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EHMTI-0096. Efficacy of sumatriptan: assessment of a possible biomarker

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Introduction

Sumatriptan is a frequently applied anti-migraine treatment, yet ≈20-30% migraineurs are nonresponders. TRPV1 channels on trigeminal nerve endings release CGRP during migraine attacks, while, supposedly, stimulation of the presynaptic 5-HT(1B)/1D receptor by sumatriptan inhibits this release. Capsaicin (CAP) stimulates TRPV1 channels, causing CGRP-dependent vasodilatation, whereas electrical stimulation (ES) induces vasodilation without direct TRPV1 activation.

Aim

To assess a possible biomarker for the efficacy of sumatriptan.

Methods

We investigated the effect of sumatriptan on the rise of dermal blood flow (DBF) of the forehead skin (innervated by the trigeminal nerve) by CAP application (0.6 mg/ml) and ES (0.2-1.0 mA) before and after subcutaneous placebo and sumatriptan in a randomized, double-blind, placebo controlled cross-over study, including healthy male (n = 11, age ± SD: 29 ± 8 yrs) and female (n = 11, 32 ± 7 yrs) subjects.

Results

DBF responses to CAP (mean ± SEM: 313 ± 16 A.U.) were significantly attenuated after sumatriptan (mean decrease DBF: 82 ± 18 A.U., $p < 0.001$) but not after placebo (mean decrease DBF: 21 ± 12 A.U., $p = 0.1026$), whereas DBF responses to ES were not affected by sumatriptan or placebo. Sumatriptan, but not placebo, increased blood pressure by 6 ± 2/11±2 mmHg, $p < 0.001$. In 23% of the subjects, sumatriptan did not attenuate the DBF response.

Conclusions

Sumatriptan may inhibit the release of CGRP via the stimulation of the presynaptic 5-HT(1B)/1D receptor and/or by a direct effect on TRPV1 channels. ES appears to be a nonspecific stimulus, most likely releasing other neuropeptides besides CGRP. Future studies should indicate whether nonresponse in our model correlates with clinical nonresponse to sumatriptan.

No conflict of interest.

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