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CLINICAL VIGNETTE

Anabolic Steroid Use Causing Extreme Hyperlipidemia in a Young, Healthy Male

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Introduction

Anabolic steroids increase serum LDL, reduce serum HDL, and increased risk of cardiovascular disease, particularly coronary artery disease. They can also elevate liver enzymes and hemoglobin, and can cause hypogonadism. Prevalence of anabolic steroid use varies globally, but most estimates are lower than the actual prevalence, due to the reluctance of anabolic steroid users to share their supplement history with their providers. It is estimated that anabolic steroid use is more common in men than women, and in recreational athletes than professional athletes. We describe a patient who presented to establish primary care, who had extremely abnormal initial labs, due to anabolic steroid use.

Case Description

A 24-year-old male with past medical history of sleep apnea presented to establish primary care. Routine labs prior to his appointment were notable for an LDL of 497 mg/dL, HDL of 18 mg/dL, total cholesterol of 576 mg/dL, and triglycerides of 307 mg/dL. Liver enzymes were also elevated, with an ALT of 173 units/L, AST of 69 units/L, alkaline phosphatase of 95 IU/L, and total bilirubin (TBili) of 2.76 mg/dL. Complete blood count and chemistry panels were otherwise within normal limits (Hgb 16.2 g/dL). Review of his military records from five months prior showed a total cholesterol of 220 mg/dL, LDL of 150 mg/dL, HDL of 50 mg/dL, and triglycerides of 96 mg/dL. LFTs five months prior showed ALT 78 U/L, AST 32 U/L, alkaline phosphatase 110 U/L, and TBili 1.00 mg/dL.

His initial evaluation, included a thorough history. He denied any prior history of hyperlipidemia and was very surprised to learn about his abnormal lab results. He confirmed that the labs were fasting and denied any angina, dyspnea on exertion, or family history of hyperlipidemia or cardiovascular disease. He eats 2-3 "healthy" meals each day, with only occasionally eating fast food. He denied taking any chronic medications and he had no right upper quadrant pain, diarrhea, recent sick contacts, or incarceration history. He drank alcohol 2-3 times a month with a maximum of 4 drinks. He had no known contacts with hepatitis.

There were no remarkable findings on his physical exam. He was alert and well-appearing. He had normal behavior, speech, thought process, and thought content. He had no scleral icterus or jaundice. There were no xanthomas or xanthelasmas, and there was no lymphadenopathy or thyromegaly. He was breathing comfortably on room air and his heart had regular rate and rhythm. His abdomen was soft, nontender, and nondistended without hepatosplenomegaly. He had no gynecomastia.

Upon further questioning, he revealed that he had taken a supplement called "4midable mass" for one month, but had stopped taking it three months prior to the initial visit. He also took N-acetylcysteine for "liver protection" The supplement dymethazine, methylstenbolone, methyl-1-alpha, methoxygonadiene was recommended by a friend although he had lost contact, he thought his friend may also have had some liver dysfunction.

After the initial visit, we repeated labs to confirm the results. LDL was 567.9 mg/dL, HDL was 18 mg/dL, triglycerides were 283 mg/dL, and his total cholesterol was 604 mg/dL. LFTs were notable for a persistently elevated ALT at 159 units/L, AST of 65 units/L, TBili of 2.18 mg/dL, and gamma-glutamyl transferase of 143 units/L. At this time, our differential included hypothyroidism, viral hepatitis, HIV, nephrotic syndrome, familial hyperlipidemia, and anabolic steroid use. His TSH returned within normal limits, and his hepatitis A, B, C, and HIV serologies were negative for acute infection. His urinalysis showed no proteinuria. Due to concern for anabolic steroid use, we checked his sex hormones. His testosterone was low at 180.88 ng/dL, as were his FSH at 0.5 IU/L (reference range 1.0-18.0 IU/L) and LH at 1.2 IU/L (reference range 1.42-15.4 IU/L). We counseled him to avoid the supplement and we repeated labs over the following months. His cholesterol and LFTs normalized over time, and his hypogonadism resolved. Seven months after his initial appointment, his LDL was 161 mg/dL, HDL was 42 mg/dL, total cholesterol was 233 mg/dL, and triglycerides were 150 mg/dL. His LFTs showed ALT 61 units/L, AST 30 units/L, alkaline phosphatase 97 IU/L, and TBili 2.18 mg/dL. His gonadal hormones normalized as well, with testosterone 389 ng/dL, FSH 2.2 IU/L, and LH 3.2 IU/L.

Discussion

Our patient presented with new findings of extreme hyperlipidemia, mild transaminitis, and hypogonadism on his initial visit to primary care. The differential was broad and only with thorough history taking was the likely cause elucidated as anabolic steroid use.

Anabolic steroids increase LDL by 20-150%, decrease HDL by 20-70%, and increase cardiovascular risk by 3 to 6 times

baseline risk.[1-3] Our patient's LDL was 330% of his baseline, significantly higher than the average reported increase. Mechanism and timing vary by route of administration. Anabolic steroids can also increase systolic and diastolic blood pressure and cause left ventricular hypertrophy, which may not resolve with cessation, and can rarely cause myocardial infarction and ventricular arrhythmias.[1] Additional questioning regarding anabolic steroid use should be conducted when a young person presents with cardiovascular or lipid abnormalities.

Anabolic steroid use can cause a range of hepatotoxicity, including toxic hepatitis and drug induced liver injury.[4] Other hepatotoxic effects include cholestatic icterus and hepatocellular carcinoma.[5] One particular type of anabolic steroids, 17-alpha-alkylated anabolic steroids, appear to be more hepatotoxic.[5] The alkylated component appears to slow the hepatic metabolism of anabolic steroids and results in oxidative stress.[5] Our patient's supplement had two 17-alpha alkylated components, dymethazine and methylstenbolone, which could explain his elevated transaminases and bilirubin.

Endocrine effects of anabolic steroids include hypogonadism, which our patient manifested.[6] Anabolic steroids are usually derived from testosterone, and which is converted to estradiol and estrone by aromatase. Chronic use of anabolic steroids by males can decrease testosterone production through a negative feedback loop mechanism, resulting in hypogonadotropic hypogonadism.[6] This can result in azoospermia and reduced fertility, which can persist for months to years.[6] Physical exam findings suggestive of hypogonadism include male-pattern baldness, acne, and gynecomastia, which our patient did not manifest, despite laboratory evidence of hypogonadism.[6]

Anabolic steroid use can also increase hemoglobin, leukocytes, and platelets. Anabolic steroids increase renal synthesis of erythropoietin with potential erythrocytosis.[6] Additionally, steroids can be thrombogenic and result in thrombocytosis.[6] There are also neuropsychiatric effects, such as irritability and aggressiveness, that can increase suicide rates in anabolic steroid users.[6] Experts suggest monitoring lipid panel, LFTs, hemoglobin, and hematocrit yearly for patients who are forthcoming about anabolic steroid use and continue to use despite counseling about cessation.[7]

Labs will usually trend back to baseline within 12 weeks of anabolic steroid cessation, although some patients have residually elevated ALT and decreased testosterone synthesis.[8] Our patient's liver enzymes, sex hormones, and lipids gradually returned to normal levels with cessation of anabolic steroid use. While physicians usually diligently inquire about medication use, it is important to routinely inquire about supplement use, as these can have significant physiologic consequences.

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