A 69-Year-Old Man with Progressive Dyspnea, Purpuric Rash, Bilateral Distal Lower Extremity Numbness, and Acute Kidney Injury

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Abstract

Cryoglobulinemic vasculitis is a systemic disease that involves multiple organ systems but remains diagnostically challenging. We report the case of a 69-year-old man who presented with progressive dyspnea over 1 week, followed by the development of a purpuric rash, bilateral distal lower extremity numbness, and acute kidney injury (AKI). Extensive evaluation by a multidisciplinary team excluded malignancy and infectious causes. Further investigations revealed evidence of systemic vasculitis, consistent with a diagnosis of cryoglobulinemic vasculitis.

Key words: cryoglobulinemia, cryoglobulinemic vasculitis, rituximab

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Statement of ethics

The above study involving human participants was planned, conducted, and reported in accordance with the World Medical Association Declaration of Helsinki.

Patient informed consent

Written informed consent was obtained from the patient to publish this case report and all accompanying images.

Disclosure statement

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Conflict of interest

The authors declare that there is no conflict of interest.

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Introduction

Cryoglobulinemic vasculitis is a multisystem, immune complex–mediated small-vessel vasculitis. It is characterized by the deposition of cryoglobulins—immunoglobulins that precipitate at temperatures below 37 °C and redissolve upon warming. It is a rare disorder, with an estimated prevalence of fewer than 5 cases per 10,000 people.[1] Organ involvement is typically multisystemic, with common manifestations including palpable purpura and non-blanching erythematous papules on the skin, membranoproliferative glomerulonephritis (MPGN) in the kidneys, mononeuritis multiplex or sensorimotor polyneuropathy affecting the peripheral nervous system, and arthralgia or non-erosive arthritis in the joints. Pulmonary or cardiac involvement may occur in severe or fulminant cases.

Case summary

A 69-year-old Thai man presented with progressive dyspnea that had worsened over 1 week prior to hospital admission. One month prior to admission, the patient developed exertional dyspnea, orthopnea, bilateral pedal edema, and oliguria. He was diagnosed with acute decompensated heart failure precipitated by salt and water retention and hypertensive emergency, along with AKI secondary to cardio-renal syndrome. Two weeks prior to admission, after being discharged home, he developed a productive cough with whitish sputum occasionally streaked with fresh blood. He denied chest pain, palpitations, dizziness, or syncope. Urinary output was reduced. One week prior to admission, he developed low-grade fever, bilateral pedal swelling, and worsening dyspnea with orthopnea. He denied chest pain, hemoptysis, joint pain, or skin rash at that time. His urine was noted to be foamy, without visible hematuria or dysuria. Three days into hospitalization, the patient developed a non-pruritic, non-ulcerative rash over his arms and thighs, without skin discoloration or other cutaneous changes.

The patient had a complex medical history, including colonic adenocarcinoma (status post left hemicolectomy with adjuvant chemotherapy in 2005), chronic hepatitis B virus (HBV) infection, hepatic hemangioma, triple-vessel coronary artery disease, and paroxysmal atrial fibrillation. Current medications included low-dose aspirin, clopidogrel, folic acid, atorvastatin, isosorbide mononitrate, metoprolol tartrate, losartan, omeprazole, spironolactone, empagliflozin, and tenofovir alafenamide. There was a history of cannabis usage for 30 years, which ceased 4 months prior. A remote history of intermittent usage of amphetamines, cocaine, and kratom over a decade ago was also reported, with no recurrence since.

The blood pressure was elevated at 161/85 mmHg, pulse rate of 90 beats per minute, body temperature of 36.7°C, and oxygen saturation of 92% on room air. He was alert, fully conscious, and cooperative. Physical examination of the head and neck revealed no facial rash, periorbital edema, or alopecia. The auricular cartilage and ear lobules were normal in contour, and no saddle nose deformity was observed. Cardiovascular examination showed elevated jugular venous pressure (5 cm above the sternal angle) with no murmurs or bruits. Peripheral pulses were full and symmetric. On respiratory examination, fine crepitations were heard at the bilateral lower lung fields. Abdominal examination was unremarkable, with no hepatosplenomegaly. Examination of the extremities revealed bilateral pedal edema. There were no joint swellings or deformities. Cutaneous examination showed non-pruritic, non-blanchable erythematous papules and macules over the chest, abdomen, back, and both thighs. A cluster of vesicular lesions was observed on the left lower back. No petechiae, ecchymoses, or purpura were noted. No lymphadenopathy was palpable. Neurological examination revealed bilateral extensor hallucis longus grade IV. Pinprick sensation was preserved, and no cerebellar signs were noted.

