Histopathology

Histopathology 2016 DOI: 10.1111/his.12932

Clinical relevance of cyclooxygenase 2 and peroxisome proliferator-activated receptor γ in eyelid sebaceous gland carcinoma

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Date of submission 12 September 2015
Accepted for publication 14 January 2016
Published online *Article Accepted* 21 January 2016

Jayaraj P, Sen S, Bhattacharya T, Arora J, Yadav S, Chhoker V, Kumar A, Dhanaraj PS, Yadavilli KS & Verma M (2016) *Histopathology*. DOI: 10.1111/his.12932

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Aims: Sebaceous gland carcinoma (SGC) is a malignancy associated with the pilosebaceous unit, and occurs at ocular or non-ocular sites. Cyclooxygenases (COXs) are enzymes that are crucial for lipid metabolism. COX-2 is overexpressed in various cancers, and its inhibition by non-steroidal anti-inflammatory drugs is known to reduce the risk of many cancers. Peroxisome proliferator-activated receptor (PPAR)-γ is a transcription factor involved in adipogenesis. PPAR-γ is a potential therapeutic target for the treatment of malignant tumours, including colon carcinoma. The aim of this study was to explore the status of COX-2 and PPAR-γ as prognostic markers in human eyelid SGC.

Methods and results: The immunohistochemical expression of COX-2 and PPAR-γ was evaluated in 31 SGC cases. Cytoplasmic expression of COX-2 was detected in 80% of the SGC cases, and nuclear expression of PPAR-γ in 87%. There were significant correlations of PPAR-γ expression with well-differentiated SGC [19/21 (90%)] and of COX-2 overexpression with reduced disease-free survival ($P = 0.0441$, log rank analysis). COX-2 expression [odds ratio (OR) 3.82, 95% confidence interval (CI) 1.02–14.33, $P = 0.046$] and lymph node metastasis (OR 0.17, 95% CI 0.04–0.65, $P = 0.009$) emerged as significant risk factors in the univariate analysis. However, COX-2 expression did not emerge as a significant independent prognostic factor in multivariate analysis.

Conclusions: COX-2 is a potential marker for identifying high-risk SGC patients. Expression of PPAR-γ in eyelid SGC cases reflects terminal sebaceous differentiation. Inhibitors of COX-2 signalling and PPAR-γ agonists are both prospective novel therapeutic targets in the management of eyelid SGC patients.

Keywords: cyclooxygenase 2, eyelid, peroxisome proliferator-activated receptor γ, sebaceous gland carcinoma

Introduction

Sebaceous gland carcinoma (SGC) is a malignancy associated with the pilosebaceous unit, and occurs both at ocular and non-ocular sites.[1] Ocular SGC is

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