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Concurrent Thyrotoxicosis-Induced Atrial Fibrillation and Seropositive Rheumatoid Arthritis Masquerading as Urinary Sepsis: A Case Report

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ABSTRACT

Autoimmune thyroid disease and rheumatoid arthritis (RA) share overlapping immunogenetic pathways, but their simultaneous, fulminant onset is rarely reported and can be mistaken for sepsis. A 68-year-old previously healthy woman presented with fever, chills, polyarthralgia, and an irregular pulse of 160 beats/min, initially diagnosed as urinary sepsis with hyponatremia. Work-up revealed suppressed thyroid-stimulating hormone (TSH) with elevated free T3/T4 and atrial fibrillation with fast ventricular response, alongside positive anti-cyclic citrullinated peptide antibody, rheumatoid factor, and elevated erythrocyte sedimentation rate, confirming synchronous thyrotoxicosis and seropositive RA. She improved with rate control, anticoagulation, carbimazole, and disease-modifying antirheumatic drugs (DMARDs). Autoimmune emergencies can closely mimic infection, a high index of suspicion and multidisciplinary management are essential to prevent cardiovascular and metabolic decompensation.

INTRODUCTION

Endocrine emergencies and systemic rheumatologic disease frequently intersect, particularly in older adults, where presentations are often atypical. Autoimmune thyroid disease and RA co-exist more often than expected by chance, reflecting a shared immunogenetic architecture involving HLA-DRB1 and PTPN22 susceptibility loci [1,2]. However, reports of simultaneous, hyperacute onset remain scarce, and most literature instead addresses how pre-existing

thyroid dysfunction affects RA treatment response [3]. This gap is clinically important as the hypermetabolic, hyperinflammatory state produced by concurrent autoimmune surges can closely mimic systemic inflammatory response syndrome, misdirecting clinicians toward infectious work-ups such as urinary sepsis [4,5]. We report a case where combined thyrotoxic and rheumatoid crisis was initially concealed behind a presumed infectious diagnosis, underscoring the need for broad diagnostic vigilance in geriatric emergency presentations.

CASE DESCRIPTION

A 68-year-old woman with no comorbidities presented with a seven-day history of fever with chills, bilateral knee and wrist pain, and generalized weakness, provisionally diagnosed as urinary sepsis with hyponatremia. She had no prior thyroid, cardiac, or autoimmune illness and took no regular medication. She was febrile with an irregular pulse of 160 beats/min, blood pressure 110/70 mmHg, and SpO_2 95% on room air; cardiorespiratory, abdominal, and neurological examinations were otherwise unremarkable (GCS 15/15).

Electrocardiography showed absent P waves with an irregularly irregular rhythm, confirming atrial fibrillation with fast ventricular response. Laboratory findings are summarized in Table 1. These findings established synchronous thyrotoxicosis and seropositive RA fulfilling ACR/EULAR criteria. Chest radiograph, echocardiography, and fundoscopy (hazy media, drusen, normal macula) were unremarkable beyond a cataract referral.

She received intravenous amiodarone for rate control and heparin for thromboembolic prophylaxis, followed by carbimazole, and methotrexate, leflunomide, and hydroxychloroquine after rheumatology review. Fever resolved, heart rate normalized, and joint pain improved over six days and she was discharged on carbimazole, bisoprolol, methotrexate, leflunomide, and supportive therapy, with follow-up advised after one week.

Table 1: Laboratory test values of the patient with reference ranges during admission.

Parameter	Patient value	Reference range
TSH	0.005 µIU/mL	0.4–4.0 µIU/mL
Free T3	4.8 pg/mL	2.3–4.2 pg/mL
Free T4	3.6 ng/dL	0.8–1.8 ng/dL
ESR	55 mm/hr	<20 mm/hr
Anti-CCP antibody	Positive	Negative
Rheumatoid factor	Positive	Negative
Haemoglobin	10.5 g/dL (microcytic, hypochromic)	12–15 g/dL
Lymphocyte count	0.9 $\times 10^3$/µL	1.0–4.8 $\times 10^3$/µL
Serum sodium	128 mmol/l	135–145 mmol/L

DISCUSSION

This presentation shows how a genuine dual autoimmune emergency can be misread as sepsis. Fever, tachycardia, and weakness are common to thyroid storm and acute RA flares alike, while hyponatremia is driven here by non-osmotic antidiuretic hormone release from a hyperdynamic thyrotoxic circulation and by cytokine-mediated impairment of renal free-water excretion reinforced the infectious impression [6,4]. Lymphocytopenia and anaemia of chronic disease, mediated by IL-6-driven hepcidin upregulation, further blurred the picture [1].

Mechanistically, RA and Graves' disease cluster through shared HLA-DRB1 and PTPN22 susceptibility, explaining their bidirectional risk relationship [1,2]. Cytokine release (IL-1, IL-6, TNF-α) from synovitis can synergize with thyrotoxic hypermetabolism, while excess thyroid hormone upregulates cardiac β1-adrenoceptor density, precipitating atrial fibrillation [7]. Amiodarone, though effective acutely, carries iodine-related risks of worsening thyrotoxicosis and interacts with hydroxychloroquine to prolong the QTc interval and selective β1-blockade (bisoprolol) and heparin-to-DOAC transition are therefore preferred once stable, particularly given a CHA_2DS_2-VASc score ≥2 [7,8]. Pre-existing macular drusen further justified deferring hydroxychloroquine. Emerging evidence suggests low-dose methotrexate may accelerate remission in Graves' disease by suppressing pathogenic TRAb-producing clones, offering a rational dual-purpose anchor DMARD in such overlap cases [9,10].

CONCLUSION

Simultaneous thyrotoxic and rheumatoid crises can convincingly mimic sepsis in older adults. Recognizing shared immunogenetic and inflammatory mechanisms, anticipating drug interactions, and coordinating endocrinology, rheumatology, cardiology, and ophthalmology input are essential to averting cardiovascular and metabolic decompensation.

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