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CASE REPORT

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Fulminant hepatitis and multi-organ failure following varicella zoster virus infection in an adult with G6PD deficiency: a case report

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Abstract

Background Varicella zoster virus (VZV) infection in adults can lead to severe complications, particularly in those with underlying comorbidities. This case report presents a rare instance of VZV-associated fulminant hepatitis progressing to multi-organ failure in a 27-year-old female with glucose-6-phosphate dehydrogenase (G6PD) deficiency.

Case presentation The patient presented with a generalized rash and rapidly developed acute liver failure, coagulopathy, and renal dysfunction. Despite aggressive management, including intravenous immunoglobulin therapy and supportive care, the patient's condition deteriorated, ultimately resulting in mortality.

Conclusions This case highlights the potential for severe hepatic complications in adult VZV infections, especially in patients with G6PD deficiency, and underscores the importance of early recognition and aggressive management. It also raises awareness about the risks associated with concomitant medication use, including over-the-counter drugs and herbal supplements, in the context of acute viral infections.

Clinical trial number Not applicable.

Keywords Varicella zoster virus, Hepatitis, G6PD deficiency, Case report

Introduction

Varicella zoster virus (VZV) infection, commonly known as chickenpox, is typically a self-limiting disease in children but can lead to severe complications in adults [1]. While cutaneous manifestations are the hallmark of VZV infection, visceral involvement, particularly hepatitis, is a rare but potentially life-threatening complication [2]. This case report presents an unusual instance of VZV-associated fulminant hepatitis progressing to multi-organ failure in an adult patient with glucose-6-phosphate dehydrogenase (G6PD) deficiency.

Hepatic involvement in VZV infection is uncommon, with an estimated incidence of 0.3–3% in immunocompetent adults [3]. However, when it occurs, it can rapidly progress to acute liver failure, especially in patients

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with underlying risk factors [4]. G6PD deficiency, the most common enzymatic disorder of red blood cells, has been associated with increased susceptibility to oxidative stress and may potentially exacerbate liver injury in the context of viral infections [5]. It is the most common enzymatic disorder of red blood cells, affecting approximately 400 million people worldwide, with a prevalence ranging from 5 to 10% in our country depending on the region and population group [5, 6]. While mortality directly attributable to G6PD deficiency is rare, it is primarily associated with complications such as hemolysis triggered by infections, certain drugs, or foods like fava beans [6]. In the context of infections, individuals with G6PD deficiency are particularly vulnerable to severe oxidative stress and inflammation, which can exacerbate organ damage [7]. However, data on the specific mortality associated with varicella-zoster virus (VZV) in G6PD-deficient patients remain scarce.

The management of VZV-associated hepatitis remains challenging, with limited evidence-based treatment options available [8]. While antiviral therapy is the mainstay of treatment for VZV infections, its efficacy in cases of fulminant hepatitis is not well established [9]. In severe cases, liver transplantation may be the only life-saving option, but the rapid progression of the disease often precludes this intervention [10].

This case report highlights the potential for severe hepatic complications in adult VZV infections, particularly in patients with underlying conditions such as G6PD deficiency. It underscores the importance of early recognition, prompt diagnosis, and aggressive management of VZV-associated hepatitis to prevent progression to multi-organ failure [11]. Furthermore, it raises awareness about the potential risks associated with concomitant medication use, including over the counter and herbal supplements, in the context of acute viral infections [12].

Case presentation

A 27-year-old female presented to our hospital with a chief complaint of generalized reddish skin papules and vesicles that had persisted for 6 days. The patient reported concurrent onset of fever and pruritus, with the rash appearing uniformly across her body. She disclosed recent contact with a family member diagnosed with VZV infection. Initially, the patient sought care from an infectious disease specialist, who prescribed acetaminophen 500 mg twice daily for symptomatic relief of pain and fever, along with acyclovir 800 mg five times daily. Outpatient management was deemed appropriate at that time. However, despite adhering to the prescribed regimen for 6 days, the patient experienced no significant improvement in her symptoms, prompting her visit to our facility. Upon admission, given the severity of her condition and the lack of response to oral antiviral therapy, intravenous acyclovir therapy was initiated immediately at a dose of 10 mg/kg every 8 h. This decision was made in accordance with current guidelines for the management of severe VZV infections.

