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The intersection of obesity, testosterone therapy, and atrial fibrillation: A case report

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Abstract

This report describes a 22-year-old man with morbid obesity, exogenous testosterone therapy, opioid use, and erythrocytosis who developed Atrial Fibrillation (AF) with Rapid Ventricular Response (RVR). He presented with acute palpitations and chest pain, and his rhythm converted to sinus with medical management, including a diltiazem infusion. Diagnostic evaluation excluded alternative causes of AF and confirmed erythrocytosis attributed to testosterone therapy. This case illustrates how overlapping public health concerns, obesity, opioid exposure, and widespread testosterone replacement can converge to create a pro-arrhythmic substrate in young adults. It highlights the need for careful monitoring in this population.

Keywords: Obesity; Hypogonadism; Testosterone replacement therapy (TRT); Erythrocytosis; Atrial fibrillation (AF).

Abbreviations: AF: Atrial Fibrillation; ARIC: Atherosclerosis Risk in Communities; BNP: B-Type Natriuretic Peptide; BMI: Body Mass Index; BP: Blood Pressure; CRP: C-Reactive Protein; CTA: Computed Tomography Angiography; DCCV: Direct Current Cardioversion; ECG: Electrocardiogram; EPO: Erythropoietin; EAT: Epicardial Adipose Tissue; FSH: Follicle-Stimulating Hormone; GnRH: Gonadotropin-Releasing Hormone; Hb: Hemoglobin; Hct: Hematocrit; HPG Axis: Hypothalamic-Pituitary-Gonadal Axis; IL-6: Interleukin-6; LH: Luteinizing Hormone; OSA: Obstructive Sleep Apnea; PT: Prothrombin Time; RBC: Red Blood Cell; REGARDS: Reasons for Geographic and Racial Differences in Stroke; RVR: Rapid Ventricular Response; TEE: Transesophageal Echocardiography; TRT: Testosterone Replacement Therapy; TSH: Thyroid-Stimulating Hormone; WBC: White Blood Cell.

Introduction

Obesity affects more than 40% of U.S. adults [1] and is projected to impact nearly half the global population by 2035 [2]. Beyond its metabolic and cardiovascular consequences, obesity is strongly associated with male hypogonadism, with prevalence estimates exceeding 30% in affected men [3,4]. Testosterone

Replacement Therapy (TRT) is frequently prescribed to manage obesity-related hypogonadism and offers well-documented benefits [5]; however, it also carries important risks. One of the most established dose-dependent adverse effects is erythrocytosis [6], occurring in approximately 13% of men treated with TRT and more commonly in those with comorbidities such as obesity-related obstructive sleep apnea [7].

Atrial Fibrillation (AF), the most common sustained cardiac arrhythmia, has been independently associated with both obesity and elevated hematocrit levels [8,9]. Opioid use, another growing public health concern affecting 3.7% of U.S. adults [10], may further increase AF susceptibility by altering autonomic tone and impairing ventilatory drive, thereby destabilizing atrial electrophysiology [11]. Although obesity [12], TRT-related erythrocytosis [5], and opioid exposure [11] are individually recognized contributors to AF risk, their combined effect is rarely discussed, particularly in young adults.

We present a young man with morbid obesity–related hypogonadism treated with TRT who developed erythrocytosis and subsequently experienced paroxysmal AF in the setting of concurrent opioid use. This case highlights the underrecognized arrhythmic vulnerabilities in young obese men receiving testosterone therapy and underscores the importance of vigilant monitoring when multiple risk factors coexist.

Case Presentation

A 22-year-old man presented with sudden-onset palpitations and severe chest pain that awakened him from sleep. He denied dyspnea, fever, or chills. His medical history included type 2 diabetes mellitus treated with semaglutide (Ozempic); chronic back pain managed with long-acting buprenorphine (Belbuca) and intermittent hydrocodone–acetaminophen; morbid obesity with substantial weight reduction (BMI decreased from 66 kg/m^2 to 47 kg/m^2); and ongoing testosterone cypionate therapy for symptomatic hypogonadism. He had been receiving intramuscular testosterone 100 mg weekly for approximately three years before the dose was reduced to 80 mg following the atrial fibrillation episode and subsequently discontinued upon endocrinology evaluation.

