Reversible Central Hypogonadism in a Young Male Alcohol Binge Drinker

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Case Report

A 29-year-old man presented to primary care clinic with complaints of erectile dysfunction. He reported impotence since 2005 and the absence of nocturnal erections. He denied depressive symptoms, penile discharge, skin lesions or rashes in the groin area, polyuria, or dysuria. He also denied changes in testicular size, body hair or voice. He reported normal sexual function until two years ago. Family history was unremarkable. Social history was positive for alcohol binge drinking over the weekends consisting of 10-20 drinks including vodka. He also smoked 10 cigarettes per day for the past 9 years. He smoked marijuana in the past, but denied current use. He also denied taking any anabolic steroids.

On exam his blood pressure was normal at 123/76, weight was 190 pounds, and height was 69 inches. Physical exam demonstrated a well-developed, well-nourished muscular Hispanic man with normal hair pattern and no gynecomastia. Examination of his lungs, heart, and abdomen was normal. Genital exam showed an uncircumcised male with normal-sized testes without tenderness, erythema, or swelling. Laboratory studies included normal urine analysis, chemistries, glucose, and liver enzymes, albumin and INR. Total cholesterol was 72 mg/dl, triglycerides 249 mg/dl, high-density lipoprotein (HDL) 12.9 mg/dl, low-density lipoprotein (LDL) calculated at 9 mg/dm. Total testosterone level was low at 190 ng/dL, with free testosterone at 4.8 pg/ml, normal prolactin level of 6.88 ng/mL, and normal FSH and LH levels of 20.6 mIU/mL and 3.17 mIU/mL respectively. Cholesterol panel showed total cholesterol of 78 mg/dl, triglycerides of 99 mg/dl, HDL 22 mg/dl, LDL calculated at 36 mg/dl. MRI demonstrated no acute intracranial process or any pituitary lesions, with normal cavernous sinuses.

Discussion

The combined effect of alcohol and tobacco on serum testosterone is complex and has not been studied enough to date. Eriksson in 1994 showed that alcohol intake leads to acute increase in blood testosterone in postmenopausal women, and Sarcola in 2003 showed an acute increase in plasma testosterone after intake of alcohol corresponding to 2-3 drinks in healthy non-alcoholic men. It has also been shown by Ylikahri in 1974 and Valimaki in 1984 that alcohol induced an acute testosterone decrease in men during or after short term heavy drinking and Adler in 1992 stated that alcoholic men often present with symptoms of decreased sexual function like impotence and infertility.

Regarding tobacco, in 2003 Svartrberg showed that smoking was positively and significantly associated with total and free testosterone and sex hormone binding globulin (SHBG) after adjustments for age, BMI and other covariates. The one controlled study done by Walter in 2007 on the combined effect of alcohol and tobacco smoking on testosterone in alcohol dependent men showed that higher levels of alcohol and tobacco consumption was associated with higher levels of testosterone before and after alcohol withdrawal.

With regard to cardiovascular effects, alcohol at low doses tends to confer protective effects, while at high doses negative effects emerge. This is the so-called J-shaped curve that most studies have shown with regards to alcohol consumption and outcomes such as coronary artery disease, hypertension, stroke, and all-cause mortality. Studies of light to moderate drinking have shown coronary heart disease risk...
reductions of 30% to 35%. A recent metanalysis showed a reduction of 18% for all cause mortality in males who drank 1-2 alcoholic drinks per day. The main benefit of alcohol with regards to cardiovascular disease appears to be associated with the increase in high-density lipoprotein (HDL) cholesterol and insulin sensitivity. However binge drinkers have higher rates of myocardial infarctions and all-cause mortality. One reason appears to be how long the effects of alcohol last. The changes in HDL cholesterol, insulin sensitivity, and inflammation only last about 24 hours. Therefore, daily consumption provides the most protection6.

This young man with a history of smoking and binge drinking had secondary hypogonadism supported by a low testosterone level and inappropriately normal LH and FSH levels. He also had markedly low total cholesterol, HDL and LDL. At follow up one year later, six months after he stopped drinking and smoking, his testosterone level had normalized, his HDL level had increased, and his triglyceride level had decreased. The only change during that time was his abstinence from alcohol binge drinking and smoking and the positive effect on libido and erectile function. This case supports the effects of binge drinking on testosterone levels. The effect on total cholesterol and its components is unclear since the patient had very low levels at the start and they did not completely correct after stopping. We feel that further work to clarify the relationship between the frequency of alcohol consumption and cholesterol levels in men with hypogonadism is needed.

REFERENCES


