

New-Onset Seizures and Acute Renal Failure: An Atypical and Severe Sarcoidosis Presentation

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Abstract

Sarcoidosis is a multisystem granulomatous disease with highly variable clinical expression. Although pulmonary involvement predominates, extrapulmonary presentations may be severe and diagnostically challenging, particularly in the absence of typical thoracic findings.

This case report describes a male in his early 30s, with mild cognitive impairment, admitted with generalized tonic-clonic seizures and encephalopathy. Clinical, laboratory, imaging, and histopathological findings were retrospectively analyzed. The patient presented with a three-month history of progressive constitutional symptoms, including weight loss, insomnia, xerosis, and lower limb edema. On admission, he developed recurrent seizures requiring sedation and mechanical ventilation. Investigation revealed severe metabolic acidosis, acute kidney injury, and pancytopenia. Neuroimaging showed nonspecific white matter changes, and electroencephalogram demonstrated epileptiform activity. Thoracoabdominal computed tomography identified splenomegaly, lymphadenopathy, and hepatic and renal lesions, without typical pulmonary involvement. Cerebrospinal fluid analysis was non-inflammatory. Further workup showed lymphocytic alveolitis with an elevated CD4/CD8 ratio and increased serum angiotensin-converting enzyme levels. Renal biopsy demonstrated granulomatous interstitial nephritis, establishing the diagnosis of systemic sarcoidosis with multiorgan involvement. High-dose corticosteroid therapy resulted in rapid clinical improvement, with recovery of renal function and resolution of encephalopathy. Temporary renal replacement therapy was required. Seizures were effectively controlled with levetiracetam.

This case illustrates a rare and severe presentation of sarcoidosis, in which life-threatening neurological and renal involvement occurred in the absence of classic pulmonary features. It emphasizes the need for early recognition of atypical multisystem presentations and the pivotal role of histological confirmation. Prompt immunosuppressive therapy can be life-saving and lead to significant clinical recovery.

Categories: Other, Emergency Medicine

Keywords: generalized tonic-clonic seizures, granulomatous interstitial nephritis, multisystem disease, severe acute kidney injury, systemic sarcoidosis

Introduction

Sarcoidosis is a multisystemic granulomatous disease of unknown cause with a wide range of clinical manifestations. The lungs, lymphatic system, skin, and eyes are predominantly affected, but the presentation of patients with sarcoidosis is highly variable, with many known clinical phenotypes [1-3]. While pulmonary involvement occurs in the majority of cases and typically guides diagnosis, nonpulmonary manifestations at presentation may lead to challenging management decisions, often resulting in diagnostic delay [4-6]. We report a case of multisystemic sarcoidosis presenting with severe acute kidney injury, seizures, and hematological involvement. It illustrates the diagnostic and therapeutic challenges of a nonpulmonary presentation in the intensive care setting and the difficulty of attributing manifestations to the disease when metabolic and disease-specific causes coexist.

Case Presentation

We present a case of a male in his early 30s with mild cognitive impairment, cared for by a first-degree relative, with no significant past medical history and no regular medication. Family and epidemiological history were non-contributory. He developed a three-month history of insomnia, anorexia with significant weight loss, cutaneous xerosis, and bilateral lower limb edema. One week before admission, he noted a progressive increase in abdominal girth.

He was brought to our emergency department following new-onset generalized tonic-clonic seizures, terminated with midazolam, without full neurological recovery and with risk of airway compromise, necessitating endotracheal intubation and mechanical ventilation. Physical examination revealed generalized edema and ichthyosiform xerosis with predominant involvement of the upper and lower limbs and flexural regions.

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Initial investigations (Table 1) revealed severe metabolic acidemia (pH = 6.8, bicarbonate = 4 mmol/L, lactate = 5.43 mmol/L), non-oliguric acute kidney injury (Kidney Disease: Improving Global Outcomes (KDIGO) stage 3, creatinine = 13.65 mg/dL) with leukocyturia, proteinuria (1.5 g/day), and uremia (321 mg/dL), and pancytopenia comprising leukopenia, normocytic normochromic anemia, and thrombocytopenia, with non-measurable haptoglobin and elevated lactate dehydrogenase [7].

