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Original ARTICLE

VENO-ARTERIOLAR REFLEX VASOSPASM OF SMALL SAPHENOUS ARTERY COMPLICATING SCLEROTHERAPY OF THE SMALL SAPHENOUS VEIN

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ABSTRACT

A 54-year-old woman underwent ultrasound-guided sclerotherapy of the left small saphenous vein (SSV). The vein measured 10.1 mm in diameter and was treated with 4 ml of sodium tetradecyl sulphate 3% liquid.The procedure was completed successfully with no unusual symptoms or signs present at the time of the procedure. On follow-up, the patient developed reticulate purpura and eventually mild fat atrophy. This was associated with the presence of a small artery running along the SSV in its fascial envelope. Treatment included low molecular weight heparin, non-steroidal anti-inflammatory drugs, foam padding and class two compression stockings. At 6 months follow up, the left SSV was fully sclerosed along its entire length but fat atrophy was clinically present.

Veno-arteriolar reflex is a well-known physiological vasospastic response of arteries in response to rapid dilatation of their associated veins. Rapid injection of sclerosants into confined venous lumens can induce a corresponding reflex vasospam in the associated arteries or arterioles. Our patient did not develop necrosis. However, rapid arteriolar vasospasm in association with sludge formation and occlusion of the affected vessels can cause cutaneous necrosis. We propose veno-arteriolar reflex as a possible mechanism responsible for the initial reticulate purpura and the subsequent atrophy in our patient.

Key words: *sclerotherapy, small saphenous artery, veno-arteriolar reflex.*

Introduction

Ultrasound guided sclerotherapy (UGS) is a well-established form of varicose vein therapy with a high safety profile. However complications such as necrosis may arise when venturing into areas that are highly vascularised or where variable arterial anatomy exists. The mechanism of post-sclerotherapy necrosis is poorly understood and various possible causes have been suggested. These include simple extravasation, intra-arterial injection, sludge formation and subsequent closure of arterio-venous shunts. Experienced sclerotherapists report an immediate blanching followed by hyperemia and eventual cyanosis and purpura preceding necrosis in some patients. The reticulate (multi-angulated) pattern is diagnostic of an arteriolar event.

We are reporting reticulate purpura and fat atrophy following uneventful UGS of a small saphenous vein (SSV).

Case History

A 54-year-old female presented with symptomatic post surgical recurrence of her left leg varicose veins with symptoms of pain, tiredness, heaviness and restlessness. Her left great saphenous vein (GSV) had been stripped from the saphenofemoral junction (SFJ) to the knee. Following the operation, she had residual parasthesia affecting her right medial ankle due to surgical damage to the saphenous nerve. The rest of her medical history was unremarkable. She was gravida 3, para 3, on no regular medications and not allergic to any drugs.

Duplex scanning demonstrated major incompetence of her left saphenopopliteal junction (SPJ) and major reflux of the SSV extending to the lateral foot. The distal GSV was also incompetent.

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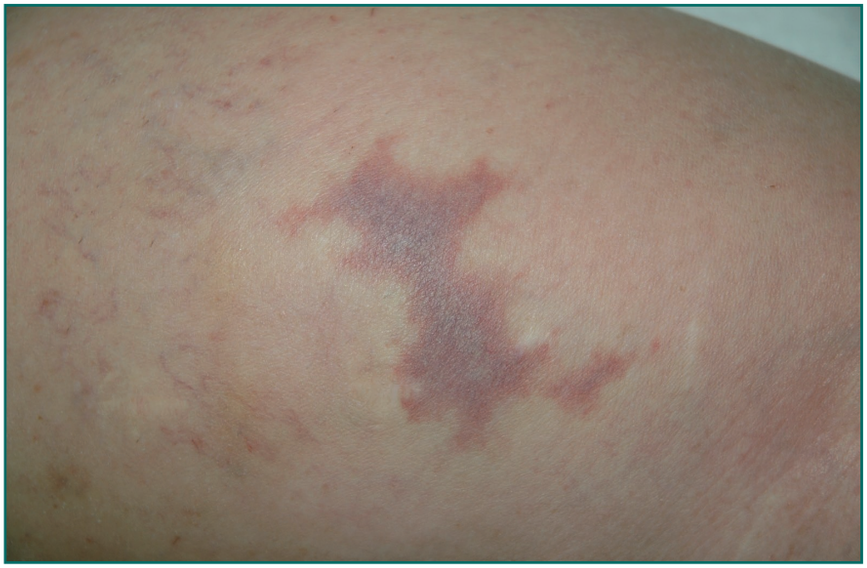

