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Sequential Progression from Hypertension to Chronic Kidney Disease with Anaemia, Followed by Ankylosing Spondylitis and Secondary Herpes Zoster Infection; A Case Report

Meghana Liman
Dr. D.Y. Patil Vidyapeeth, Pune
Snehalata Pawar
sneha.ayurved@gmail.com

Dr. D.Y. Patil Vidyapeeth, Pune
Vaishali Chaudhari
Dr. D.Y. Patil Vidyapeeth, Pune
Shruti Kulkarni
Dr. D.Y. Patil Vidyapeeth, Pune

Case Report

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Abstract

Background

Chronic kidney disease (CKD) is a systemic disorder that affects multiple organ systems, particularly the immune system. Patients with long-standing hypertension are at increased risk of developing CKD, which may subsequently be complicated by chronic inflammation and altered immune responses. These changes predisposed to both autoimmune conditions and infections, such sequential occurrences in same patient are rarely documented, timely diagnosis and investigation plays an important role to prevent prognosis of disease along with intervention.

Case Presentation

A 49-year- old married Indian female with a 20-year of hypertension had history of generalized weakness, dizziness, Shortness of breath on exertion, pallor and was reported with severe anaemia treated with blood transfusion. Further evaluation revealed underlying Stage IV chronic kidney disease. During the course of illness, she developed accelerated hypertension with left ventricular failure and metabolic acidosis, requiring hospitalization and medical management. Two months later, she presented again with persistent severe low back pain and stiffness. Investigations showed markedly elevated inflammatory markers, and imaging revealed sacroiliac joint inflammation. HLA-B27 positivity confirmed a diagnosis of Ankylosing Spondylitis. Due to chronic inflammation and reduced immunity, she developed a herpes zoster infection, thus highlighting an underlying state of immune dysregulation due to chronic inflammatory changes in CKD.

Conclusion

Although CKD-associated immune dysfunction is well recognized, the sequential occurrence chronic hypertension and advanced CKD, anaemia, HLA-B27-positive Ankylosing spondylitis followed by Herpes zoster infection has been rarely reported in same patient. This case highlights the importance of early recognition and multidisciplinary approach to prevent complications and improve patient outcomes.

Background

Chronic Kidney Disease (CKD) is defined as abnormalities of kidney structure, present for more than three months, with health implications, including a decreased glomerular filtration rate (< 60 mL/min/1.73 m²) [1]. Chronic Kidney Disease (CKD) is major global public health problem, affecting approximately 11–13% with the majority stage III population and contributing significantly to the morbidity and mortality worldwide. [2] hypertension is emerging leading causes of CKD with the Global Burden of Disease (GBD) 2021 study reporting over 24 million prevalent cases and more than 454,000 deaths worldwide in 2021 [3]. The progression from hypertension to CKD is driven by chronic

hemodynamic stress ultimately resulting in irreversible renal impairment. Anaemia is a frequent and clinically significant complication of CKD, primarily due to reduced erythropoietin production and chronic inflammation with prevalence increases with disease severity, affecting approximately 40–50% of patients and rising to 80–90% in end -stage renal disease, particularly in resource-limited settings such as India [4].

CKD is also recognized as a state of complex immune dysregulation involving both innate and adaptive immunity due to inflammation this condition, often described as “chronic inflammatory immune depression” is mediated by the accumulation of uremic toxins. This altered immune state predisposes patients to increased susceptibility to infections, including viral reactivation such as herpes, while also contributing to the unmasking of autoimmune diseases in genetically predisposed individuals. [5]

This case highlights rare clinical sequence linking of long-standing hypertension to CKD with anaemia, followed by immune dysregulation leading to HLA-B27 positive Ankylosing Spondylitis and subsequent herpes zoster infection. It underscores the dual impact of CKD on the chronic inflammation and immune system also impairing host defence leading to infection and emphasizes the need for early recognition and a multidisciplinary approach to prevent systemic complications.

Case Presentation

A 49-year-old woman with a 20-year history of hypertension presented with generalized weakness, dizziness, Shortness of breath on exertion, pallor on initial evaluation, she was reported to have severe anaemia, with a haemoglobin level of 4.7 g/dL. She subsequently received transfusion of two units of Packed red blood cells on 13 September and 17 September 2025, following which her symptoms improved.

