



Case Report

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A Confluence of Autoimmunity in a Male Patient: Severe Hypothyroidism with Massive Pericardial Effusion, Sjögren's Syndrome, and Vasculitis

Mohak Jain^{*}, Yash Patel[®], Ajay Parmar[®]

Department of General Medicine, Baroda Medical College, Vadodara, Gujarat, India.

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Running Title Polyautoimmunity in a Male Patient

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ABSTRACT

We report a rare case of polyautoimmunity in a 40-year-old male presenting with massive pericardial effusion, severe primary hypothyroidism, primary Sjögren's syndrome (SS), and leukocytoclastic vasculitis. While hypothyroidism and SS are predominantly female disorders, their confluence in a male patient presents unique diagnostic challenges. The patient exhibited progressive dyspnoea, generalized weakness, xerosis, and purpuric rashes. Diagnostic workup revealed massive pericardial effusion requiring pericardiocentesis, TSH >100 mIU/L, and positive anti-SSA/Ro and anti-SSB/La antibodies. He was treated with levothyroxine, corticosteroids, and supportive care, resulting in the resolution of the effusion, vasculitic lesions, and renal impairment. This case highlights the importance of screening for multiple autoimmune pathologies in patients presenting with complex, multisystemic illness to ensure timely and appropriate multidisciplinary management.

Introduction

Hypothyroidism is a common endocrine disorder, significantly more prevalent in females than males [1]. Pericardial effusion is a known complication of severe hypothyroidism, reported in 3–37% of cases; however, massive effusions causing hemodynamic compromise are relatively uncommon, occurring in less than 30% of such cases [2]. Sjögren's syndrome (SS) is a systemic autoimmune connective tissue disease primarily characterized by xerostomia and xerophthalmia, also exhibiting

a strong female preponderance with a ratio of approximately 9:1 [3]. SS is associated with cutaneous vasculitis in 5–10% of cases, with histopathology typically suggestive of leukocytoclastic vasculitis [4].

The co-occurrence of these three conditions—namely, severe hypothyroidism, SS, and vasculitis presenting with a life-threatening massive pericardial effusion in a male patient—is exceptionally rare. This case of polyautoimmunity poses a significant diagnostic and therapeutic challenge [3]. We report this rare confluence to highlight the diagnostic complexity and the necessity of screening for coexisting autoimmune

*** Corresponding Author:****Mohak Jain****Address:** Department of General Medicine, Baroda Medical College, Vadodara, Gujarat, India.**E-mail:** mohakjaind@gmail.com

pathologies in atypical presentations.

Case presentation

A 40-year-old male painter presented with a one-year history of generalized weakness and easy fatigability. Over the preceding six months, he developed non-pitting pedal edema, facial puffiness, hoarseness of voice, dry scaly skin on his lower limbs, hair loss, and constipation. One month prior to presentation, he experienced progressive exertional dyspnea, worsening from NYHA Class II to III, accompanied by a dry cough. He also reported significant dry mouth and polyarthralgia. There was no history of fever, oliguria, chest pain, or Raynaud's phenomenon. He had a 20-year history of tobacco chewing and had been treated for pulmonary tuberculosis nine years prior.

On examination, the patient was obese (BMI 31

kg/m²), apathetic, and pale, with periorbital and generalized facial puffiness (myxedema facies), coarse sparse hair, and xerotic skin (Figure 1). He had a hoarse voice with decreased pitch. Musculoskeletal examination revealed suprapatellar swelling over the right knee with mild tenderness. His blood pressure was initially 90/60 mmHg (rising to 110/60 mmHg post-pericardial tapping), pulse 98 bpm, and respiratory rate 18/min. Bilateral lower limbs showed non-pitting edema and palpable, non-blanching, maculopapular purpuric lesions over the thighs and legs (Figure 2). Cardiovascular examination revealed muffled heart sounds, and lung auscultation showed fine crepitations at the bases. Neurological examination demonstrated delayed deep tendon reflexes (Woltman sign).

Diagnostic workup included an electrocardiogram showing low-voltage QRS complexes with widespread T-wave inversion. Chest radiograph revealed marked



Fig. 1. Facial Puffiness with dry skin, sparse hair, and wrinkles over the neck.