Upon presentation, the patient reported a new onset of epigastric abdominal pain that had developed over the preceding 48 h. She characterized the pain as constant and non-colicky, without radiation to other abdominal regions. Accompanying her abdominal discomfort, the patient experienced a significant loss of appetite, coupled with nausea and vomiting. Importantly, she denied experiencing diarrhea, or constipation. She also reported no recent consumption of suspect foods or contact with individuals exhibiting similar gastrointestinal symptoms. Specifically, there was no history of ingestion of fava beans, legumes, or other foods known to trigger hemolysis in individuals with G6PD deficiency.

Review of the patient's past medical history revealed glucose-6-phosphate dehydrogenase (G6PD) deficiency diagnosed in childhood but was otherwise unremarkable. She denied any history of diabetes mellitus, hypertension, cardiovascular disease, hepatic disorders, or other chronic medical conditions. The patient had no prior surgical interventions and reported no known allergies to foods, medications, or environmental factors. She affirmed abstinence from tobacco, alcohol, and illicit substances.

The patient's family history was non-contributory, with no reported hereditary or congenital disorders.

The patient reported strict adherence to her prescribed medications. She denied the use of any other prescription medications, over-the-counter drugs, or dietary supplements.

Physical examination revealed a young woman in mild distress. Vital signs were as follows: temperature 38.9 °C, heart rate 92 beats per minute, respiratory rate 18 breaths per minute, blood pressure 118/76 mmHg, and oxygen saturation 98% on room air.

Positive findings included generalized icterus, scleral icterus, conjunctival pallor, mild tenderness in the epigastric region and right upper quadrant, and a diffuse erythematous rash with papules and vesicles in various stages of evolution. The lesions were distributed across the trunk, extremities, face, and scalp.The rest of the systemic examination, including respiratory, cardiovascular, neurological, and musculoskeletal evaluations, was unremarkable (Fig. 1).

These findings, particularly the evidence of jaundice and abdominal tenderness, suggest a potential hepatobiliary involvement that warrants further investigation in the context of the patient's presenting symptoms and history.

Following the initial assessment and physical examination, we proceeded with a comprehensive diagnostic

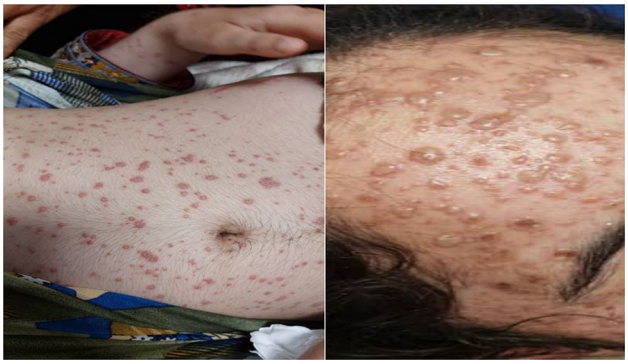

Fig. 1 Papules and Vesicles Spread All Over the Body Due to Varicella Zoster Infection

Table 1 Laboratory examination results at admission

Test	Result	Reference Range	Unit
White Blood Cell Count	14.3	4.5–11.0	x10^9/L
Hemoglobin	11.8	12.0–15.5	g/dL
Platelet Count	142	150–450	x10^9/L
ALT	2001	7–56	U/L
AST	6332	10–40	U/L
ALP	681	44–147	U/L
GGT	180	9–48	U/L
Total Bilirubin	4.2	0.1–1.2	mg/dL
Direct Bilirubin	2.8	0.0–0.3	mg/dL
PT	14.5	11.0–13.5	seconds
INR	1.3	0.8–1.1	ratio
aPTT	48	25–35	seconds
Fibrinogen	269	200–400	mg/dL
Lipase	45	13–60	U/L
Amylase	70	28–100	U/L
HAV IgM	Negative	Negative	-
HBsAg	Negative	Negative	-
Anti-HCV	Negative	Negative	-
HEV (IgM and PCR)	Negative	Negative	-

workup. Laboratory investigations included a complete blood count, liver function tests, coagulation profile, pancreatic enzymes, and viral hepatitis markers. Testing for Hepatitis A, B, C, and E viruses was performed. The results for Hepatitis A, B, and C were negative. Hepatitis E virus (HEV) serology and PCR testing were also conducted and returned negative, ruling out HEV as a contributing factor in this case. This comprehensive testing confirmed that the acute hepatitis was not attributable to other common viral pathogens, further substantiating the diagnosis of VZV-associated fulminant hepatitis. The results of laboratory exams are summarized in Table 1.

