ΑΝΑΤΟΜΙΚΗ ΜΕΛΕΤΗ

ΕΛΛΗΝΙΚΗ ΔΕΡΜΑΤΟΧΕΙΡΟΥΡΓΙΚΗ
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Etiopathogenetic hypothesis about venous ulcer

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As it can be noticed from anatomical preparations (in succession during preparation), in the medial malleolar anatomical district up to the inferior third of the leg, two small satellite arteries for each perforating vein (figures 1-3) are evidenced or a single arterial trunk, greater and straight away out of the fascia, that gets entangled, in spiral, around the perforating vein (figure 2) and divides (figure 3) into two or more small arteries, in a crawling way on the external surface of the fascia; subsequently they become superficial, branching out heavily, at first in the subcutis (figure 4) and subsequently in the hypodermis and dermis (figure 5) and at last assuming a terminal distribution. In the two superior districts (thigh and leg), otherwise every perforating vein has a unique perforating artery (figure 8).

A perforating vein is unique for the outer muscular fascia, but it originates from the union of two trunks which depart from two satellite veins of the deep system (figure 6).

Nowadays, there is no theory available to explain the reason why varicose ulcers develop in the same anatomic region; a hypothesis which is more accredited is that of the increased hydrostatic pressure, while another theory suggests that varicose ulcers develop due to the presence of many perforating veins in the same region.

Nevertheless, these two hypotheses don't explain the reason why a patient, who suffers from varicose veins for many years, suddenly develops an atrophic lesion; evidently, the pathology of this acute event may not be a chronic situation.

The described anatomical study, experienced in various post mortem examinations, was the first to demonstrate the alteration of the two small satellite arteries for each perforating vein in the upper malleolar region and in the inferior third of the leg-medial face.

The reason of this situation is yet unknown, but it can be supposed with a certain confidence that primum movens of the atrophic lesions may be an ischemic event caused by a progressive compression of the

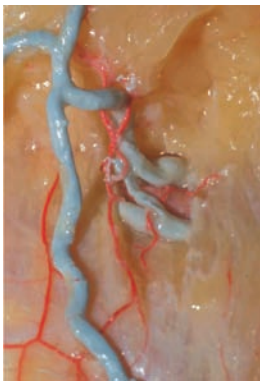

Figure 1.

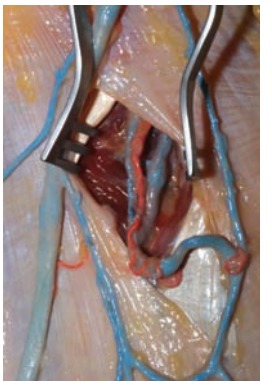

Figure 2.

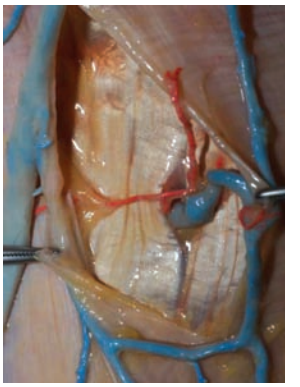

Figure 3.

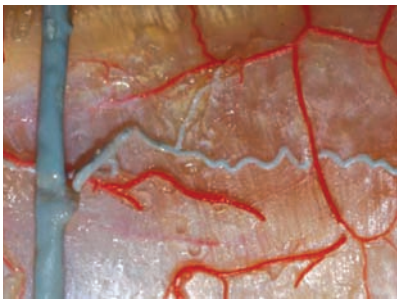

Figure 4.

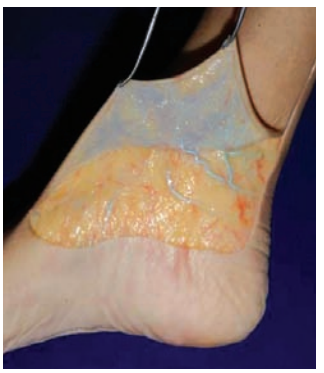

Figure 5.

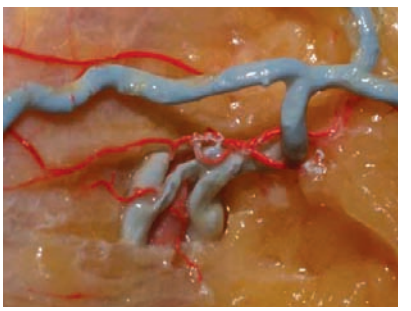

Figure 6.

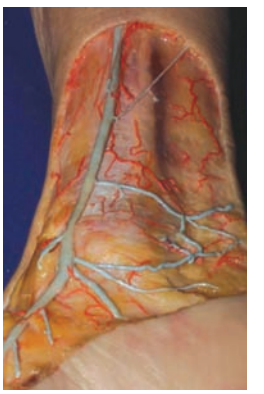

Figure 7.

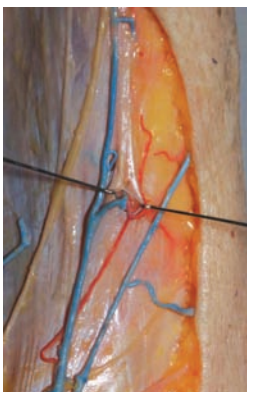

Figure 8.

dilated veins on arteries: these arteries are trapped and strangled by the dilated perforating vein and between the vein and the fibrous ring of muscular fascia, which is non-stretch and adherent.

These small but numerous vessels are terminal and they feed skin wall in the medial back malleolar region, so that a low arterial flow can create an ischemic state and consequently the lesion.

It is important to observe that the described hypothesis doesn't represent an annulment of the hypertensive microangiopathy hypoxia, but it is its integration: in fact hypoxia is present in all the distal part of the leg, whereas ischemic hypothesis concerns specifically the preferred critic point of ulcus cruris.

Therefore, Anatomy, although little studied or not widely accepted and referred to in oldest studies, gives interesting data which may open new pathways for more appropriate therapies. This theory must be validated further on with thorough researches and studies but, for the moment, it can be a stimulus for serious consideration.