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ANABOLIC-ANDROGENIC STEROID–INDUCED PNEUMONITIS AND MYOCARDIAL INJURY IN A YOUNG BODYBUILDER

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ABSTRACT

Introduction: Anabolic-androgenic steroids (AAS) are frequently misused by athletes to increase muscle mass and performance, and their toxicity affects multiple organ systems. This case describes cardiopulmonary complications.

Case description: We report a 29-year-old competitive bodybuilder who presented with cough, haemoptysis, exertional dyspnoea, and fatigue after escalating AAS use before a competition. Investigations revealed systemic inflammation, bilateral pulmonary ground-glass opacities, pleural effusions, and myocardial oedema with pericardial involvement. Anti-glomerular basement membrane antibodies were tested to exclude Goodpasture's syndrome, tuberculosis was excluded (GeneXpert and multiplex biomarker test negative), and polymerase chain reaction testing for coronavirus disease-19 was negative. Lab results showed mildly elevated troponin T (0.08 ng/ml, normal <0.014), supporting myocardial injury. Cultures of blood and sputum showed no pathogenic growth, and antinuclear antibodies (ANA) were negative.

The patient received empirical antibiotics, corticosteroids, and diuretics, with marked clinical improvement within 72 hours. Oral corticosteroid therapy was tapered over 7 days. Follow-up computed tomography scan of the chest 7 days later showed complete resolution of pleural effusions and ground-glass opacities. Echocardiography revealed resolution of pericardial effusion and normalization of myocardial appearance coinciding with sustained cessation of AAS. At 6-month follow-up, the patient had discontinued anabolic steroid use, remained clinically well, and resumed recreational sports.

Conclusion: This case demonstrates that AAS misuse may cause combined pulmonary and cardiac complications. Although infection cannot be fully excluded due to concurrent antibiotics, negative cultures, ANA, and Anti-GBM results support the interpretation that AAS was the primary cause of pneumonitis and myocardial injury. Improvement after cessation of AAS strengthens the causal link, supported by existing literature.

KEYWORDS

Anabolic-androgenic steroids, adverse effects, chemically induced pneumonia, bodybuilding

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LEARNING POINTS

- Anabolic-androgenic steroids (AAS) can cause serious multisystem toxicity, including pulmonary and cardiac complications, even in young athletes.
- AAS misuse should be considered in the differential diagnosis of unexplained ground-glass opacities and myocardial injury in otherwise healthy individuals.
- Detailed substance history is often difficult to obtain from patients, underscoring the need for a high index of suspicion.
- Early recognition and cessation of AAS use can lead to clinical and radiological improvement.

INTRODUCTION

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone widely misused by athletes to enhance muscle mass and performance. While cardiovascular, hepatic, and endocrine complications of AAS use are well recognized, pulmonary toxicity remains rarely described in the literature[1–3].

Reports of AAS-induced pneumonitis and diffuse alveolar haemorrhage are limited to a few isolated case studies. The mechanisms proposed include endothelial dysfunction, oxidative stress, and direct chemical injury from oil-based preparations.

Here, we present a case of a young competitive bodybuilder who developed simultaneous pulmonary and myocardial injury temporally related to escalating AAS use. The case underscores the importance of considering AAS misuse in the differential diagnosis of unexplained cardiopulmonary symptoms in otherwise healthy young adults.

CASE DESCRIPTION

A 29-year-old male bodybuilder presented with a 2-week history of productive cough with haemoptysis, progressive dyspnoea, and fatigue. He denied fever, chest pain, or recent infection. The patient reported AAS use for bodybuilding purposes but did not disclose specific agents or dosages. He had used AAS for 3 years, with recent escalation in the last 4 months for competition preparation.

Physical examination revealed tachycardia (heart rate 110 bpm), tachypnoea (respiratory rate 24/min), and oxygen saturation of 92% on room air. Bibasilar crackles and dullness on percussion were noted at the lung bases. Heart sounds were muffled with absent murmurs, and ankle oedema was present.

Laboratory tests showed leucocytosis (18.5 $\times 10^3$/µl), neutrophilia, C-reactive protein 77.5 mg/l, lactate 2.15 mmol/l, and hypoxemia with respiratory alkalosis (partial pressure of oxygen 58 mmHg). Blood and sputum cultures revealed no growth, and ANA testing was negative. High-sensitivity troponin T was mildly elevated (0.08 ng/ml), supporting myocardial injury. Anti-glomerular basement membrane antibodies and *Mycobacterium tuberculosis* testing were negative.

Computed tomography (CT) scan of the chest revealed bilateral ground-glass opacities and small pleural effusions *(Fig. 1)*. Echocardiography demonstrated diffuse myocardial hyperechogenicity ("bright myocardium") with trivial pericardial effusion, consistent with myocardial oedema.

The patient was admitted with suspected AAS-induced pneumonitis and myocardial injury. Treatment included ceftriaxone, moxifloxacin, methylprednisolone, and diuretics. Symptoms improved within 72 hours. After discharge, oral corticosteroid therapy was tapered over 1 week. The patient abstained from further AAS use. Follow-up chest CT scan 7 days after discharge showed near-complete resolution of pulmonary findings *(Fig. 2)*, and echocardiography confirmed normalization of myocardial appearance. At 6-month follow-up, the patient remained asymptomatic and had resumed recreational sports.

