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Favorable Response to Systemic Corticosteroids as Supportive Diagnostic Evidence in Sarcoidosis: A Case Report

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Abstract

Sarcoidosis can be difficult to diagnose when salivary and lacrimal gland involvement resembles Sjögren disease or IgG4-related disease. A 58-year-old woman with a history of previously treated papillary thyroid carcinoma presented with subacute bilateral mandibular pain, parotid enlargement, and periorbital swelling. Computed tomography showed enlargement of the parotid and lacrimal glands, proptosis, and mediastinal and mesenteric lymphadenopathy. Serum angiotensin-converting enzyme was elevated, serum calcium was normal, and the reported autoimmune serological tests were negative. Parotid biopsy showed lymphocytic inflammation with epithelioid and multinucleated giant cells. Cervical lymph node biopsy demonstrated non-necrotizing granulomatous lymphadenitis without evidence of epithelial malignancy in the sampled tissue. The reported microbiological investigations were negative. These findings favored sarcoidosis, although the salivary gland histology raised the possibility of seronegative Sjögren disease. Oral prednisolone at 30 mg daily was followed by rapid improvement in glandular swelling and pain during one week of inpatient observation. Outpatient outcomes were unavailable. The response increased confidence in the working diagnosis when considered alongside the biopsy and imaging findings. This case describes the contribution of treatment response to diagnostic assessment while acknowledging its limited specificity.

Categories: Rheumatology, Internal Medicine
Keywords: heerfordt syndrome, lofgren syndrome, sjogren syndrome , systemic sarcoidosis, systemic steroids

Introduction

Sarcoidosis is a systemic inflammatory disease of unknown etiology, characterized by non-necrotizing granulomatous inflammation [1]. In most cases, diagnostic assessment combines a compatible clinical presentation, histological evidence of non-necrotizing granulomatous inflammation, and investigation of alternative causes; histology must be interpreted in context because granulomas are not specific to sarcoidosis [2]. It has a global prevalence ranging from 4.6 to 64 cases per 100,000 inhabitants; in Colombia, the estimated prevalence is 12.8 per 100,000 inhabitants [3]. It affects women two times as often as men. The peak incidence occurs between the ages of 35 and 45 [1].

Acute presentation is uncommon and manifests through two syndromes: Löfgren and Heerfordt syndromes, with the latter being the less frequent. Sarcoidosis presents as Löfgren syndrome in 5-10% of cases, whereas it presents as Heerfordt syndrome in 1.5-3% of cases [1]. Clinical phenotypes vary geographically; in a Swedish referral cohort, 383 of 1,000 patients with sarcoidosis had Löfgren syndrome [4]. Lupus pernio is described in the European Respiratory Society (ERS) guideline as a chronic cutaneous manifestation and should be distinguished from acute syndromic presentations [5]. Löfgren syndrome is characterized by fever, mediastinal lymphadenopathy, ankle arthralgia, and erythema nodosum, while Heerfordt syndrome presents with fever, bilateral symmetrical parotid enlargement, peripheral facial paralysis, and anterior uveitis [6]. Salivary involvement may be unilateral or bilateral, as illustrated in a Heerfordt-associated cohort and a population-based parotid study [4,7].

Most patients exhibit chronic, nonspecific symptoms, which often lead to a delayed diagnosis [1]. Other findings supporting a diagnosis of sarcoidosis include hypercalcemia and elevated angiotensin-converting enzyme (ACE) levels, but these should not be used independently [2]. Diagnosing sarcoidosis poses a clinical challenge because infectious and noninfectious causes of granulomatous inflammation may overlap in the differential diagnosis [2]. The educational contribution of this case is the interpretation of overlapping glandular and nodal pathology in a patient with bilateral salivary and lacrimal enlargement, alongside documentation of the early treatment response. Corticosteroid responsiveness cannot independently distinguish sarcoidosis from IgG4-related disease [2,8].

