CLINICAL VIGNETTE

Androgenic-Anabolic Steroid Drug-Induced Liver Injury

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Case

A 39-year-old male body builder with a past medical history of opioid use disorder, post-traumatic stress disorder (PTSD), intravenous drug use, and prolonged anabolic steroid use presented with new onset painless jaundice and pruritus of his lower extremities for six days. He also noticed stool discoloration, diarrhea and weight loss. Patient denied current intravenous drug use. Patient reported a body building regimen of intramuscular stanozolol, and oral sustanon, testosterone-cypionate, trenbolone, mass-sterone, provirone, cabergoline, and tamoxifen. Patient had taken testosterone supplementation very frequently for five months with innumerable intramuscular and oral doses.

In the Emergency Department, the patient was afebrile with a pulse of 83/min, blood pressure 145/89 mm Hg, respiratory rate of 16/min and oxygen saturation 100% on room air. He appeared well, in no distress with unremarkable cardiac, pulmonary, and abdominal exams. His exam was notable for jaundice and scleral icterus. Labs were significant for elevated alanine aminotransferase 164 U/L, and aspartate aminotransferase 125 U/L with total bilirubin of 11.37 mg/dL and direct bilirubin of 6.68 mg/dL. His creatinine was 1.67. Acetaminophen level was <10 μg/mL and hepatic viral panel was negative. His testosterone level ion was 1408.44 ng/dL. Ultrasound revealed an enlarged liver measuring 19cm. Poison control was contacted, and patient was started on N-Acetyl Cysteine (NAC) and admitted to the hospital.

The patient was hospitalized for four days with improvement in his liver tests and imaging. He received five oral doses of NAC. His bilirubin decreased and his prothrombin time improved to 12.6. Repeat liver ultrasound improved after complete cessation of androgenic-anabolic steroids (AAS), and liver biopsy was deferred. At follow up two weeks later he remained off AAS and confirmed improvement in symptoms.

Discussion

AAS are frequently abused by bodybuilders and recreational weightlifters for improved athletic performance.[1] Although AAS is rarely associated with jaundice, the proliferation of illicit use and supratherapeutic doses has increased steroid induced liver injury and jaundice. Abuse of AAS has metabolic and physiologic consequences. AAS bind to the intracytoplasmic androgen receptor leading to the production of proteins within muscle cells and inhibit the catabolism of muscle. In addition to their anabolic effects, they have virilizing properties. AAS use can lead to endocrine disorders, myocardial dysfunction, cerebral vascular accidents, and hepatocellular injury.[2]

Our patient primarily used stanozolol, a 17α-AAS, which can cause proliferation of hepatocytes and direct hepatotoxicity.[3] AAS can also affect drug metabolism and cause cholestasis of the liver.[4,5] The pattern of liver dysfunction appears to be a mixed cholestatic and direct hepatic derangement, based on published reports.[4,5] Cessation of AAS, supportive care and empiric usage of ursodeoxycholic acid can improve drug induced cholestasis.[6]

This case of AAS induced hepatotoxicity was noted after five months of AAS use and improved with NAC administration and cessation of the offending agent. The improvement was primarily due to cessation of all AAS and NAC administration. Ursodeoxycholic acid was not given. The patient did have slightly elevated prothrombin time that improved after intervention. AAS-induced hepatocellular damage can be difficult to diagnose given generally covert use. Fortunately, our patient was forthcoming about supplements he was taking. Continued cessation was advised to prevent progressive hepatocellular toxicity.

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