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Atypical Hemorrhagic Varicella in a Patient With End-Stage Renal Disease: A Case Report

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Abstract

Hemorrhagic varicella (HV) is a rare and severe complication of varicella-zoster virus (VZV). This case describes an unusual delayed hemorrhagic transformation.

A 46-year-old male with end-stage renal disease (ESRD) on hemodialysis, hypertension, chronic liver disease (CLD), abdominal tuberculosis, and pancytopenia presented with intermittent dry cough, dyspnea, anuria, and scattered erythematous papules on both the upper and lower limbs without vesicles or purpura. On admission, the initial labs revealed thrombocytopenia (83,000 cells/mm^3), severe anemia (hemoglobin 6.1 g/dL), elevated inflammatory markers with a low WBC count of 3,800 cells/mm^3, and elevated serum creatinine of 4 mg/dL.

Following initial clinical improvement, he was readmitted to the intensive care unit four weeks later with new erythematous rashes and tense blisters on the left upper limb, which progressed to clear vesicles and later to hemorrhagic bullae and purpuric eruptions. Initially, fluid-filled blebs appeared on the left shoulder margin and then spread to bullae that extended distally to the forearm. The arm remained neurovascularly intact. A Tzanck smear of the vesicle fluid showed multinucleated giant cells consistent with a herpesvirus infection. Despite a course of intravenous acyclovir, followed by oral valacyclovir, the patient developed worsening thrombocytopenia (40,000 cells/mm^3) and hemorrhagic necrosis. This presentation was clinically consistent with HV with complications.

This case demonstrates the atypical progression of HV in an immunocompromised patient, with a delay of early hemorrhagic features. The underlying comorbidities of this patient may have contributed to impaired cell-mediated immunity, which altered the typical disease course. This case serves as an important learning point in recognizing atypical presentations of varicella in high-risk patients.

Categories: Internal Medicine, Nephrology, Infectious Disease
Keywords: atypical varicella, chronic kidney disease, hemorrhagic varicella, treatment of varicella in immunosuppressed patients, varicella in immunosuppressed patients

Introduction

Chronic kidney disease (CKD) is a major global health burden, and patients with end-stage renal disease (ESRD) are especially prone to infections because of profound immune dysfunction. Uremic toxins, chronic inflammation, and abnormalities in both innate and adaptive immunity contribute to this susceptibility [1]. These alterations make ESRD patients highly vulnerable to severe outcomes following viral infections.

Varicella, caused by the varicella-zoster virus (VZV), an alpha-herpesvirus of the *Herpesviridae* family, continues to be a common primary infectious disease worldwide. Transmission occurs mainly via airborne droplets and direct contact with vesicular fluid. Although most cases occur in children and present with characteristic vesicular eruptions, the disease can be severe in immunocompromised adults, leading to significant morbidity and mortality [2-3].

Other populations at increased risk of severe VZV infection include those with HIV, autoimmune diseases, malignancies, and transplant recipients, while comorbidities such as diabetes and chronic obstructive pulmonary disease are associated with a heightened risk of infection rather than true immunocompromised status [3].

Hemorrhagic varicella (HV) is among the rarest and most severe forms of VZV infection, usually seen in immunocompromised hosts, and is associated with high mortality. Such atypical presentations, including hemorrhagic and necrotic lesions, are increasingly recognized in these groups [4-13].

We report a case of HV with atypical cutaneous evolution in a patient with ESRD on maintenance

hemodialysis and with multiple comorbidities. This case emphasizes the importance of early recognition, aggressive management, and preventive strategies in managing varicella among immunocompromised patients.

Case Presentation

A 46-year-old male with ESRD on maintenance hemodialysis for two years presented with acute-onset breathlessness, intermittent dry cough for two months, anuria, and rash over his limbs for one week. The etiology of his ESRD was not established prior to this admission.