Post transfusion laboratory evaluation revealed haemoglobin of 9.1 g/dL, packed cell volume of 28.80%, and RBC of 2.80 million/mm³. Red cell indices demonstrated macrocytosis (mean corpuscular volume [MCV] 102.9 fL) along with markedly elevated red cell distribution width (RDW 26.4%). Peripheral smear examination showed a dimorphic red cell population with microcytic hypochromic erythrocytes and macrocytes, suggestive of mixed anaemia. biochemical evaluation revealed Serum creatinine of 2.91 mg/ dL and Glomerular filtration rate (eGFR) of 19 mL/min/1.73 m², consistent with Stage Four IV chronic kidney disease.

After 1 month, she had acute onset breathlessness associated with excessive sweating and a sense of uneasiness for approximately 12 hours, for which she was admitted. At presentation, her blood pressure was markedly elevated at 200/130 mmHg, pulse rate was 140/min, oxygen saturation was 85%, and blood glucose was 136 mg/dL also known case of hypertension for 20 years with off regular Antihypertensive medicine. On examination, she was conscious and oriented. Cardiovascular examination revealed normal heart sounds, while respiratory system examination showed reduced bilateral air entry. The abdomen was soft and non-tender on palpation and no abnormal bowel sound.

Arterial blood gas (ABG) analysis reported pH 7.288, p CO_2 19.9 mm Hg, pO_2 121 mm Hg, HCO_3^- 9.5 mmol /L). Lactate levels were within normal limits. Laboratory investigations showed haemoglobin of 10.2 g/dL, Total leukocyte count of 14,760/cu mm, and normal platelet count. Renal parameters were deranged, with blood urea of 66 mg/dL and serum creatinine of 2.58 mg/dL and Albuminuria- 203 mg/l was noted (eGFR) of 22. Liver function tests were within normal limits except mild elevation in alkaline phosphatase. Serum calcium was low (6.9 mg/dL).

Echocardiography revealed a preserved Left ventricular ejection fraction (LVEF) of 55%, with mild diastolic dysfunction, mitral annular calcification, and trivial valvular regurgitation. Based on the clinical presentation and investigations, a diagnosis of accelerated hypertension with left ventricular failure, lower respiratory tract infection, and chronic kidney disease was made.

Two months later, the patient presented with complaints of severe low back pain and stiffness. Inflammatory markers were significantly elevated, with ESR of 107 mm/hr and CRP of 49.13 mg/dL. MRI of the lumbosacral spine showed sacroiliac joint effusion with inflammatory changes suggestive of spondyloarthropathy. Bone aspiration was performed to exclude infection and was negative. Further evaluation reported HLA-B27 positivity, confirming the diagnosis of Ankylosing Spondylitis. (Appendix 1)

In February 2026, the patient developed red rash appearing in a unilateral dermatomal pattern cluster of fluid- filled vesicles on back region herpes zoster infection, suggesting an underlying state of immune compromise, possibly related to chronic disease and systemic inflammation. Figure 1

Timeline

The sequence of clinical events which were observed is summarized in Table 1.

Table 1
Timeline of Clinical Events

Date	Clinical Event	Laboratory Findings	Diagnosis /Clinical Outcome
September 2025 (Initial Presentation)	Presented with generalized weakness, dizziness, dyspnoea on exertion, pallor, easy fatigability	Hb: 4.7 g/dL	**Severe anaemia diagnosed**
13 & 17 September 2025	Blood transfusion administered	Two units of packed red blood cells transfused	Symptomatic improvement
Post-transfusion (September 2025)	Laboratory evaluation	Hb: 9.1 g/dL, PCV: 28.8%, RBC: 2.8 million/mm^3, MCV: 102.9 fL, RDW: 26.4%, dimorphic smear	Mixed (dimorphic) anaemia
	Renal evaluation	Creatinine:2.91 mg/dL, eGFR: 19 mL/min/1.73 m^2	**Chronic Kidney Disease**
October 2025	Acute breathlessness, sweating, uneasiness	BP: 200/130 mmHg, PR: 140/min, SpO_2: 85%	Hypertensive emergency
	Past History	Known case of hypertension (20 years, irregular treatment)	Risk factor established
	Clinical Examination	Reduced bilateral air entry	Respiratory involvement
	ABG analysis	pH 7.175, p CO_2 26.9 mm Hg, pO_2 121 mm Hg, HCO_3^- 9.9 mmol /L).	Metabolic acidosis
	Laboratory investigations	Hb: 10.2 g/dL, TLC: 14,760, Urea: 56 mg/dL, Creatinine: 2.58 mg/dL, Ca: 6.9 mg/dL, Albuminuria: 203 mg/L	CKD with systemic complications
	2D Echo	LVEF: 55%, mild diastolic dysfunction	Cardiac involvement
	Diagnosis		Accelerated hypertension with left ventricular failure, lower respiratory tract infection, and chronic kidney disease
December 2025	Severe low back pain & stiffness	ESR: 107 mm/hr, CRP: 49.13 mg/dL	Inflammatory pathology suspected