DISCUSSION

Systemic and cardiovascular toxicity of AAS is well documented[1,2]. However, pulmonary complications are less frequently recognized[3–5].

The myocardial injury in this patient is supported by mildly elevated troponin T and CK-MB, along with echocardiographic evidence of diffuse myocardial oedema. Although cardiac

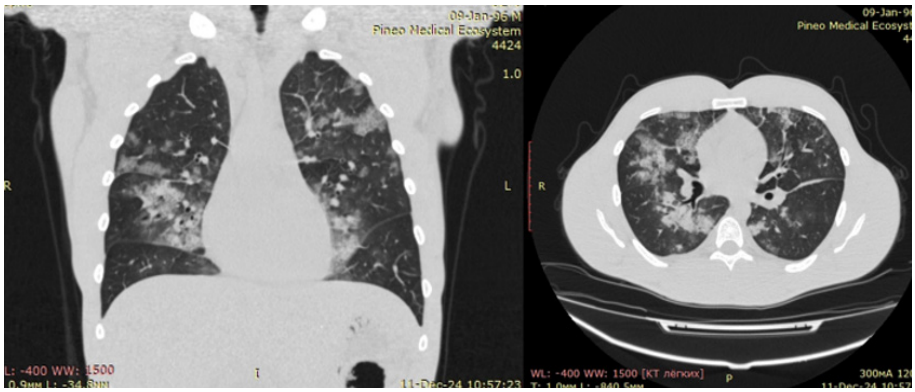


Figure 1. Computed tomography scan of the chest showing bilateral ground- glass opacities and small pleural effusions.

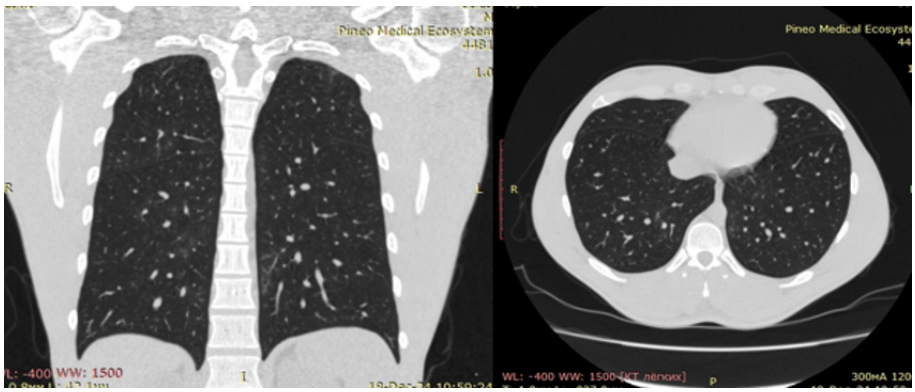


Figure 2. Follow up computed tomography scan of the chest demonstrating improvement of pulmonary ground-glass opacities and resolution if pleural effusions.

magnetic resonance imaging was not performed, these findings are consistent with previous reports of AAS-related myocardial injury and cardiotoxicity[1,2].

Importantly, blood and sputum cultures were negative, and ANA and anti-GBM antibodies were negative, reducing the likelihood of infectious or autoimmune causes. Nevertheless, the patient received broad-spectrum antibiotics, so infection cannot be entirely excluded. Sustained clinical and radiologic improvement after cessation of AAS supports a steroid-induced mechanism.

Pulmonary findings—bilateral ground-glass opacities and small pleural effusions—are consistent with previously reported cases of diffuse alveolar haemorrhage (DAH) and pneumonitis in AAS users[4–6]. Hvid-Jensen et al. reported two young bodybuilders with DAH and haemoptysis associated with AAS abuse[4]. Rodríguez et al. described a similar case with negative autoimmune serology[5]. Nomura et al. noted corticosteroid therapy led to rapid improvement in AAS-induced pneumonitis and ground-glass opacities[6].

Corticosteroid therapy was used in this patient to reduce pulmonary inflammation and stabilize alveolar-capillary injury. Rapid symptomatic and radiologic improvement suggests that steroids, in combination with AAS cessation, played a key role in recovery. Similar outcomes have been reported in other AAS-induced pneumonitis and DAH cases[4,6].

Mechanistically, pulmonary and myocardial injury may result from endothelial dysfunction, capillary leakage, oxidative stress, and direct steroid toxicity. Injectable oil-based steroid preparations may also cause chemical pneumonitis resembling fat or silicone embolism syndromes[7].

From a clinical perspective, cardiopulmonary complications in young athletes should raise suspicion for AAS use. Early recognition, discontinuation of AAS, and supportive therapy, including corticosteroids in moderate-to-severe cases, can lead to full recovery[8].

CONCLUSION

This case demonstrates that AAS misuse can cause combined pulmonary and myocardial injury in otherwise healthy young athletes. Objective evidence includes elevated troponin, echocardiographic myocardial oedema, negative infectious and autoimmune testing, and radiologic pulmonary abnormalities. Improvement after cessation of AAS and corticosteroid therapy supports causality, highlighting the importance of clinician awareness.

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