An abdominal ultrasound was performed to evaluate the hepatobiliary system. The imaging revealed increased echogenicity of the liver parenchyma with slight enlargement, measuring 16 cm in the midclavicular line. Mild periportal echogenicity was noted, suggesting edema around the portal tracts. The gallbladder appeared normal without evidence of stones or wall thickening, and the common bile duct measured 5 mm in diameter with no signs of obstruction. The pancreas was unremarkable, while the spleen showed mild enlargement at 13 cm in length.

These findings, combining laboratory results and imaging studies, strongly suggest acute hepatitis, likely associated with the recent VZV infection. While drug-induced liver injury is a potential consideration, the minimal dosage of acetaminophen (500 mg twice daily) prescribed to the patient makes this an unlikely contributor to the observed fulminant hepatitis. Instead, the clinical presentation and findings strongly support varicella zoster virus infection as the primary etiology, compounded by the patient's underlying G6PD deficiency.

Following the initial assessment and diagnostic workup, a polymerase chain reaction (PCR) test for VZV was performed. The test revealed a strongly positive result with a high viral load, indicating active and severe VZV infection. This finding confirmed the diagnosis of VZV-associated fulminant hepatitis.

A liver biopsy was also performed to further evaluate the etiology of the acute liver failure. Autoimmune hepatitis (AIH) was considered as a potential differential diagnosis due to the rapid progression of the patient's condition. Although AIH-related serology was not performed due to logistical constraints, histopathological examination of the liver tissue showed no lymphocytic infiltration or features suggestive of AIH, effectively ruling out this diagnosis. The absence of serological and histological markers of autoimmunity, combined with the positive PCR result for VZV, confirmed VZV-associated fulminant hepatitis as the etiology. Histopathological examination of the liver tissue showed extensive hepatocellular necrosis without significant lymphocytic infiltration, granulomas, or features suggestive of autoimmune hepatitis. Additionally, PCR analysis of the liver biopsy specimen confirmed the presence of VZV DNA. Although serum PCR had already established systemic VZV infection, the liver biopsy PCR was performed to directly demonstrate the presence of VZV DNA within the liver tissue. This step was critical in confirming the liver as the site of active viral replication and damage, thereby substantiating the diagnosis of VZV-associated fulminant hepatitis. The absence of histological and serological markers of autoimmunity, combined with the positive PCR result for VZV, excluded autoimmune hepatitis as the underlying cause of liver failure in this patient.

Given the severity of the patient's condition and the potential for infectious complications, she was promptly isolated and admitted to the Intensive Care Unit (ICU) for close monitoring and aggressive management. This decision was made to ensure optimal care and to prevent potential nosocomial spread of the VZV infection.

Upon admission to the ICU, the patient was immediately placed on a comprehensive conservative management protocol. This included the initiation of intravenous acyclovir therapy at a dose of 10 mg/kg every 8 h, in addition to intravenous hydration with balanced crystalloid solutions to maintain adequate fluid balance and support renal function. The decision to escalate to intravenous antiviral therapy was based on the severity of her condition and the lack of response to oral acyclovir therapy during the preceding 6 days. Vital signs were monitored closely, with assessments performed every six hours to detect any early signs of clinical deterioration or hemodynamic instability.

N-acetyl cysteine (NAC) was not administered in this case, as the primary etiology of the acute liver failure was identified as varicella zoster virus (VZV)-associated fulminant hepatitis, compounded by the patient's underlying G6PD deficiency [13]. The prescribed dosage of acetaminophen (500 mg twice daily) was minimal and well below the threshold for hepatotoxicity, making acetaminophen-induced liver injury an unlikely contributor. Additionally, there was no clinical or laboratory evidence of oxidative liver injury that would have necessitated the use of NAC.