Parameter	Patient value	Reference range
Arterial blood gas		
pH	6.8	7.35–7.45
O2 arterial pressure	165 mmHg	>60 mmHg
CO2 arterial pressure	26 mmHg	35–45 mmHg
Bicarbonate	4 mmol/L	22–26 mmol/L
Lactate	5.43 mmol/L	0.5–2.0 mmol/L
Renal function, electrolytes, and general chemistry		
Glucose	61 mg/dL	75–110 mg/dL
Creatinine	13.65 mg/dL	0.67–1.17 mg/dL
Urea	321 mg/dL	10–50 mg/dL
eGFR (CKD-EPI)	4 mL/min/1.73m ²	KDIGO 2022 categories
Sodium	133 mEq/L	135–145 mEq/L
Potassium	4.9 mEq/L	3.5–5.1 mEq/L
Phosphorus	9.3 mg/dL	2.7–4.5 mg/dL
Magnesium	1.60 mEq/L	1.55–2.05 mEq/L
Amylase	54 U/L	22–80 U/L
Lipase	73 U/L	7–60 U/L
Creatine kinase	58 U/L	10–172 U/L
C-reactive protein	14.2 mg/L	<3.0 mg/L
Urinalysis		
Density	1.010	1.002–1.030
pH	5.0	5.0–8.0
Ketone bodies	Absent	<10 mg/dL
Bilirubin	Absent	Absent
Urobilinogen	Absent	<1.0 mg/dL
Protein (urinalysis)	0.30 g/L	<0.15 g/L
Albumin (urinalysis)	150 mg/L	<30 mg/L
Nitrites	Absent	Absent
Leukocyturia	Present	Absent
Hematology and hemolysis workup		
Leukocyte count	1,110 cells/mm ³	4,000–11,000 cells/mm ³
Neutrophils (absolute)	740 cells/mm ³	~1,800–7,700 cells/mm ³
Lymphocytes (absolute, differential)	180 cells/mm ³	~1,000–4,000 cells/mm ³
Hemoglobin	7.3 g/dL	13.0–17.0 g/dL
Platelet count	87,000/mm ³	150,000–400,000/mm ³

Haptoglobin	Undetectable	30–200 mg/dL
Lactate dehydrogenase	357 U/L	125–220 U/L
Others		
Toxicology screen	All negative	Negative

TABLE 1: Initial investigations.

CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration (equation); eGFR, estimated glomerular filtration rate; KDIGO, Kidney Disease: Improving Global Outcomes.

Cerebral computed tomography demonstrated nonspecific white matter changes. Electroencephalography revealed right frontal epileptiform activity without active seizures or status epilepticus. Cerebrospinal fluid analysis (Table 2) excluded acute central nervous system infection.

Parameter	Patient value	Reference range
CSF appearance	Clear, no visible particulate matter	Clear
CSF glucose	73 mg/dL*	~60–70% of concurrent serum glucose
CSF protein	0.57 g/L	0.15–0.45 g/L
CSF erythrocytes	200 cells/mm³	0 (none/rare)
CSF leukocytes	3 cells/mm³ (1 PMN, 2 mononuclear)	0–5 cells/mm³
CSF Gram stain, culture, and multiplex PCR panel ^a	All negative	Negative

TABLE 2: Cerebrospinal fluid analysis.

^a Chlamydia pneumoniae, Mycoplasma pneumoniae, CMV, enterovirus, parvovirus B19, adenovirus, parechovirus, BK/JC polyomavirus, HHV-6, HSV-1, HSV-2, and VZV.

* Concurrent serum glucose was 85 mg/dL.

BK/JC, BK and JC polyomaviruses; CMV, cytomegalovirus; CSF, cerebrospinal fluid; HHV-6, human herpesvirus 6; HSV-1/HSV-2, herpes simplex virus 1/2; PCR, polymerase chain reaction; PMN, polymorphonuclear cells; VZV, varicella-zoster virus.

Thoracoabdominal computed tomography (Figure 1) identified hepatomegaly with mild portal space edema, splenomegaly (16.3 cm), retroperitoneal, iliac and inguinal lymphadenopathy, diffuse reduction in cortical contrast enhancement of both kidneys with perinephric fat stranding and bilateral parenchymal heterogeneity, a non-obstructive calculus in the right kidney, and a moderate amount of free pelvic fluid without organized collections.