Initial laboratory investigations revealed normocytic anemia, with a hemoglobin level of 7.9 g/dL and a hematocrit of 23.5%. The white blood cell count was 8,500 cells/μL, comprising 82% neutrophils and 11.4% lymphocytes. The platelet count was within normal limits at 200,000 cells/μL. Blood chemistry demonstrated markedly elevated blood urea nitrogen at 71 mg/dL and serum creatinine at 3.23 mg/dL, consistent with azotemia. Serum electrolytes were as follows: sodium 142 mEq/L, potassium 3.5 mEq/L, chloride 110 mEq/L, and bicarbonate 21 mEq/L. These findings were indicative of impaired renal function with mild metabolic acidosis.

The clinical presentation suggested a systemic inflammatory process involving multiple organ systems, including the pulmonary, renal, dermatologic, and neurologic systems. This constellation of findings was highly suggestive of a systemic vasculitic syndrome, with possible differential diagnoses including antineutrophil cytoplasmic antibody–associated vasculitis such as granulomatosis with polyangiitis, microscopic polyangiitis, and eosinophilic granulomatosis with polyangiitis; immune complex–mediated vasculitis such as cryoglobulinemic vasculitis and immunoglobulin A (IgA) vasculitis; and systemic autoimmune diseases such as systemic lupus erythematosus–associated vasculitis. Additionally, pulmonary infection with acute decompensated heart failure should be considered in light of the patient's known triple-vessel coronary artery disease, atrial fibrillation, and bilateral pulmonary crepitations.

Diagnostic testing revealed mild hypoalbuminemia (albumin 2.64 g/dL) with preserved hepatic synthetic and excretory functions. Urinalysis demonstrated a specific gravity of 1.014 and a pH of 5.0, with 3+ protein, 3+ blood, >100 red blood cells/high-power field (HPF), and 3–5 white blood cells/HPF. The urine protein-to-creatinine ratio was 2.9, consistent with significant proteinuria and active urinary sediment. Cardiac and pulmonary evaluations revealed a normal sinus rhythm on a 12-lead electrocardiogram. Chest radiography showed bilateral blunting of the costophrenic angles, suggesting pleural effusions. Transthoracic echocardiography performed 5 months after the previous study showed mildly impaired left ventricular systolic function with a left ventricular ejection fraction of 42.5%, not significantly changed from 38.4% previously. Regional wall motion abnormalities persisted, with hypokinesia of the basal lateral wall and akinesia of the inferior wall from basal to mid segments, mid lateral wall, and apical inferolateral segment. Right ventricular size and function remained normal. Mild–moderate secondary mitral regurgitation was present, slightly increased from the previously mild degree. Pulmonary pressures remained within the low-probability range for pulmonary hypertension. Chest computed tomography with computed tomography angiography of the aorta identified a 0.5 × 1.0 cm penetrating atherosclerotic ulcer at the aortic arch, diffuse emphysematous changes, moderate bilateral pleural effusions, fibrosis with traction bronchiectasis in the right upper lobe (likely post-infectious), and multiple mediastinal and hilar lymph nodes suggestive of reactive lymphadenopathy. Microbiologic studies showed no growth on blood cultures. Sputum culture grew extended-spectrum β-lactamase (ESBL)-producing *Escherichia coli*, and urine culture grew *Enterococcus faecalis*. Tests for influenza A/B and severe acute respiratory syndrome coronavirus 2 were negative. Viral serologies, including human immunodeficiency virus antibody, hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV) antibody, and rapid plasma reagin test, were also negative, and HBV deoxyribonucleic acid (DNA) viral load was < 10 IU/mL. Tzanck smear showed no multinucleated giant cells. Diagnostic thoracentesis revealed pleural fluid consistent with a transudative effusion based on Light's criteria. Microbiological studies, including Gram stain and culture, showed no evidence of organisms, and cytological examination was negative for malignancy. Serologic evaluation showed low complement levels (complement component 3 [C3] 48 mg/dL, complement component 4 [C4] <2.9 mg/dL) and a positive rheumatoid factor (RF) 1:64. Antinuclear antibody, anti–double-stranded DNA, anti–proteinase 3, and anti–myeloperoxidase were negative. Cryoglobulin testing was positive (cryocrit <1%) and the sample was sent to an outside laboratory. Antiphospholipid antibodies were negative. Immunoglobulin profiling revealed elevated immunoglobulin M (IgM) 317 mg/dL with normal IgA 298 mg/dL and immunoglobulin G (IgG) 1,261 mg/dL. Immunofixation identified monoclonal IgM–kappa, though no M-spike was seen on serum protein electrophoresis. Skin biopsy from the left thigh demonstrated regular acanthosis and superficial perivascular lymphocytic infiltration, with rare neutrophils in the dermis, consistent with an old lesion of leukocytoclastic vasculitis (LCV). Bone marrow aspiration and biopsy showed reactive marrow with cellularity of 40%, myeloid-to-erythroid ratio of 2:1, and no evidence of hematologic malignancy on flow cytometry. Nerve conduction studies indicated moderate to severe sensorimotor axonal polyneuropathy, predominantly affecting the lower limbs, with the possibility of confluent mononeuritis multiplex. Sural nerve biopsy revealed small lymphoid infiltrates within epineurial vessels, suspicious for vasculitic neuropathy. No granulomas or atypical cells were identified. Kidney biopsy revealed IgM-dominant MPGN, with histopathologic features detailed in **Figure 1**.

