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Renal Artery Thrombosis in a Bodybuilder using Anabolic Steroid - Case Report

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Abstract

Acute renal infarction (ARI) is a rare condition associated with poor prognosis in most cases. Due to non-specific clinical symptoms and abnormalities in lab results, as well as equivocal results of imagining scans, diagnosis of ARI is often delayed. Anabolic-androgenic steroids (AAS) intake in contribution to heavy weight-lifting workout are among the risk factors for hypercoagulable state. AAS are responsible for a number of haemostatic defects, including higher platelet number, enhanced platelet aggregation, increased synthesis of procoagulant factors and impaired fibrinolysis. Increasing number of AAS intake among amateur and professional athletes observed in recent years require consideration of these drugs as possible cause for thrombotic events in young patients. Yet, only few case studies have been published describing an occurrence of venous or artery thrombosis in athletes who had been using anabolic steroids. In this case report, authors present a case of right renal artery thrombosis in a 34-old bodybuilder who had been using unknown anabolic steroids to increase his performance. Due to bleeding complications after selective intra-arterial thrombolysis treatment, he required urgent nephrectomy and was released from hospital with significantly elevated azotemia parameters. This highlights the importance of careful medical history being taken with respect to AAS and illicit drug abuse for the prevention and fast, accurate diagnosis of thrombotic episodes in young athletic patients.

Keywords Anabolic-androgenic steroids; Weight-lifting; Athletes; Renal thrombosis

Introduction

Acute renal infarction (ARI) is an uncommon disease which refers to ischemic damage of renal parenchyma caused by interruption of blood flow [1]. Due to its non-specific clinical presentation, it is usually misdiagnosed, or accurate diagnosis is delayed. Recent studies have reported estimated incidences of 0,004%-0,007% based on admissions to ER departments [2].

The most common clinical symptoms of RI are: persistent abdominal and/or flank pain with nausea, vomiting and fever [1]. These symptoms can often be misdiagnosed as other common acute abdomen diseases, including: urolithiasis, diverticulitis or acute appendicitis. There are no characteristic lab results abnormalities of RI, however studies demonstrate that hematuria, proteinuria, leukocytosis, impairment of kidney function and elevated lactate dehydrogenase (LDH) support the diagnosis of renal infarction [1,3].

The basic tool for diagnosis of renal infarction is radiologic imaging [4]. Due to excellent availability and ability to out rule other common diseases, the abdominal ultrasonography should be performed in the first place. Further Doppler imaging might suggest vascular etiology of presented symptoms, but this method requires skilled radiologist and is usually not available in emergency conditions. In case of vascular disease suspicion, a wide array of radiological methods may be applied, including: computed tomography angiography, magnetic resonance angiography and conventional angiography. Conventional angiography is preferred if intravascular treatment is considered [5–9]. Although all of those methods prove to be highly specific and sensitive, they all require use of contrast which might be challenging in case of acute kidney injury. Some authors suggest that contrast enhanced ultrasonography would be superior in those cases, but this method is still rarely used which limits its feasibility [10].

Studies have revealed, that the most common risk factor for occurrence of ARI is thromboembolism secondary to cardiac diseases, including: atrial fibrillation, mitral stenosis and dilated cardiomyopathy [3,11,12]. Other causes for ARI are: hereditary thrombophilia, injury, neoplasms and birth-control pills [2–4,12]. In 30% of the cases, the etiology of ARI remains unknown [2,4].

Acute renal infarction secondary to thromboembolism of cardiac origin has been widely reported in the literature. However, we have managed to find only two described cases of ARI secondary to anabolic-androgenic steroids (AAS) consumption. With the increasing use of anabolic steroids, AAS must be considered in differential diagnosis of ARI in bodybuilders. Here, we report the case of right renal artery thrombosis in a middle-age amateur bodybuilder who had been using AAS to improve his performance.