His comorbidities included hypertension, chronic liver disease (CLD), abdominal tuberculosis treated with antitubercular therapy, chronic empyema with a history of tuberculous pleural effusion, mild portal hypertensive gastropathy, and pancytopenia. The underlying etiology of the patient's CLD was not established in the available records. Although mild portal hypertensive gastropathy was documented, no abdominal ultrasonography report or other relevant hepatic imaging was available for review. Consequently, the presence or absence of cirrhosis and the compensated or decompensated status of the liver disease could not be determined.

On admission (Day 0), he was malnourished, conscious, and oriented with a blood pressure of 200/80 mmHg, a pulse of 72 beats/min, and a respiratory rate of 20 breaths/min. His oxygen saturation was 99% while receiving supplemental oxygen at 5 L/min through a face mask. Respiratory examination revealed inspiratory crepitations, raising suspicion of an underlying lower respiratory tract infection.

Dermatological examination revealed scattered, discrete, non-pruritic erythematous papules, a few millimeters in size, distributed symmetrically over both upper and lower limbs, with no associated vesicles, bullae, or purpura at this stage. At this stage, the differential diagnosis for the papular eruption included uremic dermopathy, a drug reaction related to antitubercular therapy, and a petechial process related to his thrombocytopenia; a viral exanthem was not initially suspected given the absence of vesicular or hemorrhagic features.

Laboratory investigations revealed severe macrocytic anemia (hemoglobin 6.1 g/dL), thrombocytopenia (83 × 10^9/L), leukopenia (3.8 × 10^9/L), and elevated serum creatinine (4 mg/dL post-dialysis). He had hypoalbuminemia and elevated alkaline phosphatase and gamma-glutamyl transferase with normal transaminases. The coagulation profile and dengue serology were within normal limits. HIV, hepatitis B surface antigen, and hepatitis C testing were nonreactive during this admission. The initial chest radiograph performed on admission was unremarkable.

The summary of the baseline investigations is provided in Table *1*.

Investigation	Result	Reference range
Hemoglobin	6.1 g/dL	13–17 g/dL
PCV	21%	40–50%
MCV	108 fL	80–100 fL
RDW	25.1%	11.5–14.5%
Platelet count	83 × 10^9/L	150–450 × 10^9/L
Total leukocyte count	3.8 × 10^9/L	4–11 × 10^9/L
Differential count	Neutrophils (N) 86%; Lymphocytes (L) 6%; Monocytes (M) 7%; Eosinophils (E) 1%; Basophils (B) 0%.	Neutrophils (N) 40–70%; Lymphocytes (L) 20–40%; Monocytes (M) 2–8%; Eosinophils (E) 1–4%; Basophils (B) 0–1%.
ESR	42 mm/hr	0–15 mm/hr (male)
Serum creatinine	4 mg/dL	0.7–1.3 mg/dL
Serum albumin	2.4 g/dL	3.5–5.0 g/dL
Total protein	5.9 g/dL	6.0–8.3 g/dL
PTH	138 pg/mL	15–65 pg/mL
Serum phosphate	6.9 mg/dL	2.5–4.5 mg/dL

Serum calcium	8.5 mg/dL	8.5–10.5 mg/dL
Total bilirubin	0.6 mg/dL	0.2 – 1.2 mg/dL
Direct bilirubin	0.1 mg/dL	0.0-0.3 mg/dL
Indirect bilirubin	0.5 mg/dL	0.2-0.9 mg/dL
ALP	184 U/L	44–147 U/L
AST	25 U/L	10–40 U/L
ALT	5 U/L	7–56 U/L
GGT	55 U/L	9–48 U/L
LDH	262 U/L	140–280 U/L
Reticulocyte count	7.2%	0.5–2.5%
PT (Patient/Control Time)	13.4/11.9 sec	11–14 sec
APTT (Patient/ Control Time)	30.1/30.7 sec	25–35 sec
INR	1.13	0.8–1.2
HIV	Nonreactive	Nonreactive
HBsAg	Nonreactive	Nonreactive
HCV	Nonreactive	Nonreactive
Dengue serology IgM	Nonreactive	Nonreactive
AFB smear (Sputum)	Negative	Negative