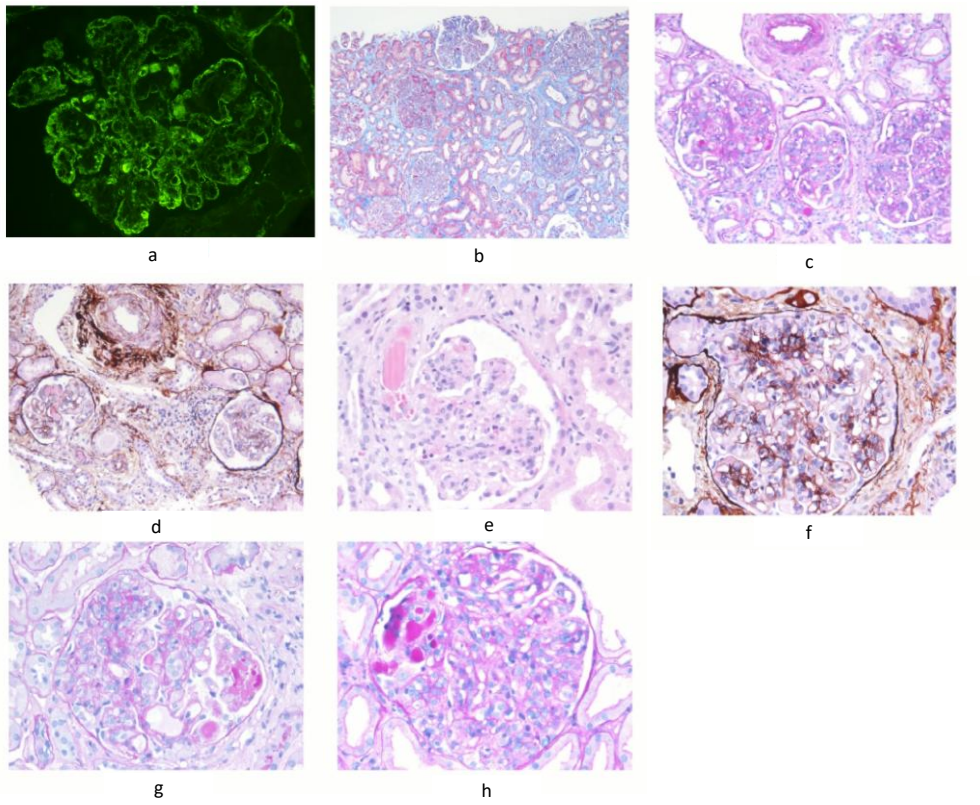

Figure 1 Kidney biopsy consisted of 5 cores: 3 cortical, 1 corticomedullary, and 1 fibrous tissue. Among 38 glomeruli, 4 were globally sclerotic. The mesangium showed mild matrix expansion and mild to moderate hypercellularity without nodular sclerosis (c). The glomerular basement membranes exhibited diffuse segmental to global splitting, with no spikes or holes (d, f). Endocapillary hypercellularity was present in 34 glomeruli, with occasional eosinophilic intracapillary plugs (g). No segmental sclerosis, crescents, fibrin, necrosis, adhesion, or hyalinosis was seen. Bowman's spaces were preserved. The tubulointerstitial compartment showed 5–10% interstitial fibrosis and tubular atrophy (IFTA), with mild lymphocytic infiltration in fibrotic areas and focal edema (b). Acute tubular injury was widespread, with scattered proteinaceous casts (e). Vessels showed mild arteriolar medial thickening and focal eosinophilic deposits; interlobular arteries were unremarkable (h). Large arteries were not sampled. Congo red staining was negative. Immunofluorescence demonstrated 2+ granular mesangial and capillary wall staining for IgM, kappa, and lambda light chains, with trace IgG and C3 (a). IgA and C1q were negative. Findings supported polyclonal immune complex deposition, predominantly IgM. The biopsy showed mild to moderate active glomerular injury with diffuse endocapillary hypercellularity and intracapillary immune deposits, along with mild chronicity (4/38 glomerulosclerosis, 5–10% IFTA). The absence of crescents, necrosis, and significant complement deposition argued against lupus nephritis or crescentic glomerulonephritis. Findings were consistent with immune complex–mediated glomerulonephritis in the setting of cryoglobulinemic vasculitis. The pathological diagnosis was IgM-dominant MPGN.

Discussion

This patient was diagnosed with cryoglobulinemic vasculitis, based on evidence of multiorgan involvement affecting the kidneys, skin, and peripheral nervous system. Renal biopsy revealed IgM-dominant MPGN, consistent with immune complex-mediated glomerular injury. A skin biopsy demonstrated LCV, and nerve conduction studies supported the presence of sensorimotor axonal polyneuropathy, further corroborated by sural nerve biopsy findings suspicious for vasculitic neuropathy. Serological testing revealed a positive RF, monoclonal IgM kappa on immunofixation, and