Date	Clinical Event	Laboratory Findings	Diagnosis /Clinical Outcome
	MRI findings	Sacroiliac joint effusion, inflammation	Suggestive of spondyloarthropathy
	Further evaluation	HLA-B27 positive	**Ankylosing Spondylitis**
February 2026	Painful unilateral vesicular rash (back region)	Dermatomal fluid filled vesicles	**Herpes Zoster Infection**

Diagnostic Assessment

Laboratory investigations revealed severe anaemia, elevated serum creatinine, reduced eGFR, metabolic acidosis, elevated inflammatory markers (ESR and CRP), and HLA-B27 positivity. MRI of the lumbosacral spine showed sacroiliac joint inflammation suggestive of spondyloarthropathy, while echocardiography revealed mild diastolic dysfunction. Infective causes were excluded by negative bone aspiration findings.

Diagnosis

Stage IV chronic kidney disease with anaemia, hypertension with left ventricular failure, HLA-B27 positive Ankylosing spondylitis, and secondary Herpes zoster infection.

Therapeutic Intervention

The patient was initially managed conservatively with transfusion of two units of packed red blood cells for severe anaemia. Subsequently, she developed a hypertensive emergency and was diagnosed with accelerated hypertension, left ventricular failure, and chronic kidney disease. She was managed under Nephrology supervision with antihypertensive therapy, correction of metabolic acidosis, antibiotics for lower respiratory tract infection, and supportive renal care and no dialysis required. Detailed Hospitalisation treatment information is provided in the discharge summary (Appendix 1). Following the diagnosis of Ankylosing spondylitis, the patient was treated conservatively with Etoricoxib 60 mg once daily after food and Thiocolchicoside 4 mg twice daily for 7 days, along with physiotherapy and posture correction exercises. Subsequently, the Herpes zoster infection was managed with oral Acyclovir 800 mg three times daily with dose adjustment according to renal function, Calamine lotion application over the lesions, maintenance of local skin hygiene, adequate hydration, and supportive care. NSAIDs were prescribed cautiously with renal monitoring

Discussion

The present case illustrates a clinically prognosis involving long standing hypertension progressing to chronic kidney disease, followed by HLA-B27 associated autoimmune manifestation and subsequent

development of herpes zoster infection. This progression reflects a pathophysiological event driven by chronic inflammation, immune dysregulation, genetic susceptibility and infection.

Hypertension and Progression to Chronic Kidney Disease

Hypertension is an established risk factor for CKD, primarily through sustained hemodynamic stress on the renal vasculature. Chronic elevation of blood pressure leads to the endothelial dysfunction, and activation of the Renin angiotensin aldosterone system (RAAS), oxidative stress and progressive nephron loss, ultimately resulting in glomerulosclerosis and tubulointerstitial fibrosis [6]. These structural changes contribute to the gradual decline in renal function and the systemic complications observed in CKD.

Anaemia in Chronic Kidney Disease

Anaemia is an established complication of CKD which is arising from a multifactorial pathophysiological mechanism. The primary mechanism involves reduced production of Erythropoietin by the damaged kidneys, leading to decreased red blood cell synthesis. In addition, chronic inflammation associated with CKD increases cytokine levels and Hepcidin activity, resulting in impaired iron utilization and functional iron deficiency. Other contributing factors include nutritional deficiencies (iron, vitamin B12, and folate), reduced red blood cell lifespan due to uremic toxins, and occasional blood loss, especially in advanced stages or during dialysis. This combination often leads to mixed (dimorphic) anaemia, reflecting both deficiency and chronic disease states, as also observed in this case. Anaemia itself can exacerbate CKD progression by causing tissue hypoxia and increasing cardiovascular strain, thereby creating a vicious cycle that worsens overall patient outcomes [7, 8].

