

Comparing the effects of propranolol and ergotamine-c in prophylaxis treatment of migraine in Iranian patients

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Migraine is a brain's vascular disease. During the pre headache phase of migraine there is a decrease in cerebral blood as vasoconstriction of serotonin may have occurred.¹⁻⁴ During the following headache phase, the cerebral blood flow is increased because of the vasodilation in this step. The platelet aggregation and plasma concentration of serotonin maybe decreased. There is a sterile inflammation after vasodilation of vessels that sensitizes the pain receptors in the vessel walls of the brain. In this step we must try to inhibit vasodilation and platelet aggregation. One of the most effective therapeutic agents for acute attacks is ergotamine (with or without caffeine).^{5,6} During the last few years several controlled trials have shown or indicated a beneficial effect with propranolol. Usage of propranolol plus ergotamine in cardiac patients reduces the dosage of ergotamine and side effects of this drug, such as myocardial infarction. The majority of patients reported nausea without vomiting (85%). Fifty percent of women reported that the intensity of headaches amplified in time. In 55-90% of patients, ergotamine acts as an analgesic, and propranolol has a beneficial effect in 60-80% of patients with migraine as a prophylactic agent. The present results suggest that the time of the maximum plasma drug level is an important determinant of the clinical effects of ergotamine, and a good therapeutic response may be expected if a plasma ergotamine level of 0.2ug/ml or more is achieved within one hour after oral or rectal administration. Determination of plasma propranolol concentration demonstrated that

different responses to the same oral dose do not depend on different plasma level of the drug.

Propranolol has been used in migraine prevention on the assumption that this drug blocked the peripheral vasodilator B-receptors and has a stereo specific affinity for brain serotonin receptors and an inhibition of thromboxane synthesis and platelet aggregation, while the effect of ergotamine is vasoconstriction of vessels; this therapeutic agent has an agonistic effect on peripheral vessels. Ergotamine affects the emetic center so nausea and vomiting may occur, and the dosage must therefore be limited to no more than 10 mg per week to minimize toxicity. In a patient with migraine, the recommended dosage of this therapeutic agent would be 2 mg ergotamine plus 200 mg caffeine before acute attacks and 80-240 mg propranolol 3 times per day.

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