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When Infection Mimics Vasculitis: Hydralazine-Induced ANCA-Associated Crescentic Glomerulonephritis in the Setting of Active Infective Endocarditis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABDEF Mohmmed Saleh
ABDF Shahrzad Zavoshi
ADF Paul Fozo

Department of Internal Medicine, Henry Ford St. John Hospital, Detroit, MI, USA

Corresponding Author: Mohmmed Saleh, Department of Internal Medicine, Henry Ford St. John Hospital, 22101 Moross Rd, Detroit, MI 48236, USA
e-mail: Mksaleh14@gmail.com
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Patient: Male, 58-year-old
Final Diagnosis: Hydralazine-induced ANCA-associated crescentic glomerulonephritis in the setting of active infective endocarditis
Symptoms: Fatigue • anemia • acute kidney injury
Clinical Procedure: —
Specialty: General and Internal Medicine
Objective: Rare coexistence of disease or pathology

Background: Hydralazine-induced antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis is a rare cause of pauci-immune crescentic glomerulonephritis that can closely mimic infection-related renal disease, particularly in the setting of active infective endocarditis. Distinguishing between infection-related glomerulonephritis and drug-induced ANCA-associated vasculitis is clinically important because management strategies differ substantially. We present a case supporting the hypothesis that persistent renal dysfunction despite microbiologic control of infective endocarditis, together with pauci-immune biopsy findings and chronic hydralazine exposure, should prompt consideration of hydralazine-induced ANCA-associated vasculitis rather than infection-related glomerulonephritis.

Case Report: We report the case of a 58-year-old man with a history of bicuspid aortic valve who presented with fatigue, anemia, and acute kidney injury. Blood cultures grew *Streptococcus mutans*, and echocardiography demonstrated aortic valve vegetations consistent with subacute infective endocarditis. Laboratory evaluation revealed hypocomplementemia, proteinuria, hematuria, and elevated MPO and PR3 ANCA titers. Despite appropriate antimicrobial therapy and clearance of bacteremia, renal dysfunction persisted. Renal biopsy demonstrated pauci-immune crescentic glomerulonephritis consistent with ANCA-associated vasculitis. Further review of the patient's medications revealed long-term hydralazine therapy, raising concern for hydralazine-induced ANCA-associated vasculitis. Hydralazine was discontinued, and the patient was initiated on high-dose corticosteroid therapy with plans for additional immunosuppressive therapy following recovery from cardiac surgery. The patient subsequently underwent successful bioprosthetic aortic valve replacement with gradual stabilization of renal function.

Conclusions: This case highlights the diagnostic overlap between infective endocarditis-associated glomerulonephritis and hydralazine-induced ANCA-associated vasculitis. In patients with infective endocarditis who develop persistent renal dysfunction despite appropriate antimicrobial therapy, particularly in the setting of ANCA positivity and chronic hydralazine use, renal biopsy and medication review are essential to establish the correct diagnosis and guide appropriate management.

Keywords: Vasculitis • Endocarditis • Hydralazine • Glomerulonephritis
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954110

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Introduction

Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis is a pauci-immune, small-vessel vasculitis characterized by autoantibodies directed against myeloperoxidase (MPO) and proteinase 3 (PR3), frequently leading to rapidly progressive glomerulonephritis and significant renal morbidity if not promptly recognized and treated [1]. While primary forms of ANCA-associated vasculitis are well described, drug-induced variants have increasingly been reported, particularly in association with medications such as hydralazine, propylthiouracil, and minocycline [2,3]. Hydralazine-induced ANCA-associated vasculitis is often associated with high titers of MPO antibodies and can present with multi-organ involvement, most notably affecting the kidneys [2,3].

Infective endocarditis is another important cause of renal dysfunction and is commonly associated with immune complex-mediated glomerulonephritis [4]. This process is typically characterized by hypocomplementemia and deposition of immune complexes within the glomeruli, leading to hematuria, proteinuria, and varying degrees of renal impairment [4]. Notably, infective endocarditis has also been associated with positive ANCA serologies, further complicating the clinical picture and creating significant diagnostic uncertainty [5].