Immune Dysregulation and Autoimmune Manifestations in CKD

CKD has been reported in patients with Ankylosing Spondylitis, often secondary to chronic inflammation, amyloidosis and long-term use of NSAID [9]. HLA-B27 is a well-recognized genetic marker associated with Ankylosing Spondylitis. The pathogenic mechanisms involve aberrant antigen presentation, molecular mimicry, and misfolding of the HLA-B27 molecule, leading to activation of the unfolded protein response and stimulation of the IL-23/IL-17 inflammatory axis. In the presence of CKD-related immune dysregulation, these mechanisms may have been amplified, facilitating the onset of autoimmune disease in genetically susceptible individuals in presented case. [10, 11].

A central feature of CKD is Immune dysfunction characterized by chronic inflammation and immunosuppression. Accumulation of the uremic toxins such as Indoxyl sulphate and p-cresyl sulphate, because of impaired renal clearance, plays a significant role in this process. These toxins induce oxidative stress, endothelial activation and further dysregulation of immune cells, including impaired phagocytic activity of neutrophils and dysfunction of an antigen presenting cells. Additionally, adaptive

immunity is compromised due to the T-cell exhaustion and impaired lymphocyte proliferation, hence leading to reduced host defence mechanisms. This altered immune state is often described as “Chronic Inflammatory Immune depression” due to persistent elevation of pro-inflammatory cytokines i.e. Interleukin-6 and Tumour necrosis factor-alpha, further involving systemic inflammation, this acts as a trigger for the expression of latent genetic predispositions to Ankylosing Spondylitis [12].

CKD-Associated Immune Dysfunction and Susceptibility to Infection

Furthermore, CKD is associated with increased susceptibility to infections due to impaired cell-mediated immunity. Effective control of latent viral infections, including varicella-zoster virus, depends on intact T-cell responses. In CKD, T-cell dysfunction and impaired cytokine signalling predispose patients to viral reactivation. Epidemiological studies have demonstrated that patients with CKD have a significantly higher risk of developing herpes zoster compared with the general population [13, 14].

The coexistence of an Autoimmune activation and increased risk of infection reflects a complex immunological paradox. The persistent inflammation promotes autoimmunity, while immune exhaustion leads to increased vulnerability to infections. This phenomenon aligns with the concept of “Inflammaging” where chronic immune activation coexists with progressive immune decline [15].

Strengths and Limitations

A major strength of this case is the documentation of sequential progression from long-standing hypertension to chronic kidney disease with associated anaemia, followed by autoimmune manifestation and viral reactivation, thereby highlighting the role of immune dysregulation in CKD. However, the limitation of this report is that it represents a single case observation with limited long-term follow-up. Further clinical studies are required to better understand the association between CKD-related immune dysfunction, autoimmune disorders, and opportunistic infections.

Conclusion

This case demonstrates the progression from long-standing hypertension to chronic kidney disease with associated anaemia, followed by immune dysregulation contributing to the development of ankylosing spondylitis and secondary herpes zoster infection. It highlights that CKD is a systemic disorder involving a complex interplay of inflammation, immune imbalance, and increased susceptibility to both autoimmune and infectious conditions. Early control of hypertension and a multidisciplinary approach are essential to prevent disease progression and timely diagnosis and appropriate investigations play an important role in the effective management of these complications.

Abbreviations

CKD Chronic Kidney Disease

eGFR Estimated Glomerular Filtration Rate

LVEF Left Ventricular Ejection Fraction

ESR Erythrocyte Sedimentation Rate

CRP C-reactive Protein

RAAS Renin-Angiotensin-Aldosterone System

NSAIDs Non-Steroidal Anti-Inflammatory Drugs

Declarations

Ethics approval and consent to participate

As this is a single case report, formal ethical approval was not required according to institutional guidelines. The case was managed using standard clinical protocols.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and clinical details.

Availability of data and materials

All relevant clinical information has been included within the manuscript. Additional details can be shared by the corresponding author on reasonable request.

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Authors' contributions

ML: Contributed to the conception and design of the study, data collection, interpretation of clinical findings, manuscript drafting, and overall preparation of the manuscript.

SP: Contributed to data collection, analysis and interpretation of clinical findings, and review of the manuscript.

VC: Contributed to conceptualization, manuscript editing, critical review of the manuscript, and approval of the final version for publication.

SK: Contributed to study conceptualization, supervision, interpretation of findings, critical review of the manuscript, and final approval of the version to be published.

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Figures



Figure 1

Unilateral dermatomal pattern cluster of fluid-filled vesicles on back region of herpes zoster infection

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