

Cutaneous Lesions, Severe Hypercalcemia and Acute Kidney Injury Revealing Sarcoidosis: A Case Report with Atypical Histology and Corticosteroid-Induced Diabetes

 Ghalia Khellaf

University Health of Sciences Dr. Youcef El Khatib, Faculty of Medicine,
Department of Nephrology, Bab El Oued University Hospital, Algiers, Algeria

Houria.Sahel

University of Health Sciences Dr Youcef El Khatib, Faculty of Medicine,
Private Laboratory, Bab El Oued, Algiers, Algeria

Mohamed.Rachid. Bahriz

University of Health Sciences Dr Youcef El Khatib, Faculty of Medicine,
Private Laboratory, Bab El Oued, Algiers, Algeria

Lamis.Debchi

University of Health Sciences Dr Youcef El Khatib, Faculty of Medicine, Department of Nephrology,
Bab El Oued University Hospital Center, Algiers, Algeria

Habiba. Rafa-Debbah

Department of Nephrology, Bab El Oued University Hospital Center, Algiers, Algeria

Yasser. Rahou

Department of Nephrology, Bab El Oued University Hospital Center, Algiers, Algeria

Ali Benziane

University of Health Sciences Dr. Youcef El Khatib, Faculty of Medicine,
Department of Nephrology, Bab El Oued University Hospital, Algiers, Algeria

Corresponding author: Prof. G. Khellaf

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Abstract

A 55-year-old woman with a 4-year history of mediastinal adenopathy of unknown etiology presented with progressive cutaneous lesions, severe hypercalcemia (120 mg/L), and acute kidney injury (serum creatinine 70 mg/L). Angiotensin-converting enzyme levels were markedly elevated (98 U/L). Kidney biopsy revealed acute tubulointerstitial nephritis without granulomas. The diagnosis of sarcoidosis was established, and prompt corticosteroid therapy led to spectacular improvement of renal function, normalization of serum calcium, and complete resolution of cutaneous lesions. Corticosteroid-induced hyperglycemia required transient insulin therapy. This case illustrates that the absence of

granulomas on kidney biopsy does not exclude sarcoidosis, and that early recognition can prevent progression to chronic kidney disease.

Keywords: sarcoidosis; acute kidney injury; hypercalcemia; interstitial nephritis; corticosteroid-induced diabetes.

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1. Introduction

Sarcoidosis is a multisystem granulomatous disorder of unknown etiology [1]. While pulmonary involvement is most common, renal involvement occurs in up to 30% of cases, primarily as acute interstitial nephritis (AIN) [2, 3]. Hypercalcemia, resulting from unregulated 1,25-dihydroxyvitamin D production by activated macrophages, affects 10-20% of patients [3, 4]. The classic triad of cutaneous lesions, hypercalcemia, and acute kidney injury (AKI) is highly suggestive of sarcoidosis but remains rare as a presenting manifestation [5]. Symptomatic hypercalcemia occurs in less than 5% of sarcoidosis patients [6].

We report a case of sarcoidosis diagnosed in a 55-year-old woman presenting with this triad, with the additional challenges of atypical histology (absence of granulomas on kidney biopsy) [2] and corticosteroid-induced diabetes requiring insulin therapy [7].

2. SHORT METHODS

We conducted a retrospective case review of a patient diagnosed with sarcoidosis in the nephrology department of Bab El Oued University Hospital Center, Algiers, Algeria. Data were collected from electronic medical records, including clinical history, physical examination findings, laboratory results, imaging studies, and histopathological reports. Kidney biopsy was performed under ultrasound guidance and examined using light microscopy, immunofluorescence, and Congo red staining. The patient provided written informed consent for

publication. This case report followed the CARE guidelines.

3. RESULTS

3.1 Clinical presentation

A 55-year-old woman was referred for evaluation of AKI associated with severe hypercalcemia and progressive cutaneous lesions. Her past medical history included mediastinal adenopathy diagnosed four years earlier, treated empirically with corticosteroids without definitive etiological diagnosis (no biopsy performed). She also had type 2 diabetes mellitus for 12 years and hypertension for 10 years treated with amlodipine.