A rigorous laboratory monitoring schedule was implemented to track the progression of hepatic dysfunction and assess for potential complications. This included:

1. Complete blood count (CBC) performed daily to monitor for signs of bone marrow suppression or developing pancytopenia.
2. Liver function tests (LFTs) conducted twice daily (BID) to closely track the progression of hepatocellular damage and synthetic function.
3. Coagulation profile, including prothrombin time (PT), international normalized ratio (INR), and activated partial thromboplastin time (aPTT), assessed daily to monitor for developing coagulopathy.
4. Renal function tests, electrolytes, and arterial blood gases performed daily to assess for signs of multi-organ dysfunction.

As the patient's LFTs continued to deteriorate, her blood pressure was closely monitored. Transient episodes of borderline hypotension were observed, but her blood pressure remained largely within a manageable range and did not necessitate vasopressor support. These hemodynamic changes were attributed to the systemic inflammatory response and evolving multi-organ dysfunction.

Despite supportive measures, the patient's condition continued to decline precipitously. Approximately 48 h post-admission, she began to exhibit signs of altered mental status, progressing rapidly to frank confusion. This neurological deterioration was consistent with the development of hepatic encephalopathy, signaling the onset of acute liver failure.

Concurrently, coagulation studies demonstrated significant abnormalities, with marked elevations in PT, INR, and aPTT. These findings were indicative of developing coagulopathy, a hallmark of severe hepatic dysfunction.

Further complicating the clinical picture, the patient's renal function parameters showed a sharp decline. Elevated serum creatinine and blood urea nitrogen (BUN) levels signaled the onset of acute kidney injury, likely due to hepatorenal syndrome or direct viral-induced nephropathy.

During the progression of her condition, the patient developed oliguria as part of the evolving acute kidney injury (AKI) associated with multi-organ failure. Renal replacement therapy (RRT) was not initiated, as the oliguria was managed conservatively with aggressive intravenous hydration and close monitoring of fluid balance. Despite the reduced urine output, the patient's metabolic parameters, including electrolyte levels and acid-base status, remained largely stable, and there were no signs of severe volume overload that would necessitate urgent RRT.

The constellation of multi-organ dysfunction—encompassing acute liver failure, coagulopathy, hepatic encephalopathy, and acute kidney injury—was consistent with a diagnosis of multi-organ failure syndrome. This rapid progression from isolated hepatitis to multi-organ failure underscores the potential severity of VZV infection in adults, particularly in the context of underlying G6PD deficiency and possible drug-induced liver injury.

In response to the patient's critical condition, a multidisciplinary team was assembled, comprising hepatologists, infectious disease specialists, critical care physicians, hematologists, and transplant surgeons. After careful consideration of the limited therapeutic options available in the face of fulminant hepatic failure, the team implemented a multi-faceted approach to management.

Firstly, intravenous immunoglobulin (IVIG) therapy was initiated as a potential immunomodulatory intervention, given its reported efficacy in severe viral infections. Following IVIG administration, a transient improvement in liver function tests was observed, with decreases in both aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels. However, this biochemical improvement did not correlate with the patient's clinical status, which continued to deteriorate. This discordance between laboratory values and clinical presentation likely indicated the completion of hepatic failure, suggesting that the remaining functional hepatic tissue had been exhausted.

Concurrently, the worsening coagulopathy necessitated hematology consultation. The hematologist

recommended the administration of cryoprecipitate to ameliorate the clotting dysfunction. This intervention aimed to mitigate the risk of spontaneous bleeding and to prepare the patient for potential invasive procedures.

Given the rapid progression of hepatic failure, an urgent surgical consultation was sought to evaluate the patient's candidacy for liver transplantation. Following a comprehensive assessment, the patient was placed on the urgent liver transplant list. The decision to pursue transplantation underscored the severity of the patient's condition and the team's recognition of the limited efficacy of conservative management in the face of fulminant hepatic failure.

Despite these aggressive interventions, including IVIG therapy, correction of coagulopathy with cryoprecipitate, and initiation of the transplant evaluation process, the patient's multi-organ failure continued to progress. The deterioration of the patient's clinical condition, despite the transient improvement in liver function tests following IVIG administration, highlighted the irreversible nature of the hepatic damage and the limitations of current therapeutic modalities in managing fulminant liver failure.

Tragically, before a suitable donor liver could be procured for transplantation, the patient succumbed to the complications of fulminant hepatic failure and multi-organ dysfunction. Despite the initiation of IVIG and maximal supportive care, including management of cerebral edema, correction of coagulopathy, and renal replacement therapy, the patient's condition continued to deteriorate. Tragically, despite these aggressive interventions, the patient succumbed to her illness and died.