The coexistence of infective endocarditis and ANCA positivity presents a well-recognized diagnostic challenge, as infection itself can induce autoantibody formation and mimic primary or drug-induced vasculitis [5]. Differentiating between infection-related glomerulonephritis and ANCA-associated vasculitis is critical, as management strategies differ substantially. Infection-related glomerulonephritis is primarily treated with appropriate antimicrobial therapy, whereas ANCA-associated vasculitis requires immunosuppressive treatment, which can be detrimental if initiated in the setting of active infection [6].

We hypothesize that persistent renal dysfunction despite microbiologic control of infective endocarditis, together with pauci-immune biopsy findings and chronic hydralazine exposure, should raise suspicion for hydralazine-induced ANCA-associated vasculitis rather than infection-related glomerulonephritis. In patients with pauci-immune biopsy findings, ANCA positivity, and medications associated with drug-induced vasculitis such as hydralazine, hydralazine-induced ANCA-associated vasculitis should be strongly considered. This distinction is clinically important because treatment strategies differ substantially and delayed recognition can contribute to progressive renal injury.

We present a case of hydralazine-induced ANCA-associated crescentic glomerulonephritis initially attributed to infective endocarditis-associated glomerulonephritis, highlighting the diagnostic complexity and emphasizing the importance of renal biopsy, medication review, and multidisciplinary management in guiding appropriate therapy [7,8].

Case Report

A middle-aged man with a history of bicuspid aortic valve presented with symptoms consistent with subacute infective endocarditis and was found to have *Streptococcus mutans* bacteremia. Transthoracic and transesophageal echocardiography revealed an 11-mm mobile vegetation attached to the aortic valve with associated valvular dysfunction. Infective endocarditis was diagnosed on 2/7/2026. The patient subsequently underwent bioprosthetic aortic valve replacement on 2/25/2026.

During hospitalization, he developed acute kidney injury with a peak creatinine of approximately 2.6 mg/dL from a baseline of 1.4 mg/dL. Laboratory evaluation demonstrated hematuria, proteinuria, low complement levels, and elevated MPO and PR3 ANCA titers. Serial laboratory trends are summarized in **Table 1**. A clinical timeline of the patient's hospital course is presented in **Figure 1**. Given the presence of infective endocarditis, the initial working diagnosis was post-infectious glomerulonephritis.

Despite appropriate antibiotic therapy and clearance of bacteremia, renal dysfunction persisted. A renal biopsy was performed and demonstrated crescentic glomerulonephritis, consistent with ANCA-associated vasculitis. Representative renal biopsy findings are shown in **Figures 2-4**. Further review of the patient's medications revealed long-term hydralazine therapy, raising concern for hydralazine-induced ANCA-associated vasculitis.

Hydralazine was discontinued, and the patient was initiated on high-dose corticosteroid therapy with a planned taper. Renal biopsy was performed on 2/13/2026, 12 days before bioprosthetic aortic valve replacement on 2/25/2026, establishing the diagnosis of pauci-immune crescentic glomerulonephritis prior to surgical intervention. At the time of surgery, renal function had improved from a peak creatinine of 2.78 mg/dL to 1.49 mg/dL preoperatively. Following surgery, creatinine remained relatively stable at 1.60 mg/dL on 2/28/2026 and at discharge on 3/5/2026. Following surgery, renal function stabilized with improvement in clinical status, and outpatient follow-up was arranged for consideration of additional immunosuppressive therapy.

Discussion

This case highlights the diagnostic complexity of differentiating infection-related glomerulonephritis from drug-induced

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Table 1. Serial laboratory values during hospitalization demonstrating acute kidney injury, inflammatory markers, and ANCA trends. Serial laboratory data from admission through discharge showing initial acute kidney injury with elevated creatinine and BUN, associated anemia, and inflammatory response (elevated CRP), followed by gradual improvement. Complement levels (C3, C4) remained low, and both MPO and PR3 ANCA titers were initially elevated with a downward trend after treatment and discontinuation of hydralazine. Urine studies demonstrated significant proteinuria.