In the emergency department, he was found to be in atrial fibrillation with Rapid Ventricular Response (RVR) at 180 beats per minute. The arrhythmia was refractory to intermittent oral and intravenous diltiazem but responded to continuous diltiazem infusion, which stabilized his heart rate in the 80s. Laboratory testing demonstrated erythrocytosis and mild leukocytosis. Chest CTA ruled out pulmonary embolism, and toxicology screening and thyroid studies were unremarkable. He was admitted for further management. Although cardioversion via Direct Current Cardioversion (DCCV) or transesophageal echocardiography (TEE) was initially considered, these interventions were ultimately unnecessary, as he spontaneously converted to normal sinus rhythm and remained stable with medical therapy alone.

Investigations

Initial electrocardiography demonstrated atrial fibrillation with rapid ventricular response at 180 beats per minute. Laboratory testing revealed erythrocytosis, with hemoglobin 19 g/dL, hematocrit 58.3%, and RBC count $6.69\times10^6/\mu L$, along with mild leukocytosis ($14\times10^3/\mu L$). Red cell distribution width was

slightly elevated at 14.6%. Serum sodium was mildly decreased (131 mEq/L) but corrected spontaneously to 137 mEq/L. Blood glucose was initially elevated at 177 mg/dL but later normalized to 86 mg/dL; HbA1c was 5.7%. Lipoprotein(a) was normal at 31 nmol/L.

Cardiac biomarkers were negative, including troponin I (<0.04 ng/mL) and BNP (<100 pg/mL). Thyroid- stimulating hormone was within normal limits. Urine toxicology was negative for stimulants and opioids; buprenorphine was not detected, as it is not included in routine screening assays. Blood cultures showed no growth.

Imaging studies included a chest CTA, which excluded pulmonary embolism, and a transthoracic echocardiogram demonstrating preserved ejection fraction without structural abnormalities. Key laboratory and imaging results are summarized in Table 1.

Key laboratory and imaging findings are summarized in Table 1.

Test	Result	Reference Range	Interpretation
Hemoglobin	19 g/dL	13.5–17.5 g/dL	Erythrocytosis
Hematocrit	58.30%	41–50%	Erythrocytosis
RBC count	6.69×10^6/μL	$4.7–6.1 \times 10^6$/μL	Elevated
WBC count	14×10^3/μL	$4.5–11 \times 10^3$/μL	Mild leukocytosis
Sodium	131 mEq/L (→137)	135–145 mEq/L	Mild hyponatremia that corrected spontaneously
Troponin I	Negative	<0.01–0.014 ng/mL	Within normal limits
BNP	Negative	<100 pg/mL	Within normal limits
TSH	1.68 mIU/L	0.4–4.0 mIU/L	Rules out thyroid etiology

Differential diagnosis

Several potential etiologies for atrial fibrillation in this young patient were considered. Infection was initially suspected given the presence of leukocytosis; however, the absence of fever, negative blood cultures, lack of an identifiable infectious source, and spontaneous normalization of the white blood cell count made infection unlikely. Drug-induced arrhythmia was also considered due to his chronic analgesic use, but a comprehensive toxicology screen was negative for stimulants and opioids. The absence of detected opioids was consistent with his intermittent oxycodone use, and buprenorphine was not assessed, as it is not included in standard screening assays.

Thyroid dysfunction, a common reversible cause of atrial fibrillation, was excluded by normal thyroid-stimulating hormone levels. Pulmonary embolism, another important cause of new-onset arrhythmia, was ruled out by a negative chest CTA. Other causes of erythrocytosis were also evaluated.

