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SHORT COMMENTARY

Extensive Tissue Necrosis: A Rare but Catastrophic Complication of Sclerotherapy

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Introduction

Both sclerotherapy and open surgical treatment have been used for varicose veins for over 100 years [1]. Increasingly in the last 20 years the non-surgical treatments, such as endovenous ablation (EVA) and sclerotherapy, have became popular and largely replaced open surgical treatments in international guidelines [2]. In my practice, sclerotherapy has developed from a cosmetic treatment for superficial veins, into a commonly used therapy for saphenous trunks and tributaries [1]. Ultrasound guided foam sclerotherapy (UGFS) has become an established modality for the treatment of varicose veins, either alone or in combination with other non-surgical treatments.

While sclerotherapy may seem safe, catastrophic complications have been reported, including local and systemic adverse reactions. For example, extensive tissue necrosis and cerebrovascular accident (stroke) [3].

Patient selection is vital and treating physicians should be aware of all potential complications and discuss them with their patients, prior to treatment, to obtain informed consent. Physicians also need to be prepared to manage catastrophic complications that could lead to amputation or death.

Tissue Necrosis

Intra-arterial injection represents the most feared complication of sclerotherapy for varicose veins [4].

Ulceration or local tissue necrosis is rare following sclerotherapy and in the region of 0.2 to 1.2% [5]. The aetiology is poorly understood and thought to be due to extravasation of sclerosant during injection. It is my experience that sclerotherapy ulcers heal by secondary intention and good wound management to encourage granulation. They are usually quite painful and warrant simple analgesia and reassurance.

Extensive tissue necrosis has been ascribed to direct intra-arterial injection and is extremely rare with < 100 cases reported worldwide. However, despite several different treatment approaches, amputation could not be prevented in > 50% of cases. Inadvertent intra-arterial injection represents a limb threatening complication of sclerotherapy [4]. Ischaemia of 6 hours duration [6] will lead to extensive tissue necrosis, there by necessitating early recognition and treatment to promote revascularisation.

The signs and symptoms of acute limb ischaemia include the six p's. These are pain, pallor, pulselessness, perishing cold, paraesthesia and paralysis [6]. Painful injection associated with immediate pallor of the affected limb should raise the suspicion of a possible inadvertent arterial injection. Over the following hours the pallor turns to a demarcated, mottled area associated with the affected peripheral artery. However, this is variable and sometimes there is no preceding pain or pallor.

The evidence for direct intra-arterial injection is circumstantial and there are other theories to explain the flow of sclerosant into the arterial arborisation. Arteriovenous malformations and thermoregulatory

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shunts (damaged by venous hypertension) have been postulated [7]. Equally the Venoarteriolar Reflex, where arteries constrict in response to rapid dilatation of their corresponding veins, has been put forward as an explanation of how arterial ischaemia can occur despite obvious intravenous injection of sclerosant on duplex ultrasound [8]. This effect has also been shown to occur in the contralateral limb [9] and it is not hard to imagine that if the patient's microcirculation is already compromised [10] this could lead to devastating consequences. Never more so than a recent patient of mine in her 70's, with strong cardiovascular risk factors, who developed bilateral ischaemic feet and suffered bilateral below knee amputation. I had treated her with bilateral UGFS as an adjunct to bilateral radio-frequency thermal ablation (RFA.)

So how can these potentially devastating complications be avoided?

The first area of concern is patient selection. There is varying opinion around the world regarding peripheral arterial disease (PAD) as a contra-indication for sclerotherapy. However professional bodies in India in 2011 and the USA in 2014 determined severe PAD to be an absolute contra-indication to sclerotherapy treatment [2]. My own patient had several cardiovascular risk factors such as age (> 70 yrs), smoking, hypertension, hypercholesterolaemia and strong family history of ischaemic heart disease but was asymptomatic for PAD. With years of experience and success, treating all comers for varicose veins, it is possible for phlebologists to overlook the risks involved with treating arteriopaths and patients with other chronic diseases. I now believe that asymptomatic patients with risk factors and symptomatic PAD patients should be further investigated prior to sclerotherapy. Investigations to be considered, in the office setting, are evaluation of the peripheral pulses [11] and duplex scanning of Doppler waveforms and pulse volume recordings [12,13]. A normal lower extremity arterial Doppler velocity tracing is triphasic. A biphasic signal is considered abnormal if there is a clear transition from triphasic to biphasic along the vascular tree and monophasic waveforms are always considered abnormal [12]. The ankle-brachial (ABI) and toe-brachial (TBI) indices are also easily performed and have high sensitivity and specificity for PAD.

The next area to consider is the sclerotherapy treatment itself. Unfortunately, the data associated with sclerotherapy treatment guidelines is heterogeneous and there is huge variation in technique between practitioners and in different countries. I believe that one important area to consider is the maximum volume of sclerosant per session. As regards foam volume, 10 ml is the upper limit for European guidelines and 20 ml for the Australian guidelines [2]. It would seem prudent to use the minimum possible dose, volume and concentration required per session. Consideration should be given to treating unilaterally rather than bilaterally as a measure to reduce potential complications and keeping the volume of foam to < 10 ml per session.

When it comes to treating saphenous, axial incompetence and especially in large veins (> 10 mm diameter at the saphenofemoral or saphenopopliteal junctions) I am increasingly combining thermal and non-thermal EVA techniques to reduce the reliance on UGFS. For example, in a large great saphenous vein I will combine RFA proximally with cyanoacrylate glue ablation distally and delay any further treatment for a few weeks to allow resolution of tributaries. That way minimalistic sclerotherapy or UGFS is required at follow up for cosmesis only.

Finally, recognition of an ischaemic attack can be difficult but prompt action for a suspected intra-arterial injection maybe the difference when it comes to saving the limb. Practitioners should develop a protocol for "**suspected**" arterial injection. However, no prospective human studies have shown that any specific treatment is superior [14] and the only universally recommended therapy is heparinisation, while steroids, acetyl-salicylic acid and peripheral vasodilators may all have their place.

Conclusion

It seems for now that sclerotherapy and UGFS will continue to be a mainstay of modern non-surgical varicose vein treatment. However, infrequent as tissue necrosis may be, it is truly devastating to both patient and physician when it occurs. Minimising the risk of complications by good patient selection, cautious treatment and developing adequate emergency protocols is vital and should not be forgotten.

Unfortunately complying with relevant guidelines can be difficult due to the heterogeneity of recommendations. In the future communication and collaboration between interested colleges and societies should be encouraged to improve the quality of data and development of more authoritative guidelines for sclerotherapy treatment.

Conflicts of Interest

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