One year prior to admission, she developed violaceous infiltrated plaques on the face, upper back, and forearms. Concurrently, she reported fatigue, 8 kg weight loss, polyuria, and polydipsia. Physical examination revealed well-demarcated erythematous to violaceous plaques (Figure 1A-D). No peripheral lymphadenopathy or hepatosplenomegaly was noted.

Figure 1A-D: Cutaneous lesions at presentation. (A) Facial plaques in periorbital and nasolabial regions. (B) Upper back. (C) Forearm extensor surface. (D) Close-up view.

3.2 Laboratory findings

On admission, laboratory investigations revealed severe AKI with serum creatinine at 70 mg/L (normal 5-12 mg/L)

and blood urea nitrogen at 0.60 g/L. eGFR (MDRD) was 8 mL/min/1.73m². Severe hypercalcemia was noted with serum calcium at 120 mg/L (normal 85-105 mg/L). Angiotensin-converting enzyme (ACE) levels were markedly elevated at 98 U/L (normal < 40 U/L). C-reactive protein was 48 mg/L. Intact PTH was suppressed at 8 pg/mL (normal 15-65 pg/mL), while 1,25-dihydroxyvitamin D was elevated at 85 pg/mL (normal 25-65 pg/mL). Immunological workup (ANA, ANCA, anti-GBM) was negative. Quantiferon-TB gold was negative.

3.3 Imaging

Chest computed tomography scan showed stable mediastinal lymphadenopathy (right paratracheal, subcarinal, and hilar nodes, largest 18 mm in short axis), unchanged from four years prior, without active pulmonary parenchymal involvement. Abdominal ultrasound revealed normal kidney size with no nephrocalcinosis or hydronephrosis.

3.4 Kidney biopsy

A percutaneous ultrasound-guided kidney biopsy was performed. Light microscopy (Figure 2) revealed diffuse interstitial edema with mononuclear cell infiltration composed of lymphocytes. Glomeruli were normal. Importantly, no non-caseating granulomas were identified despite examination of multiple sections (15 levels examined). Immunofluorescence was negative for immunoglobulins and complement. Congo red staining was negative for amyloid deposits.

Figure 2: Kidney biopsy findings. H&E x100: diffuse interstitial mononuclear infiltrate with normal glomeruli.

3.5 Diagnosis

The diagnosis of sarcoidosis was established based on: (1) clinical triad of cutaneous lesions, hypercalcemia, and AKI; (2) elevated ACE and 1,25-dihydroxyvitamin D with suppressed PTH; (3) mediastinal adenopathy compatible with sarcoidosis; (4) histological evidence of acute tubulointerstitial nephritis; (5) exclusion of other causes (tuberculosis, drug-induced, autoimmune, multiple myeloma); and (6) dramatic response to corticosteroid therapy.

The absence of granulomas on kidney biopsy, while atypical, does not exclude sarcoidosis as granulomas are absent in 30-40% of renal sarcoidosis cases. In a series of native kidney biopsies from patients with sarcoidosis, non-granulomatous AIN was observed in 14-44% of cases.

3.6 Treatment and outcome

The patient was hospitalized and managed with intravenous hydration, furosemide, and calcitonin for hypercalcemia. Corticosteroid therapy was promptly initiated: intravenous methylprednisolone 500 mg/day for 3 consecutive days followed by oral prednisone 60 mg/day (approximately 1 mg/kg/day).

The clinical and biological response was spectacular, as detailed in Table 1. Serum calcium normalized rapidly within 3 days. Serum creatinine progressively declined from 70 mg/L (Day 0) to 12 mg/L (Day 60), with eGFR improving from 8 to 68 mL/min/1.73m² at 12 months. ACE levels normalized by Day 60 (32 U/L). Cutaneous lesions completely resolved by Day 30.