Laboratory test	Units	Reference range	2/7/26 (Day 1)	2/11/26	2/15/26	2/20/26	2/24/26	2/28/26	3/5/26 (Discharge)
Creatinine	mg/dL	0.74-1.35	2.78	2.35	2.11	2.25	1.49	1.60	1.60
BUN	mg/dL	7-20	45	52	45	40	49	46	40
eGFR (CKD-EPI)	mL/min/1.73 m^2	≥90	25	31	35	32	55	50	50
Hemoglobin	g/dL	13.5-17.5 (M), 12.2-16.0 (F)	7.2	7.1	7.4	7.3	7.6	7.9	7.9
WBC	K/µL	4.0-10.0	8.66	11.36	10.44	6.59	5.52	5.60	5.60
Platelets	K/µL	150-400	221	248	266	298	312	330	335
CRP	mg/L	<5.0	118	96	74	52	28	18	10
Albumin	g/dL	3.5-5.0	2.9	2.8	2.7	2.6	2.8	2.9	3.0
C3 complement	mg/dL	90-180	62 (L)	58 (L)	55 (L)	57 (L)	63 (L)	69 (L)	75 (L)
C4 complement	mg/dL	10-40	6 (L)	5 (L)	5 (L)	6 (L)	7 (L)	8 (L)	10 (low-normal)
MPO antibody	U/mL	≤2.0	7.8 (H)	6.9 (H)	6.1 (H)	5.7 (H)	5.5 (H)	4.2 (H)	3.8 (H)
PR3 antibody	U/mL	≤2.0	4.5 (H)	4.1 (H)	3.2 (H)	2.8 (H)	1.8 (N)	1.4 (N)	1.2 (N)
Urine ACR	mg/g	<30	3440 (H)	—	—	—	—	—	—
Urine PCR	g/g	<0.2	0.70 (H)	—	—	—	—	—	—

Abbreviations: BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein; MPO, myeloperoxidase; PR3, proteinase 3; ACR, albumin-to-creatinine ratio; PCR, protein-to-creatinine ratio; L, low; H, high; N, normal.

antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis in the setting of active infective endocarditis. Both entities can present with acute kidney injury, hematuria, proteinuria, and overlapping serologic findings, including hypocomplementemia and positive ANCA titers, making clinical distinction particularly challenging [1,3,6].

Infective endocarditis-associated glomerulonephritis is typically mediated by immune complex deposition, leading to complement activation and glomerular inflammation [1]. Patients often demonstrate low complement levels and may have circulating immune complexes, with renal function frequently improving following appropriate antimicrobial therapy and source control [1]. However, ANCA positivity has been increasingly recognized in the setting of infective endocarditis, further complicating the diagnostic picture and raising the potential for misclassification as primary or secondary vasculitis [1,6].

In contrast, ANCA-associated vasculitis is characterized by a pauci-immune necrotizing glomerulonephritis, most commonly associated with antibodies directed against MPO or PR3 [2,8]. Drug-induced ANCA-associated vasculitis, particularly related to hydralazine, has been well described and is often associated with high titers of MPO antibodies, although dual positivity for MPO and PR3 can also occur [4,5,7]. These cases may also demonstrate hypocomplementemia, further overlapping with infection-related processes and complicating clinical interpretation [3,7].

Hydralazine-induced ANCA-associated vasculitis is thought to result from drug-induced immune dysregulation, leading to autoantibody formation and small-vessel inflammation [4,5]. Risk factors include prolonged exposure and higher cumulative doses, although presentations can vary [4]. Renal involvement is common and can manifest as rapidly progressive glomerulonephritis, necessitating prompt recognition and intervention [5].

