Introduction: Aggressive fibromatosis (desmoid tumor) is a locally invasive soft tissue lesion, composed of a mononclonal proliferation of benign-appearing spindle (fibroblast like) cells. A subset of lesions contain a somatic mutation in the adenomatous polyposis coli (APC) gene (1). Familial Adenomatosis Polyposis (FAP) is an inherited pre-neoplastic condition that predisposes to colonic neoplasia and occasional fibromatoses, that is caused by a germ-line mutation in the APC gene. A large percentage of sporadic colonic polyps and cancers also harbor somatic APC mutations.

Cyclooxygenases (COX) are enzymes involved in prostaglandin synthesis. COX-1 is ubiquitously expressed, and plays a role in normal physiology, while COX-2 is expressed in only select sites, usually by cells that are involved in an inflammatory or proliferative process. Normal fibroblasts do not express COX-2 (2). Colonic neoplasms express COX-2 and in mouse model of FAP, COX-2 blockade significantly decreases the number of colon tumours formed (3).

Current treatments for aggressive fibromatosis are less than satisfactory, with high recurrence rates, loss of function, or local complications being not uncommon outcomes (4). A pharmacological agent to retard tumor growth would be a useful in this lesion. COX-2 blockade is one such potential treatment. The purpose of this study is to determine if aggressive fibromatosis expresses COX-2, and if COX-2 blockade in-vitro will alter cellular proliferation or apoptosis rates.

Methods: Eighteen cases of sporadic aggressive fibromatoses were studied. Cryopreserved tumor tissue for RNA analysis was available from all 18, and primary explant cell cultures were available from four. For eight of the cases, cryopreserved normal "normal" tissues also were available, and in six of the cases, large enough quantities of cryopreserved normal tissues were available for Northern and Western blot. In four cases primary cell cultures were established.

RT-PCR using specific oligonucleotide primers for COX-2 and GAPDH (housekeeping control) were used to test for COX-2 expression. Northern blot, using a PCR generated digoxigenin labeled probe was performed in six cases of tumor tissue, and corresponding "normal" marginal tissues. Western blot was also performed, using a commercially available polyclonal antibody (Oxford Biomedical, PG 27) on six of the tumor tissues.

Primary cell cultures were grown to near confluence and then divided into several culture flasks. As a control, an additional primary cell culture was available from four. For eight of the cases, normal fibrocytes were grown to near confluence and then divided into several culture flasks. As a control, an additional normal cell culture was available from four. For eight of the cases, normal fibrocytes were grown to near confluence and then divided into several culture flasks.

Proliferation and apoptosis were determined after twenty-four hours in culture by a germ-line mutation in the adenomatous polyposis coli (APC) gene (1). Familial Adenomatosis Polyposis (FAP) is an inherited pre-neoplastic condition that predisposes to colonic neoplasia and occasional fibromatoses, that is caused by a germ-line mutation in the APC gene. A large percentage of sporadic colonic polyps and cancers also harbor somatic APC mutations.

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