2. Table 1: Evolution of clinical and biological parameters under corticosteroid therapy

Parameter	Day 0	Day 3	Day 7	Day 14	Day 30	Day 60	Normal range
Serum creatinine (mg/L)	70	52	38	24	16	12	5-12
eGFR (mL/min/1.73m ²)	8	12	18	32	48	62	> 90
Serum calcium (mg/L)	120	98	88	86	84	85	85-105

ACE (U/L)	98	—	—	62	45	32	< 40
CRP (mg/L)	48	—	22	12	6	3	< 5

Corticosteroid-induced hyperglycemia

The patient had pre-existing type 2 diabetes mellitus well-controlled on metformin (HbA1c 6.8% at baseline). Following initiation of high-dose corticosteroids, significant hyperglycemia developed. Fasting glucose rose from 1.08 g/L at baseline to 1.85 g/L at Day 3, and postprandial glucose reached 2.60 g/L. Basal-bolus insulin therapy was initiated: glargine 10 units at bedtime and aspart 4-6 units before meals (total daily dose 24-30 units). As prednisone was tapered (60 → 40 mg/day at Day 14 → 25 mg/day at Day 30 → 15 mg/day at Day 60), insulin requirements decreased. At Day 60, insulin was discontinued, and glycemic control was maintained on metformin alone [7].

At last follow-up (12 months), the patient remained well with serum creatinine 11 mg/L, eGFR 68 mL/min/1.73m², serum calcium 84 mg/L, ACE 28 U/L, and HbA1c 6.7% on metformin alone. Prednisone was tapered to 5 mg/day for maintenance.

4. DISCUSSION

We report a case of sarcoidosis presenting with the classic triad of cutaneous lesions, severe hypercalcemia, and AKI in a 55-year-old woman. Several aspects of this case merit discussion. **4.1**

Delayed Diagnosis

The patient had a four-year history of mediastinal adenopathy treated empirically with corticosteroids without a definitive diagnosis. This delay is not uncommon in sarcoidosis, especially when pulmonary symptoms are absent [1]. In a retrospective series of 47 patients with renal sarcoidosis, Mahévas and colleagues reported a mean diagnostic delay of 24 months from symptom onset [2]. The diagnostic process can be prolonged due to the absence of classic systemic sarcoidosis findings [8]. A recent multicenter cohort study emphasized that there is no good test to diagnose

renal involvement in sarcoidosis other than kidney biopsy, and the heterogeneous presentation of the disease underscores the need for better biomarkers to guide treatment decisions[8]. This highlights the importance of tissue biopsy (lymph node or other accessible site) in patients with unexplained mediastinal adenopathy [1].

4.2 Severe Hypercalcemia

Severe hypercalcemia (≥ 120 mg/L or 3.0 mmol/L) occurs in less than 5% of sarcoidosis patients [9]. The mechanism—unregulated production of 1,25-dihydroxyvitamin D by activated macrophages within granulomas[4] was confirmed by elevated 1,25-dihydroxyvitamin D (85 pg/mL) with suppressed PTH (8 pg/mL). This pathophysiology is well-described in the literature: hypercalcemia in sarcoidosis occurs due to the uncontrolled synthesis of 1,25-OH D₃ by macrophages in the sarcoid granulomata, resulting in increased calcium absorption in the intestine and calcium resorption in the bone[10]. Calcitriol-mediated hypercalcemia in sarcoidosis is driven by extrarenal 1- α -hydroxylase activity, a key pathophysiological feature that differentiates it from other causes of hypercalcemia[9].

The differential diagnosis includes primary hyperparathyroidism (elevated PTH), malignancy (multiple myeloma, solid tumors), vitamin D intoxication, and other granulomatous diseases (tuberculosis, fungal infections). In our patient, the elevated ACE level (98 U/L) and mediastinal adenopathy pointed strongly toward sarcoidosis [1]. Symptomatic hypercalcemia in sarcoidosis is unusual, presenting in <5% of patients, and rarely results in kidney damage [9]. A case series reported that hypercalciuria (90%) was more common than hypercalcemia (74%) in patients with renal sarcoidosis [2]. It is essential to recognize that patients with cutaneous sarcoidosis may develop hypercalcemia without systemic disease, and long-

term follow-up is advised as some may develop systemic involvement subsequently [11].