Discussion

This case report presents a rare and severe complication of varicella zoster virus (VZV) infection in an adult patient with glucose-6-phosphate dehydrogenase (G6PD) deficiency, resulting in fulminant hepatitis and multi-organ failure. The case highlights the potential for life-threatening complications in adult VZV infections, particularly in patients with underlying conditions, and underscores the importance of early recognition and aggressive management.

Hepatic involvement in VZV infection is uncommon but can be severe when it occurs. A retrospective study by Tunbridge et al. found that VZV-associated hepatitis can lead to significant morbidity and mortality in adults, emphasizing the need for clinicians to consider this etiology in patients presenting with acute hepatitis [8]. The rapid progression to multi-organ failure observed in our patient is consistent with the findings of Maggi et al. who reported a case of fulminant multiorgan failure due to VZV in an immunocompromised adult [4].

To better contextualize our case, we conducted a comprehensive review of the literature and summarized some reported cases of varicella hepatitis in adults. Table 2 provides an overview of these cases, highlighting patient demographics, comorbidities, clinical presentation, treatment modalities, and outcomes. This table underscores the variability in clinical outcomes and the critical role of early recognition and intervention in managing this rare but severe complication.

The presence of G6PD deficiency in our patient may have contributed to the severity of the hepatic injury. Cappellini and Fiorelli, demonstrated that G6PD-deficient cells are more susceptible to oxidative stress-induced damage, which could potentially exacerbate liver injury in the context of viral infections [5]. This suggests that patients with G6PD deficiency may be at higher risk for severe complications during viral infections, including VZV, due to the critical role of G6PD in maintaining redox homeostasis and modulating the inflammatory and immune responses. G6PD deficiency results in impaired cellular response to oxidative stress, which can exacerbate inflammation and tissue damage during infections [7]. Studies have demonstrated that G6PD-deficient individuals exhibit altered cytokine profiles, and an enhanced pro-inflammatory response mediated through pathways such as NF-κB activation and increased reactive oxygen species (ROS) production [22, 23]. In the context of viral infections, this dysregulated inflammatory response may amplify organ-specific damage, particularly in the liver, where elevated ROS levels can induce hepatocellular injury. Furthermore, G6PD-deficient granulocytes show diminished respiratory burst activity, impairing bactericidal and antiviral defenses, and predisposing individuals to secondary infections and prolonged disease severity [24]. These mechanisms collectively suggest that the absence of adequate G6PD activity disrupts the balance between pro- and anti-inflammatory pathways, rendering patients more vulnerable to severe complications, as evidenced in our case of fulminant hepatitis and multi-organ failure following VZV infection. These findings underscore the need for heightened vigilance and tailored therapeutic strategies in managing viral infections in G6PD-deficient patients.

Furthermore, Tomar et al. reported a case of acute viral hepatitis E presenting with hemolytic anemia and acute renal failure in a patient with G6PD deficiency, highlighting the potential for severe complications in viral infections in these patients [11].

The management of VZV-associated hepatitis remains challenging, with limited evidence-based treatment options available. Gnann and Whitley emphasized the importance of timely initiation of antiviral therapy in adult VZV infections to prevent severe complications but noted that its efficacy in established severe complications

