

## APOPTOSIS IN VARICOSE VEINS OF WOMEN OF DIFFERENT AGE GROUPS

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Varicosis is a complex pathology characterized by venous hypertension, blood stagnation, and reflux leading to progressive venous wall remodelling. The primary cause is still unknown, but it is likely that defect is in the wall of the lower limb veins. One of the factors influencing vascular wall remodelling is cell apoptosis. The apoptosis of endothelial cells (EC) may promote infiltration of inflammatory cells into the intima and increase smooth muscle cells (SMC) proliferation [1] and changes in the cells ultrastructure. Damages of endothelial and smooth muscle cells can destroy the structure of the vascular wall.

The aim of this study was to detect apoptotic endothelial cells and smooth muscle cells in varicose veins wall. Material obtained from 168 patients was analyzed using light microscopy, immunohistochemistry and electron microscopy. Women (n=147) undergoing the excision of varicose veins were divided into 3 groups: younger than 35 years (Group I, 34 patients), 36-50 years (Group II, 54 patients), older than 50 years (Group III, 59 patients). Control group form 21 women. Apoptosis in EC and SMC was determined by the TUNEL method. Cell apoptosis was assessed by scoring the TUNEL-positive cells in three layers of veins, i.e. in intima, media and adventitia. TUNEL-positive staining was graded from 0 to 4.

Results: In most patients veins were stretched out with highly varying wall thickness. In most specimens endothelium was either damaged or missing. In media and adventitia smooth muscle cells were destroyed or showed altered morphology, the amount of connective tissue was increased together with irregular organization of collagen fibers and disruption of the elastic network around SMC bundles. In Group II and especially in Group III the percentage of apoptotic EC and SMC in the layers of varicose vein wall was increased compared to Group I and Control group.

In conclusion, the study demonstrates that aging is associated with increased sensitivity of EC and SMC to apoptotic stimuli.

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### References

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