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Cardiovascular Parameter Changes in Patients With Erectile Dysfunction Using Pde-5 Inhibitors: A Study With Sildenafil and Vardenafil

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Sildenafil is the most prescribed oral agent for patients with erectile dysfunction (ED). Vardenafil is a new phosphodiesterase type 5 (Pde-5) inhibitor that was approved by the US Food and Drug Administration last year to treat patients with ED of various causes. Both of these Pde-5 inhibitors have vasodilating properties and effects on blood pressure (BP), and like nitrates, they work through the nitric oxide cyclic guanosine monophosphate pathway. The aim of this study was to investigate the influence of these Pde-5 inhibitors on BP and heart rate (HR) in normotensive men with ED by a crossover comparison. Thirty-five patients with ED were enrolled to evaluate and compare the effect of sildenafil (50 mg) and vardenafil (10 mg) on BP and HR. At the screening (baseline [B]) visit, sitting systolic blood pressure (B-SBP), diastolic blood pressure (B-DBP), and HR were measured. We performed a multiple administration for both drugs and, therefore, multiple measurements of BP and HR changes, 3 doses a week, on alternate days, late in the afternoon, and on an empty stomach. B-SBP, B-DBP, and HR were recorded before each 50-mg sildenafil dosing and after 30, 60, 120, and 240 minutes. Data were averaged over the 4 time points and compared with the baseline values obtained before each dosing. After a 3-week wash-out period, patients were crossed over to vardenafil (10 mg) with the same study design. After administration of both drugs, we observed a statistically significant decrease of BP and an increase of HR. On average, sildenafil caused a decrease of SBP ranging from 5.1 ± 3.9 mm Hg during the first dosing to 4.7 ± 4.2 mm Hg during the third dosing, DBP ranged from 4.4 ± 4.9 to 4 ± 4.1 mm Hg, and HR increased 1.8 ± 2.0 bpm (first dose) and 1.2 ± 0.9 bpm (third dose). With vardenafil, we recorded a greater variation for SBP and DBP. SBP decreased from 8.02 ± 8.0 mm Hg during the first dosing to 5.4 ± 5.5 mm Hg during the third dosing, whereas DBP decreased from 6.6 ± 7.2 to 5.0 ± 5.3 mm Hg, respectively. Recorded HR showed an increase of 3.1 ± 3.2 bpm (first dose) and 2.4 ± 2.3 bpm (third dose). After the first vardenafil administration, we recorded fainting episodes in 3 patients because of a decrease in BP greater than 20 mm Hg. Two of the patients were in therapy with doxazosin for benign prostatic hyperplasia (BPH).

Cardiovascular response was not significantly different after the first dose between the 2 treatments. Vardenafil demonstrated clinically significant differences (fainting) with respect to sildenafil only during the first doses. We suggest that before starting therapies with Pde-5 inhibitors, particularly with the newer ones, that baseline cardiovascular parameters are measured and monitored, especially during the first dose, because of the presence of a "first dose effect." Moreover, it is necessary to pay particular attention to those patients in treatment with other drugs that could have a synergistic hypotensive effect as a result of vasodilation potentiation.

Key words: hypotensive effect, side effect, crossover study

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