Case Report

A 34-year-old man with irrelevant medical history was admitted to surgical emergency department with right mid abdominal pain that was radiating to the right flank area. The onset of the pain was sudden and present for few hours before admission. The patient described the pain as dull and persistent; he had no other complaints-he denied nausea, vomiting or fever. He admitted chronic consumption of AAS to improve his muscle mass (he did not mention the name or dose of the substances he used) and regular weight-lifting five times a week. Physical examination revealed tenderness on palpation in the right

mid abdominal area with no other marked abdominal signs of rebound or guarding. The examination of other systems was unremarkable. Lab tests performed in ER revealed leukocytosis, normocytic anemia, thrombocythemia, azotemia with serum creatinine level of 1,82 mg/dl (normal range: 0,72-1,25) and GFR 47 ml/min/1,73 m^2, urea level of 64 mg/dl (normal range: 19,0-44,0); elevated D-dimer level-988 ng/ml (normal range: 0-500); urinalysis showed red blood cell 5-10/hpf, proteinuria (100 mg/dl in single urine sample), the coagulation profile and inflammatory parameters were within normal limits. Abdominal ultrasound was obtained, which showed a hyperechoic right kidney with disturbed blood flow in the proximal segment of the right renal artery - renal infarct was suspected and therefore CT angiography was performed. It revealed a large wedge-shaped area of absent enhancement in the right kidney and upon this, kidney infarction was diagnosed. The patient underwent selective intra-arterial thrombolysis. Arteriography performed after the procedure showed an unobstructed right renal artery but only slightly enhancement of the parenchyma of the kidney. Antithrombotic treatment was ordered – single dose of alteplase 20 ml and enoxaparin 2 x 100 mg. In the first 24 hours after procedure, patient's general condition has been getting worse-he complained of abdominal pain and has been periodically confused. Lab results revealed a significant elevation in azotemia parameters with serum creatinine level of 3,82 mg/dl and GFR 19 ml/min/1,73 m^2, urea level of 84 mg/dl, hyperkalemia; total WBC count was 49,50 G/l, total RBC count was 2,54 T/l, and CRP was 200 mg/l. Another abdominal ultrasound was performed and a perirenal hematoma about 102 mm was revealed near the lower pole of the right kidney. Due to intra-abdominal bleeding and suspicion of sepsis, a urologist arranged for a life-saving nephrectomy. After operation, patient required CRRT and pressor amines therapy and therefore was admitted to an intensive care unit. 2 days later, he was transported back to the Department of Urology for further treatment. He was there consulted by an Internist and a Cardiologist. An echocardiogram was done, and it revealed left ventricular hypertrophy with no sign of thrombus or right ventricle pressure-overload. Further screening tests for hypercoagulability was ordered - factor V Leiden, fibrinogen level, protein C and S, anti-thrombin III, antiphospholipid antibody, homocysteine, anti–double stranded DNA were all within normal limits. Taking everything into consideration, infarction of the right kidney due to hypercoagulable state secondary to anabolic – androgenic steroids use was diagnosed. Patient was released from the hospital in a general good condition; he did not require renal replacement therapy. Serum creatinine level at discharge was 3 mg/dl. After being released, he has never showed up for a follow-up in an outpatient clinic.

Discussion

Anabolic - androgenic steroids are a group of testosterone derivatives, both natural and synthetic which are responsible for the development of male secondary sexual characteristics and increase in protein synthesis. AAS have been commonly used by bodybuilders and athletes at all levels to enhance performance and improve muscle mass and strength. Studies focusing on AAS have shown a significant rise in steroids intake since their discovery in late 1930s – the prevalence of a lifetime of anabolic steroids use is estimated to be 1% in the male population in the United States [13]. The uncontrolled misuse of these substances can result in many adverse effects including: impotence, liver and kidney disfunction, coagulopathy and vascular events [14].

The pathogenesis of vascular events and coagulopathy in AAS users is not yet well established and is probably multifactorial. The studies have proved that AAS intake is associated with higher blood pressure, endothelial disfunction and cholesterol abnormalities: decrease in high-density lipoprotein (HDL) cholesterol and increase in low density (LDL) cholesterol resulting in higher risk for atherosclerotic vascular disease [15]. Acute myocardial infarction has been reported in a 41-year old bodybuilder who had been using oxymetholone and methenolone for 15 years. 5 hours after successful PCI with metal stenting he was diagnosed with renal infarction in the right kidney. It was concluded that anabolic steroids were a contributing factor to vascular events in this patient [11].

