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Anabolic steroids: The biomarker and treatment for Crohns disease

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Abstract

The finding that the reduction within the Estrogen Receptor ER-beta/ ER-alpha ratio may be a pathologic biomarker for flairs in Crohn???s Disease has been scientifically linked, retrospectively, to 1) reduced bioavailable testosterone, 2) hypothalamic-pituitarygonadal axis dysregulation and 3) environmental toxins as probable causation. Estrogen turns off the ER-beta and must be avoided. Bioavailable testosterone is recognized because the biomarker, the Free Androgen Index (FAI). Decreased bioavailability is calculated because the ratio of decreasing Total Testosterone levels and increasing sex-hormone-binding globulin (SHBG). The FDA medication that increases serum total testosterone without increasing estrogen is nandrolone. The FDA medication that decreases SHBG is stanozolol. Using weekly intramuscular injections, the FAI is used because the drug-related biomarker. Increases in FAI parallel the recovery and potential remission seen with 5 of seven Crohn???s patients followed for up to five years. Each had exhausted all medication and surgical options; 2 had all the complications related to their Short Bowel Syndrome. The FAI is the scientific serum drug-related biomarker that increases with treatment directed improvement in disease. These two available anabolic steroids offer a paradigm shift beyond biologics and surgical resection; these patients may now realize the compassionate relief from the devastation of inflammatory bowel disease.

Case presentation: Our patient was a 31-year-old man who presented to a regional hospital with the chief complaint of substernal hurting. His past case history was significant for Crohn's disease treated with infliximab every six weeks. He was in his normal state of health until he suddenly developed substernal pain related to nausea and vomiting after he finished his routine workout within the morning. He reported similar episodes of hurting together with shortness of breath on exertion over the past two years. He was cited a cardiologist who opted to perform an exercise treadmill check with unremarkable results. He denied any case history of cardiovascular diseases. He regularly followed a gastroenterologist for his Crohn's disease that was complicated by anal fistula. Unfortunately, he had been experiencing daily chronic diarrhea because of Crohn's disease. He was also scheduled for an abdominal ultrasound for his chronically elevated liver enzymes within the upcoming months. Otherwise, he denied alcoholic abuse, smoking history, or illicit medication use. He worked as a guard and was also an important weight lifter. He used chronic anabolic steroids in three-to-four months cycles for roughly the past ten years.

Electrocardiogram (EKG) revealed anterior wall ST-segment elevations together with an elevated cardiac troponin level . The patient was transferred to our hospital status post administering of aspirin 324 milligrams (mg) via helicopter transportation service so as to undergo emergent coronary angiography that exposed a standard left main arteria coronaria, but a thrombotic occlusion was found within the left anterior descending artery (LAD). There was thrombolysis in infarction (TIMI) grade flow of 1-2 from collateral arteries of the distal right arteria to the septal cascade. The patient underwent balloon angioplasty related to intracoronary thrombolysis of tissue proteolytic enzyme (tPA) 10 mg over 10 minutes. Insertion of a 4.5 x 3.8 millimeters of the bare-metal stent was deployed at high air pressure. the following angiography demonstrated TIMI II/III flow to the anterior lateral wall with respective correlation found on the left ventriculogram that showed severe anterior lateral, anterior apical, inferior apical hypokinesis with an approximate ejection fraction (EF) of 30%. He remained to be hemodynamically stable throughout the procedure without significant arrhythmias apart from a transient accelerated idioventricular rhythm (AIVR) episode.

The patient was subsequently transferred to the cardiac care unit (CCU) status post the intervention for further management. He remained to be stable overnight. On day two of hospitalization, he was hemodynamically stable with normal sinus rhythm while on telemetry monitoring. Laboratory results were unremarkable apart from elevated troponin levels that peaked at 440 NG/ML, reactive leukocytosis of 18.0 K/UL without a clinical impression of infection together with elevated aspartate aminotransferase (AST) of 521 U/L and alanine aminotransferase (ALT) of 186 U/L with a standard level of alkaline phosphatase. He had a standard hemoglobin A1c of 5.4% and elevated lipoprotein cholesterol (LDL) of 158 MG/DL despite a traditional cholesterol level of 198 MG/DL. Echocardiogram showed a moderate to severely reduced left ventricular systolic function with an estimated EF of 25%-30%. He had no signs of acute congestive cardiopathy or arrhythmias. He remained to be stable overnight. On day three of hospitalization, he continued to stay hemodynamically stable without signs of arrhythmia or acute congestive heart disease. He was asymptomatic with negative reviews of the system. He was fitted for a wearable cardiac defibrillator to potentially reduce the danger of sudden cardiopulmonary arrest. He was subsequently downgraded to the telemetry floor from the CCU. He was discharged from the hospital and recommended against taking further anabolic

steroids because it was believed to be the reason for his MI without obvious cardiovascular risk factors. He was advised to follow up along with his primary cardiologist within the outpatient setting for the continuation of care. He eventually underwent an automatic implantable cardioverter-defibrillator (AICD) implantation three months later.

Conclusions: It is important to boost awareness of the potential side effects of chronic AAS because it may cause the event of MI. an in depth social history within the young patient population is also a game-changer when investigating the underlying causes of MI.

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