Cyclooxygenase-2 expression and recurrence of colorectal adenomas: effect of aspirin chemoprevention

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Résumé en anglais	Background Low-dose aspirin reduces the incidence of colorectal cancer and recurrence of adenomas. Cyclooxygenase-2 (COX-2), one of its main target enzymes, is reportedly over-expressed in colorectal adenomas.Aim To assess COX-2 expression, in relation to adenoma recurrence and the protective effect of aspirin, in a large series of colorectal adenomas, recruited from a double-blind randomised controlled trial comparing recurrences after low-dose aspirin or placebo. Methods Follow-up colonoscopies were performed after 1 and 4 years to assess adenoma recurrence. COX-2 expression was assessed by immunohistochemistry for each adenoma obtained at baseline colonoscopy, separately for epithelium, deep stroma and overall. Architecture, grade of dysplasia, K-ras mutation, p53 and cyclin D1 expression were studied. Results COX-2 expression could be assessed in 219 adenomas from 136 patients: 128 adenomas (58%) from 59 patients strongly expressed COX-2. Strong COX-2 expression predominated in adenomas larger than 10 mm (84/129 vs 44/90; p=0.02) and in adenomas showing high-grade dysplasia (22/29 vs 104/188; p=0.04). Deep stromal but not epithelial initial expression of COX-2 predicted adenoma recurrence in the whole population (30/72 patients or 42% strongly expressed deep stromal COX-2 compared with16/64 or 25% without recurrent adenoma; p=0.04). The protective effect of aspirin was mainly observed in patients in whom COX-2 initial expression was low (RR for recurrence in patients taking aspirin with low COX-2 expression: 0.59; 95% CI 0.39 to 0.90; p=0.02). There was no significant effect of aspirin at the end of the trial. Conclusion Over-expression of COX-2 was frequent and predominated in large and high-grade dysplasia adenomas. Deep stromal but not epithelial initial expression of COX-2 predicted recurrence of adenomas. Aspirin did not act preferentially on patients whose initial adenomas strongly expressed COX-2.
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