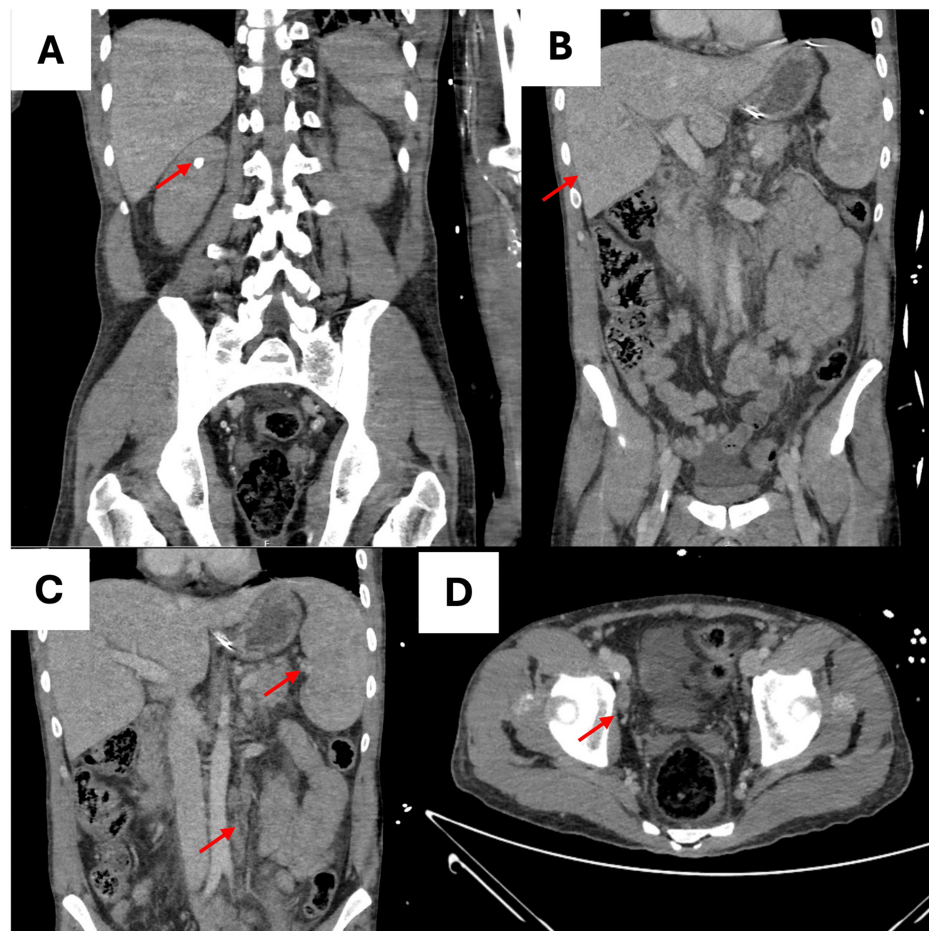


FIGURE 1: Thoracoabdominal computed tomography findings.

(A) Coronal CT section at the level of the kidneys showing a right renal calculus; the arrow indicates the calculus in the right kidney. (B) Coronal CT section at the level of the liver showing hepatomegaly; the arrow indicates the enlarged liver. (C) Coronal CT section at the level of the abdominal aorta showing splenomegaly and retroperitoneal lymphadenopathy; the upper arrow indicates the enlarged spleen and the lower arrow indicates an enlarged para-aortic retroperitoneal lymph node. (D) Axial CT section at the level of the pelvis showing iliac and inguinal lymphadenopathy; the arrow indicates an enlarged right iliac lymph node.

The patient was admitted to our intensive care unit for ongoing management and etiological investigation.

During the initial days in the intensive care unit (ICU), renal replacement therapy was initiated in the setting of uremic syndrome with significant nitrogenous waste retention, with uremic encephalopathy considered a contributing etiology. Sustained low-efficiency daily dialysis was commenced and maintained until adequate renal recovery was achieved.

