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Case report

The cost of seeking an edge: Recurrent renal infarction in setting of recreational use of anabolic steroids

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HIGHLIGHTS

- Consideration of earlier CT study with contrast.
- Thorough history taking including gym supplements.
- The use of NOACs.
- The need for further understanding of how anabolic steroids affect coagulation.
- Educating patients using anabolic steroids regarding the risks of continued use.

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ABSTRACT

Introduction: Anabolic-androgenic steroid (AAS) use and testosterone therapy have been well established risk factors for the creation of a pro-thrombotic state, and to precipitate formation of thromboemboli in individuals already predisposed to thrombosis.
Case report: Here, we present the case of an amateur bodybuilder, with a negative thrombophilia workup, who experienced primary renal infarction while using the AAS trenbolone acetate and testosterone, as well as a subsequent renal infarction while anticoagulated with apixaban.
Discussion: The development of subsequent infarctions in an anticoagulated patient with discontinued recreational steroid use poses a unique situation and challenges the current understanding of a thrombophilic state associated with steroids. The lifetime prevalence of anabolic steroid use is estimated to be 1% in the male population in the United States which is significant.
Conclusion: Further understanding and recommendations of appropriate anticoagulant should be further elucidated to appropriately medically manage patients from this confounding social and medical history.

1. Introduction

Recreational anabolic-androgenic steroid (AAS) use have been highlighted in media with the overwhelming use in professional and amateur bodybuilders. Muscular hypertrophy proceeds with the desired effects, however, multiple medical side effects ensue – impotence, severe acne, gynecomastia, atrophy of testicles, increased agitation, liver and kidney dysfunction. One of the side effects includes coagulopathy from a multifactorial pathophysiology. In a multifactorial proposed mechanism, AAS induce thrombosis through increased platelet activity, increased production of coagulation factors, increased LDL and decreased HDL cholesterol, and increased inflammation as measured by C-reactive protein [1,2]. Furthermore, testosterone therapy has been shown to promote a state of increased viscosity due to similar effects on HDL cholesterol metabolism and increases in hematocrit [3].

2. Case report

A 43-year-old male with a past medical history of obsessive compulsive disorder (OCD) and prior appendectomy presented to

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the emergency department reporting 2 days of left flank pain described as severe, sudden onset, sharp, constant, and without radiation. At the time of presentation, he denied fever, chills, dysuria, hematuria, change in urinary frequency, or changes in his bowel movements. He worked as a financial advisor and his only reported medication was escitalopram 20mg. During initial evaluation, he had an abdominal exam which revealed a soft and non-tender abdomen, no guarding or rebound elicited, no discoloration, no costovertebral angle tenderness, and no genitourinary abnormality. Urine analysis was unremarkable and urine electrolytes were not ordered. Blood work displayed a hemoglobin of 17.4 gm/dL, hematocrit of 49.5%, platelets of 186 K/μL, and creatinine of 1.7mg/dL (baseline <1.2mg/dL). A non-contrast CT was performed, which revealed no change in the renal parenchyma, and no evidence of nephrolithiasis. The patient was hemodynamically stable overnight with no signs of infection, and was sent home with opioid analgesics. His acute kidney injury was attributed to poor hydration in the setting of pain. The documentation did not reveal what the likely cause of the pain was.

The following day, the patient returned to the emergency department due to non-resolution of his pain. No changes in urination were mentioned. He underwent a contrast CT, which indicated a new, wedge-shaped hypodensity in the superolateral pole of the left kidney, measuring 5.2 × 2.5cm with adjacent fat stranding (Fig. 1). Additionally, the anterior segmental branch of the left renal artery demonstrated dilatation to 7mm with severe luminal narrowing due to occlusion by hypodense material. Serum creatinine was measured to be 1.7mg/dL again. At this time, the patient was diagnosed with left renal parenchymal infarct and acute kidney injury (AKI). He was admitted and started on a continuous heparin drip for anticoagulation.

Further work-up during that hospitalization included a transthoracic echocardiogram, which showed no evidence of thrombus or vegetations as potential etiologies for embolization. Extensive hypercoagulable workup was ordered: Factor II mutation - normal; Antithrombin III deficiency - 96% activity (normal 80–120); Factor V Leiden - normal; Cryoglobulin - none detected, ANA - negative; Lupus Anticoagulant – none detected; Anti-Cardiolipin IgG <14 (negative is < 14); Anti-Cardiolipin IgM < 12 (negative <12); Total Complement – 56U/mL (31–60); Complement C4 - 22mg/dL (12–38); Complement C3 – 78.2mg/dL (59–152); Homocysteine levels 8 μmol/L (normal 5–16). Normal ranges are mentioned above in parentheses. None of these disorders were detected. A lipid panel revealed a total cholesterol of 126mg/dL, LDL 65.8mg/dL, VLDL 15mg/dL, HDL 45mg/dL, and triglycerides of 76mg/dL. CRP was 3.57mg/L.

