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PROCEDURAL DERMATOLOGY (DIRECT VIA EEO)

Treatment of facial hypertrophic capillary malformations with tumescent-assisted sclerotherapy

Mina Kang[1,2] | **Kurosh Parsi**[1,2,3]

[1]*Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst, New South Wales, Australia,* [2]*University of New South Wales, Sydney, Australia, and* [3]*Australasian Vascular Anomalies Centre, New South Wales, Australia*

ABSTRACT

Facial capillary malformations (CMs) become hypertrophic and nodular overtime and pose great therapeutic challenge. Here, we describe safe and effective use of tumescent-assisted sclerotherapy (TAS) in conjunction with yellow vascular laser (577 nm) for the treatment of HFCMs. Three patients underwent TAS were included in the case series, and complete resolution in nodularity was achieved in all patients with TAS, with no major complications such as skin necrosis, distal embolisation, blindness and neurological adverse events such as stroke or TIA occurred in any patients.

Key words: Capillary malformation, CM, facial vascular malformation, sclerotherapy, tumescent anaesthesia.

Learning points

- Tumescent-assisted sclerotherapy is effective in treating nodular and hypertrophic areas of facial capillary malformations.
- Comprehensive understanding of head and neck anatomy, extensive experience in vascular interventional techniques, adjuvant tumescent anaesthesia and intra-operative ultrasound guidance are all critical to perform this safely.

Correspondence: A/Prof Kurosh Parsi, Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St. Vincent's Centre for Applied Medical Research, Level 8, Lowy-Packer Building, 405 Liverpool Street, Darlinghurst, NSW 2010, Australia. Email: kurosh.parsi@svha.org.au

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Mina Kang, MD. Kurosh Parsi, PhD.

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INTRODUCTION

Capillary malformations (CMs) are common congenital vascular malformations affecting the dermal vessels. CMs do not spontaneously involute or regress. Rather, they grow commensurate with the child's growth. The lesions most commonly affect the head and neck, in particular the V1 and V2 dermatomes. Facial CMs initially appear as pink patches that evolve into darker and thicker plaques that may exhibit soft tissue hypertrophy and nodularity during adulthood. Hypertrophic CMs cause cosmetic disfigurement and may even cause vision and other functional impairments.[1]

Treatment options for hypertrophic facial CMs (HFCMs) have included surgical excision, dermabrasion, radiotherapy, electrocautery, cryotherapy, photodynamic therapy (PDT), intense pulsed light (IPL) and various laser modalities.[2–4] Current treatment of choice for CMs is pulse dye laser (PDL) therapy, but the treatment outcome is suboptimal in 40%–50% of patients with complete resolution in 10% of patients and recurrence rates of 35%.[5] In hypertrophic CMs, the outcomes are even poorer.[6] Improved results have been reported with other lasers such as Nd:YAG and Alexandrite systems both of which achieve deeper penetration than PDL. Nonetheless, hypertrophic CMs remain a therapeutic challenge and complete resolution is difficult to achieve.

Sclerotherapy is commonly used in the treatment of venous malformations (VMs) and less frequently arteriovenous (AVM) and lymphatic malformations (LMs). Detergent sclerosants such as sodium tetradecyl sulfate (STS) and polidocanol (POL) may be injected as liquids or prepared as foams by mixing the liquid agent with a gas such as room air. The agent is then injected into the target lesions to cause vessel occlusion. To our knowledge, foam sclerotherapy has not been reported in the treatment of CMs. Here, we discuss our treatment technique using tumescent-assisted sclerotherapy (TAS)[7] to treat the nodular regions of hypertrophic CMs in

combination with yellow vascular laser (577 nm) for non-nodular plaques.

MATERIALS AND METHODS

Materials

STS (FIBRO-VEIN, STD Pharmaceuticals, Hereford, UK); POL (Aethoxysklerol®, Chemische Fabrik Kreussler, Wiesbaden, Germany); Aplio™ 500 Duplex Ultrasound (Canon, Tokyo, Japan); tumescent anaesthesia (TA) prepared by adding 20 mL of lignocaine 2%, 5 mL of adrenaline 1:10 000, 5 mL of sodium bicarbonate 8.4% to 250 mL of normal saline 0.9%; 577 nm vascular laser (QuadroStarPRO, Asclepion, Jena, Germany); Minims Amethocaine (Tetracaine) Hydrochloride 1% w/v (Bausch+Lomb, New York, USA); EMLA® (Aspen Pharmacare, Durban, South Africa); 5-micron filter (Filter-hub, B.Braun, Melsungen, Germany); three-way tap (BD Connecta, New Jersey, USA); luer-lock syringes (Terumo, Leuven, Belgium).

