HIV-ASSOCIATED KAPOSI’S SARCOMA SPINDLE CELLS: SMOOTH MUSCLE ORIGIN AND IMMUNOLOGICAL LINK

Here I will focus on (i) a possible smooth muscle cell (SMC) origin of HIV-associated Kaposi’s sarcoma (KS) spindle cells, and (ii) their growth control by activated immune cell-derived cytokines (1-3). The following is considered:

(i) if KS spindle cells, or a subpopulation of them, derived from SMC (1), they should then also reveal other features of SMC that are not explored in KS spindle cells. One of these "other features" is the readiness of vascular SMC to shape-change, from spindle to stellate, in response to beta-adrenergic stimulation (4,5) and to an actin-destroying agent, cytochalasin B (4). Further, this process is (a) cytoskeleton-dependent, and (b) pharmacologically preventable by antitubulins, such as colchicine (4) and by protein kinase C activators (5). Meanwhile, this SMC shape-change was first described in 1866 by Langhans (6), but neglected by Rudolph Virchow, in human atherosclerosis.

(ii) because tumor necrosis factor (TNF)'s growth stimulation index over KS spindle cells is one of the highest between other growth factors tested (1, their Table 5), the following recent data should be kept in mind (a) TNF production is regulated by beta-adrenergic agonists (7), (b) antitubulins downregulate TNF receptors (8), (c) colchicine prevents TNF-induced toxicity (9), and (d) the shared actions of endotoxin and the microtubule-stabilizing agent taxol on TNF receptors and TNF release (10). Hence a possible pharmacological control over hyperplasia in HIV-associated KS spindle cells, using anti-TNF agents proposed for research in AIDS therapy (11-13), seems to be a promising approach together with some folklore’s "counter irritants" with antioxidant activity, e.g. turmeric (see 14) and some drugs known to inhibit SMC’s secretion, e.g. colchicine (15), and possibly brefeldin A (see 16).

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