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Bradydcardia Associated with Ophthalmic Beta-Blockers

Sir,
The systemic adverse effects of ophthalmic beta-blockers including bradydcardia may be overlooked. Beta-blockade can be highly hazardous to aged patients suffering from cardio-pulmonary diseases. This communication describes two patients with bradydcardia associated with ophthalmic beta-blockers therapy.

Case 1:
A 73-year-old man with essential hypertension and chronic open-angle glaucoma, who was receiving atenolol (50 mg/day) and carteolol 1% (one drop in both eyes twice daily) for two years developed syncope. The patient took no other medications. An electrocardiogram at admission to the emergency service revealed sinus bradydcardia (20 beats/min). Atropine (0.5 mg intravenously) was administered and the heart rate increased to 70 beats per minute. Atenolol and carteolol were discontinued and the heart rate ranged from 50 beats per minute to 80 beats per minute during the following 72 hours. An ambulant 24-hours electrocardiogram Holter demonstrated a heart rate ranging from 50 beats per minute to 95 beats per minute without pauses. He is asymptomatic after six months of follow-up. Enalapril was prescribed for his hypertension and ophthalmic latanoprost for his glaucoma.

Case 2:
A 53-year-old man with history of chronic open-angle glaucoma, who was receiving timolol 0.25% (one drop in both eyes twice daily) for four months. He took no other medications. In a check-up, an electrocardiogram showed a sinus bradydcardia (50 beats per minute). He denied history of syncope, diziness or other clinical manifestations associated with bradydcardia. Timolol was continued and an ambulant 24-hours electrocardiogram Holter demonstrated a heart rate ranging from 45 beats per minute to 85 beats per minute without pauses. He is asymptomatic after 16 months of follow-up. Enalapril was prescribed for his hypertension and ophthalmic latanoprost for his glaucoma.

Ophthalmic beta-blockers may resemble intravenous beta-blockers in terms of systemic bioavailability, plasma kinetics, and cardiopulmonary effects. These two cases illustrate the variable severity of the bradydcardia associated with ophthalmic beta-blocker therapy. Use of the Naranjo ADR Probability Scale indicated a possible relationship in case 1 and a probable relationship in case 2 between bradydcardia and ophthalmic beta-blockers therapy. No evidence of sinus-node dysfunction was found in an ambulant 24-hour-Holter electrocardiogram in either of them.

In one study, 14 patients treated with ophthalmic timolol only showed a slight decrease in the heart rate. Dickstein et al. reported similar results in a recent clinical trial with a 7.5% decrease (amounting approximately to 6 beats per minute) in the mean heart rate in the daytime in patients treated with ophthalmic timolol. Timolol seems to cause bradydcardia more frequently than carteolol. The intrinsic sympathomimetic activity of carteolol probably compensates for its beta-blocking activity, thereby decreasing the probability of occurrence of bradydcardia with its use.

Case 1 demonstrates that symptomatic bradydcardia may occur many months after beginning ophthalmic beta-blockers therapy. It seems evident that the concomitant use of oral beta-blockers may increase the risk and the severity of the bradydcardia associated with ophthalmic beta-blockers, as seen in case 1. When ophthalmic beta-blockers therapy is hampered by adverse effects, it is advisable to change to another agent such as ophthalmic latanoprost or carbonic anhydrase inhibitors for the control of glaucoma.

References