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Title: Tachykinin 1 receptor antagonist FK888 suppresses angiotensin converting enzyme inhibitors (ACEIs)-induced tracheal inflammation, an experimental study

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Body: Introduction Several mechanisms have been proposed to explain angiotensin converting enzyme (ACE) inhibitor-induced cough, but the exact mechanism is not known. ACEIs has been shown to induce the production of PGE2 and NO, which have been implicated as a mediator of inflammation in experimental models. Aim of the work To study the effect of acute and chronic treatment with ACE inhibitors (perindopril and fosinopril) each alone and in combination with tachykinin 1 antagonist FK888 on tracheal Nitrate/Nitrite (N/N) and PGE2 levels Material and Methods About 60 rats of adult rats, of both sexes, weighing 120-150 gm were used in the study. Four groups in acute treatment, four groups in chronic treatment and two control groups (6 rats in each group). The control group was injected with carboxymethyle cellulose.the treated groups were injected with perindopril or fosinopril either alone or in combination with FK888,three hours later in acute inflammation groups and 21 days later in chronic treatment groups, rats were sacrificed and their tracheae were extracted, homogenized and the supernates were used for of total nitrite/nitrate and PGE2 assay. Results Both acute and chronic treatment with perindopril and fosinopril had insignificant increase in tracheal N/N and PGE2 levels ($P < 0.01$). Combination of FK888 lead to a significant decrease in tracheal N/N and PGE2 levels ($P < 0.01$). Conclusion The study proved that treatment with the studied ACEIs increased tracheal N/N and PGE2 levels, however, if these mechanisms contribute to ACEIs induced cough or not requires further studies.