Case Presentation

This case report describes a 58-year-old woman from a rural area with a history of papillary thyroid

carcinoma treated with total thyroidectomy, neck dissection, and radioiodine approximately five years before the current presentation. The oncology summary described classic-variant tumors measuring 1.1 cm in the left lobe and 2.7 cm in the right lobe, with involvement of surrounding adipose tissue and no reported lymphovascular or perineural invasion. She presented with subacute bilateral mandibular pain, bilateral parotid enlargement, and periorbital edema. She was afebrile at the recorded assessment (36.5 °C). The documented inpatient analgesics were acetaminophen 1 g every eight hours and metamizole (dipyrone) 1 g every eight hours.

Physical examination revealed bilateral parotid induration, a soft, mobile left supraclavicular lymph node, and pruritic lesions over the hands and pretibial region. Dermatology described two erythematous-violaceous plaques with central hemorrhagic crusting over the anterior tibial prominence, considered them nonspecific inflammatory lesions, and prescribed topical corticosteroid treatment with outpatient review. Arthralgia of the left first metacarpophalangeal joint was also documented.

Ophthalmology attributed the conjunctival congestion to dry eye and conjunctivitis. The corneas showed no staining, and the anterior chambers were deep. Corrected visual acuity was 20/50 in both eyes in the setting of myopia, and intraocular pressure was 14 mmHg bilaterally. Topical corticosteroid-containing drops were prescribed for 10 days and lubricating drops for one month. The initial parotid swelling and subsequent reduction are illustrated in Figure *1*.

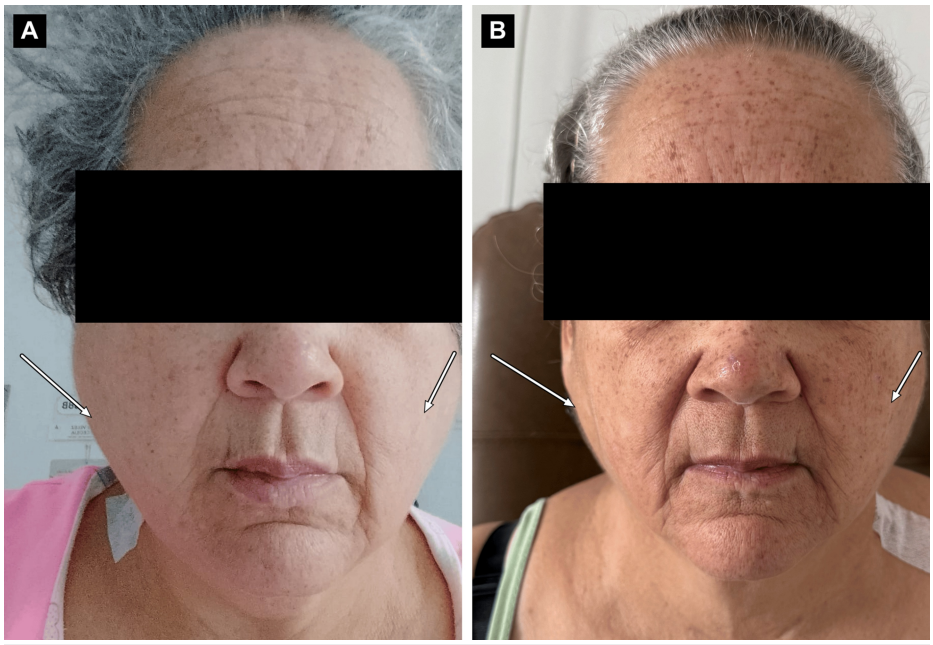


FIGURE 1: Reduction in bilateral parotid swelling on follow-up

(A) Photograph obtained on the day of cervical lymph-node biopsy (Day 11), showing bilateral parotid-region swelling (arrows). (B) Photograph obtained at hospital discharge (Day 26), 15 days later, showing reduced swelling in the corresponding regions (arrows). Black boxes cover the eyes and eyebrows for patient privacy.

Serum calcium was normal at 9.5 mg/dL (laboratory reference interval, 8.4-10.2 mg/dL), while serum angiotensin-converting enzyme was mildly elevated at 79.4 U/L (reference interval, 12-68 U/L). Antinuclear antibody (ANA) and the measured extractable nuclear antigen (ENA) antibodies were negative, and serum IgG subclasses were within the laboratory reference intervals. Selected laboratory results are summarized in Table *1*.