TABLE 1: Baseline investigations

Abbreviations: AFB, acid-fast bacilli; ALP, alkaline phosphatase; ALT, alanine aminotransferase; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; ESR, erythrocyte sedimentation rate; HBSAg, hepatitis B surface antigen; HCV, hepatitis C virus; HIV, human immunodeficiency virus; INR, international normalized ratio; LDH, lactate dehydrogenase; MCV, mean corpuscular volume; PCV, packed cell volume; PTH, parathyroid hormone; RDW, red cell distribution width.

During the hospital course, blood cultures grew *Klebsiella pneumoniae*, which was treated as the causative organism for his systemic bacterial infection, supported by an elevated procalcitonin level of 8 ng/mL; however, the number of positive bottles and full culture details were not available for review. Although the respiratory symptoms, oxygen requirement, and inspiratory crepitations raised suspicion of a pulmonary infective process, the initial chest radiograph did not demonstrate a radiological infiltrate. He developed a worsening sensorium and refractory hypertension, requiring mechanical ventilation and vasopressor support. Supportive management, including antibiotics and hemodialysis, was continued. The patient showed partial clinical improvement over the following weeks.

By the fourth week, his condition deteriorated again with vomiting, anuria, and declining consciousness, necessitating readmission to the intensive care unit. Given his worsening sensorium, a lumbar puncture was performed with CSF analysis, which was essentially within normal limits, arguing against VZV-related CNS involvement. Head imaging was not performed, as there were no focal neurological deficits to suggest a structural intracranial process.

After brief stabilization, new erythematous rashes developed over the trunk and extremities, accompanied by tense blisters confined to the left upper limb. The blisters progressed to large, clear, fluid-filled blebs, and a Tzanck smear from a fresh vesicle showed multinucleated giant cells, consistent with herpesvirus infection. The distribution of lesions, initially on the left shoulder and subsequently spreading to the forearm, trunk, and face in a non-dermatomal pattern, was consistent with disseminated VZV infection rather than

localized herpes zoster (shingles). The patient had no documented prior history of varicella infection or VZV vaccination.

The corresponding laboratory findings on the day of readmission are summarized in Table 2.

Investigation	Result	Reference range
Hemoglobin	7.4 g/dL	13–17 g/dL
PCV	25%	40–50%
MCV	99 fL	80–100 fL
RDW	22.3%	11.5–14.5%
Platelet count	60 × 10^9/L	150–450 × 10^9/L
Total leukocyte count	7.1 × 10^9/L	4–11 × 10^9/L
Differential count	Neutrophils (N) 89%; Lymphocytes (L) 6%; Monocytes (M) 4%; Eosinophils (E) 1%; Basophils (B) 0%.	Neutrophils (N) 40–70%; Lymphocytes (L) 20–40%; Monocytes (M) 2–8%; Eosinophils (E) 1–4%; Basophils (B) 0–1%.
ESR	95 mm/hr	0–15 mm/hr (male)
Serum creatinine	6.6 mg/dL	0.7–1.3 mg/dL
Serum albumin	1.9 g/dL	3.5–5.0 g/dL
Total protein	6.1 g/dL	6.0–8.3 g/dL
ALP	224 U/L	44–147 U/L
AST	28 U/L	10–40 U/L
ALT	5 U/L	7–56 U/L
GGT	45 U/L	9–48 U/L
PT (Patient/Control Time)	12.6/11.9 sec	11–14 sec
APTT (Patient/Control Time)	26/30.7 sec	25–35 sec
INR	1.06	0.8–1.2
Procalcitonin	9.01 ng/mL	<0.5 ng/mL

TABLE 2: Laboratory findings on readmission to the renal ICU at four weeks

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; ESR, erythrocyte sedimentation rate; GGT, gamma-glutamyl transferase; INR, international normalized ratio; MCV, mean corpuscular volume; PCV, packed cell volume; PT, prothrombin time; RDW, red cell distribution width.