4.3 Absence of Granulomas on Kidney Biopsy

Classically, renal sarcoidosis is characterized by non-caseating granulomatous interstitial nephritis. However, granulomas may be absent in a significant proportion of cases, especially in early or mild disease [2, 5]. As noted in recent reviews, "amid various forms of glomerulonephritis, granulomatous interstitial nephritis is the most common one, but it rarely leads to renal impairment. Histologically, granulomas can be absent" [8]. Similarly, Bergner and Löffler stated that "the most common histological form of renal sarcoidosis is the granulomatous interstitial nephritis; however, granulomas can be absent" [12].

In a series of 43 patients with biopsy-proven renal sarcoidosis, 84% exhibited interstitial nephritis, with granulomatous interstitial nephritis in only 33% of cases [13]. This confirms that non-granulomatous interstitial nephritis is a frequent finding and does not exclude the diagnosis. In a retrospective multicenter cohort study, the authors emphasized that "in patients with known sarcoidosis, hypercalcemia and renal insufficiency it is important to perform a kidney biopsy because the treatment of hypercalcemia is different from the treatment of interstitial nephritis and waiting for the renal effect of hypercalcemia correction causes valuable time loss when interstitial nephritis is also present" [8].

In our patient, kidney biopsy showed non-specific acute tubulointerstitial nephritis without granulomas. This finding does not exclude sarcoidosis [2, 5]. The diagnosis was supported by the clinical context, elevated ACE levels, elevated 1,25-dihydroxyvitamin D with suppressed PTH, exclusion of other causes (tuberculosis, drug-induced, autoimmune), and the dramatic response to corticosteroids [1, 9].

Drug-induced interstitial nephritis was considered. The patient had been taking amlodipine for 10 years. Drug-induced AIN is typically associated with antibiotics, NSAIDs, proton pump inhibitors, and allopurinol [14]. Calcium channel blockers are rarely implicated. The absence of fever, rash, eosinophilia, and the temporal relationship (symptoms after 10

years of stable treatment) make drug-induced AIN unlikely [14].

4.4 Prognosis and Treatment

Corticosteroids are the mainstay of treatment for renal sarcoidosis [14]. Early initiation is associated with better renal outcomes. In a study of 35 patients with sarcoidosis-associated AIN, Rajakariar and colleagues reported that patients treated within 6 months of symptom onset had significantly better renal recovery compared to those with delayed treatment (mean final eGFR 58 vs 32 mL/min, $p < 0.01$) [5]. Our patient achieved near-complete renal recovery (eGFR from 8 to 68 mL/min at 12 months), emphasizing the importance of prompt diagnosis [9].

Recent studies have provided valuable data on treatment outcomes in renal sarcoidosis. In a cohort of 43 patients with biopsy-proven renal sarcoidosis, 86% achieved recovery within 6 months, with 74% achieving complete recovery [13]. Steroid therapy was strongly associated with renal recovery and improved long-term outcomes [13]. The starting dose of prednisolone of 0.5-1.0 mg/kg/day is congruent with standard recommendations for renal sarcoidosis [5, 13].

Severe hypercalcemia management in sarcoidosis requires a multifaceted approach, including intravenous hydration and corticosteroids as the cornerstone of therapy [9]. Glucocorticosteroids act by inhibiting the 1 α -hydroxylase activity of macrophages, with prednisone 20 to 40 mg/day followed by a taper being the recommended dose [14]. In patients with impaired renal function, bisphosphonates may carry a risk of nephrotoxicity, and corticosteroids directly target the underlying pathophysiology [9].

Predictors of poor renal prognosis include preexisting CKD, elevated serum creatinine at biopsy, and severe interstitial fibrosis and tubular atrophy [13]. As noted in the literature, "early detection and management of renal involvement in sarcoidosis are critical to preventing irreversible kidney damage" [8].