Etiological investigation was pursued systematically (Table 3). Peripheral blood smear demonstrated anisocytosis with mild anisochromia, target cells, and very rare schistocytes. ADAMTS13 functional activity was mildly decreased at 43.7%, and the direct antiglobulin test was negative. Nutritional assessment identified folate deficiency. Serum immunoglobulins were within normal limits. Lymphocyte immunophenotyping revealed marked lymphopenia affecting T CD4+ and T CD8+ subsets with an inverted CD4/CD8 ratio, alongside B-cell and natural killer cell lymphopenia. The proportions of memory (CD45RO+) and naïve (CD45RA+) T-cell subsets were within normal limits. Serum angiotensin-converting enzyme was markedly elevated at 197.4 U/L. All serological and autoimmune studies were negative (Table 4). Further laboratory studies identified new-onset hypothyroidism, and levothyroxine was initiated.

Parameter	Patient value	Reference range
Lymphocyte immunophenotyping		
Total lymphocyte count	104 cells/mm ³ (11.65%)	1500–3000 cells/mm ³ (28-39%)
CD3+ T cells	68 cells/mm ³ (72.38%)	1100–1700 cells/mm ³ (67-76%)
CD4+ T cells	23 cells/mm ³ (22.22%)	700–1100 cells/mm ³ (38-46%)
CD8+ T cells	47 cells/mm ³ (44.90%)	500–900 cells/mm ³ (31-40%)
CD4/CD8 ratio	0.49	
B cells (CD19+)	11 cells/mm ³ (13.41%)	200–400 cells/mm ³ (11-16%)
NK cells (CD56+/CD16+)	9 cells/mm ³ (10.61%)	200–400 cells/mm ³ (10-19%)
Serum angiotensin-converting enzyme	197.4 U/L	8–52 U/L
ADAMTS13 activity	43.7%	>67%
Direct antiglobulin test	Negative	Negative
Endocrine and nutritional studies		
TSH	4.28 mIU/L	0.3-3.18 mIU/L
Free T4	0.89 ng/dL	1.01-1.65 ng/dL
Free T3	1.57 pg/mL	2.66-4.33 pg/mL
Red blood cell folate	145.1 ng/mL	235-788 ng/mL
Vitamin B12	640 pg/mL	191–663 pg/mL
Immunity		
IgA	288.0 mg/dL	114.0–457.0 mg/dL
IgG	1482.0 mg/dL	793.0–1590.0 mg/dL
IgM	46.6 mg/dL	29.0–226.0 mg/dL
Complement C3	76.6 mg/dL	81.0–167.0 mg/dL
Complement C4	25.4 mg/dL	11.0–42.0 mg/dL

TABLE 3: Additional laboratory workup: Hematological, endocrine, and immunological studies.

ADAMTS13, a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13; C3/C4, complement components 3 and 4; CD, cluster of differentiation; Ig, immunoglobulin; NK, natural killer (cells); TSH, thyroid-stimulating hormone.

Parameter	Patient value	Reference range
Autoimmune workup		
ANA (pattern/titer)	Speckled nuclear (AC-4,5), 1/80	<1/160 (negative)
Cytoplasmic pattern	Negative	Negative
Mitotic pattern	Negative	Negative
Anti-dsDNA	11.0 IU/mL	<10 negative; 10–15 equivocal; >15 positive
Anti-Sm, Anti-SSA, and Anti-SSB	<7 U/mL	<7 U/mL
Anti-PR3	2.3 UQ	<20 UQ
Anti-MPO	11.8 UQ	<20 UQ
Anti-GBM	3.7 U/mL	<7
Anticardiolipin (IgM, IgG, IgA)	<20 UQ	<20 UQ
Anti-β2-glycoprotein I (IgM, IgG, IgA)	<20 UQ	<20 UQ
IgA anti-transglutaminase	0.4 U/mL	<7
Serological workup		
Anti-treponema pallidum antibodies	Negative	Negative
Rickettsia conorii (IgM/IgG)	0.0/0.0	Negative
Leptospira PCR (blood)	Negative	Negative
Influenza A/B, RSV, SARS-CoV-2 PCR (blood)	Negative	Negative
CMV PCR (blood)	Negative	Negative
CMV IgM/IgG	0.140 IU/mL/426.0 IU/mL	IgM negative; IgG positive (past exposure)
HBsAg/Anti-HBs/Anti-HBc	Negative	Negative
Anti-HCV	Negative	Negative
Anti-HIV 1/2	Negative	Negative
EBV PCR (blood)	Negative	Negative
Parvovirus B19 PCR (blood)	Negative	Negative
Leishmania IgG+IgM	0.3	Negative

TABLE 4: Autoimmune and serological workup.