The patient was started on renally adjusted intravenous acyclovir 100 mg every 24 hours, which was later switched to oral valacyclovir 500 mg every 24 hours. Although the vesicular lesions began crusting, new hemorrhagic vesicles and purpuric eruptions appeared on the face and trunk. The new erythematous rashes on the trunk and face were similar in morphology to the earlier limb lesions, though they occurred at a more advanced stage, with progression to hemorrhagic vesicles and purpuric eruptions.

Serial hematological, renal, and electrolyte parameters during the hospital course are presented in Figure 1.

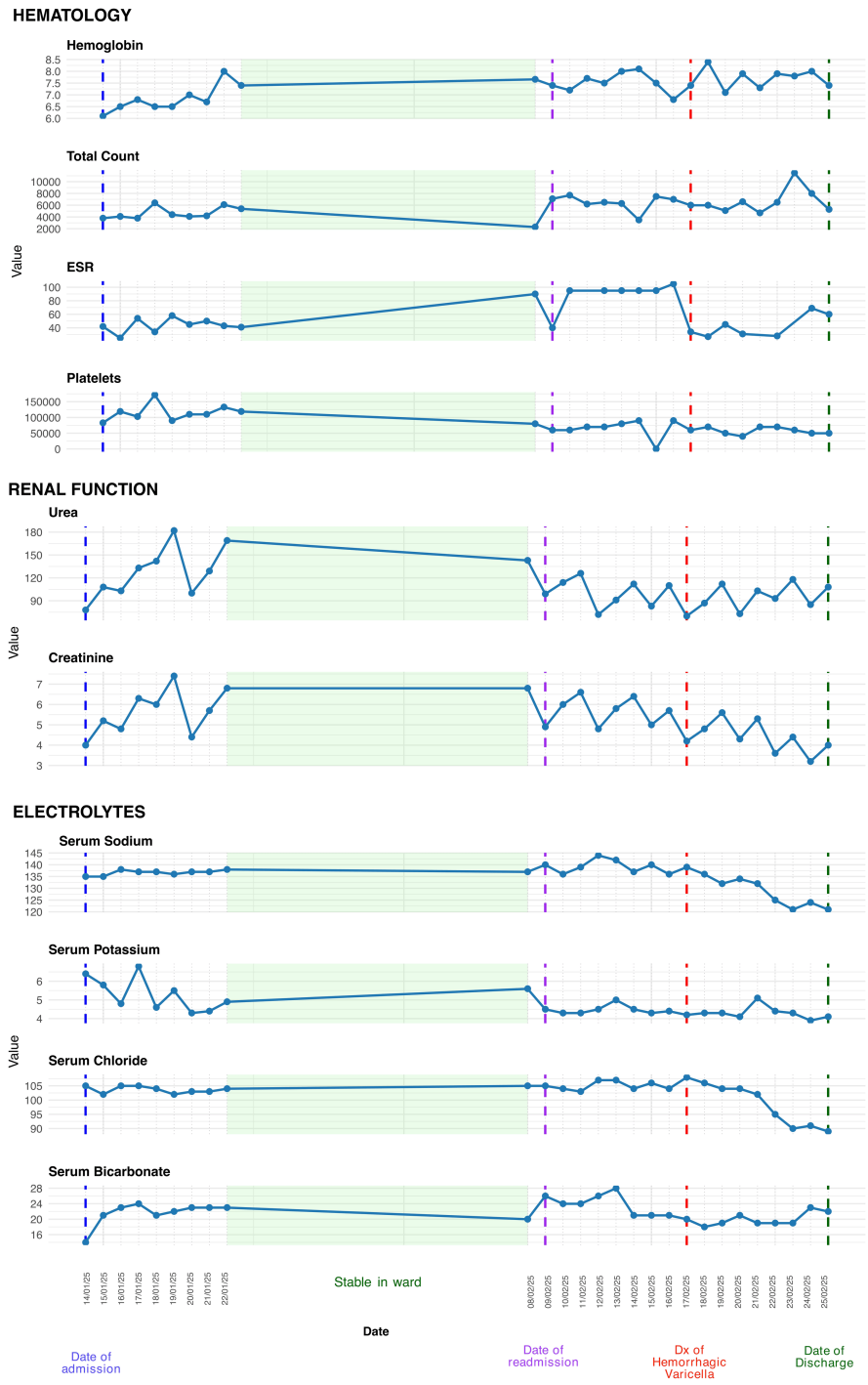


FIGURE 1: Serial hematological, renal, and electrolyte trends during Renal ICU admission showing anemia, thrombocytopenia, fluctuating counts, persistent renal dysfunction, and electrolyte derangements.

The shaded region indicates stability in the ward; dashed lines mark admission, readmission, diagnosis of hemorrhagic varicella, and discharge.

Figure generated using jamovi (v2.6; The jamovi project, Sydney, Australia) and R (v4.4; R Foundation for Statistical Computing, Vienna, Austria).

Platelet count dropped to 40 × 10^9/L with mucosal bleeding, consistent with immune-mediated thrombocytopenia, for which intravenous dexamethasone was administered. The patient remained uremic at this time (creatinine 5.6 mg/dL), and uremic platelet dysfunction likely contributed to the mucosal bleeding in addition to the immune-mediated thrombocytopenia.

Coagulation studies showed a mildly prolonged prothrombin time (14.0/11.9 sec) and international normalized ratio (1.18), with activated partial thromboplastin time within the reference range (33.8/30.7 sec) and preserved fibrinogen (281 mg/dL), arguing against disseminated intravascular coagulation. No additional testing for immune thrombocytopenia (e.g., platelet antibodies) was performed; the coagulopathy workup was limited to the parameters listed in Table 3. Key diagnostic and follow-up investigations in the subsequent weeks are summarized in Table 3.