This case supports the hypothesis that persistent renal dysfunction despite microbiologic control of infective endocarditis should prompt diagnostic reassessment for hydralazine-induced

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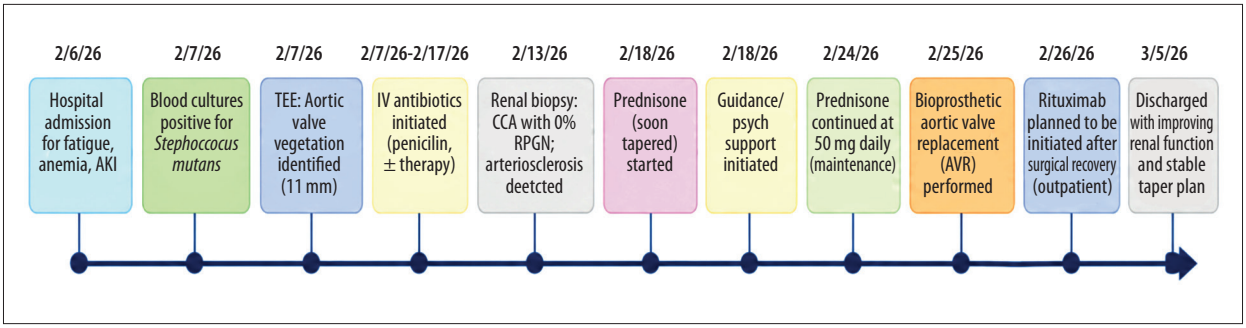

Figure 1. Clinical timeline illustrating key diagnostic and therapeutic events during hospitalization. The patient was admitted on 2/6/26 with fatigue, anemia, and acute kidney injury. On 2/7/26, blood cultures grew *Streptococcus mutans* and transesophageal echocardiography identified an 11-mm aortic valve vegetation. Intravenous antibiotic therapy was initiated. Renal biopsy performed on 2/13/26 demonstrated pauci-immune crescentic glomerulonephritis consistent with ANCA-associated vasculitis. Long-term hydralazine therapy was identified and subsequently discontinued. High-dose prednisone therapy was initiated with a planned taper and later continued at 50 mg daily. The patient underwent bioprosthetic aortic valve replacement on 2/25/26 and was discharged on 3/5/26 with improving renal function, a stable taper plan, and outpatient follow-up for planned rituximab initiation after recovery from cardiac surgery. CCA, cellular crescents absent; RPGN, rapidly progressive glomerulonephritis; TEE, transesophagea/ echocardiogram; IV, intravenous; AKI, acute kidney injury. Note: Immunosuppression during hospitalization consisted of prednisone only. Rituximab was planned to be initiated after recovery from cardiac surgery.