Carboxyhemoglobin Mia was unlikely due to the absence of smoking or relevant environmental exposures, and no cardiac, pulmonary, or renal abnormalities were identified on examination or laboratory evaluation.

After exclusion of these potential etiologies, testosterone-induced erythrocytosis in the context of obesity- related hypogonadism was considered the most plausible contributor to his arrhythmia. The potential additional influence of chronic opioid therapy is explored further in the Discussion.

Treatment

In the emergency department, the patient was started on metoprolol tartrate for rate control and apixaban for stroke prevention, given his diagnosis of atrial fibrillation. His CHA_2DS_2-VASc score was 1, indicating a low-to-intermediate risk of thromboembolic events. Anticoagulation was initiated in anticipation of possible direct current cardioversion.

Discharge diagnoses

At discharge, the patient's presentation was attributed to paroxysmal atrial fibrillation likely precipitated by multiple interacting factors, including morbid obesity, chronic opioid exposure, and exogenous testosterone therapy. His markedly elevated hemoglobin and hematocrit levels supported the diagnosis of testosterone-induced erythrocytosis. These underlying conditions were identified as significant contributors to his arrhythmia and informed recommendations for long-term management and specialist follow-up.

Discussion

Several factors likely contributed to the development of paroxysmal Atrial Fibrillation (AF) in this patient, including obesity, exogenous testosterone use, erythrocytosis, and chronic opioid exposure.

Obesity and AF risk

Obesity promotes AF through multiple interrelated mechanisms involving structural, inflammatory, and electrophysiologic alterations. Excess adiposity leads to atrial remodeling, oxidative stress, autonomic imbalance, and metabolic disturbances that together weaken atrial stability [8,12]. Epicardial Adipose Tissue (EAT) is particularly influential; it can infiltrate the atrial myocardium, slow conduction, and facilitate reentrant circuits [12]. In addition to its mechanical effects, EAT secretes proinflammatory and profibrotic adipokines, including chemerin and resistin, that alter ion channel function, promote fibrosis, and increase susceptibility to arrhythmia [8,12].

Obesity is also characterized by elevated circulating inflammatory cytokines such as interleukin-6 (IL-6), C-Reactive Protein (CRP), and Tumor Necrosis Factor-alpha (TNF-α), which contribute to oxidative stress, collagen deposition, and impaired calcium handling, all of which disrupt atrial electrophysiology [14,15]. Furthermore, obesity is strongly associated with Obstructive Sleep Apnea (OSA), which heightens sympathetic activation, promotes atrial stretch and fibrosis, and further increases the risk of AF [4,13].

Collectively, these mechanisms create a vulnerable atrial substrate in which additional triggers can more easily initiate arrhythmia (Figure 1).