Investigation	Result	Significance
Blood culture	Klebsiella pneumoniae	Systemic bacterial infection
Procalcitonin	27.92 ng/mL	Progressive systemic inflammation/infection
Tzanck smear	Multinucleated giant cells	Supports herpesvirus infection.
Platelet count	40 × 10^9/L	Severe thrombocytopenia accompanying hemorrhagic lesions
PT (Patient/Control time); INR	PT 14.0/11.9 sec; INR 1.18	Mild coagulation abnormality
APTT (Patient/Control time)	33.8/30.7 sec	Within reference range
Fibrinogen	281 mg/dL	Preserved; argues against overt DIC
CSF analysis	Essentially within normal limits	No significant CNS inflammatory abnormality

TABLE 3: Key Diagnostic and Follow-up Investigations

Abbreviations: APTT, activated partial thromboplastin time; CSF, cerebrospinal fluid; DIC, disseminated intravascular coagulation; INR, international normalized ratio; PT, prothrombin time; VZV, varicella-zoster virus.

The cutaneous lesions evolved into necrotic patches with slough formation over the volar aspect of the left forearm and wrist, requiring surgical debridement and wound care (Figure 2).



FIGURE 2: Dermatological progression of hemorrhagic varicella during the Week 4 readmission, showing hemorrhagic bullae, purpuric eruptions, and necrotic patches with slough formation over the left upper limb and chest. No photographic documentation of the initial Day 0 papular rash is available; both images depict lesions from the Week 4 readmission onward.

Despite antiviral therapy, the patient's condition continued to worsen, marked by progression of the cutaneous lesions to necrotic bullae requiring surgical debridement and persistent thrombocytopenia, as described above.

Given the poor prognosis associated with HV, the family opted for discharge against medical advice after a total hospital stay of 42 days.