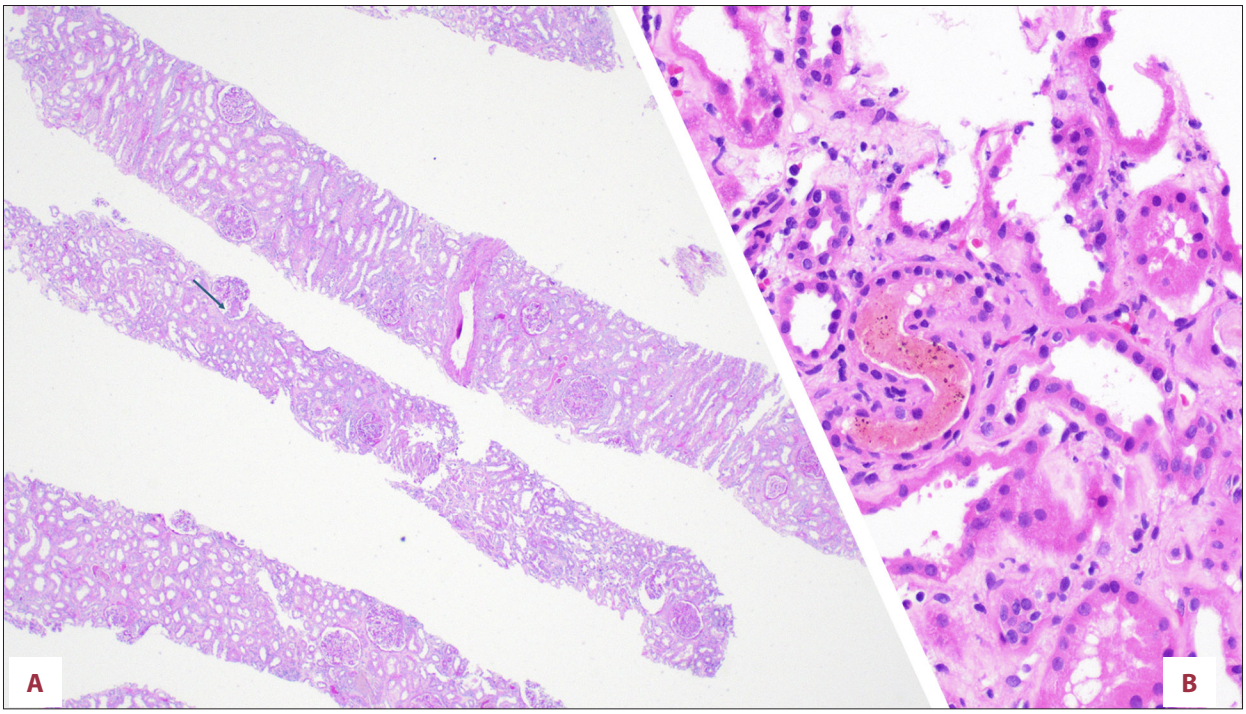

Figure 2. Renal biopsy demonstrating crescentic glomerulonephritis on light microscopy. (**A**) Low-power view of renal cortex showing glomeruli with crescent formation and surrounding inflammatory changes. (**B**) High-power view demonstrating cellular crescent formation with glomerular injury and inflammatory infiltrates, consistent with pauci-immune crescentic glomerulonephritis seen in ANCA-associated vasculitis.

ANCA-associated vasculitis when pauci-immune biopsy findings and chronic hydralazine exposure are present. Although infective endocarditis itself can produce ANCA positivity and glomerulonephritis-like findings, several features in this case favored hydralazine-induced ANCA-associated vasculitis as the most likely explanation for the patient's persistent renal dysfunction. These included chronic hydralazine therapy, dual MPO/PR3 ANCA positivity, persistent kidney injury despite microbiologic clearance of bacteremia, and pauci-immune crescentic findings on renal biopsy. Together, these findings supported

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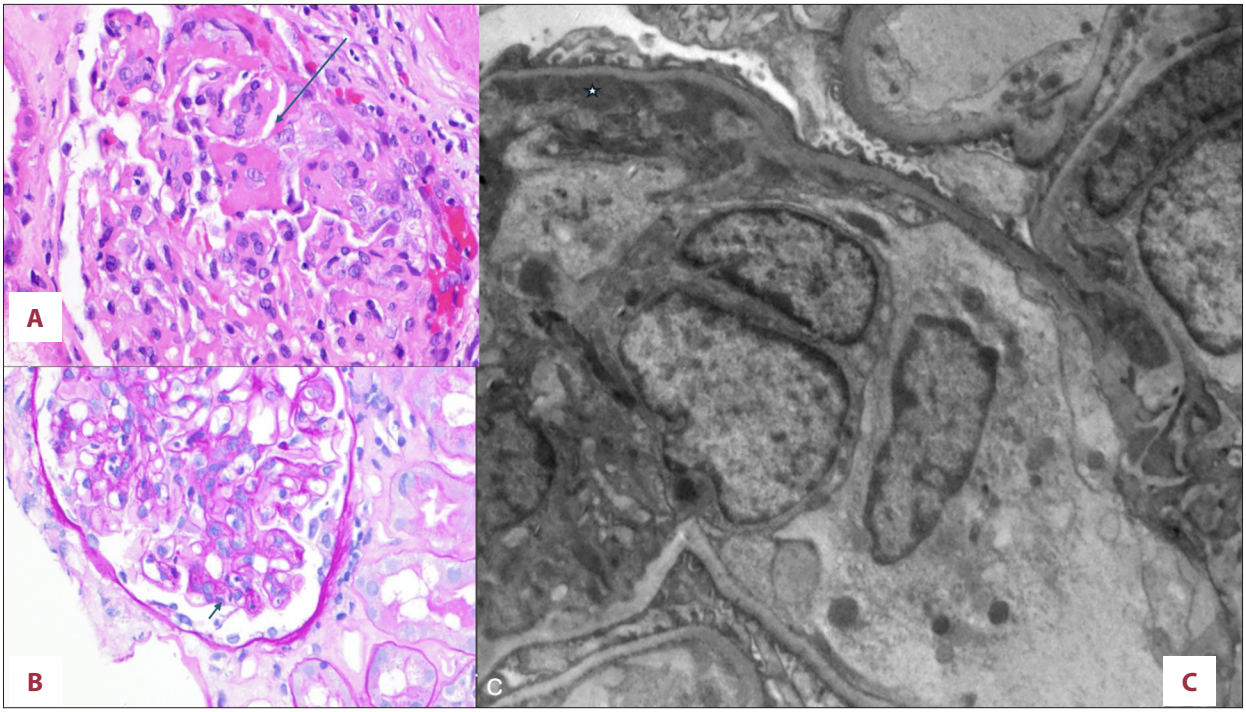