AC, anti-cell pattern; ANA, antinuclear antibody; ANCA, anti-neutrophil cytoplasmic antibody; Anti-HBc, antibody to hepatitis B core antigen; Anti-HBs, antibody to hepatitis B surface antigen; dsDNA, double-stranded DNA; EBV, Epstein-Barr virus; GBM, glomerular basement membrane; HBsAg, hepatitis B surface antigen; HCV, hepatitis C virus; HIV, human immunodeficiency virus; Ig, immunoglobulin; MPO, myeloperoxidase; PCR, polymerase chain reaction; PR3, proteinase 3; RSV, respiratory syncytial virus; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; Sm, Smith antigen; SSA/SSB, Sjögren's syndrome-related antigens A and B; UQ, chemiluminescent units.

Brain magnetic resonance imaging (Figure 2) demonstrated subtle T2 and fluid-attenuated inversion recovery hyperintensities adjacent to the frontal horns of the lateral ventricles, of nonspecific nature, and a small hypointense focus on T2* at the genu of the corpus callosum. No restricted diffusion, abnormal intraparenchymal or meningeal enhancement, or cranial nerve involvement was identified.

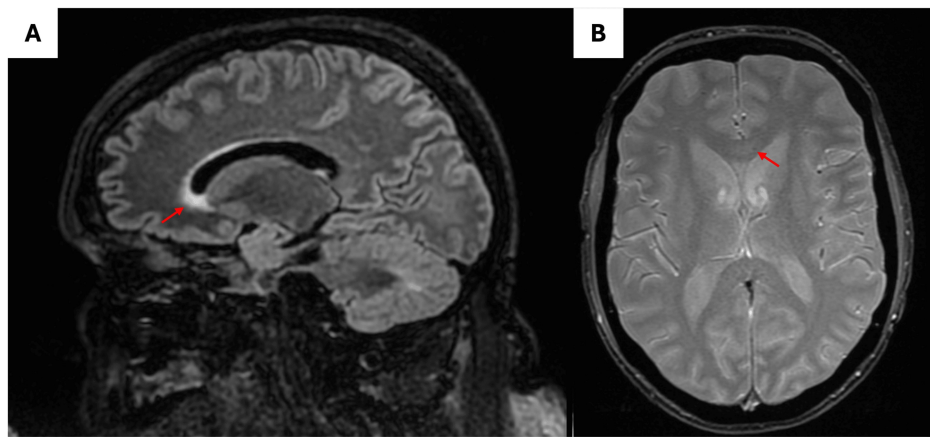


FIGURE 2: Brain magnetic resonance imaging findings.

(A) Sagittal fluid-attenuated inversion recovery (FLAIR) section showing subtle hyperintensities, also visible on T2-weighted sequences, adjacent to the frontal horns of the lateral ventricles; the arrow indicates the periventricular hyperintensity at the frontal horn. (B) Axial T2*-weighted section at the level of the basal ganglia demonstrating a small hypointense focus at the genu of the corpus callosum; the arrow indicates this focus.

Due to the severity of the clinical presentation and imaging findings, early renal biopsy was performed, revealing a polymorphic inflammatory infiltrate with non-necrotizing granulomas, consistent with granulomatous interstitial nephritis, with additional evidence of tubulitis and tubular injury.

Cervical ultrasound (Figure 3) was performed as part of the diagnostic evaluation of hypothyroidism and confirmed a palpable thyroid nodule as a solid isoechoic left lobe lesion with coarse peripheral and central calcifications, for which ultrasound-guided fine-needle aspiration cytology was performed. Cytological analysis was suggestive of granulomatous thyroiditis, correlating with the broader clinical context.