Methods

Patient selection

Three patients with HFCMs are described. All patients were clinically assessed and photographed. Duplex ultrasound (DUS) scans were performed to confirm the clinical diagnosis and exclude underlying or associated VMs, AVMs or LMs that may be found with CMs in combined lesions.

Treatment approach

Non-nodular thinner plaques were treated with the yellow laser (577 nm) in the scanning mode. Hypertrophic plaques were treated with TAS using STS 1.5% foam for nodules and POL 0.5% foam for the less nodular but thicker plaques deemed to be unresponsive to laser therapy.

Foam preparation

Sclerosant foam was prepared with room air using a modified Tessari technique. 1 mL and 3 mL lure-lock syringes were each connected *via* a 5-micron filter to a three-way stopcock. Terumo syringes were selected over other brands to ensure minimal rubber content and stable foam production with a reasonable half-life.[8] Macro-bubbles were avoided following techniques previously described by this group.[9–11]

Tumescent-assisted sclerotherapy technique

EMLA® was applied to the target area 60 min prior to the procedure. Ultrasound guidance was used to assess the target lesion and identify normal structures such as nerves and arteries to avoid inadvertent injury. Normal draining veins in the region of interest were compressed through perivascular ultrasound-guided injection of TA prior to sclerotherapy to obstruct the outflow and systemic dissemination of foam-derived bubbles. In the peri-orbital region, these included the infraorbital, angular, supratrochlear and supraorbital veins. TA was also infiltrated by direct injection in the tissue plane immediately below the malformation to compress the target lesion, reduce its size and minimise the neutralisation of the sclerosants that occurs following mixing with intravascular blood content.[7] TA was drawn up in a 10 mL syringe and injected *via* a 25 guage (1.5 inch) needle.

Foam sclerosants were percutaneously injected intralesionally into the vascular space of the target lesion until mild blanching was observed. Extreme caution was exercised to deliver the injections with minimal pressure, minimal volume of sclerosants and at a slow rate. When lesions were extensive, small areas were treated at a time. External compression tapes were applied for 4 h.

Laser technique

A 577-nm laser system (7 W, 6 ms, continuous wave, scanning mode, 14–32 J) was used following laser safety regulations. To treat the CM involving the upper and lower eyelids, an ophthalmic shield was used after the application of topical anaesthetic eye drop.

RESULTS

Three male patients with moderate-to-severe facial regional hypertrophy were treated. Two had prominent nodularity. All had failed conventional therapy and one had a significant recurrence following previous surgical excision of nodules (Table 1).

On average, each patient underwent 9 procedures in a 12-month period (Table S1). The interval between treatment sessions ranged between 2.5 and 5 weeks. The sclerosant volume ranged between 5 and 24 mL of foam in an individual session. TA was used in all sclerotherapy sessions at volumes ranging between 20 and 100 mL.

The treatments were well tolerated in all patients with none complaining of pain or discomfort during or after the procedure. No other significant complications such as distal embolisation, blindness and neurological adverse events occurred in any patients. Small areas of skin necrosis occurred at the injection site in two patients which resolved within 14 days with no scarring.

Complete resolution of nodularity was achieved in all patients (Figs 1,2). On one-year follow-up, no recurrence in lesion hypertrophy, nodularity or colour darkening was observed in patient one (Fig. 1a,b). No recurrence of nodularity or hypertrophy was detected in patient two on one-year follow-up, although mild darkening could be observed (Fig. 1c,d). On three-year follow-up, mild recurrence of hypertrophy and darkening of colour without nodularity was seen in patient three (Fig. 2). All three patients were extremely satisfied with the clinical outcome.

Table 1 Patient demographics and lesion characteristics

Patient	Age	Gender	Distribution	Previous Tx	CM Morphology			
						Hypertrophy	Nodules	Colour
1	66	M	Left V1/V2/V3 distribution	Argon and IPL	Before	Moderate	Nil	Dusky red – violaceous
					After	Nil	Nil	Normal skin colour – pale salmon
2	63	M	left v1 distribution	Candela V beam	Before	Severe	Moderate	Violaceous
					After	Nil	Nil	Dusky red
3	74	M	Left V1/V2 distribution	PDL and surgical excision of nodules	Before	Severe	Severe	violaceous
					After	Nil	Mild	Pink

CM, capillary malformation; Tx, treatment.