Figure 3. Multimodal renal biopsy findings including light microscopy and electron microscopy in ANCA-associated crescentic glomerulonephritis. (A) Light microscopy showing glomerular injury with crescent formation and inflammatory cell infiltration (arrow). (B) Periodic acid–Schiff (PAS) stain demonstrating glomerular structural changes without significant immune complex deposition, supporting a pauci-immune pattern. (C) Electron microscopy revealing absence of electron-dense immune deposits, consistent with ANCA-associated pauci-immune crescentic glomerulonephritis.

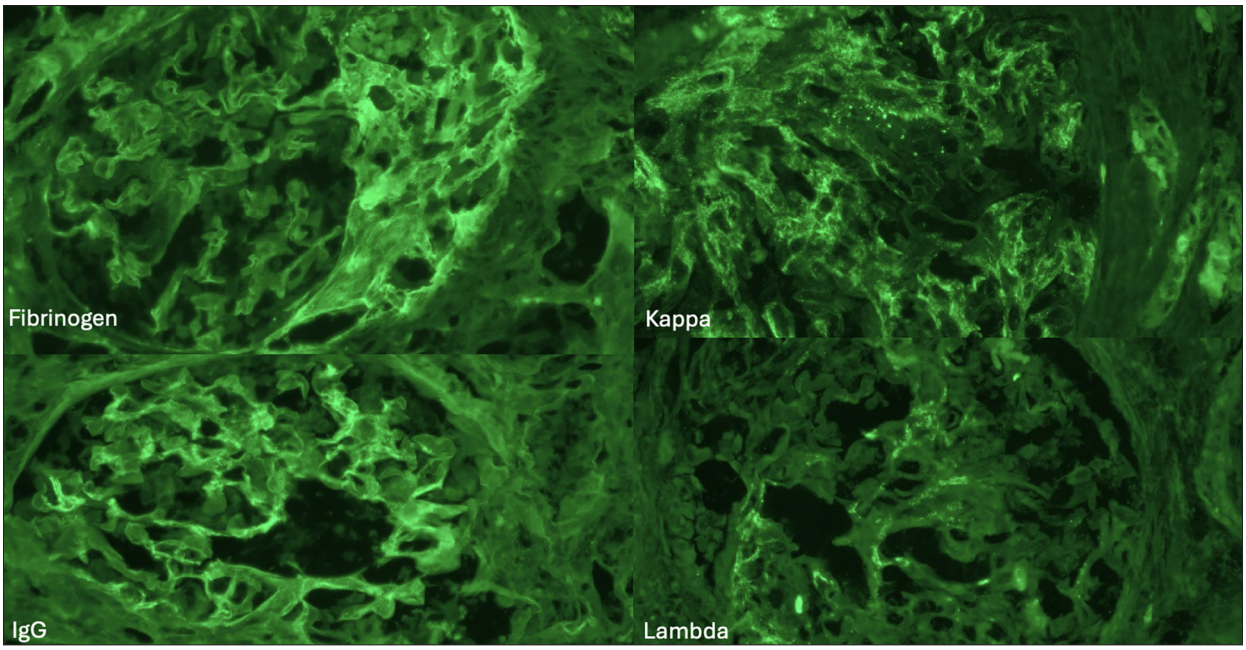

Figure 4. Immunofluorescence findings in renal biopsy demonstrating pauci-immune pattern. Immunofluorescence microscopy showing staining for fibrinogen, IgG, kappa, and lambda light chains without significant immune complex deposition, consistent with pauci-immune crescentic glomerulonephritis seen in ANCA-associated vasculitis.

