	303 U/L	7 - 56 U/L
6.	Aspartate aminotransferase	249 U/L	8 - 45 U/L
7.	Alkaline phosphatase	231 U/L	44 - 147 U/L
8.	Gamma-glutamyl transferase	406 U/L	5 - 40 U/L

3. Management and Outcome

The patient was started on intravenous acyclovir 10 mg/kg every eight hours to treat the VZV infection. Supportive care, such as intravenous fluids, electrolyte balance, and antipyretics, was initiated. Due to her hepatic involvement, liver function tests were monitored closely, and a hepatology consultation was obtained. She was also initiated on prophylactic proton-pump inhibitors to avoid stress-related mucosal injury.

Neurology and infectious diseases teams took care of her, because there was fear of VZV encephalitis. Lumbar puncture was performed, with findings of cerebrospinal fluid analysis as described (Table 2); PCR detected the presence of VZV DNA in cerebrospinal fluid. Brain magnetic resonance imaging (MRI) did not show hemorrhage or ischemia.

Table 2. General examination of cerebrospinal fluid.

General Examination (CSF)	Result	Reference Range
Volume	10 ml	
Appearance	Clear	
Colour	Colourless	
Clot/Clot Web	Absent	
pH	8.5	7.35 - 7.44
Chemical Examination		
COB - WEB	Absent	
CSF Sugar	70.5	40 - 80
CSF Protein	33.8	20 - 40
Microscopic examination		
Erythrocytes	No cells seen	
Nucleated cells	No cells seen	
Polymorphs	No cells seen	
Lymphocyte	No cells seen	

During her hospitalization, her mental status normalized progressively, with crusting of the vesicular lesions. Her liver function tests became normal within two weeks, and she was hemodynamically stable. As per standard local protocol, she was switched to oral valacyclovir after 14 days of intravenous acyclovir for an additional 10-day course.

4. Discussion

Disseminated VZV is a severe disease primarily seen in immune-compromised hosts, such as patients with rheumatoid arthritis, human immunodeficiency virus infection, cancer, or those on immunosuppressive therapy. Below, we have sum-

marized the potential mechanisms correlating rheumatoid arthritis [3] to an immune-compromised state (Table 3).

Table 3. Mechanisms resulting in an immune-compromised state in rheumatoid arthritis.

Mechanism	Effect on Immune Status
Chronic autoimmunity	Immune dysregulation and altered host defence
Intrinsic immune defects	Impaired cell-mediated responses
Comorbidities & lifestyle	Additional infection risk factors
Immunosuppressive therapy	Iatrogenic immune-compromise
Clinical outcomes	Elevated risk of infections

VZV encephalitis is the most dreaded of disseminated VZV complications, as it can cause severe neurological impairment and high mortality, if left untreated. It also occurs primarily in elderly or immune-compromised patients. The diagnosis is often delayed; risk factors for unfavourable outcome are age, cerebral vasculitis, and Glasgow Coma Scale score < 15 [5]. The mechanism of pathogenesis includes viral reactivation in the dorsal root ganglia with subsequent spread to the central nervous system. This leads to inflammation, neuronal damage, and, in some instances, vasculopathy causing strokes [4].

Psychiatric symptoms, seen as subtle mental changes in our patient, can be associated with systemic and central nervous system infection and they can be the initial presenting symptoms, occurring in the absence of neurological symptoms. A 2001 study done by Caroff *et al.* reported delusions, hallucinations and affective disorders as the initial symptoms, although it is possible that the hallucinations can be attributed to the febrile nature of the illness [5]. According to a comprehensive review by Hokkanen L *et al.* most patients with cognitive decline due to Herpes Zoster encephalitis eventually returned to their prior cognitive status provided they received timely treatment. However, persisting cognitive symptoms such as memory impairment, dysphasia, difficulty in interpreting proverbs, and, in some cases, dementia have been reported. Neuropsychiatric symptoms such as psychosis, irritability and affect labiality ranging from euphoria to dysphoria have frequently been described both at the acute stage and as residual symptoms [6]. In our case, early detection and vigorous antiviral treatment avoided severe neurologic deterioration.

Untreated VZV encephalitis carries a high mortality rate, stressing the urgency of early treatment. According to a study by Venkatesan *et al.* (2013), systemic treatment of encephalitis in the form of early empirical antiviral therapy until confirmatory test results are available is advocated [7]. Our case followed suit, illustrating the urgency of an early initiation of acyclovir. The role of adjunctive therapy in disseminated VZV is uncertain. Although corticosteroids have been found to decrease inflammation in some viral encephalitis, corticosteroid use in VZV is controversial secondary to the fear of exacerbating viral replication [8]. In some

cases, intravenous immunoglobulin (IVIG) has been suggested to benefit immune-compromised patients with fulminant VZV infections, although this is based on limited studies (Merck Manual) [9].

Vaccination is the most effective measure against VZV complications. The recombinant zoster vaccine (RZV) has been effective in preventing herpes zoster and its complications, even in immune-compromised patients [10]. Our patient was recommended to get vaccinated after recovery, to minimize the chances of reactivation of VZV in the future.

5. Conclusion

This case highlights the need to consider disseminated varicella zoster virus (VZV) infection as a serious complication in immune-compromised patients, including those with rheumatoid arthritis (RA). Although not on a biologic immunosuppressant, our patient showed profound immune dysfunction, which emphasizes the need for increased awareness in such patients. Early detection and timely institution of antiviral therapy with intravenous acyclovir were important in avoiding further complications, especially encephalitis and hepatic impairment.

Key Takeaways

- Immune dysregulation in rheumatoid arthritis, even if not an active immunesuppression, poses a significant risk in the development of varicella zoster dissemination.
- Subtle behavioural shifts and cognitive difficulties can often be the only presenting symptoms hinting towards VZV encephalopathy.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. Patient confidentiality and anonymity have been maintained.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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List of Abbreviations

VZV	Varicella Zoster Virus
RA	Rheumatoid Arthritis
IgM	Immunoglobulin M
IgG	Immunoglobulin G
PCR	Polymerase Chain Reaction
DNA	Deoxyribonucleic Acid
CSF	Cerebrospinal Fluid
MRI	Magnetic Resonance Imaging
ED	Emergency Department
IVIG	Intravenous Immunoglobulin
RZV	Recombinant Zoster Vaccine