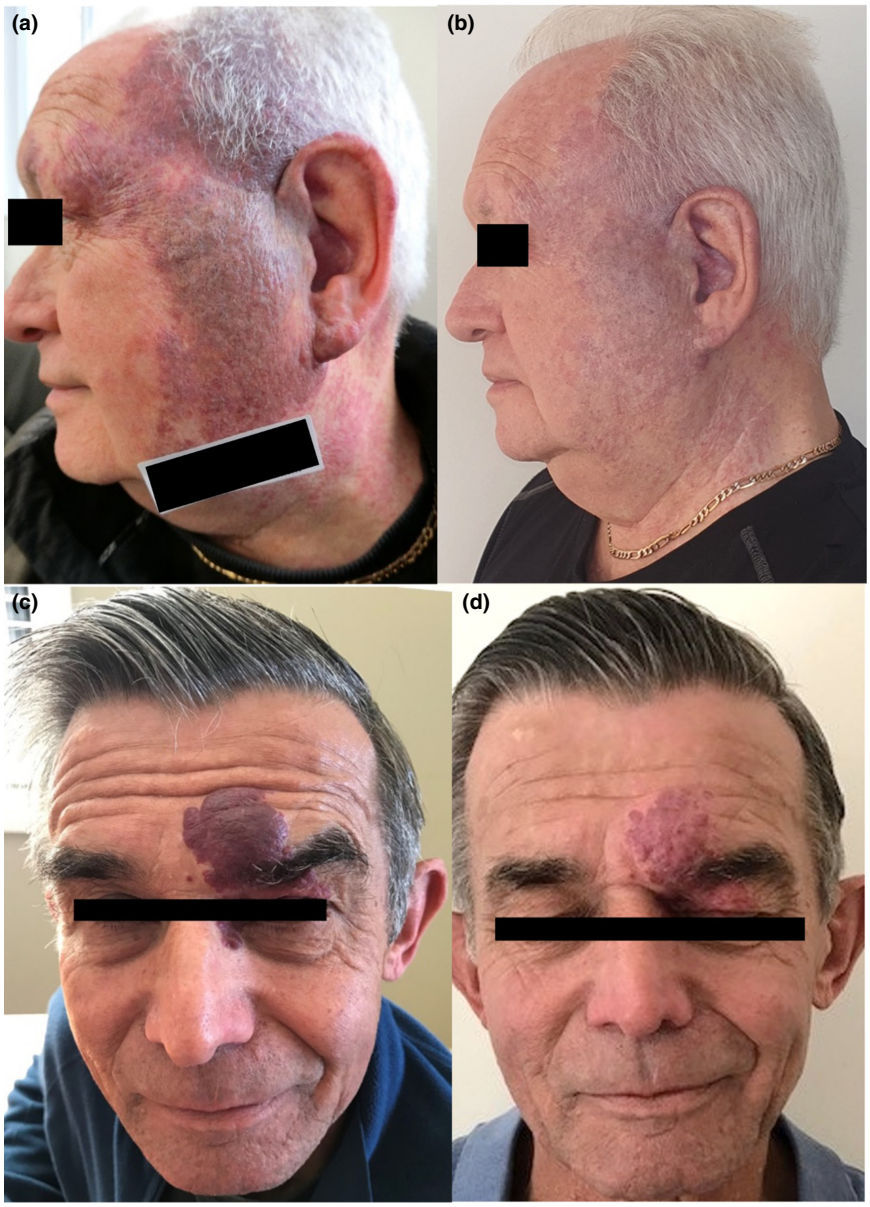

Figure 1 Patient 1 – (a) Before treatment; note the extensive V1-3 involvement with mild hypertrophy and violaceous colour. (b) Photograph taken on one-year follow-up. Note significant lightening in colour as well as reduction in hypertrophy that is maintained without recurrence on one-year follow-up. Patient 2 – (c) before treatment; note the hypertrophic and violaceous CM involving V1 dermatome. (d) Photograph taken on one-year follow-up, complete resolution of nodularity, significant reduction in lesion hypertrophy and lightening in colour were achieved and maintained with no recurrence.

