Introduction
Glaucoma is the first cause of irreversible blindness. Early diagnosis and treatment are crucial to stop the disease.

Methods
139 eyes from 72 patients affected with glaucoma under BB and PGA treatment were included in a prospective study. Single nucleotide polymorphism (SNP), were analyzed using real-time PCR assays: prostaglandin F2α receptor (PTGRF) rs3766355 and 765380; cytochrome-p450 2D6 (CYPD2) rs416947 and 760298; beta 2 adrenergic receptor (ADRB2) rs1042714. The other variables of the study were mean deviation (MD) of visual field (VF), glaucoma surgery background, medication side effects, medical treatment, baseline and treated intraocular pressure (IOP).

Results
From a total of 139 eyes, 68 (48.9%) were right eyes. The main diagnosis was primary open angle glaucoma (56.2%). Additionally, 57(41.4%) eyes had some kind of glaucoma surgery and also 57(41.4%) eyes were under 3 or more medications (PGA+BB+others). Side effects were reported in 12 eyes (8.9%). IOP for baseline and treated patients were 26.55 ± 7.59 and 20.98 ± 8.63. Comparing the mean of MD in rs3766355 wildtype HZ and mutated homozygous (HZ) was primary open angle glaucoma (66.2%). Additionally, 57(41%) eyes had some genetic variants in relation with the disease. Recently some genetic variants have been described in relation with the disease. Significant differences were found in the treated IOP in HZ and HT patients carrying rs3766355 allele with regard to HT wildtype patients. Therefore, poor response to treatment may be associated with being a carrier of this mutated allele.

Conclusions
Significant differences were found when comparing the MD in wildtype HZ, HT and mutated KC carrying rs3766355 and rs7318189 variants; greater number of mutated alleles correlated with a better outcome in the visual field (VF). Likewise significant differences were found in the treated IOP in HZ and HT patients carrying a mutated rs3766355 allele with regard to HT wildtype patients. Therefore, poor response to treatment may be associated with being a carrier of this mutated allele.

References