markedly decreased C4 levels. Taken together, these findings were characteristic of type II mixed cryoglobulinemia, with a likely secondary etiology—most plausibly triggered by previous or occult infectious processes. Given the patient's history of chronic HBV infection, its possible contribution to cryoglobulinemic vasculitis warranted consideration. HBsAg was negative and HBV DNA remained <10 IU/mL, indicating effective viral suppression and absence of active infection, although occult infection or immune complex formation from prior exposure could not be excluded. When initiating strong immunosuppressive therapy, close monitoring of liver function test, HBsAg, and HBV DNA levels, together with continuation of antiviral treatment, was essential to minimize the risk of viral reactivation.[1] In this patient, the secondary causes of cryoglobulinemic vasculitis were identified as *Escherichia coli* (ESBL–producing) pneumonia and *Enterococcus faecalis* urinary tract infection. The distinguishing features of each cryoglobulinemia type are summarized in **Table 1**.

Table 1 Characteristics of Cryoglobulinemia by Type, adapted from Cacoub et al. (2024).[2]

Characteristic	Type I Cryoglobulinemia	Type II Cryoglobulinemia	Type III Cryoglobulinemia
Range of cryoglobulin levels in serum — g/liter	1–30	0.5–2	0.05–0.5
Findings on serum protein electrophoresis	Monoclonal spike	Monoclonal spike and polyclonal elevation of gammaglobulins	Polyclonal elevation of gammaglobulins
Serum protein immunofixation	IgG (most frequent), IgM, IgA (least frequent)	Typically IgM kappa	None
RF activity	Very rare	Frequent	Variable
Low C4 level	Very rare	Frequent	Variable
Skin biopsy	Noninflammatory thrombotic lesions, with downstream infarction or hemorrhage	LCV, hyaline thrombi	LCV, hyaline thrombi
Peripheral-nerve biopsy	Pauci-inflammatory occlusive lesions with neuronal ischemia	• Lymphocytic infiltrate around epineurial vessels, with axonal degeneration of affected nerves (vasa vasorum); • Necrotizing vasculitis or demyelination might be present	• Lymphocytic infiltrate around epineurial vessels, with axonal degeneration of affected nerves (vasa vasorum); • Necrotizing vasculitis or demyelination might be present
Kidney biopsy	• Thrombotic and hypocellular glomerular lesions; • Type I MPGN may occur	• Type I MPGN, endocapillary proliferation, deposits of subendothelial or intraluminal immune complexes (or both); • Electron microscopy: double-contour pattern of the glomerular basal membrane, microtubular immunoglobulin deposits; • Mesangial proliferative glomerulopathy, intraglomerular hyaline thrombi, and vasculitis	• Type I MPGN, endocapillary proliferation, deposits of subendothelial or intraluminal immune complexes (or both); • Electron microscopy: double-contour pattern of the glomerular basal membrane, microtubular immunoglobulin deposits; • Mesangial proliferative glomerulopathy, intraglomerular hyaline thrombi, and vasculitis