Fig. 2. Non blanchable, multiple small reddish coloured, diffuse, macule to papule to plaque rashes present over bilateral thighs, legs and sides of abdomen suggestive of small vessel vasculitis.

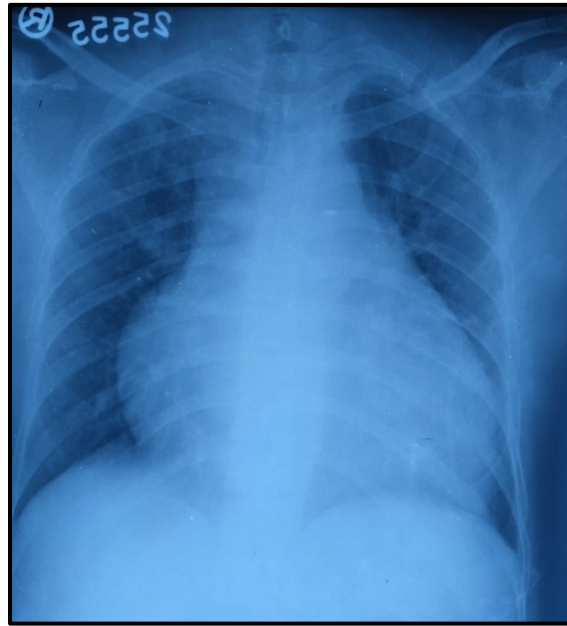


Fig. 3. Chest Radiograph showing enlarged cardiac silhouette (Money Bag appearance)

cardiomegaly with a “money bag” appearance (Figure 3). 2-D echocardiography demonstrated a large circumferential pericardial effusion (maximum 30 mm) with a swinging heart, mild mitral and tricuspid regurgitation, and a dilated inferior vena cava. HRCT chest showed moderate left and minimal right pleural effusion with patchy consolidations. Thyroid ultrasonography revealed a normal thyroid gland size with a homogeneous echopattern.

Laboratory investigations (Table 1) revealed severe primary hypothyroidism and acute renal dysfunction with hyperuricemia. Inflammatory markers (ESR, CRP) were elevated. LDH was markedly high at 1636 U/L. The hemogram showed severe microcytic, hypochromic anaemia. Autoantibody screening was positive for Sjögren’s syndrome (anti-SSA/Ro, anti-SSB/La) and autoimmune thyroiditis (anti-thyroglobulin). ANA was positive. Rheumatoid factor, ANCA, and cryoglobulins were negative. The Schirmer’s test was positive. Skin biopsy was not performed as the patient did not give consent initially, as well as due to rapid resolution of changes on starting therapy. Based on these findings, the patient was diagnosed with:

- Large pericardial effusion.
- Severe primary hypothyroidism (Hashimoto thyroiditis).
- Primary Sjögren’s syndrome (ACR/EULAR 2016 criteria [5]).

- Vasculitis (small vessel non-cryoglobulinaemic, probable leukocytoclastic vasculitis). Therapeutic pericardiocentesis was performed, draining the exudative effusion (glucose 74.0 mg/dL, protein 7.4 g/dL, ADA 14.30 IU/mL), which led to immediate symptomatic improvement. He was treated with oral L-thyroxine 100 mcg daily and low-dose corticosteroids. Supportive therapy included antacids, multivitamins, folic acid, laxatives, and iron supplementation. Following treatment, renal and liver functions normalized within a week (Table 1, Figure 4), and vasculitis lesions resolved.