Panel/test	Result (units)	Laboratory reference/threshold
Red-cell indices	Hb 13.2 g/dL; Hct 40%; RBC 5.04 ×10⁶/μL	Hb 12–15 g/dL; Hct 36–45%; RBC 4.2–5.4 ×10⁶/μL
MCV	78.8 fL ↓	86–100 fL
White cells (×10³/μL)	WBC 7.0; neutrophils 3.43; lymphocytes 2.24; monocytes 1.05 ↑	WBC 3.8–11.8; neutrophils 1.71–7.67; lymphocytes 1.14–4.72; monocytes 0–0.94
Platelets	298 ×10³/μL	150–400 ×10³/μL
Creatinine	1.00 mg/dL	0.52–1.04 mg/dL
Thyroid tests	Free T4 1.69 ng/dL; TSH 0.02 mIU/mL ↓; thyroglobulin 0.32 ng/mL	Free T4 0.77–2.18 ng/dL; TSH 0.400–4.049 mIU/mL; thyroglobulin <0.1 ng/mL after thyroidectomy
ANA	Negative (AC-0)	Negative at 1:80
ENA antibodies	Anti-Sm, anti-RNP, anti-SSA/Ro and anti-SSB/La: each <1.0 U/mL	Negative: <1.0 U/mL for each antibody
IgG subclasses (g/L)	IgG1 8.59; IgG2 2.35; IgG3 0.13; IgG4 0.59	IgG1 4.05–10.11; IgG2 1.69–7.86; IgG3 0.11–0.85; IgG4 0.03–2.01
Calcitriol	42.6 pg/mL	15.2–90.1 pg/mL
Infection screen (nonreactive)	HIV 0.13 S/CO; anti-HBc 3.25 S/CO; anti-HCV 0.01 LU; RPR nonreactive	Nonreactive: HIV ≤0.90 S/CO; anti-HBc ≥1.20 to <4.80 S/CO; anti-HCV <0.90 LU; RPR nonreactive
Anti-HBs	0.07 mIU/mL	Absent: <8 mIU/mL
Coagulation	PT 11.3 s; INR 1.08; aPTT 29.4 s	PT 9.6–13.6 s; INR 0.84–1.19; aPTT 25.1–36.5 s

TABLE 1: Selected laboratory results

Results and local reference intervals or assay thresholds were transcribed from the patient's laboratory reports. ↑ and ↓ indicate values above and below the stated interval. Laboratory findings are interpreted alongside the clinical, imaging and pathological assessment [2].

Abbreviations and terms: Hb, hemoglobin; Hct, hematocrit; RBC, erythrocyte count; WBC, leukocyte count; MCV, mean corpuscular volume; ENA, extractable nuclear antigens; PT, prothrombin time; aPTT, activated partial thromboplastin time; LU, luminescence units; ACE, angiotensin-converting enzyme; ANA, antinuclear antibodies (tested by indirect immunofluorescence); AC-0, laboratory code for a negative ANA result; anti-Sm, anti-Smith antibodies; anti-RNP, anti-ribonucleoprotein antibodies; anti-SSA/Ro and anti-SSB/La, antibodies to Sjögren syndrome-related antigens A/Ro and B/La, respectively; IgG, immunoglobulin G; calcitriol, 1,25-dihydroxyvitamin D; T4, thyroxine; TSH, thyroid-stimulating hormone; HIV, human immunodeficiency virus; anti-HBc, antibodies to hepatitis B core antigen; anti-HBs, antibodies to hepatitis B surface antigen; anti-HCV, antibodies to hepatitis C virus; RPR, rapid plasma reagin; INR, international normalized ratio; S/CO, signal-to-cutoff ratio

Units: g, gram; mg, milligram; ng, nanogram; pg, picogram; L, liter; dL, deciliter; mL, milliliter; μL, microliter; fL, femtoliter; U, unit; mIU, milli-international unit; s, second

Contrast-enhanced CT on Day 2 demonstrated symmetric lacrimal gland enlargement measuring 20 × 12 × 24 mm, with proptosis. The parotid glands were heterogeneously enlarged, measuring 50 × 38 mm on the left and 50 × 23 mm on the right, without a dominant focal lesion. Mediastinal and bilateral hilar lymphadenopathy involved stations 4R, 5, 6, 7, 10R/L and 11R/L, including nodes measuring 13 × 22 mm at station 4R and 15 × 30 mm at station 7. Abdominal nodes included in the scan, described around the hepatic hilum, measured up to 10 mm in short axis. No pulmonary parenchymal lesions or suspicious thyroid-bed recurrence were reported.