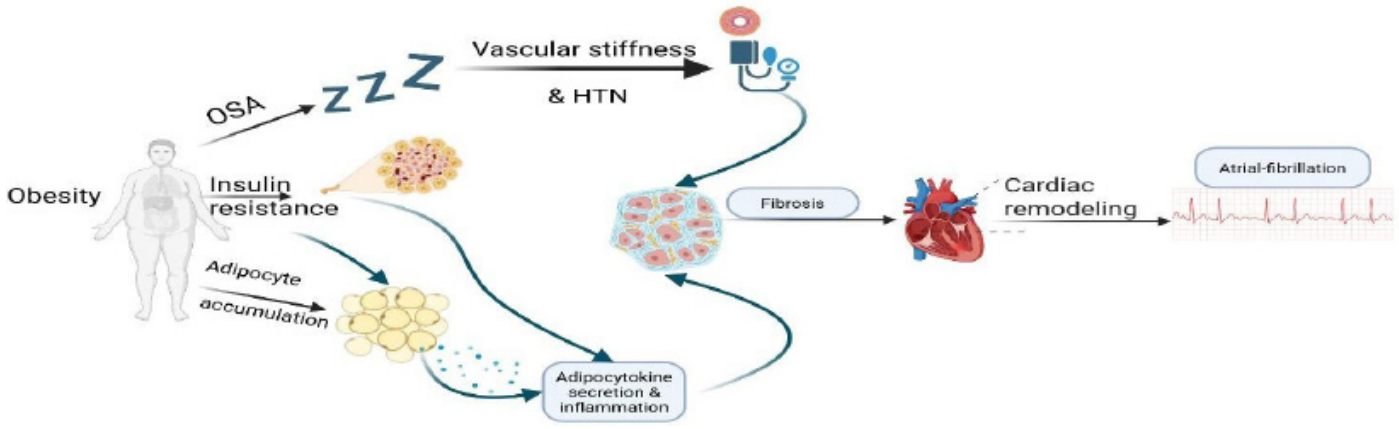

Figure 1: Mechanistic pathways linking obesity to atrial fibrillation.

Obesity induced hypogonadism risk:

Beyond its direct arrhythmogenic impact, obesity plays a central role in the development of secondary male hypogonadism through disruption of the Hypothalamic–Pituitary–Gonadal (HPG) axis. Increased aromatase activity within adipose tissue accelerates the conversion of testosterone to estradiol, which suppresses Gonadotropin-Releasing Hormone (GnRH) and Luteinizing Hormone (LH) secretion through negative feedback, thereby reducing testicular testosterone production [16,17]. Obesity-associated hyperleptinemia, leptin resistance, chronic low-grade inflammation, and oxidative stress further impair hypothalamic signaling and worsen central suppression of the HPG axis [3].

This dynamic has been described in Cohen's "hypogonadal–obesity cycle," in which increasing adiposity drives aromatase activity and estradiol production, perpetuating hormonal suppression and promoting additional fat accumulation [18]. The resulting decline in endogenous testosterone contributes not only to progressive adiposity but also to physiological disturbances that may predispose to atrial arrhythmia. As a result, many obese men are treated with exogenous testosterone to address symptomatic hypogonadism, introducing additional considerations relevant to AF risk.

TRT induced erythrocytosis and AF risk

Testosterone Replacement Therapy (TRT) is widely used to treat hypogonadism and is effective in improving energy, libido, and muscle mass [19]. However, TRT exerts potent hematologic effects that can become clinically significant. Testosterone increases hemoglobin and hematocrit through several complementary mechanisms: stimulation of renal Erythropoietin (EPO) transcription, enhanced responsiveness of erythroid progenitors to EPO, suppression of hepatic hepcidin leading to increased intestinal iron absorption, expansion of erythroid precursor populations, and prolonged red blood cell survival [5]. These coordinated pathways predictably result in erythrocytosis.

Beyond hematologic effects, testosterone influences cardiac electrophysiology by altering ion channel expression and augmenting adrenergic signaling, creating a more excitable atrial substrate [20]. TRT can also exacerbate or induce Obstructive Sleep Apnea (OSA) by increasing upper-airway collapsibility and blunting ventilatory responses to hypoxia and hypercapnia, thereby heightening nocturnal sympathetic

activation and oxygen demand [20]. For these reasons, clinical guidelines recommend monitoring for new or worsening OSA in patients receiving TRT. Together, these mechanisms link testosterone therapy to increased susceptibility to Atrial Fibrillation (AF) and are summarized in Figure 2.