4.5 Corticosteroid-Induced Diabetes

High-dose corticosteroids frequently induce hyperglycemia, even in patients without preexisting diabetes [6]. In patients with pre-existing type 2 diabetes, corticosteroid-induced hyperglycemia is almost universal and often requires insulin therapy. The mechanisms include increased hepatic gluconeogenesis, decreased peripheral insulin sensitivity, and impaired insulin secretion [6].

A retrospective review of 170 sarcoidosis patients found that glucocorticoid therapy was associated with worsening or new-onset diabetes (relative risk 3.0; 95% CI 1.0-8.8; $p < 0.05$) [15]. Management requires frequent glucose monitoring and a basal-bolus insulin regimen, as glucocorticoids predominantly affect post-prandial glucose [6]. Consensus guidelines recommend monitoring blood glucose QID and initiating insulin therapy when blood glucose exceeds 11.1 mmol/L on two or more occasions [6]. In patients with pre-existing diabetes, insulin therapy should be adjusted in tandem with reductions in glucocorticoid dosage [6].

Our patient required insulin for approximately six weeks, with successful discontinuation after prednisone was tapered below 20 mg/day. This underscores the need for close collaboration between nephrologists and endocrinologists [15].

5. LIMITATIONS

We acknowledge several limitations. First, tissue confirmation from an extrarenal site (e.g., lymph node biopsy) was not obtained, as the mediastinal adenopathy was stable and the patient declined invasive diagnostic procedures. Second, the retrospective nature of this case report limits the generalizability of our findings. Third, the diagnosis of sarcoidosis was made based on a combination of clinical, biological, and histological findings, without the gold standard of granuloma identification on tissue biopsy [2, 5]. Finally, the absence of long-term follow-up data beyond 12 months limits our ability to comment on the patient's long-term prognosis and potential for relapse.

6. CONCLUSION

This case illustrates several important lessons for clinicians managing patients with suspected sarcoidosis.

First, the classic triad of cutaneous lesions, hypercalcemia and acute kidney injury should raise strong suspicion for sarcoidosis, even in the absence of pulmonary symptoms. Early recognition of this triad can lead to prompt diagnosis and treatment.

Second, the absence of granulomas on kidney biopsy does not exclude sarcoidosis. As demonstrated in this case and supported by the literature, non-specific acute tubulointerstitial nephritis can be the only histological finding, especially in early disease [2]. Clinicians should not dismiss the diagnosis of sarcoidosis based solely on the absence of granulomas on renal biopsy.

Third, a high index of suspicion is needed in patients with unexplained mediastinal adenopathy, even when pulmonary symptoms are absent. A four-year diagnostic delay, as seen in our patient, is not uncommon and can be avoided with appropriate tissue biopsy and investigation [1].

Fourth, early recognition and prompt corticosteroid therapy can lead to spectacular clinical and biological improvement, preventing progression to chronic kidney disease [7]. The dramatic renal recovery observed in our patient (eGFR from 8 to 68 mL/min/1.73m² at 12 months) underscores the importance of timely diagnosis and treatment.

Finally, physicians should be vigilant for corticosteroid-induced metabolic complications, particularly hyperglycemia, which may require insulin therapy [6]. Patients with pre-existing diabetes are at particularly high risk and require close glucose monitoring and collaboration between nephrologists and endocrinologists [12].

Multidisciplinary collaboration involving nephrologists, dermatologists, pathologists, and endocrinologists is essential for optimal management of this complex disease.

7. ETHICS STATEMENT

The patient provided written informed consent for publication. Institutional review board approval was not required for this single-case report.

8. AUTHOR CONTRIBUTIONS

GK: Conceptualization, data collection, writing - original draft, review and editing.

HS: Clinical management, data collection, review and editing.

MRB: Pathology analysis, data collection, review and editing.

LD: Clinical management, data collection, review and editing.