The radiological impression favored a lymphoproliferative process, with IgG4-related disease identified as the principal alternative diagnosis. Representative CT findings are shown in Figure *2*.

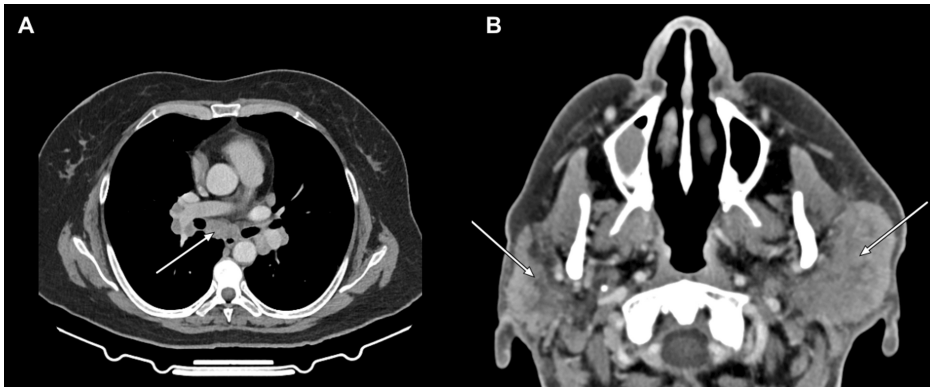


FIGURE 2: Thoracic lymphadenopathy and bilateral parotid enlargement on computed tomography

(A) Axial chest CT: the arrow indicates prominent subcarinal lymph node tissue. (B) Axial neck CT: the arrows indicate bilateral heterogeneous parotid gland enlargement.

The pathology report from the percutaneous biopsy of the left parotid gland described dense lymphocytic inflammation with epithelioid and multinucleated giant cells, reported as Chisholm-Mason grade 4. Plasma cells were demonstrated by CD138 and CD38 staining. IgG and IgG4 immunostains were reported as completely negative, and the reporting pathologist interpreted the findings as excluding IgG4-related disease. Numerical IgG4-positive cell counts and an IgG4/IgG ratio were not reported.

An excisional biopsy of a deep cervical lymph node reported non-necrotizing granulomatous lymphadenitis. Cytokeratin AE1/AE3 staining did not identify infiltrating epithelial malignant cells in the sampled lymph node. The parotid report also described no epithelial neoplasm, with CK7, p63, and smooth-muscle-actin findings interpreted in the glandular structures.

Microbiological testing of deep cervical lymph-node tissue collected on Day 11 showed negative Gram, Ziehl-Neelsen, and potassium hydroxide examinations, and no routine bacterial growth after 72 hours. *Mycobacterium (M.) tuberculosis* PCR was negative. Final cultures showed no fungal growth after 30 days and no *M. tuberculosis*-complex growth in Mycobacteria Growth Indicator Tube (MGIT) after 42 days. The final fungal and mycobacterial reports were validated on Days 39 and 55, respectively. Pathology reports also described negative Ziehl-Neelsen and methenamine-silver stains in both parotid and nodal tissue, with negative modified Ziehl-Neelsen staining in the parotid specimen.

Sarcoidosis, Sjögren disease [9], IgG4-related disease, infection, and recurrent malignancy were considered. Dry-eye symptoms and lymphocytic parotid inflammation prompted consideration of Sjögren disease. The combination of nodal non-necrotizing granulomatous inflammation, systemic lymphadenopathy, and bilateral glandular involvement nevertheless favored sarcoidosis.