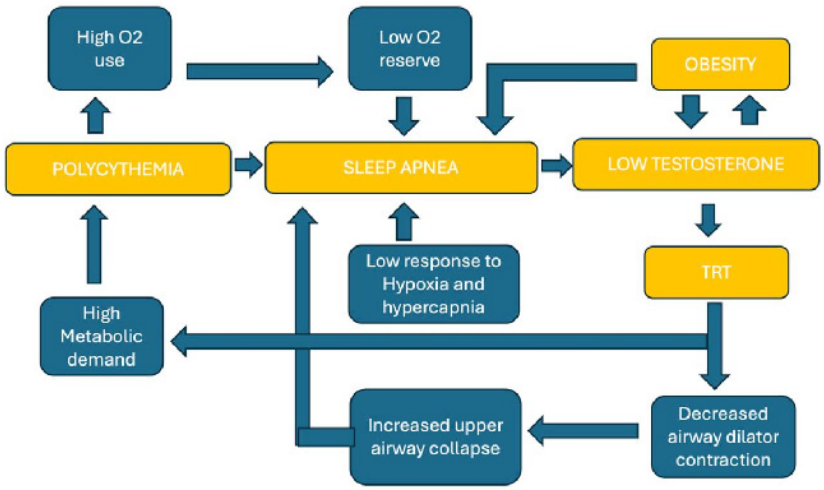

Figure 2: Relationship between obesity, OSA, testosterone and polycythemia.

Clinical evidence supports these mechanistic concerns. In the TRAVERSE trial, which followed approximately 5,200 men aged 45–80, testosterone therapy did not increase major adverse cardiovascular events overall; however, the incidence of atrial fibrillation was significantly higher in the testosterone group compared with placebo (3.5% vs 2.4%, P=0.02) [21]. Although reassuring for older men, this trial excluded younger adults, leaving unanswered whether supraphysiologic testosterone exposure in younger individuals with severe obesity or metabolic dysfunction may confer greater arrhythmic vulnerability [21]. In our patient, 22 years old, with structurally normal cardiac anatomy, the development of marked erythrocytosis (hemoglobin 19 g/dL, hematocrit 58.3%) strongly implicates testosterone-induced hematologic stress as a key contributor to his AF presentation.

Further complexity arises from population studies such as FINRISK and ARIC, which suggest a U-shaped association between endogenous testosterone levels and AF risk, with both low and high testosterone concentrations linked to increased arrhythmia incidence [22,23]. This duality underscores the importance of balanced androgen signaling untreated hypogonadism may elevate arrhythmic risk, whereas excessive or prolonged testosterone exposure may similarly destabilize atrial conduction. These findings highlight the narrow therapeutic window within which androgen levels support, rather than impair, cardiovascular stability.

Erythrocytosis and AF risk:

Erythrocytosis contributes to cardiovascular and arrhythmic risk through hemodynamic and endothelial mechanisms that destabilize atrial electrophysiology [24]. Elevated hematocrit increases blood viscosity and shear stress, imposing chronic mechanical strain on the atrial myocardium and impairing endothelial function. Data from high-altitude Andean populations with excessive erythrocytosis (hemoglobin ≥21 g/dL) demonstrate significantly reduced flow-mediated dilation that improves following isovolumic hemodilution, highlighting the direct vascular impact of hyper-viscosity [24]. Similarly, animal

models of chronic erythropoietin overexpression show that markedly elevated hematocrit can lead to ventricular dilation, myocardial edema, and reduced survival—even in the absence of hypertension [25].

Together, these studies indicate that erythrocytosis itself can drive endothelial dysfunction, cardiac remodeling, and electrical instability [24,25]. In our patient, whose hematocrit reached 58.3%, these mechanisms likely compounded the pro-arrhythmic effects of obesity and testosterone therapy, thereby lowering the threshold for atrial fibrillation.

Opioid- Induced AF risk:

Chronic opioid therapy may contribute to Atrial Fibrillation (AF) risk through autonomic dysregulation, ventilatory depression, and disruption of endogenous cardioprotective signaling pathways [26]. Under normal physiologic conditions, endogenous opioid peptides activate potassium channels that protect atrial tissue during oxidative stress; chronic exposure to exogenous opioids may diminish this protective effect and increase atrial vulnerability [11]. Opioids also exert complex vagotonic and vagolytic influences, disturbing sympathetic–parasympathetic balance and creating an electrophysiologic environment conducive to arrhythmogenesis [26]. Altered autonomic tone can destabilize atrial conduction, while direct inhibition of sodium, potassium, and calcium channels interferes with repolarization, potentially resulting in QT prolongation or increased susceptibility to triggered activity [26].