Figure 1: *Reticulae purpura following UGS of the SSV*

The patient underwent UGS. Her SSV measured 10.1mm in diameter. It was treated with 4ml of sodium tetradecyl sulphate (STS) (Australasian Scientific & Medical, Artarmon, NSW, Australia) 3% liquid. Class II compression stockings were applied. The procedure was uneventful with no immediate adverse reactions. The patient did not complain of excessive pain during or after the treatment. No blanching or immediate hyperemia or cyanosis was noticed at the time of the treatment.

Two days later she reported erythema and pain with hardening of the skin overlying the injection sites to the skin of her medial posterior calf.

On examination she presented with an irregular patch of dusky erythema measuring 5cm by 3cm over the left medial calf (Figure 1). The patient was treated with fractionated low molecular weight heparin (enoxaparine), oral ibuprofen, foam padding and Class II compression stockings. Two days later she had developed reticulate purpura in the left medial calf area indicating impending skin necrosis but the pain had improved.

Detailed duplex scanning of the affected area revealed a small artery running alongside the SSV in its fascial envelope. This vessel was not visualized previously during the initial incompetence mapping nor during the UGS. The artery was 1.25mm away from the vein (Figures 2, 3a and 3b). It arose from a gastrocnemius perforator artery at the level of mid-calf, traveled with SSV in the fascial envelope for a short distance, then left the facsial compartment to travel along an intersaphenous vein in the subcutaneous fat. This artery was found to supply the corresponding area of fat atrophy and the cutaneous reticulate purpura.

The skin did not ulcerate and returned to normal within two weeks of treatment. The induration persisted and was replaced by fat atrophy.

At six months follow up, the left saphenopopliteal junction was found to be fully sclerosed. The SSV was fully sclerosed along its entire length to the level of the lateral malleolus. The small saphenous artery was still present and patent. There was fat atrophy in the left medial calf area.

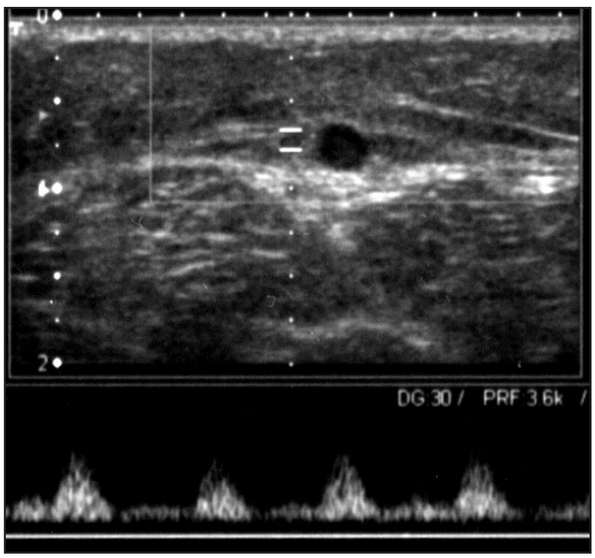

Figure 2: *Cross-section of the small saphenous vein with its accompanying artery.*