Upon further questioning, the patient revealed that he had been using both testosterone and the injectable anabolic steroid, trenbolone acetate, intermittently over a period of 5 years, with last use 2 weeks prior to initial admission. Consistent with anabolic steroid use, his testosterone level was 14ng/dL, DHEA-S 124 mg/dL, 17b estradiol <20 pg/mL.

As the patient's renal function and flank pain showed consistent improvement, the patient was started on apixaban and discharged home on hospital day 3.

Four days post discharge, this patient presented again to the emergency department with recurrence of severe left flank pain radiating to the left lower quadrant and groin with associated nausea. The patient again denied gross hematuria, dysuria, fever, or chills. Follow-up CT with contrast was ordered, at which time a new renal infarct was detected at the inferior pole of the left kidney (Fig. 2). The patient was again placed on a continuous heparin drip for anticoagulation. Doppler imaging revealed <60% stenosis of left renal artery with reduced flow to the superior pole. The renal vein was patent. Follow-up radioisotope renography (MAG3 scan) estimated differential renal function of 33% on the left and 67% on the right (Fig. 3). A urine drug screen was negative for all substances except opiates, which he had been prescribed.

The patient experienced gradual improvement in pain and renal function. On hospital day 3, enoxaparin was started as a bridge to therapeutic INR on warfarin. He was discharged on hospital day 4, following stabilization of creatinine (~1.3mg/dL) and acceptable pain control. Extensive counseling was provided regarding the risk of devastating thromboembolic events with continued use of anabolic steroids.

3. Discussion

The lifetime prevalence of anabolic steroid use is estimated to be 1% in the male population in the United States [4]. The breadth of complications from these agents, as well as the treatment algorithms for managing complications, are not well characterized. In this case, we cared for a 43 year old male with no predisposing pro-

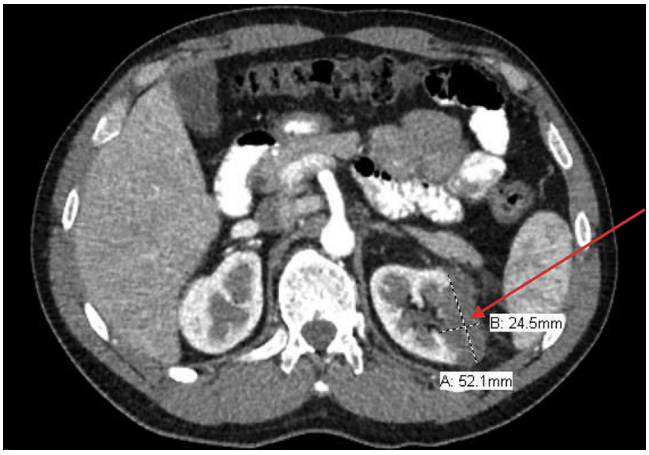

Fig. 1. Computed tomography scan of the abdomen with contrast indicating a new, wedge-shaped hypodensity in the superolateral pole of the left kidney (see arrow). Measuring 5.2 cm × 2.5 cm.

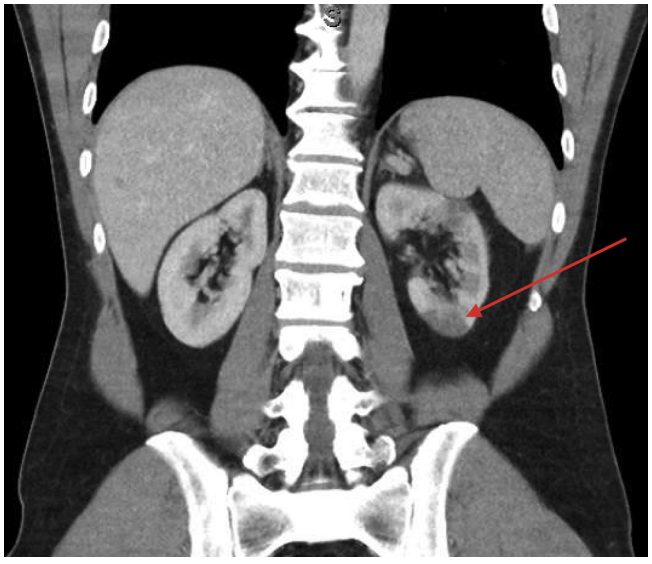

Fig. 2. Computed tomography scan of the abdomen with contrast revealed a new area of infarction at the inferior pole of the left kidney (indicated by the arrow).