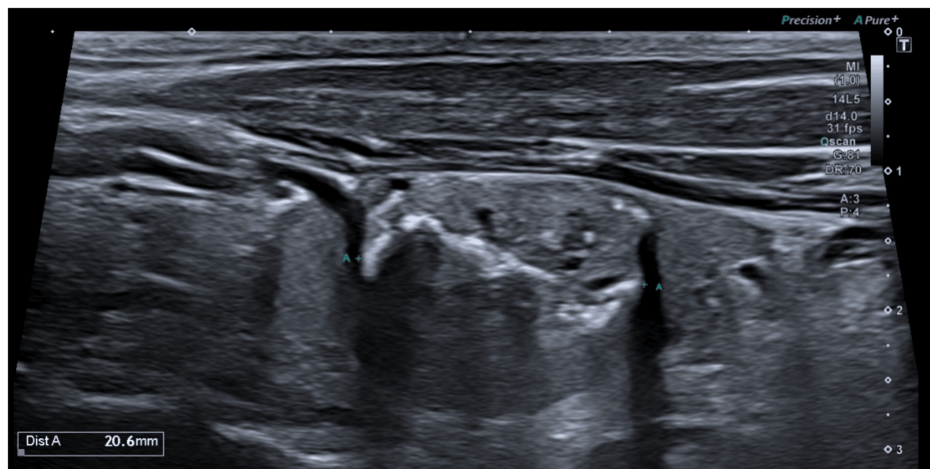


FIGURE 3: Solid isoechoic left thyroid lesion at cervical ultrasound with coarse peripheral and central calcifications.

Following the diagnosis of systemic sarcoidosis, intravenous methylprednisolone pulses (500 mg daily for three consecutive days) were administered, followed by oral prednisolone (1 mg/kg/day). Prophylactic treatment was initiated, comprising atovaquone, a proton pump inhibitor, vitamin D, and nystatin. Seizures were controlled with levetiracetam.

During the subsequent clinical course, mild hypoxemic respiratory insufficiency developed. Bronchoalveolar lavage (Table 5) revealed a lymphocytic alveolitis (28%) with a CD4/CD8 ratio greater than 4, reinforcing the diagnosis.

Parameter	Patient value	Reference range
Cell concentration	2.58 ×10 ⁵ cells/mL	<1.8 ×10 ⁵ cells/mL
Macrophages/monocytes	40.0%	80–90%
Lymphocytes	28.0%	10–15%
Neutrophils	32.0%	1–2%
Eosinophils	0.0%	<1%
Mast cells	0.0%	<1%
Other cells	Numerous erythrocytes (hemorrhagic BAL)	—
CD3+ T cells	97.0%	63–83%
CD4+ T cells	75.0%	40–45%
CD8+ T cells	16.0%	30–35%
CD4/CD8 ratio	4.69	—
CD19+ B cells	1.0%	0–4%
NK cells	2.0%	2–14%

TABLE 5: Bronchoalveolar lavage: Cytology and immunophenotyping.

BAL, bronchoalveolar lavage; CD, cluster of differentiation; NK, natural killer.

Reassessment thoracic computed tomography demonstrated faint ground-glass opacities and micronodularities with a peribronchovascular distribution, predominantly in the bilateral lower lobes and along the superior aspect of the major fissure, consistent with pulmonary sarcoidosis. Septic screening identified fully sensitive *Staphylococcus aureus* at fewer than 100,000 colony-forming units per milliliter, for which antibiotic treatment was initiated.

The patient demonstrated a progressive recovery of renal function, achieving dialysis independence after 12 days of renal replacement therapy (serum creatinine = 2.54 mg/dL and proteinuria = 0.5 g/day at ICU discharge). Encephalopathy resolved fully, with return to pre-admission cognitive baseline. Cutaneous xerosis resolved in parallel with the resolution of uremia. The patient was discharged after approximately six weeks, with outpatient follow-up arranged for ongoing sarcoidosis management and prednisolone tapering.

Discussion

This case illustrates a rare and severe presentation of systemic sarcoidosis, in which life-threatening neurological and renal involvement occurred in the absence of classic pulmonary features at admission, posing a significant diagnostic challenge. The diagnosis of sarcoidosis rests on three elements: compatible clinical and radiological findings, histological evidence of non-necrotizing granulomas, and exclusion of alternative causes of granulomatous disease [8,9]. In this case, all three criteria were met, with renal biopsy providing the definitive histological confirmation.

Previous constitutional symptoms and the neurological, renal, and hematological involvement at admission highlighted the importance of investigating a multisystemic process, including infectious, inflammatory/infiltrative, and neoplastic disease. All invasive investigations were guided by, and correlated with, clinical findings.

