Short Report

Elevation of Serum Creatine Phosphokinase during Pindolol Treatment

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IMATAKA, K., SEKI, A., Takahashi, N. and Fujii, J. Elevation of Serum Creatine Phosphokinase during Pindolol Treatment. Tohoku J. exp. Med., 1981, 133 (3), 363-364 — We observed elevation of serum CPK in hypertensive patients under pindolol treatment. In this report, we showed a representative patient and compared serum CPK values in 56 patients under pindolol treatment to those in 64 patients under propranolol treatment. Mean serum CPK values in the patients under pindolol treatment and propranolol treatment were 117.0±12.6 (S.E.) and 50.8±3.8 IU/liter, respectively (p<0.01). Although the mechanism of elevation of serum CPK cannot be well understood, we should be careful in clinical evaluation of serum CPK during pindolol treatment.

Some of adverse effects of beta-blockers are not predictable from their pharmacological actions (Forfar et al. 1979). We experienced abnormally high values of serum creatine phosphokinase (CPK) in a hypertensive patient during pindolol treatment. The present report describes this patient with a review of serum CPK values in other hypertensive patients under pindolol or propranolol treatment. We are unaware of any previous report on the elevation of serum CPK by pindolol.

Case report

A 61-year-old Japanese man was treated for moderate hypertension with 15-30 mg of pindolol, 150 mg of hydralazine and 4 mg of trichlormethiazide daily (Fig. 1). After 6 months of treatment serum CPK was found to be elevated from the pre-treatment value of 86 to 390 IU/liter (normal range<118 IU/liter). The serum CPK remained raised during the subsequent 5 months of treatment. Temporary switching from pindolol to propranolol was followed by a fall of serum CPK to normal. Challenge tests were attempted with 15 mg of pindolol, and then the CPK value increased to 271 IU/liter in 13 days. Hydralazine and trichlormethiazide were continued throughout the treatment. These changes in serum CPK were not accompanied by any significant changes in serum glutamic oxaloacetic transaminase (GOT) or glutamic pyruvic transaminase (GPT). Serum CPK isoenzymes were measured when CPK value increased, but CPK-MB activity was only 1% of total CPK.

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Serum CPK in hypertensive patients under pindolol or propranolol treatment

To clarify the effect of pindolol on serum CPK, clinical records of 120 patients with uncomplicated hypertension who had been treated with pindolol or propranolol were reviewed. The values of serum CPK measured during treatment were 117.0±12.6 (s.e.) IU/liter in 56 patients under pindolol (2.5-30 mg/day) and 50.8±3.8 IU/liter in 64 patients under propranolol treatment (20-120 mg/day). The difference of serum CPK was significant between the two groups of patients (p<0.01). In 43 of these patients serum CPK was measured both before and during treatment. As compared with pre-treatment values serum CPK was elevated by more than 40 IU/liter in 10 of 29 patients (34%) during pindolol, but in none of 14 patients during propranolol treatment. Serum GOT, GPT and LDH remained essentially unchanged during treatment.

The present report partly supports an observation by Ueda et al. (1975) that alprenolol treatment induced a slight elevation of serum CPK in five patients. Elevation of serum CPK has usually been considered to be associated with necrosis of heart or skeletal muscles, but the present results did not disclose any evidence of muscle necrosis. Griffiths (1966) and Bloor et al. (1978) observed elevation of serum CPK during exercise in man or mini-swines and suggested that altered cellular membrane permeability was responsible for the elevation of serum CPK. The same mechanism may be involved in the elevation of serum CPK during pindolol treatment. The fact that serum CPK was increased by pindolol or alprenolol but not by propranolol treatment indicates that the mechanism of CPK elevation may be related to the intrinsic sympathomimetic action of beta-blockers. In the light of the present study, we should be careful in clinical evaluation of serum CPK during pindolol treatment.

References