Furthermore, AAS use is an established risk factor for the creation of pro-coagulative state [13]. Increased production of coagulation factors (both in intrinsic and extrinsic pathway), increase in platelets number and activity are associated with AAS consumption [16]. AAS' relation to impaired fibrinolytic activity has also been noted [16]. Other mechanisms for arterial thrombosis in AAS users include increased thromboxane A2 receptor density, decreased production of prostaglandins and impaired vasoreactivity [11,15].

In our case, an acquired prothrombotic state due to AAS intake has most probably contributed to development of the infarction of the kidney since other known risk factors for arterial thrombosis had been excluded. We have found two recently described cases of renal infarct secondary to AAS use [13,17]. In both cases, the right diagnosis was delayed due to non-specific symptoms and non-disclosure of this important aspect of patient's medical history. Both patients were middle-age athletic men with irrelevant prior medical history. They were both admitted to ER with flank pain described as severe, sharp, sudden onset and constant. Physical examination and lab results showed no specific abnormalities. In both cases, contrast CT scan was performed, and renal infarction was diagnosed. Upon further questioning, both patients admitted using anabolic steroids (Figure 1).

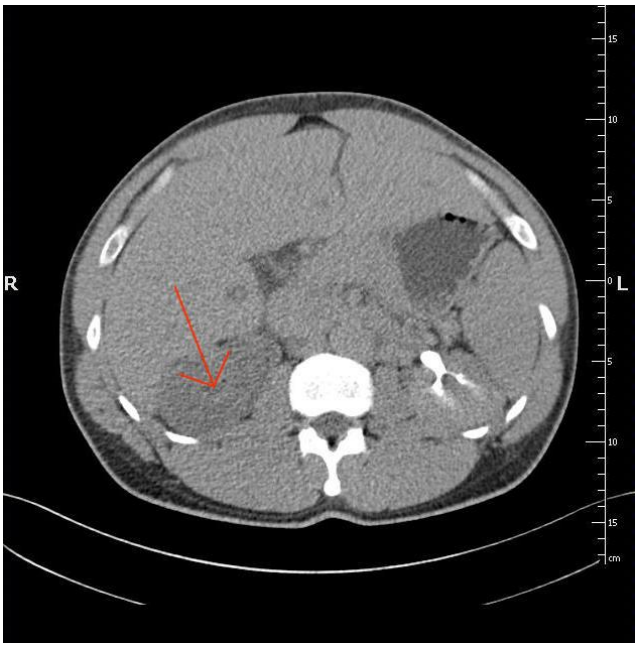

Figure 1: Radiology of Renal Artery Thrombosis.

To the best of our knowledge, our case study is the first one to present severe complications of the treatment of renal artery

thrombosis secondary to AAS intake. Due to bleeding complications of intra-arterial thrombolysis, life-saving nephrectomy had to be performed. Our patient was released from hospital with significantly elevated azotemia parameters and in the future may require renal replacement therapy in relatively young age. This highlights the importance of suspicion of renal infarct in young athletic men, especially with the history of using anabolic supplements, presented with abdominal or flank pain. We suggest a contrast CT to be performed in those patients, despite unremarkable abdominal ultrasound to exclude potential renal artery thrombosis. As for now, no clear strategies for diagnosis of ARI have been established; in Figure 2 we propose a diagnostic algorithm for ARI.

Figure 2: Diagonostic algorithm for ARI.

The limitations to our case report are: the short-time observation as our patient never showed-up for an appointment in an out-patient clinic, and secondly, the lack of information concerning the name and doses of the AAS he used.

Conclusion

Since renal infarct is a rare entity and symptoms are non-specific, a high suspicion is advised in high risk patients. It is relevant to ask all athletic men about illicit drug abuse or ASS intake when presenting with sudden onset flank pain. It could save precious time to compromise the final diagnosis.

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