Cholestasis Induced by Parabolan Successfully Treated with the Molecular Adsorbent Recirculating System

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We describe a case of a 21-year-old male bodybuilder who overdosed on Parabolan (trenbolone acetate) because of its anabolic activity. The patient, with no previous medical history, experienced pruritus and yellow discoloration of the skin and sclerae. Basic biochemical laboratory examination revealed signs of cholestasis with a serum bilirubin level of up to 65.5 mg/dl. Because supportive medical treatment was ineffective, the patient was treated with the molecular adsorbent recirculating system (MARS). Five MARS cycles lasting from 8 to 12 hours were performed every second day. The procedure was well tolerated by the patient and resulted in a sustained relief of pruritus. At the 2-month follow-up visit the plasma bilirubin level had decreased to 2 mg/dl. *ASAIO Journal* 2006; 52:117–118.

Case Report

In July 2005, a 21-year-old male bodybuilder with no medical history was admitted to the hospital with jaundice and invalidating pruritus. The serum bilirubin level was 19.43 mg/dl, the aspartate aminotransferase (AST) level was 73 IU/l, the alanine aminotransferase (ALT) level was 127 IU/l, and prothrombin time was 94.07%. About 7 weeks before admission, he self-administered one tablet of Parabolan (25 mg) daily in two cycles lasting for 3 weeks with a 7-day pause. According to the anamnesis, his friends had taken the same drug in a daily dosage of 1 to 3 tablets in a 6-week cycle. Despite symptomatic treatment with hydrocortisone acetate, phenobarbital, ursodeoxycholic acid, and phytomenadione, the bilirubin level increased to 60.6 mg/dl. The patient was finally transferred to the Medical University of Gdańsk.

At the time of admission to the clinic, the patient was conscious. Blood pressure was 90/60 mm Hg, pulse was 82 beats/min, respiratory rate was 12 breaths/min, and temperature was 36.5°C. The main symptom was intensive pruritus. The bilirubin level was 65.5 mg/dl, the AST level was 174 IU/l, the ALT level 106 IU/L, and prothrombin time was 121%. Extracorporeal albumin dialysis using the molecular adsorbent recirculating system (MARS) was started. Five sessions of MARS, which lasted from 8 to 12 hours each, were conducted every second day. The procedure was well tolerated by the patient and resulted in a sustained relief of pruritus and a decline in plasma bilirubin to 24.1 mg/dl. The patient was discharged home in good general condition. A the 2-month follow-up visit, the patient felt very well and the bilirubin level was reduced to 2 mg/dl.

Discussion

Anabolism is defined as a state in which nitrogen is differentially retained in lean body mass, either through stimulation of protein synthesis or decreased breakdown of protein anywhere in the body.[1] Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone modified to enhance anabolic rather than androgenic actions of the hormone.[1,3] The anabolic activity of AAS is primarily manifested in its myotrophic action, which results in greater muscle mass and strength; however, many scientists conclude that use of AAS for the purpose of anabolic properties is at least controversial.[1,4]

Because testosterone is rapidly metabolized by the liver, it was necessary to administer slowly absorbed testosterone esters by injection or to apply its derivatives by mouth. The latter are slowly metabolized by the liver and have deleterious side effects, as seen in our case.[2]

According to many researchers, high and multiple doses of AAS can lead to serious and irreversible organ damage. Among the most common adverse effects of AAS are reduced fertility, gynecomastia in males and masculinization in women and children, hypertension, atherosclerosis, blood clotting, tendon damage, psychiatric and behavioral disorders, and various alterations of liver function.[1,4–6] AAS may cause subcellular alterations, elevation of liver enzymes, peliosis hepatitis, Budd-Chari syndrome, hyperplasia, hepatoadenoma, hepatocarcinoma, and intrahepatic cholestasis, which usually occur after 2 to 5 months of drug intake and are probably induced by impairment of hepatocellular bile secretion.[5–10] Toxic hepatitis inducted

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by AAS is sometimes associated with coexistent renal dysfunction; however, we did not observe renal failure in our patient.[11–13]

The supportive treatment of cholestasis caused by AAS is confined to prompt AAS withdrawal, ursodeoxycholic acid administration, and correction of fat-soluble vitamin deficiency.[7,10–12] However, in some cases, MARS provides very good relief of pruritus.[14] For patients in whom supportive treatment is ineffective, MARS could be the best solution.

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