

SUMMARY

Sclerotherapy refers to the introduction of a foreign substance into the lumen of a vessel causing thrombosis and subsequent fibrosis. This procedure, when performed on telangiectasias is referred to as microsclerotherapy.

Fortunately, the majority of patients with ectatic and telangiectatic veins do not have a life-threatening problem. Therefore, treatment should be as simple as possible, with least risk of significant side effects.

The development of varicose veins is probably secondary to anatomic differences. The venous system of the lower extremities functions as a conduit for carrying deoxygenated blood from the muscles, the cutaneous and subcutaneous tissues to the heart and also functions as a reservoir of blood. It consists of three channels one within the muscular system and one superficial to it, the deep venous system, the superficial venous system and the perforating veins which connect deep veins to the superficial veins. Dysfunction in any of these three systems result in dysfunction of the other two. When the superficial veins are placed under high pressure, they varicose to accommodate the increased blood volume.

When become chronic, this may lead to valvular insufficiency, which usually causes a reversal of blood flow from the deep veins into the superficial veins through incompetent veins leading to formation of varicose veins and telangiectasias.

Telangiectasias represents an expanded venule, a capillary, or an arteriole ranging in diameter from 0.1 to 1mm. It is classified into 4 types based on clinical appearance: -

1-sinus or simple, 2-arborizing, 3-spider or star 4-puntiform (papular).

Many conditions-inherited, acquired, and iatrogenic-are known to be involved in the formation of telangiectasias. Varicose veins lead to the development to telangiectasias most likely through associated venous hypertension with resulting angiogenesis or vascular dilation, or through an associated distensibility of the telangiectatic vein wall.

For sclerotherapy to be effective without recanalization of the thrombotic vessel, the endothelial damage and resulting vascular necrosis must be extensive enough to destroy the entire blood vessel wall.

All sclerosing solutions can be placed into three broad categories based on their mechanism for producing endothelial injury: detergent, osmotic, or chemical.

External compression can benefit the patient with venous insufficiency by augmentation of the body's natural muscle pump through application of a graduation of pressure in the leg , forcing blood toward the heart .

Postsclerotherapy compression primarily eliminates a thrombo - phlebitic reaction and substitutes a sclerophlebitis with the production of a firm fibrous cord.

Unfortunately, as with any therapeutic technique, sclerotherapy carries with it a number of potential adverse sequel and complications, Postsclerotherapy coagula, temporary swelling, telangiectatic matting, pain, localized urticaria, tape compression blisters and folliculitis, and recurrence. Hazardous complications include coetaneous necrosis, arteriolar or lymphatic injection, superficial thrombophlebitis, air embolism, pulmonary embolism and deep vein thrombosis.

The permanent eradication of ectatic and telangiectatic leg veins by compression sclerotherapy should be a predictably successful endeavor.