Insulin-Like Growth Factor-I Reverts Testicular Atrophy in Rats With Advanced Cirrhosis

Inma Castilla-Cortazar, María García, Jorge Quiroga, Nieves Diez, Fernando Diez-Caballero, Alfonso Calvo, Matías Díaz, and Jesus Prieto

Abstract of:
Hepatology 2000;31:592-600

The pathogenesis of hypogonadism in cirrhosis is not completely understood. The levels of insulin-like growth factor-I (IGF-I), an anabolic factor with trophic actions on testes, are reduced in cirrhosis. This study was undertaken to evaluate whether rats with advanced cirrhosis develop hypogonadism and whether the administration of IGF-I exerts beneficial effects on testicular structure and function. Wistar rats with ascitic cirrhosis induced with CC14 were allocated into 2 groups (n = 10, each) to receive recombinant IGF-I (20 ug · kg-1 · d-1, subcutaneously) or vehicle for 3 weeks. Healthy rats receiving vehicle were used as the control group (n = 10). At baseline, both cirrhotic groups showed similar deterioration of liver function tests. Compared with controls, nontreated cirrhotic rats showed decreased serum levels of IGF-I (P < .05), reduced testicular size and weight (P < .001), and intense histopathological testicular abnormalities, including reduced tubular diameters (P < .001), loss of the germinal line (P < .001), and diminutions in cellular proliferation, spermatogenesis (P < .001), and testicular transferrin expression (P < .001). In addition, low serum testosterone (P < .01) and high serum LH (P < .01) were present in untreated cirrhotic animals. Cirrhotic rats that received IGF-I showed full recovery of testicular size and weight and of all histopathological abnormalities (P < .001 to < .01 vs. nontreated cirrhotic rats; P = ns vs. controls). Serum levels of sex hormones tended to normalize. In conclusion, IGF-I deficiency may play a pathogenetic role in hypogonadism of cirrhosis. Low doses of IGF-I for a short period of time reverse testicular atrophy and appear to improve hypogonadism in advanced experimental cirrhosis.

Skin manifestations of a case of phenylbutazone-induced serum sickness-like reactions

G. Sánchez, L. Vila, M. Pajarón and L. Diéguez

Department of Allergology and Clinical Immunology, University Clinic, Faculty of Medicine, University of Navarra, Pamplona, Spain

Abstract of:

Serum sickness consists of a systemic reaction resulting from the formation of soluble circulating immunocomplexes after the introduction of a foreign substance into the body. We studied a 38-year-old woman diagnosed with anxiety depression and right sacroileitis who was treated with phenylbutazone, ranitidine, clomipramine and levomepromazine. After taking this treatment for 1 month, she presented with fever, diarrhea, localized edemas, generalized pruritic papular and erythematous rash and lymphadenopathies. She presented the same symptoms after oral intake of metamizole. The diagnosis was confirmed following a single-blind, placebo-controlled provocation test with phenylbutazone and a biopsy of the affected skin.