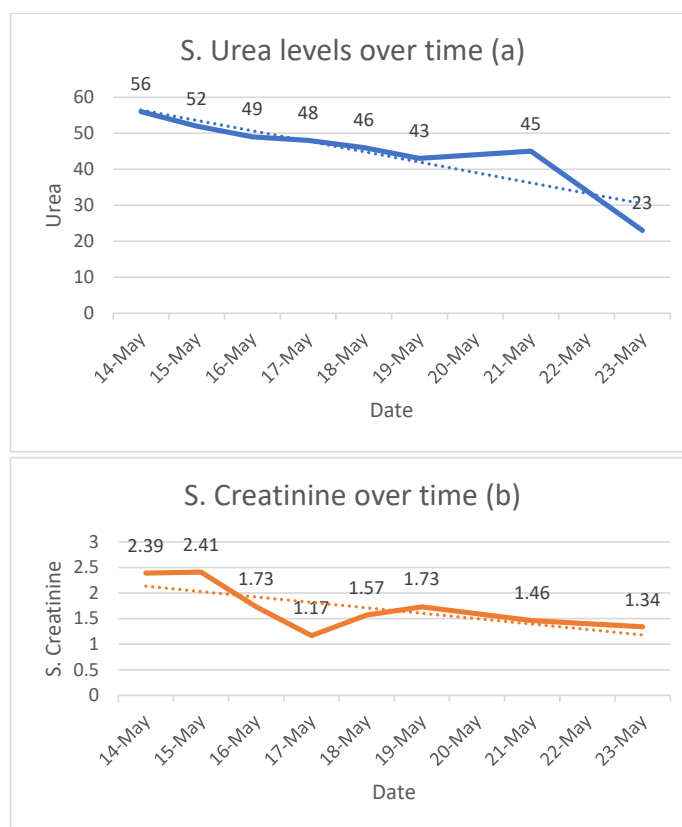
Immediate follow-up showed improvement in dyspnea following pericardiocentesis and initiation of thyroxine. The long-term management will focus on optimizing thyroid status, managing Sjögren’s syndrome, and monitoring for recurrence of vasculitis and pericardial effusion.

Discussion

This case presents a rare confluence of severe primary hypothyroidism, primary Sjögren’s syndrome, and non-cryoglobulinemic probable leukocytoclastic vasculitis in a 40-year-old male presenting with a large pericardial effusion. Each of these conditions individually is less common in males [1], and their simultaneous occurrence is highly unusual. Large pericardial effusion is an uncommon but serious complication of severe hypothyroidism [2]. The effusion in hypothyroidism is typically exudative, as

Table 1. Serial laboratory parameters on admission and follow-up

Parameter	On admission	After 1 week	Notes / Reference range	Unit
Serum Urea	56	23	14-40	mg/dL
Serum Creatinine (Cr.)	2.39	1.34	0.7-1.4	mg/dL
Sodium (Na+)	135	139	135-145	mEq/L
Potassium (K+)	4.2	4.3	3.5-5.1	mEq/L
S. Uric Acid	12.5	3.6	2.5-7.2	mg/dL
SGPT	32	21	0-40	IU/L
SGOT	58	32	0-37	IU/L
ALP	120	93	0-120	IU/L
TP	8.3	8.3	6-8.3	gm/dL
Albumin	3.5	3.6	3.5-5.5	gm/dL
CRP	122	66	0-6	mg/dL
ESR	146	37	0-15	mm/hr
Hemoglobin	8.2	8.5	13-17	gm/dL
PCV	26.7	26.3	40-50	%
MCV	76.6	71.9	83-101	fL
MCH	23.4	23.2	27-32	pg
MCHC	30.7	32.3	31.5-34.5	gm/dL
Total White Cell Count	11800	4800	4000-10000	/mm ³
Platelet Count	157 × 10 ³	160 × 10 ³	150 × 10 ³ - 450 × 10 ³	/mm ³
TSH	>100 mIU/L		0.4-4.2	mIU/L
Free T4	0.32 ng/dL		0.8-1.8	ng/dL
Free T3	0.69 pg/mL		2.3-4.2	pg/mL
Anti-Thyroglobulin Antibody	147 IU/mL		0-115	IU/mL
Anti-TPO Antibody	18.27 IU/mL		0-35	IU/mL
Cryoglobulins (Serum)	Negative		Negative	
ANA Screening (By Immunofluorescence)	+3 (Coarse Speckled, Cytoplasmic Granular)		Negative	
Anti-SSA/Ro	3		Negative	
Anti-SSB/La	3		Negative	
Schirmer's Test	7 mm/5 min (both eyes)		>10 mm/5 min	

**Fig. 4 (a,b).** Serum creatinine and urea values over 1 week of therapy with Thyroxine and supportive therapy.

seen in this patient, and develops slowly. Sjögren's syndrome, while primarily affecting exocrine glands, is a systemic disease and can have extraglandular manifestations involving skin, causing xerosis and vasculitis [4]. Cardiac involvement in SS is considered rare but can include pericarditis and mild pericardial effusion [6].