HRD: Clinical management, data collection, review and editing.

YR: Clinical management, data collection, review and editing.

AB: Clinical management, data collection, review and editing.

9. CONFLICT OF INTEREST

The authors declare no conflicts of interest.

10. FUNDING

No funding was received for this work.

12. FIGURE LEGENDS

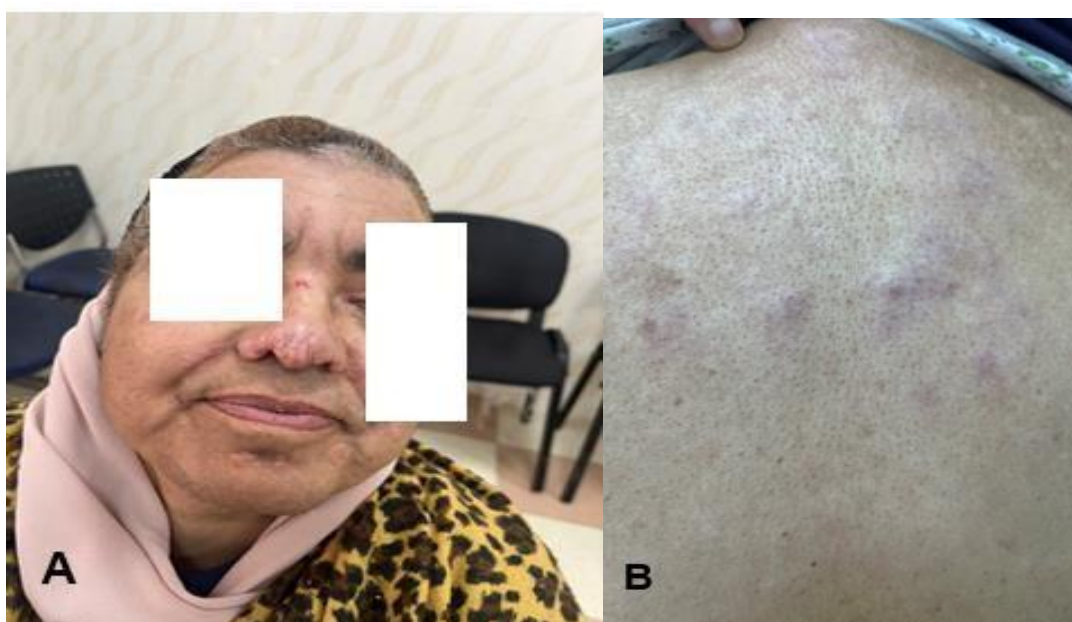




Figure 1: Cutaneous lesions at presentation. (A) Facial plaques in periorbital and nasolabial regions. (B) Upper back plaques. (C) Forearm extensor surface plaques. (D) Close-up view showing violaceous hue.