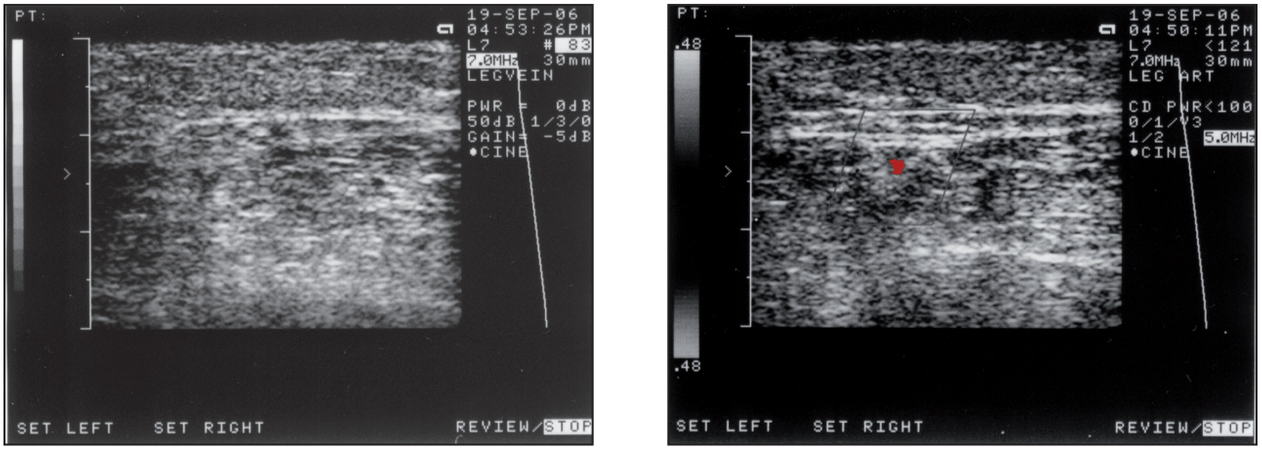

Figure 3A AND 3B: *It is almost impossible to visualise an arteriole adjacent to the sclerosed small saphenous vein on B-mode however with colour spectral analysis, arterial flow is demonstrated.*

Discussion

The posterior calf is one of the high-risk areas for sclerotherapy. A small variably placed satellite artery, small saphenous artery (SSA), may be presented adjacent to the SSV. SSA provides blood supply to the vasovasorum of the SSV.[1] Schadeck et al[2] reported a frequency of over fifty percent (50%) of such arteries in 108 limbs and de Somer-Leroy et al[3] reported an almost constant finding in 125 patients. The risk of causing arterial spasm or damage by direct intra-arterial injection or extraluminal irritation is an ever concern if this artery is present. Older ultrasound systems with low resolutions may not be able to detect the presence of this artery. High-resolution systems may also find it hard to detect this vessel and unless the operator is specifically looking for this vessel it may be easily missed.

Direct intra-arterial injection as a complication of sclerotherapy is quite rare.[4] Cases of intra-arterial complications have been reported around the popliteal and ankle regions with sequelae of cutaneous necrosis, compartmental syndrome, muscular contractures and amputations of involved limbs.[5-9]

Our patient's complication presumably arose from reflex vasospasm of the SSA secondary to a rapid intravenous injection of the SSV. The second possible mechanism is occlusion of an arterio-venous shunt between the SSV and SSA secondary to sclerosant sludge formation. The second possibility is less likely given that no such shunt was observed on detailed ultrasound scanning using colour Doppler, power Doppler and Advanced Dynamic Flow (applying ultra-high bandwidth normally used in B-mode

