INTRODUCTION

Varicose veins are an important cause of morbidity with a prevalence of 10 to 50%.

Risk factors: advanced age, female gender, diet, obesity, physical activity, standing occupations, connective tissue alterations, genetic predisposition.

The aetiology and pathogenesis: Two hypothesis have been proposed:

1. Vasculal dysfunction causing venous reflux in early state of pathology
2. A primary change in varicose vein wall (structural and biochemical changes).

We hypothesize that the formation of varicose vein is secondary to defects in cellular and extracellular matrix components, causing wall weakness and altered permeability.

METHODS

Samples

Saphenous veins from patients undergoing varicose vein surgery (CEAP-2) Segments were divided into: normal, lesions, varicose vein (VV), and altered Endothelial and distal normal (NV) normal with Eco-Doppler.

Immunohistochemistry

Varicose veins

Primary antibodies are used to detect and visualize the distribution of various fibrous proteins.

Microscopy

Methods include:

- Immunohistochemistry
- Histology and Neuroscience
- Confocal microscopy

RESULTS

Segments with normal macroscopic appearance

Dilatation

Irregular muscle fiber arrangement

Increased collagen fibrils

Intimal nodules

Segment with normal histology

Segment with initial tissue alterations

Conclusions

1. In the proximal segment, the surface of varicose vein wall is normal with minimal intimal conjunctive tissue in some regions.
2. In the proximal segment, the wall is normal with minimal intimal conjunctive tissue. Myointimal cells and adventitia are normal. Adventitia does not show any lesion.
3. In the varicose vein wall tissue of normal fibrosis are also observed. In these degenerative changes, mastocytes do not seem to play a role.