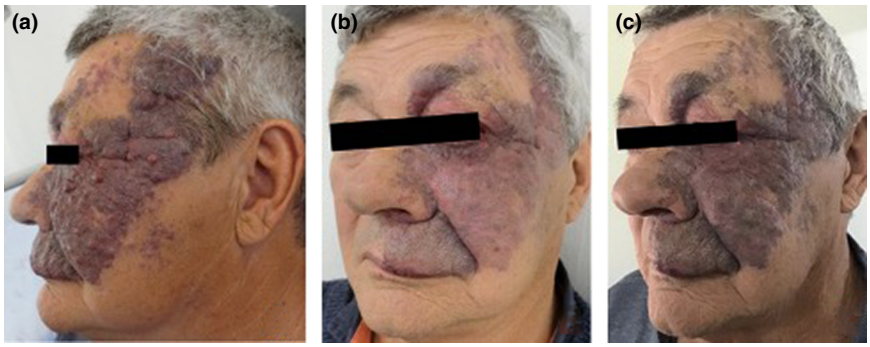

Figure 2 Patient 3 – (a) Before treatment, note the severe hypertrophy and nodularity involving V1-2 dermatomes. (b) Photograph taken 6 months after commencement of treatment course. Complete resolution of nodularity and reduction in hypertrophy as well as lightening in colour was achieved . (c) Photograph taken on three-year follow-up; note mild recurrence in hypertrophy and colour darkening but no recurrence in nodularity.

DISCUSSION

HFCMs remain a therapeutic challenge, frequently failing conventional vascular laser therapy.[12] Here, we describe successful treatment of hypertrophic and nodular CMs using our novel technique of TAS.

Considering the higher potency of STS compared with POL, the sclerosants were carefully selected depending on the size, anatomical depth and thickness of the lesion. STS foam 1.5% was selected for the treatment of larger nodular lesions as high potency sclerosant was required to achieve destruction of the entire lesion *via* cell lysis. Use of POL foam 0.5% on the other hand would have result in piecemeal lysis of the endothelial lining of the target vessels with a much larger risk of recurrence.[13,14] We were careful to use good quality foam, with microbubbles and a stable half-life. Embolisation of foam-derived macrobubbles may result in irreversible neurological adverse events.[15,16] Although CO2 has the advantage of being more soluble in blood due to its physiologic properties, it also produces a less stable foam, resulting in swift foam degradation and ultimately a reduced therapeutic effect. Given the peripheral nature of the malformation as well as the adjunctive use of tumescent anaesthesia, we preferred the use of physician-compounded foams using room air with improved foam stability and predictable efficacy.[8,17]

TAS involves the use of perilesional injection of TA to achieve compression of the target lesion and normal draining veins, thereby avoiding local or systemic dissemination of foam-derived bubbles. This technique allows the use of higher volumes of foam in highly sensitive areas with a fair degree of safety and certainty. We do not recommend such high volumes of foam without the adjunct use of TA and careful ultrasound guidance. In addition, TA provided anaesthesia and haemostasis eliminating the need for sedation.

Comprehensive ultrasound assessment of lesion characteristics and surrounding anatomy to guide treatment was paramount in optimising efficacy and avoiding significant complications such as inadvertent intra-arterial injections. Facial CMs commonly involve the V1 and V2 dermatomes and periorbital involvement is common. Though rare, there are multiple reports of orbital infarction syndrome, cavernous sinus thrombosis and ophthalmic artery occlusion leading to monocular blindness from STS sclerotherapy of periorbital veins or VMs.[18,19]

Historically, alcohol embolisation has been used for the treatment of facial vascular malformations with higher rates of complications.[20] We avoided the use of alcohol given its complication profile and potential for facial nerve damage.

Vascular lasers have been disappointing in the treatment of HFCMs. Here, we used yellow vascular laser as an adjunct to TAS to treat the flat lesional surfaces. An important utility of laser was to treat the CMs involving eyelids with a good outcome.

Two of the three patients reported here initially presented with a provisional diagnosis of 'haemangioma'. Less commonly, the nodular lesions are misdiagnosed as AVMs. While we report an effective and safe method to treat HFCMs, it is important to note that treating facial vascular anomalies requires an accurate diagnosis, comprehensive knowledge of facial anatomy and extensive training in vascular interventional techniques, especially of the head and neck region.

The limitations of this case series include the small sample size and the qualitative nature of the report. Larger studies are warranted to further consolidate these findings.

In summary, we report successful treatment of three patients with HFCMs using TAS. Though promising, the technique requires adequate training to be performed safely.

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Supporting Information

Additional Supporting Information may be found online in Supporting Information:

Table S1 Summary of treatment data. No., number; POL, polidocanol; STS, sodium tetradecyl sulfate; TA, tumescent anaesthesia; TAS, tumescent-assisted sclerotherapy; Tx, treatment.