		with fibrinoid necrosis might be found	with fibrinoid necrosis might be found
Direct immunofluorescence	Monoclonal immunoglobulin, usually without complement deposition	Deposits of IgM, IgG, or C3 (or a combination of the three)	Deposits of IgM, IgG, or C3 (or a combination of the three)

Cryoglobulinemia is classified into 3 types.[1] Type I involves monoclonal immunoglobulins, often associated with lymphoproliferative disorders.[1] Types II and III are mixed cryoglobulinemia, commonly linked to chronic infections—especially HCV—autoimmune diseases, and lymphoproliferative disorders.[1] Type II features monoclonal IgM with RF activity binding polyclonal IgG, while type III involves polyclonal IgM and IgG.[1, 2] Clinical manifestations of mixed cryoglobulinemia include Meltzer's triad—palpable purpura, arthralgia, and weakness.[1]

The current case demonstrated several classic features of mixed cryoglobulinemia, including purpuric rash, MPGN, and sensorimotor polyneuropathy, in conjunction with serologic and immunopathologic confirmation. While HCV is the most frequently implicated etiology, this patient's virologic testing was negative for both HCV and HBV, with the latter well controlled on tenofovir therapy. A thorough evaluation excluded other infectious, autoimmune, or malignant causes. The patient had a remote history of substance use—including cannabis, amphetamines, cocaine, and kratom (*Mitragyna speciosa*), plant native to Southeast Asia which contains psychoactive alkaloids with stimulant and opioid-like effects,[3] which had been discontinued for months to years. These are not established triggers of cryoglobulinemia. Although rare, drug-induced cryoglobulinemic vasculitis has been reported in a single case of amphetamine use, suggesting possible immune complex–mediated endothelial injury, though causality remains unproven. Ramos et al. described a man with long-term intravenous heroin use who developed MPGN and cutaneous vasculitis, later diagnosed as mixed cryoglobulinemia.[4] Infectious triggers of cryoglobulinemia extend beyond HCV and HBV. Case reports have linked cryoglobulinemic vasculitis to pathogens such as *Bartonella*, parvovirus B19, *Leishmania*, hepatitis E virus (HEV), *Schistosoma mansoni*, and various forms of bacterial endocarditis, as summarized in **Table 2**.[2] Many cases showed MPGN on renal biopsy, often with LCV and neurological symptoms, similar to the findings in this patient.

Table 2 Review of infectious causes

Study	Age/ Sex	Pathogen	Type of Cryoglobulinemia	Clinical Features	Biopsy Findings	Treatment	Outcome
Vivekananthan et al. (2022)[5]	17/F	*Bartonella* spp.	Type III (polyclonal IgG)	Weight loss, rash, splenomegaly, renal impairment	Focal proliferative glomerulonephritis, C3/IgM positive	Antibiotics, steroids	Full recovery
Bailly et al. (2020)[6]	57/M	Parvovirus B19	Type III (mixed)	Recurrent AKI, rash, arthralgia	Acute postinfectious glomerulonephritis, MPGN	Subcutaneous immunoglobulin, plasmapheresis, rituximab	Renal recovery
Padrón Romero et al. (2019)[7]	69/M	*Leishmania* spp.	Type II (mixed)	Renal failure, skin purpura, anemia	MPGN Type I	Amphotericin B	Full recovery
Bazerbachi et al. (2017)[8]	24-58	HEV	Type II/III (mixed)	Renal failure, thrombocytopenia	MPGN, nephrosclerosis	Ribavirin, peg-IFN	9 improved, 3 ESRD, 1 Chronic renal failure, 1 death

