IJAE
Vol. 116, n. 1 (Supplement): 152, 2011
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

# Intima neovascularization studied on epoxy-resin semithin sections in a rare case of carotid recurrent in-stent restenosis

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*Introduction*. Restenosis after carotid endarterectomy (CE) has been described in 8-19% of patients (1). In-stent restenosis (ISR) after stenting has been reported in a few cases (2). Here we report an extremely rare case of recurrent ISR after CE and wallstent.

*Materials and methods*. An atherosclerothic plaque belonging to a patient who underwent recurrent ISR after both CE and wallstent has been studied. Semithin sections (0,850 $\mu$m thick) obtained from epoxy-resin embedded samples were stained as reported (3). Light microscopy observations were performed at magnifications of 400X and 1000X, within an area of 3,8mm2 corresponding to 10 microscopic field at 400X. The morphological characteristics considered were: cells number and type; presence, number and type of neovessels; calcifications, and inflammatory cells.

*Results*. The plaque appeared mostly fibrous, with an internal rupture and hemorrhagy. Very few foam cells (N= 12/3,8mm2) were observed whereas vascular smooth muscle cells (VSMCs) were abundant (n=160/3,8mm2) and often entrapped in a thick coat of self-secreted collagen. Lipid laden VSMCs were present (n=18/3,8mm2); three of them showed intracellular microcalcifications. Marked signs of inflammation were found, macrophage (n=50/3,8mm2), mast cells (n=8/3,8mm2), lymphocytes (n=5/3,8mm2) as well as granulocytes (n=4/3,8mm2). There was a marked neovascularization, 24 sections of circular regular vessel and 6 sections of flattened but patent vessels were found in 3,8mm2. Neovessels had intact endothelium and likely derived from the underneath media.

*Discussion*. Histopathological diagnosis is usually carried out on paraffin-embedded 4$\mu$m-thick sections. The observation of semithin sections has allowed us to achieve a better morphological characterization than that routinely obtained. The cellular population observed is that of an active remodelling plaque, susceptible of rupture, both for the presence of mast cells and extensive neovascularization. Mast cells secrete effector molecules that affect endothelial cells and VSMCs activity, the presence of neovessels weakens the plaque, thus producing cleavage planes.

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Keywords: atherosclerosis, neovascularization, in-stent restenosis, carotid artery

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