Beyond their electrophysiologic effects, opioids may also contribute to structural remodeling. Preclinical studies demonstrate that prolonged opioid exposure can induce ventricular hypertrophy, collagen deposition, oxidative stress, and myocardial stiffness, collectively promoting a substrate that supports reentrant arrhythmias [27]. In our patient, chronic buprenorphine therapy, combined with intermittent oxycodone exposure, may have added to the electrophysiologic and structural vulnerabilities created by obesity and testosterone-induced erythrocytosis.

Epidemiologic data support these mechanistic observations. In the REGARDS study, AF prevalence was significantly higher among opioid users compared with non-users (12.6% vs 8.4%, P<0.001), with the association persisting in men (12.3% vs 8.7%, P=0.002) [10]. These findings reinforce the potential contribution of chronic opioid exposure to arrhythmic risk, particularly in individuals with additional predisposing factors.

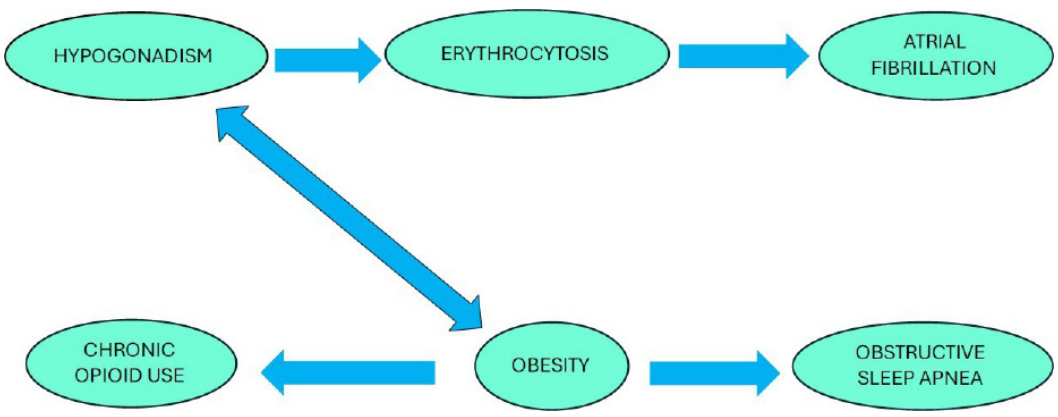

Figure 3: Mapping the pathophysiologic interactions contributing to AF in our.

Clinical guidelines emphasize the importance of regular hematologic monitoring in patients receiving testosterone therapy. A hematocrit above 50% should prompt reassessment and temporary deferral of treatment until the underlying cause is clarified, while levels at or above 54% require intervention, typically through dose reduction, adjustment of dosing interval, or temporary discontinuation of therapy [5]. In our patient, the hematocrit rose to 58.3%, exceeding guideline-based safety thresholds and likely contributing to the development of atrial fibrillation despite an otherwise structurally normal heart.

Conclusions

This case highlights the need for heightened vigilance when prescribing testosterone replacement therapy in young men with obesity and coexisting risk factors. Regular hematologic monitoring and prompt recognition of rising hemoglobin and hematocrit levels are essential to prevent avoidable cardiovascular complications. Early identification of testosterone-related erythrocytosis may allow clinicians to intervene before arrhythmia such as atrial fibrillation develops.

Relevance

This case illustrates that atrial fibrillation in young adults can arise from mechanisms that are not immediately apparent. It underscores the importance of recognizing erythrocytosis as a potential adverse effect of testosterone therapy and demonstrates how multiple risk factors, including obesity, hypogonadism, exogenous testosterone exposure, and chronic opioid use, can interact to create a pro-arrhythmic environment. Awareness of these intersecting influences supports more individualized risk assessment and management in young patients receiving androgen therapy.

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