After the clinical and pathological assessment, oral prednisolone 30 mg daily was initiated. Parotid and lacrimal swelling and local pain improved during the following week of inpatient observation (Figure *1*). No adverse events or dose taper were recorded during that interval. She was discharged on Day 26, with outpatient rheumatology reassessment planned to guide subsequent treatment; longer-term outcomes were unavailable. The documented investigation dates and observed treatment interval are summarized in Table *2*.

Day/interval	Assessment	Documented finding	Date source
Day 1	Initial laboratory tests	CBC, creatinine, and free T4 obtained	Sample date
Day 2	CT	Bilateral glandular enlargement and thoracic/abdominal nodes	Study date
Day 11	Cervical-node microbiology	Deep cervical lymph-node tissue submitted	Sample date
Day 19	Calcium/calcitriol	Calcium 9.5 mg/dL; calcitriol 42.6 pg/mL	Sample date
Day 39	Fungal culture	No growth after 30 days	Final validation
Day 55	M. tuberculosis culture	No growth after 42 days	Final validation
After diagnostic assessment	Systemic treatment	Prednisolone 30 mg orally daily	Relative phase
Following week	Inpatient response	Reduced glandular swelling and pain; no dose taper	Observation interval

TABLE 2: Documented investigation timeline and observed treatment interval

Day 1 represents the initial documented laboratory assessment; subsequent days indicate elapsed time from that assessment. Culture-report validation days are distinct from specimen-collection days. Treatment and response intervals are described relative to prednisolone initiation. The observed response is interpreted within the diagnostic assessment and is not disease-specific [2,8].

Abbreviations: CBC, complete blood count; CT, computed tomography; T4, thyroxine; M. tuberculosis, Mycobacterium tuberculosis; MGIT, mycobacterial growth indicator tube. Units: mg, milligram; mg/dL, milligrams per deciliter; pg/mL, picograms per milliliter

Discussion

This case illustrates the diagnostic difficulty of bilateral parotid and lacrimal enlargement when findings from different tissues suggest overlapping inflammatory conditions. Sarcoidosis was favored by the combination of glandular involvement, mediastinal and mesenteric lymphadenopathy, and non-necrotizing granulomatous lymphadenitis. This assessment followed the American Thoracic Society (ATS) framework of a compatible presentation, granulomatous inflammation, and investigation of alternative causes, recognizing that granulomas alone do not establish the diagnosis [2]. The glandular presentation is consistent with previous reports: Poate and colleagues described orofacial manifestations preceding recognition of systemic sarcoidosis, while Ungprasert and colleagues documented parotid involvement with varying laterality and occasional associated lacrimal disease [7,10].

The value of these findings lay in their combination. The lymph node biopsy helped interpret a presentation initially dominated by glandular swelling, while imaging demonstrated involvement beyond the head and neck. The Sarcoidosis Diagnostic Score offers a structured approach to combining biopsy and organ-specific findings, but its multicontinental validation also identified reduced discrimination from other noninfectious granulomatous disorders [11]. A score therefore requires careful assignment of the clinical findings and continued assessment of competing explanations [11]. The modest angiotensin-converting enzyme (ACE) elevation was treated as ancillary evidence because ACE has limited diagnostic sensitivity and specificity [2].

Ophthalmology's assessment of dry eye and conjunctivitis helped characterize the ocular symptoms. Uveitis and facial nerve dysfunction were not recorded, and she was afebrile at the documented examination. This presentation was therefore described as glandular sarcoidosis rather than assigned an incomplete Heerfordt syndrome, whose definitions involve combinations of uveitis, salivary gland enlargement, and cranial nerve involvement [4].

Sjögren disease was considered because of dry-eye symptoms, parotid enlargement, and lymphocytic glandular inflammation. Negative anti-SSA/Ro and anti-SSB/La results do not independently exclude a seronegative presentation [12]. However, the reported Chisholm-Mason grade in parotid tissue is not interchangeable with the labial-gland focus score used in the 2016 ACR/EULAR criteria [12]. The nodal granulomas and systemic distribution of disease favored sarcoidosis, which is itself an exclusion condition in those classification criteria [2,12].