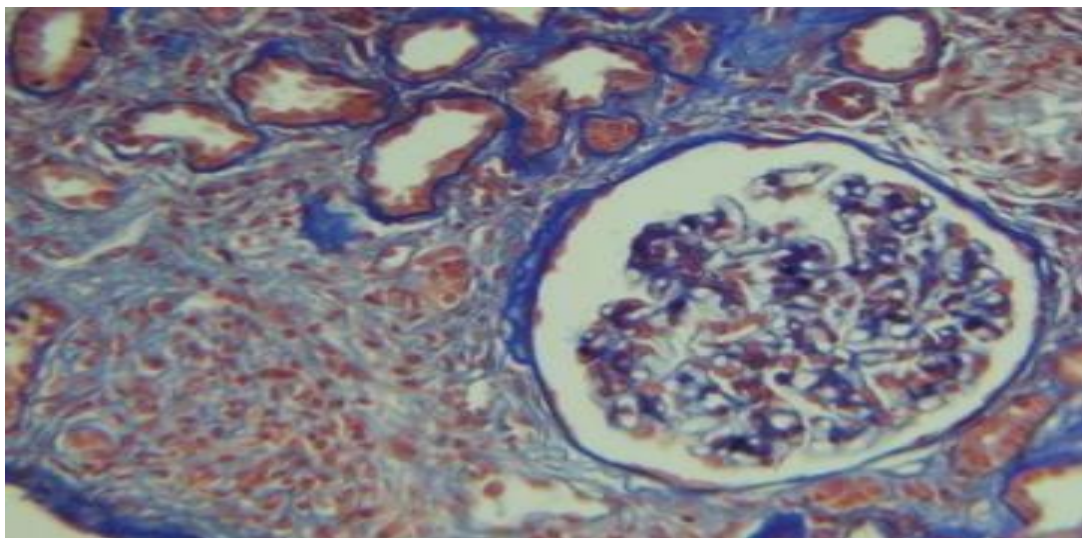


Figure 2: Kidney biopsy findings. (A) H&E x100: diffuse interstitial mononuclear infiltrate with normal glomeruli.

References

1. Iannuzzi, M.C., B.A. Rybicki, and A.S. Teirstein, *Sarcoidosis*. N Engl J Med, 2007. **357**(21): p. 2153–65.
2. Mahévas, M., et al., *Renal sarcoidosis: clinical, laboratory, and histologic presentation and outcome in 47 patients*. Medicine (Baltimore), 2009. **88**(2): p. 98–106.
3. Baughman, R.P. and I. Papanikolaou, *Current concepts regarding calcium metabolism and bone health in sarcoidosis*. Curr Opin Pulm Med, 2017. **23**(5): p. 476–481.
4. Adams, J.S., et al., *Isolation and structural identification of 1,25-dihydroxyvitamin D3*

- produced by cultured alveolar macrophages in sarcoidosis.* J Clin Endocrinol Metab, 1985. **60**(5): p. 960–6.
5. Rajakariar, R., et al., *Sarcoid tubulo-interstitial nephritis: long-term outcome and response to corticosteroid therapy.* Kidney Int, 2006. **70**(1): p. 165–9.
 6. Roberts, A., J. James, and K. Dhatariya, *Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group.* Diabet Med, 2018. **35**(8): p. 1011–1017.
 7. Judson, M.A., *The diagnosis of sarcoidosis.* Clin Chest Med, 2008. **29**(3): p. 415–27, viii.
 8. Bomhof, G., et al., *A retrospective multicenter cohort study of patients with sarcoidosis and renal involvement.* BMC Nephrol, 2026. **27**(1): p. 120.
 9. Nakada, K., et al., *Diagnosis and treatment of sarcoidosis-associated hypercalcemia requiring emergency hemodialysis: a case report.* Renal Replacement Therapy, 2026. **12**(1): p. 24.
 10. Kulkarni A, E.R., Ramesh N, Ruby E., *ODP106 Mystery of the Calcium: A Case of Exclusive Cutaneous Sarcoidosis presenting with Symptomatic Hypercalcemia.* J Endocr Soc, 2022 Nov . **2022**;6(Suppl 1):A170.
 11. Kim, J.M., et al., *Subcutaneous sarcoidosis presenting as a suprapubic mass, acute kidney injury, and hypercalcemia.* Korean J Intern Med, 2014. **29**(4): p. 535–8.
 12. Bergner, R. and C. Löffler, *Renal sarcoidosis: approach to diagnosis and management.* Curr Opin Pulm Med, 2018. **24**(5): p. 513–520.
 13. Miao, J., et al., *Clinicopathological Characteristics and Long-Term Kidney Outcomes in Biopsy-Proven Renal Sarcoidosis.* Kidney360, 2025. **6**(10): p. 1780–1789.
 14. Moledina, D.G. and M.A. Perazella, *Drug-Induced Acute Interstitial Nephritis.* Clin J Am Soc Nephrol, 2017. **12**(12): p. 2046–2049.
 15. Gostiljac, D.M., et al., [Sarcoidosis and diabetes: therapeutic aspects]. Med Pregl, 299 2005. 58 Suppl 1: p. 46–50.