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hydralazine-induced ANCA-associated vasculitis as the leading diagnosis in a complex overlapping clinical context.

The incremental contribution of this case lies in demonstrating that persistent renal dysfunction despite microbiologic control of infective endocarditis should prompt renewed diagnostic evaluation rather than continued attribution to infection alone. In particular, this case illustrates the added diagnostic value of integrating dual MPO/PR3 ANCA positivity, hypocomplementemia, pauci-immune renal biopsy findings, and careful medication review to identify hydralazine-induced ANCA-associated vasculitis despite concurrent infective endocarditis.

Nevertheless, the limitations of a single-case report should be acknowledged. Infective endocarditis can independently trigger ANCA positivity and immune-mediated renal injury, and contributory effects from infection-associated immune activation cannot be completely excluded. Therefore, the findings presented here should be interpreted as supporting hydralazine-induced ANCA-associated vasculitis as the most likely explanation within a complex overlapping clinical scenario rather than establishing definitive causality.

Renal biopsy remains the gold standard for distinguishing between these entities. Immune complex-mediated glomerulonephritis, as seen in infective endocarditis, typically demonstrates granular immune deposition on immunofluorescence, whereas ANCA-associated vasculitis is characterized by a pauci-immune pattern with minimal or absent immune deposits and the presence of crescent formation on light microscopy [2,4]. In this case, the finding of pauci-immune crescentic glomerulonephritis was critical in redirecting the diagnosis toward ANCA-associated vasculitis after renal dysfunction persisted despite microbiologic control of infection.

Accurate differentiation between these conditions is essential, as management strategies differ significantly. Infection-related glomerulonephritis is primarily treated with targeted antimicrobial therapy and supportive care, whereas ANCA-associated vasculitis may require immunosuppressive therapy, including corticosteroids and, in some cases, agents such as rituximab [3,8]. Initiating immunosuppression in the presence of uncontrolled infection can be harmful, underscoring the importance of diagnostic precision and multidisciplinary coordination.

The educational value of this case lies in the substantial overlap between infective endocarditis-associated glomerulonephritis and hydralazine-induced ANCA-associated vasculitis. The coexistence of hypocomplementemia, dual MPO/PR3 positivity, active infective endocarditis, and persistent renal dysfunction despite microbiologic control created significant diagnostic uncertainty. This case demonstrates how renal biopsy and careful medication review can help clarify the diagnosis and redirect management in complex overlap presentations.

This case underscores the importance of maintaining a broad differential diagnosis in patients with infective endocarditis who develop persistent or worsening renal dysfunction despite appropriate therapy. Early renal biopsy, careful medication review, and close collaboration among nephrology, infectious disease, and cardiology teams are essential to guide appropriate management and optimize clinical outcomes.

Conclusions

This case supports the hypothesis that persistent renal dysfunction despite microbiologic control of infective endocarditis, together with pauci-immune biopsy findings and chronic hydralazine exposure, should prompt consideration of hydralazine-induced ANCA-associated vasculitis rather than infection-related glomerulonephritis. This case highlights the importance of medication review and biopsy-driven diagnostic reassessment when clinical improvement does not parallel microbiologic control. In patients with infective endocarditis who continue to have persistent renal dysfunction despite appropriate antimicrobial therapy and biopsy findings consistent with pauci-immune disease, early recognition of hydralazine-induced ANCA-associated vasculitis may facilitate appropriate management, including consideration of immunosuppressive therapy in addition to antimicrobial treatment and source control.

Institution Where Work Was Done

Henry Ford St. John Hospital, Detroit, MI, USA.

Patient Consent

Informed patient consent was obtained for publication of this case report.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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