Discussion

This case illustrates an atypical dermatological progression of varicella in a patient with ESRD undergoing hemodialysis who developed vesicular lesions at the Week 4 readmission that later evolved into hemorrhagic and necrotic eruptions. However, ESRD alone may not account for this presentation, as most patients with ESRD do not develop HV. Coexisting CLD, malnutrition, and concurrent systemic infection may have further

contributed to impaired host defense, although the relative contribution of each factor remains uncertain.

HV is a rare complication, reported in approximately 3.3% of varicella cases [14], and is most often seen in immunocompromised individuals.

According to a review of published cases, the dermatological symptoms are diverse, ranging from segmental pigmented vesicles and widespread hemorrhagic eruptions to atypical ecthymatous crusting and purpuric dermatomal lesions [4-13].

Immune dysfunction in ESRD arises from multifactorial defects in the innate and adaptive immune systems driven by oxidative stress, intestinal barrier failure, uremic toxins, chronic inflammation, reduced antigen presentation, and B-cell function, predisposing patients to severe viral infections [1].

The patient's multiple comorbidities may have further contributed to impaired host defense. Mortality rates from varicella in renal transplant patients reach 30%-35% [15], whereas Ong et al. reported a 13.6% mortality rate among ESRD patients with varicella [16]. The delayed onset of hemorrhagic lesions in our case underscores the atypical progression of VZV infection in these hosts.

Atypical presentations, including bullous pemphigoid resemblance [17], vasculitis lesions, and pulmonary involvement, have been reported [18, 19]. Some patients recovered, while others succumbed despite adequate treatment [5, 7, 8, 13].

Few cases have reported progression of HV to disseminated intravascular coagulation, multiorgan failure, and shock, leading to morbidity [5, 8, 13, 20, 21]. Severe thrombocytopenia, as observed in this study, may result from immune-mediated destruction or direct viral cytopathy, with the latter having preserved fibrinogen levels, as in this case [22].

Early antiviral therapy is still the most important part of treating HV. In our case, acyclovir followed by valacyclovir, corticosteroids for thrombocytopenia, and local wound care were administered, mirroring the management strategies reported in similar cases [6, 13]; however, the disease progressed, reflecting the poor prognosis of HV.

Preventive measures, including providing two doses of vaccine four weeks apart, are safe for patients with CKD [23]. In summary, early recognition, prompt antiviral therapy, vigilant hematological monitoring, and preventive vaccination are crucial for reducing morbidity and mortality rates.

This report has limitations. The diagnosis was clinical, supported by a Tzanck smear; skin biopsy, VZV PCR, and VZV serology were not performed, so primary infection could not be distinguished from reactivation. Details on the extent of debridement and the wound outcome were not available.

Conclusions

VZV infection is typically a mild, self-limiting disease in children, but in adults with impaired immunity it may progress to severe, life-threatening forms. This case emphasizes the necessity of heightened clinical vigilance in immunocompromised patients presenting with varicella. Although ESRD-related immune dysfunction may have contributed to the clinical presentation, coexisting CLD, malnutrition, and concurrent systemic infection may also have influenced the disease course. The relative contribution of these comorbidities remains uncertain, and the presentation should not be attributed to ESRD alone. Early recognition of atypical cutaneous signs and supportive management are crucial to improving outcomes; however, this case illustrates that severe HV may progress to necrosis despite timely initiation of antiviral therapy. Equally important is the role of preventive strategies, especially varicella vaccination in seronegative ESRD patients before immunosuppressive therapy or transplantation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kaandeeban Mohanraj, Sampathkumar Krishnaswamy

Acquisition, analysis, or interpretation of data: Kaandeeban Mohanraj, Andrew Deepak Rajiv, Sampathkumar Krishnaswamy, Naveen Punchayil Narayanankutty

Drafting of the manuscript: Kaandeeban Mohanraj

Critical review of the manuscript for important intellectual content: Kaandeeban Mohanraj, Andrew

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Supervision: Sampathkumar Krishnaswamy

Disclosures

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