Table 2 Summary of reported cases of varicella hepatitis in adults and children

Case	Author/ year	Age/Gender	Comorbidities	Clinical Presentation	Treatment	Outcome
1	Kusne et al., 1995 [14]	19/Female	Liver transplant	Rash, abdominal pain, fulminant hepatitis	IV acyclovir	Died
2	Kusne et al., 1995 [14]	33/Male	Liver and pancreas transplant	Fever, abdominal pain, rash, hepatitis, coagulopathy	IV acyclovir, VZIG	Survived
3	Kusne et al., 1995 [14]	38/Male	Liver transplant	Rash, fever, hepatitis	IV acyclovir	Survived
4	Mantadakis et al., 2005 [15]	4/Female	Acute lymphoblastic leukemia	Rash, abdominal pain, fulminant hepatitis	IV acyclovir, VZIG	Died
5	Natoli et al., 2006 [16]	15/Male	None	Severe abdominal pain, rash, fulminant hepatitis	IV acyclovir, MARS	Died
6	Plisek et al., 2010 [17]	26/Female	Corticosteroid use for suspected MS	Rash, fulminant hepatitis, coagulopathy, ARDS, multi-organ failure	IV acyclovir, coagulation factors	Died
7	Remmerswaal et al., 2012 [18]	62/Male	Autologous HSCT, BOOP	Abdominal pain, rash, hepatitis, gastric ulcers	IV acyclovir	Survived
8	Saitoh et al., 2013 [2]	47/Male	Multiple myeloma, allo-HSCT	Fatigue, severe hepatitis, bleeding	Supportive care, postmortem diagnosis	Died
9	Chhabra et al., 2017 [19]	50/Male	Renal transplant	Abdominal pain, rash, hepatitis, pancreatitis	IV acyclovir	Survived
10	Brewer et al., 2018 [20]	66/Female	Dermatomyositis, immunosuppression	Rash, acute liver failure	IV acyclovir	Died
11	Acara et al., 2019 [21]	36/Male	Liver transplant, DM	Rash, shingles, hepatitis	IV acyclovir	Survived
12	Current case, 2025	27/Female	G6PD deficiency	Rash, jaundice, fulminant hepatitis, multi-organ failure	IV acyclovir, VZIG, cryoprecipitate, transplant evaluation	Died

ARDS: Acute Respiratory Distress Syndrome, BOOP: Bronchiolitis Obliterans Organizing Pneumonia, DM: Diabetes Mellitus, G6PD: Glucose-6-Phosphate Dehydrogenase, HSCT: Hematopoietic Stem Cell Transplantation (autologous = from the patient; allogeneic = from a donor), IV: Intravenous, IVIG: Intravenous Immunoglobulin, MARS: Molecular Adsorbent Recirculating System, MS: Multiple Sclerosis, PCR: Polymerase Chain Reaction, VZV: Varicella Zoster Virus, VZIG: Varicella-Zoster Immune Globulin

is less clear [1]. Our patient was initially treated with oral acyclovir, which is the standard antiviral therapy for VZV infections. However, upon admission to our facility, intravenous acyclovir therapy was promptly initiated due to the severity of her condition and the lack of clinical response to oral treatment. This approach aligns with current guidelines, which recommend intravenous antiviral therapy for severe VZV infections or when oral therapy fails to produce clinical improvement. Despite the timely initiation of intravenous acyclovir, as demonstrated by Dits et al., antiviral therapy alone may be insufficient in cases of fulminant hepatic failure associated with VZV infection [9]. Our multidisciplinary approach to management, including IVIG therapy, correction of coagulopathy, and consideration for liver transplantation, aligns with current best practices for managing fulminant hepatic failure. The use of IVIG in severe viral infections has shown some promise, as demonstrated by Verma et al. in their study of neonatal herpes simplex virus infection presenting as acute liver failure [10]. However, as in our case, the efficacy of IVIG in adult VZV-associated fulminant hepatitis requires further investigation.

The rapid progression to multi-organ failure in our patient, despite aggressive supportive care, highlights the potential limitations of current therapeutic modalities in managing fulminant liver failure. This is consistent with the findings of Saitoh et al. who reported a case of VZV-associated fulminant hepatitis following allogeneic hematopoietic stem cell transplantation [2].

Our case also raises important considerations regarding medication use in the context of acute viral infections, particularly in patients with underlying conditions such as G6PD deficiency. The patient's use of acetaminophen and unregulated herbal supplements may have contributed to the liver injury. A comprehensive review by Navarro et al. highlighted the potential for drug-induced liver injury in patients with viral infections, emphasizing the need for cautious medication use in these situations [12].

The step-by-step approach taken in our case included prompt isolation, comprehensive laboratory monitoring, initiation of supportive care, multidisciplinary team involvement, administration of IVIG therapy, management of coagulopathy, and evaluation for liver transplantation. This approach aligns with current best practices for managing fulminant hepatic failure, as outlined by Stravitz and Lee in their review of acute liver failure management [25].

