Effect of Meloxicam on Oral Ca. of Xenografted in Nude Mice

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INTRODUCTION

Background
2. Cyclooxygenase (COX)-2 is overexpressed in various cancers including the head and neck cancer and catalyzes the formation of prostaglandins (PGs), such PGE2, which affect cell proliferation and inhibit the immune response against malignant cell and the overproduction of PGs could favor malignant growth.

The purpose of this study is to assess the anti-tumor effect of a selective COX-2 inhibitor, Meloxicam, on human oral cavity squamous cell carcinoma xenografted in nude mice.

The study suggested that the novel applications of selective COX-2 inhibitors for adjuvant treatment of oral cavity cancer, and the further clinical study will be needed for the determination of the efficacy of selective COX-2 inhibitors for the treatment of oral cavity cancer.

METHODS AND MATERIALS

Materials
1. Cell line: KB cell (human oral cavity squamous cell carcinoma cell line)
2. Laboratory animals: 30 athymic mice, KB cells (1.0 x 10^7) injected S.C. into Rt. flank
3. Meloxicam, a selective COX-2 inhibitor (4-hydroxy-2-methyl-N-(5-methyl-2-thiazolyl)-2H-1,2-benzothiazine-3-carboxiamide-1,1-dioxide)
4. Materials and Methods

RESULTS

In Vitro – Cell proliferation (Figure 1)
1. Hemocytometer
2. Cell proliferation is suppressed dose-dependently (p<0.05)
3. PGE2 is produced by human oral cavity SCC cell line (KB cell)
4. Increased level of PGs have been detected multiple epithelial cancers, including head and neck cancers.

In Vitro – COX-2 expression (Figure 2)
1. RT-PCR
2. COX-2 expression is suppressed in 100uM and 300uM.
3. Meloxicam, a selective COX-2 inhibitor, suppressed the growth of KB cell probably through the suppression of COX-2 dose-dependently.

In Vitro, Meloxicam suppressed the proliferation and induced the apoptosis (%)(p<0.01).

In Vivo – Tumor weight (Figure 3)
1. Tumor weight is decreased dose-dependently (p<0.05).
2. Control
3. Meloxicam, a selective COX-2 inhibitor (30mg/kg), suppressed the proliferation and induced the apoptosis (%)(p<0.01).
4. Meloxicam, a selective COX-2 inhibitor (30mg/kg), suppressed the proliferation and induced the apoptosis (%)(p<0.01).

In Vivo, Meloxicam suppressed the growth of KB cell probably through the suppression of COX-2 dose-dependently and also induced the apoptosis (%)(p<0.01).

In Vivo, Meloxicam suppressed the proliferation of tumor cells dose-dependently and also induced the apoptosis (%)(p<0.01).

In Vivo, Meloxicam suppressed the growth of tumors xenografted in nude mice significantly.

CONCLUSIONS

1. In Vivo, Meloxicam suppressed the growth of KB cell probably through the suppression of COX-2 dose-dependently.
2. In Vivo, Meloxicam suppressed the proliferation and induced the apoptosis (%)(p<0.01) and suppressed the tumor growth in xenografted tumors (%)(p<0.01).
3. The effect of Meloxicam on angiogenesis was not detected in this study.

REFERENCES