The patient presented with elevated serum creatinine and uric acid, which is likely secondary to a reduction in renal plasma flow and glomerular filtration rate, a known complication of severe hypothyroidism. These renal parameters, including the borderline hyponatremia, showed significant improvement following the initiation of levothyroxine therapy [7]. Similarly, the elevated aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) levels, in the context of normal bilirubin and borderline low albumin, were attributed to hypothyroid-induced myopathy [8]. The patient's hematological profile revealed microcytic hypochromic anemia, which is atypical for hypothyroidism, as it usually causes normocytic normochromic anemia. Although serum ferritin was within the normal range, it is likely that a concomitant iron deficiency was masked, as ferritin is an acute-phase reactant and can be elevated in inflammatory states like this patient's autoimmune condition. The iron profile was also suggestive of iron deficiency. The patient's vegetarian diet is a potential contributing factor to iron deficiency. While occupational exposure to lead in the paint industry was considered as a cause for anemia, it was deemed less probable because the patient's anemia responded well to oral iron supplementation after the systemic inflammation was controlled with steroids and other immunosuppressants [9].

Leukocytoclastic vasculitis is the most common form of vasculitis seen in SS, often manifesting as palpable purpura on the lower extremities [4]. Importantly, in this case, the vasculitis was non-cryoglobulinemic. Skin vasculitis in SS is associated with more severe disease [4].

The co-occurrence of autoimmune thyroid disease and Sjögren's syndrome is well-documented, suggesting shared pathogenic mechanisms or genetic predispositions (polyautoimmunity) [3]. Hypothyroidism is one of the most common autoimmune diseases to develop in patients with primary SS [3]. The patient's occupational history in the paint industry raises the possibility of exposure to organic solvents, which have been implicated as a risk factor for autoimmune diseases, including systemic vasculitis [10]. In autoimmune thyroid disease, it

is probable that solvents may interfere with iodine transportation and induce oxidative stress that leads to an inflammatory response to the thyroid gland [10]. Tobacco chewing, as in this patient, can also exacerbate symptoms of dry mouth in Sjögren's syndrome [11]. Both factors are important points to consider in this patient, although direct causality cannot be established. The patient's neuropsychiatric symptoms, such as decreased concentration and lethargy, could be attributed to severe hypothyroidism but can also be manifestations of Sjögren's syndrome, which is known to cause cognitive dysfunction and fatigue [12].

The diagnosis required careful integration of clinical features, serological markers, and imaging. Management involved addressing each component: thyroid hormone replacement, immunosuppression for vasculitis and potentially for systemic manifestations of SS, and management of the life-threatening pericardial effusion.

Conclusions

This case shows the importance of considering multiple autoimmune diagnoses in male patients presenting with complex, multisystemic illness, even when individual conditions are more prevalent in females. Large pericardial effusion, while rare, can be a presenting feature of severe hypothyroidism, potentially compounded by coexisting autoimmune conditions like Sjögren's syndrome and associated vasculitis. A multidisciplinary approach is crucial for timely diagnosis and management of such intricate cases.

List of abbreviations

- **ADA:** Adenosine Deaminase
- **ANA:** Antinuclear Antibody
- **ANCA:** Antineutrophil Cytoplasmic Antibodies
- **BMI:** Body Mass Index
- **CRP:** C-Reactive Protein
- **ESR:** Erythrocyte Sedimentation Rate
- **HRCT:** High-Resolution Computed Tomography
- **IVC:** Inferior Vena Cava
- **LDH:** Lactate Dehydrogenase
- **LVEF:** Left Ventricular Ejection Fraction
- **NYHA:** New York Heart Association
- **SS:** Sjögren's Syndrome
- **TSH:** Thyroid Stimulating Hormone

Ethical Considerations

Consent for publication:

Written informed consent was obtained from the patient for participation.

Ethical approval and its number

Ethics approval was waived as this is a case report of a single patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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Conflict of Interests

The authors have no conflict of interest to declare.

AI Disclosure

The authors used a large language model (ChatGPT, OpenAI) solely for language editing and grammatical refinement. All clinical content, interpretation, and conclusions are original and were independently verified by the authors.

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