New-onset generalized tonic-clonic seizures without full neurological recovery warranted investigation for structural lesions, including malignancy, and for central nervous system infection, both of which were excluded by magnetic resonance imaging and cerebrospinal fluid analysis. In this patient, the seizures were most likely of multifactorial metabolic origin, given the severe uremia and acidemia at presentation. Direct neurological involvement by sarcoidosis was considered. However, neurosarcoidosis has heterogeneous presentations; the available diagnostic tests lack specificity, and nervous tissue histology was not obtained. Furthermore, because renal replacement therapy and corticosteroids were started concurrently, neurological improvement cannot be attributed to immunosuppressive therapy. Metabolic derangement therefore remains the most likely explanation [10].

Although rare, renal sarcoidosis most commonly occurs at the time of initial presentation and should be considered in patients who present with renal failure and have extra-renal features consistent with sarcoidosis [11]. Non-oliguric acute kidney injury required immediate exclusion of a post-renal cause, which was ruled out on computed tomography. A prerenal contribution was assumed, and fluid resuscitation was undertaken under close hemodynamic monitoring. The presence of leukocyturia and proteinuria warranted investigation for an intrinsic renal process. Renal biopsy was performed early, and the resulting diagnosis of granulomatous interstitial nephritis supported the initiation of immunosuppressive therapy. The additional finding of tubulitis and tubular injury provided the histological substrate for the severity of the acute kidney injury observed. The right-sided renal calculus may be related to sarcoidosis-associated disorders of calcium metabolism, although urinary calcium excretion was not assessed. Cutaneous findings were attributed at least in part to uremia. During the nephrology outpatient follow-up, renal function remained stable, with serum creatinine between 2.56 and 2.87 mg/dL and no significant proteinuria (urine protein-to-creatinine ratio <0.200 g/g).

Pancytopenia warranted a comprehensive work-up to understand the underlying mechanism. Peripheral blood smear findings, together with slightly elevated lactate dehydrogenase, non-measurable haptoglobin without reticulocytosis, a negative direct antiglobulin test, and mildly decreased ADAMTS13 functional activity, pointed toward extravascular hemolysis with inadequate compensatory bone marrow production. Lymphocyte immunophenotyping revealed the severity of the underlying disease, with marked lymphopenia and an inverted CD4/CD8 ratio. The preserved proportions of memory and naïve T-cell subsets were suggestive of a peripheral redistribution or destruction process, rather than a primary immunodeficiency. Normal immunoglobulins and negative serological and autoimmune studies helped rule out important differential diagnoses, including opportunistic infections, other autoimmune diseases, and primary immunodeficiencies. Bone marrow involvement was excluded by positron emission tomography-computed tomography, supporting a peripheral mechanism. Elevated serum angiotensin-converting enzyme reflected a high total granuloma burden, multiorgan involvement, and high inflammatory disease activity, further supporting this interpretation. Taken together with the clinical and imaging findings, these results supported a peripheral, sarcoidosis-related mechanism for the pancytopenia, attributable to splenic granulomatous infiltration in a patient with nutritional deficits.

Respiratory involvement developed later in the clinical course, delaying bronchoalveolar lavage. Nonetheless, the resulting lymphocytic alveolitis with a CD4/CD8 ratio >4 reinforced the diagnosis. Septic screening results became available only after corticosteroid therapy had been initiated, prompting subsequent antibiotic treatment.

Thyroid involvement, evidenced by granulomatous thyroiditis on fine-needle aspiration cytology, represented an additional extrapulmonary manifestation. Thyroid sarcoidosis may present with hypothyroidism, as observed in this patient, and the subsequent reduction in thyroid granuloma burden on follow-up imaging following corticosteroid therapy further supports the granulomatous etiology [12].

Corticosteroids remain the mainstay of therapy for sarcoidosis [13]. In this case, high-dose intravenous methylprednisolone pulses, followed by oral prednisolone, resulted in progressive recovery of renal function - an outcome that underscores the importance of early diagnosis and prompt immunosuppressive treatment in severe, multisystem presentations.

Conclusions

This case highlights several key diagnostic and therapeutic lessons: the importance of maintaining a high index of suspicion for sarcoidosis in young patients with acute multisystem disease, even in the absence of classic pulmonary findings; the pivotal role of early renal biopsy in establishing a definitive diagnosis when histological confirmation from more accessible sites is not immediately feasible; and the life-saving potential of timely corticosteroid therapy in severe extrapulmonary sarcoidosis.

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.

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Supervision: Rogério Gomes Silva, Maria João Ferreira da Silva

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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