IgG4-related disease was a close clinical alternative because of the salivary and lacrimal involvement. Normal serum IgG4 alone cannot exclude it; interpretation depends on tissue morphology and IgG4-positive plasma-cell assessment [13]. In this case, plasma cells were present, but parotid IgG and IgG4 immunostains

were reported as completely negative. The reporting pathologist concluded against IgG4-related disease. No numerical IgG4/IgG ratio was inferred. Primary granulomatous inflammation is also an exclusion item in the 2019 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification framework [14]. These findings were considered together rather than relying on serum IgG4 alone. Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis is another recognized cause of glandular involvement [15].

The history of thyroid carcinoma also required consideration of recurrent malignancy. CT showed no suspicious thyroid-bed lesion, and the sampled cervical node showed granulomatous lymphadenitis without epithelial malignant infiltration. These findings argued against recurrence at the evaluated sites, while recognizing that sarcoidosis and metastatic thyroid cancer may coexist [16]. Cancer-associated sarcoid-like reactions can also produce non-necrotizing granulomas and may regress with treatment or observation, so steroid response cannot distinguish those reactions from systemic sarcoidosis [17].

Infectious alternatives were investigated with tissue stains, tuberculosis PCR, and dedicated cultures. The negative 30-day fungal and 42-day mycobacterial cultures strengthened the microbiological assessment beyond the initial routine culture. These results were interpreted with the clinical and pathological findings [2]; negative routine culture alone does not exclude organisms such as *Nocardia* [18].

Prednisolone was followed by improvement in glandular swelling and local pain. Symptom relief and quality of life are recognized treatment considerations in sarcoidosis [5]. The 30 mg daily dose falls within the 20-40 mg initial range described in the BTS pulmonary statement, although that guidance does not establish an optimal dose for predominantly glandular disease [19]. SARCORT found no improvement in its principal relapse or treatment-failure outcome with an initial 40 mg regimen compared with 20 mg in pulmonary sarcoidosis; it did not compare 30 mg with 20 mg for this presentation [20].

The improvement was clinically useful because it documented an early response after the pretreatment findings had already favored sarcoidosis. Its diagnostic contribution nevertheless remained limited. In the prospective IgG4-related disease study by Masaki and colleagues, 93.2% of 44 patients with definite disease responded to glucocorticoids and 65.9% achieved complete remission [8]. That study did not compare IgG4-related disease with sarcoidosis, and it cannot establish superior responsiveness in either condition [8]. Although failure to respond to an adequate glucocorticoid course is an exclusion item in the IgG4-related disease classification framework, a favorable response remains compatible with that disease [14]. In the present report, “supportive” therefore means that improvement was consistent with the working diagnosis based on the preceding clinical, imaging, and pathological evidence; it does not mean that treatment response independently confirmed sarcoidosis or distinguished it from IgG4-related disease [2,8,14].

The observed response was limited to one week of inpatient follow-up, without an inpatient taper or available outpatient outcomes. Concurrent analgesics and topical ocular and cutaneous corticosteroids also limited attribution of pain and ocular or skin improvement to systemic treatment alone. The reduction in parotid swelling remained a useful clinical observation, although the photographs did not provide standardized measurements of change.

Conclusions

Sarcoidosis belongs in the differential diagnosis of bilateral parotid and lacrimal gland enlargement with systemic lymphadenopathy. Salivary gland inflammation suggestive of Sjögren disease requires interpretation alongside findings from other affected tissues. In this patient, improvement with corticosteroids reinforced a working diagnosis supported by granulomatous lymphadenitis and the distribution of disease. Treatment response contributed to diagnostic confidence, but its limited specificity requires continued attention to alternative diagnoses and the subsequent clinical course.

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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Critical review of the manuscript for important intellectual content: Michel Andraoos Grajales, Álvaro D. Durán Mendoza, Mauricio A. Quintero Betancur

Supervision: Mauricio A. Quintero Betancur

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Comité de I+D+i del Hospital General de Medellín Luz Castro de Gutiérrez E.S.E issued approval Acta 10_15092026. **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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