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Uso Crônico de Decanoato de Nandrolona Como Fator de Risco Para Hipertensão Arterial Pulmonar em Ratos Wistar



Chronic Use of Nandrolone Decanoate as Risk Factor for Pulmonary Arterial Hypertension in Wistar Rats

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RESUMO

Introdução: O uso indiscriminado de esterôides anabolizantes sintéticos, análogos à testosterona, im plica aumento do risco cardiovascular e hipertrofía cardíaca. Assim, o aumento da massa ventricular direita corrigido pelo peso corporal (i.é., hipertrofía ventricular direita - MVD), poderia elevar o risco para o desen volvimento de hipertensão arterial pulmonar (HAP). Objetivos: Examinar os efeitos do tratame prazo com decanoato de nadrolona na HVD e sua relação com a HAP em ratos. Métodos: 16 ratos Wistar com três meses de idade foram aleatoriamente divididos em dois grupos: 1) controle sham (CONT, n = 8); 2) tratados com decanoato de nandrolona (DECA, n ~ 8). O tratamento consistiu na aplicação intramuscular de Deco-durabolin" 6.0mg.kg.º de peso corporal durante quatro semanas. Após tratamento, os animais foram anestesiados com hidrato de cloral (4.0ml.kg⁻¹, (p.), submetidos à cateterização da artéria femoral para registro da pressão arterial media (PAM) e freguência cardíaca (PC). O coração, os rins e o figado foram retirados, pesados e avaliados os indices de hipertrofía, os quais foram calculados pela razlio da massa do órgão pelo peso corporal (mg.gr¹). Resultados: Os animais tratados com DECA apresentaram aumento (p < 0.011 do peso comoral (338 + 6g) vs. CONT (315 + 5g). Não houve alterações da PAM, embora houvese (n < 0.01) bradicardia nos animais tratados com DECA (321 ± 13bpm) vs. CONT (368 ± 11bpm). Verificou-se significativa (p < 0.01) hipertrofia dos ventrículos e rins, más não no figado. A correlação entre a HVD e PAM no grupo DECA apresentou coeficiente de Pearson positivo e maior (r² = 0,4013) quando comparado com o controle (r² = 0.0003). Conclusões: Esses dados demonstram que o uso em longo prazo de decanoato de nandroloria induz importante bradicardia e HVD, o que sugere aumento do risco para HAP.

Palavras-chave: esteróide anabolizante, pressão sanguinea, hipertrofía ventricular, hipertensão arterial pulmonat.

ABSTRACT

Introduction: The unsystematic use of anabolic steroids, synthetic analogs of testosterone, implies enha ced cardiovascular risk and cardiac hypertrophy. Thus, increased right ventricular mass corrected by the body weight learright ventricular hypertrophy -RVHI could raise the risk for development of pulmonary arterial hypertension (PAH). Objectives to examine the effects of long-term chronic treatment with nandrolone decanoate on the RVH and its relationship with PAH in rats. Methods: 16 three-month Watar male rats were treated with nandrolone decanoate (6.0 mg/kg 1 body weight; DECA, n=8) or control whicle (CONT, n=8). The drug and vehicle were administered by a single injection in the femoral muscle once a week for 4 weeks. After the treatment, rats were anesthetized with chloral hydrate (4.0mi, Agr¹, ip), and catheterized in the femoral artery. Twenty-four hours later, mean arterial pressure (MAP) and heart ratio were measured. The heart, kidneys and liver were removed, weighed and the rates of hypertrophy (RH) were measured, which were calculated by the ratio of the weight of the organs by the body weight (mg.g-1). Results: DECA nt increased body weight (338 ± 6g; p <0.01) vs. CONT (315 ± 5g). This treatment had no effect on the MAP (CONT, 110±4mmiHg, DECA, 113 ± 4mmHg). However, the bradycardia of animals treated with DECA (321 ± 13bpm, p<0.01) was significantly lower than that of CONT (368 ±11bpm). RH increased (p<0.01) the cardiac ventricles and the kidneys, but not in the liver. The correlation between the RVH and MAP in DECA showed positive and higher Pearson's coefficient (r² = 0.4013) vs CONT (r² = 0.0003). Conclusions: it was concluded that chronic nandrolone decanoate treatment induced bradycardia and RVH, which suggests increased risk for PAH.

Keywords: anabolic steroids, blood pressure, ventricular hypertrophy, pulmonary arterial hypertension.

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