NasrAllah et al. (2015)[9]	37/M	*Schistosoma mansoni*	Type II (monoclonal IgM + polyclonal IgG)	Chest pain, hemoptysis, gangrene	MPGN, hyaline thrombi	Praziquantel, steroids, cyclophosphamide	Death from sepsis
Beydon et al. (2022)[10]	47-78	*Bartonella/ Coxiella*	Type II/III	Renal failure, skin purpura, anemia, arthralgia, fever, hematuria, proteinuria	Pauci-immune glomerulonephritis, immune complex glomerulonephritis	Antibiotics, steroids	Recovery if early antibiotics
Liu et al. (2020)[11]	56/M	*S. aureus* (IE)	Type III	Purpura, mild renal dysfunction	LCV	IV cefazolin + steroids	Improvement
Josephson et al. (2022)[12]	40/M, 31/F	MRSA Endocarditis	Type II/III	Purpura, glomerulonephritis, colitis, purpuric ulcers, septic emboli	LCV, vasculitis with fibrin thrombi	Daptomycin + ceftaroline, steroids	Recovery, healing ulcers
Nassih et al. (2020)[13]	8/F	Hepatitis A virus (HAV)	Type II (mixed IgM, IgA, IgG)	Vasculitic rash, arthritis, jaundice, proteinuria	LCV	Oral prednisone	Full recovery
Reinberg et al. (2024)[14]	57/M	MSSA Endocarditis + chronic HCV	Type III (polyclonal)	Meltzer's triad (purpura, weakness, arthralgia), mild AKI, Raynaud's phenomenon	LCV	Flucloxacillin + valve replacement	Complete remission after antibiotics

The pathogenesis of cryoglobulinemic vasculitis involves circulating immune complexes that activate the classical complement pathway, leading to endothelial injury and inflammation, evidenced by low C4 and histologic features such as endocapillary proliferation, double-contour glomerular basement membranes, and subendothelial immune complex deposits. Peripheral nerve involvement may present as distal symmetric polyneuropathy or mononeuritis multiplex, with perivascular lymphocytic infiltrates on histology.[1, 2]

The treatment of mixed type cryoglobulinemic vasculitis is guided by pathophysiology, etiology, and disease severity. In HCV-related cases, viral eradication with direct-acting antivirals (DAAs) is first-line, with immunosuppressive therapy for severe or life-threatening presentations. HBV-related cases require antiviral nucleos(t)ide analogues with careful monitoring during immunosuppression. Rituximab, often with short courses of glucocorticoids, is preferred for severe manifestations, including glomerulonephritis, peripheral neuropathy, and skin ulcers, in both viral and nonviral cases. Refractory or relapsing disease may require repeat rituximab or combination regimens with alkylating agents or belimumab. Plasma exchange is reserved for life-threatening situations. Relapse can occur despite viral clearance, potentially due to persistent B-cell clones, and clinical severity along with markers like RF and complement levels help predict response. Long-term follow-up is essential to monitor relapse and progression to lymphoproliferative disorders.[1, 2]

In this case, after completing antibiotic therapy with piperacillin–tazobactam for urinary tract infection and pneumonia, immunosuppressive treatment was initiated. The patient received the first dose of rituximab 560 mg (375 mg/m²) on February 20, 2025, together with prednisolone 25 mg/day (0.5 mg/kg/day), followed by rituximab 375 mg/m² administered weekly, for a total duration of 4 weeks, with the last dose administered on March 12, 2025. Prednisolone was tapered and discontinued on July 23, 2025. The patient showed marked clinical improvement: cutaneous lesions—previously described

as non-pruritic, non-blanchable erythematous papules and macules over the chest, abdomen, back, and thighs—completely resolved. Neurologic status improved, with bilateral extensor hallucis longus strength increasing from grade IV to IV+. Biochemically, serum creatinine decreased from 3.23 to 1.41 mg/dL, urine protein-to-creatinine ratio improved from 2.92 to 0.08, and complement levels partially recovered (C3 increased from 48 to 69 mg/dL, C4 from <2.9 to 4.9 mg/dL). Heart failure symptoms—attributed to salt and water retention—improved after diuretic therapy, and a plan was made to continue guideline-directed medical therapy.

Conclusion

This case highlighted the diagnostic and therapeutic complexity of cryoglobulinemic vasculitis, even when infectious etiologies were clearly identified. A high index of suspicion is warranted in patients presenting with multisystem involvement, including renal, dermatologic, and neurologic manifestations, especially when accompanied by serological evidence of cryoglobulinemia. Early diagnosis and appropriate immunosuppressive therapy can lead to favorable outcomes, although relapses and complications remain a clinical challenge.

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