Recent studies have further emphasized the importance of early recognition and management of VZV-associated complications [26]. Pietrzak et al. investigated the incidence and course of VZV hepatitis in 216

immunocompetent children, reporting that approximately 10% of patients experienced elevated alanine aminotransferase (ALT) levels during chickenpox. However, the course of hepatitis was predominantly mild, with no cases of liver failure observed, even among the immunosuppressed patients in the study [27]. Similarly, Feldman et al. found no correlation between the severity of chickenpox and liver involvement in their study of chemically defined VZV hepatitis in children and adolescents, further supporting the rarity of severe hepatic complications in such cases [28].

In contrast, Gnann et al. highlighted that higher viral loads translate into more severe VZV infections, which underscores the need for timely initiation of acyclovir therapy, particularly in high-risk patients [29]. In our case, the lack of clinical response to initial antiviral therapy, coupled with progressively worsening laboratory parameters—such as rising ALT, AST, and coagulopathy markers—served as early indicators of the severe disease course. These findings suggest that the absence of clinical improvement on standard antiviral treatment, along with laboratory evidence of worsening hepatic dysfunction, may serve as predictive elements for fulminant VZV hepatitis.

Future studies are needed to further explore the utility of viral load quantification and laboratory markers, such as ALT, AST, and coagulation profiles, as early predictors of disease severity. Such research could help guide clinical decision-making regarding the timing and intensity of interventions, including the use of intravenous antiviral therapy and consideration for intensive care or liver transplantation in severe cases.

Le and Rothberg provided updated guidelines on the management of herpes zoster infections, including recommendations for preventing and treating severe complications [30]. These findings further highlight the urgent need for heightened vigilance in high-risk populations and for the development of tailored treatment strategies to mitigate the risk of severe outcomes.

Conclusion

This case report describes a rare and fatal complication of varicella zoster virus infection in an adult patient with G6PD deficiency, characterized by fulminant hepatitis progressing to multi-organ failure. It emphasizes the need for vigilance in monitoring for hepatic complications in adult VZV infections, particularly in patients with underlying conditions. Clinicians should maintain a high index of suspicion for severe complications in adult VZV infections and consider early, aggressive management in cases showing signs of hepatic involvement.

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Author contributions
Nazanin Zeinali Nezhad: Conceptualization, Data curation, Writing– original draft. Behnoush Heidari: Methodology, Supervision, Validation, Project administration. Aryan Mohamadi Nezhad: Data curation, Investigation, Writing– review & editing. Mohsen Nakhaie: Writing– review & editing, Supervision. Samaneh Jahangiri: Conceptualization, Methodology.

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Data availability
The data supporting the findings of this case report are available upon request from the corresponding author. Availability of the data is contingent upon journal policy and ethical considerations.

Declarations

Ethical approval
This case report was conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study was approved by the Institutional Review Board of Kerman University of Medical Sciences. Written informed consent was obtained from the patient's father, acting as her legal guardian due to her comatose state, for the publication of this case report and any accompanying images. The consent form explicitly stated that the patient's medical information and images would be used for educational and research purposes, including publication in medical journals, while maintaining patient anonymity. The authors have made every effort to ensure the patient's privacy and confidentiality throughout the reporting process. The consent process for this case report was as follows: 1. Initial patient consent: Upon the patient's admission and while she was conscious and capable of providing informed consent, we obtained written informed consent from the patient herself for the potential publication of her case details and associated images. This consent included an explanation that her medical information would be used for educational and research purposes, including possible publication in medical journals, with assurances of maintaining her anonymity. 2. Patient's clinical deterioration: Unfortunately, the patient's condition worsened, progressing to a comatose state and ultimately resulting in her death due to multi-organ failure. 3. Additional consent from legal guardian: Following the patient's death and in accordance with the recommendations of our university's ethical committee, we obtained additional written informed consent from the patient's father, who served as her legal guardian. This step was taken to ensure comprehensive ethical coverage, particularly given the patient's ultimate inability to reaffirm her initial consent. 4. Ethical review and approval: The entire case report, including our consent process, was reviewed and approved by the Institutional Review Board of Kerman University of Medical Sciences. This review ensured that our study was conducted in accordance with the ethical standards of both our institutional and national research committees, as well as the 1964 Helsinki declaration and its subsequent amendments.

Consent for publication
Each author listed assumes responsibility for the overall integrity of the work and has provided consent for the publication of this version.

Consent statement
A written informed consent was obtained from the patient to publish this report in any medical journals.

Competing interests
The authors declare no competing interests.

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