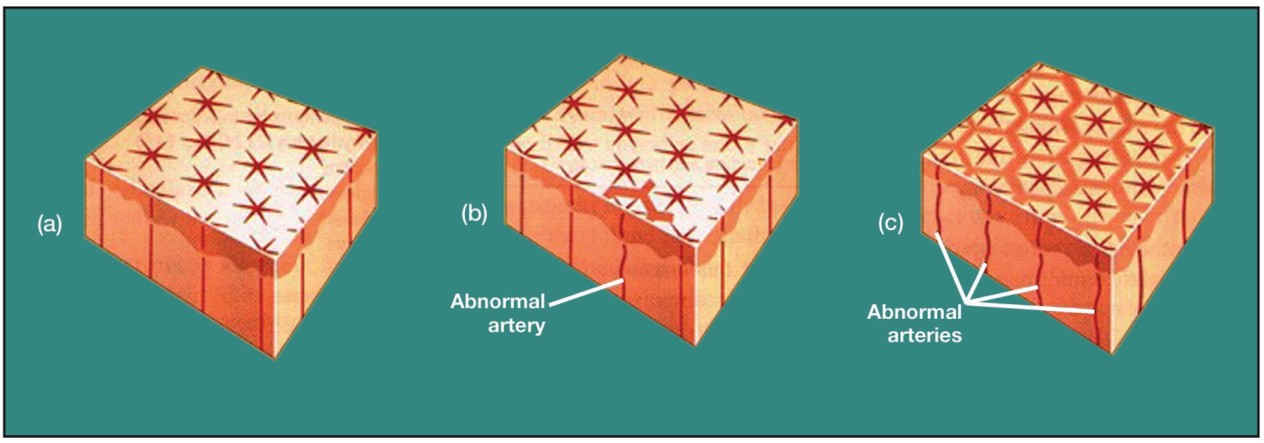

Figure 4: *Reticulate pattern of arteriolar supply of the skin. Note vasoconstriction or occlusion of these arterioles will affect the corresponding areas of the skin in that particular pattern. The size of the patch will depend on the size of the affected arteriole.*

to Doppler signal processing). Also the artery was found to be open two days after the incident maintaining normal flow. Finally, full occlusion would have been less likely given that no necrosis eventuated.

Rapid dilatation of superficial veins is thought to induce a reflex vasospasm of the associated arteries and arterioles. This reflex alters and controls the vasomotor tone of the local arterial system. The study of changes of flow in the toes when one leg only of a supine subject is elevated has provided evidence for these reflexes.[10] As expected, leg elevation causes a decrease in arterial flow. However, one would also expect that lowering the limb below the level of the heart might show either an increase in flow or no change - certainly not a reduction in flow. Yet there has been consistent evidence of a decrease in flow on rapid lowering of the limb. Convincing evidence that the stimulus came from the distention of the leg veins was provided when it was noted that, in some subjects, returning the leg to the horizontal did not restore the flow to normal until the leg was lifted momentarily and the veins were drained.

This phenomenon was further investigated in a study of the blood flow of the fingers, using both negative (in the plethysmograph) and positive tissue pressure.[11,12] Vasoconstriction occurred as a result of the increase in transmural pressure and distention of the veins.

Veno-arteriolar reflexes provide a negative feedback mechanism. Congestion of the veins would be relieved if arterial inflow was reduced. Therefore outflow from the veins would be greater than their inflow and a stable state would be quickly formed. Also, full closure of the arterioles due to venous congestion would not occur.

This phenomenon can explain the clinical events that precede necrosis. Arteriolar vasospasm causes the immediate blanching that is clinically observed. The subsequent reactive hyperemia is due to re-perfusion of the affected area. This usually starts in the periphery of the affected skin and expands slowly till the whole area is re-perfused. Dusky erythema follows. This is replaced by reticulate purpura in a day or two and eventually formation of a dark scab. The size of the reticulate patch, its shape and location depends on the size and distribution of the arterioles affected (Figure 4). If re-perfusion is adequate or if the initial insult is not significant, necrosis does not occur.

In one of the author's (KP) experience, it is also more likely to create a cutaneous ulcer if a lumen or dead space within a previously treated vein is being injected. Once again, injection of a confined space within a vein's lumen can cause rapid dilatation of the venous segment and vasospasm of the associated arteries or arterioles. Hence slow injection techniques are advocated. In conclusion, we postulate the cause of the reticulate purpura in our patient to be a vasospastic response secondary to rapid injection of sclerosant into the SSV.

Acknowledgement

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