Topic: Overview of Complications of Sclerotherapy

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Time: 1545-1600

Speaker:

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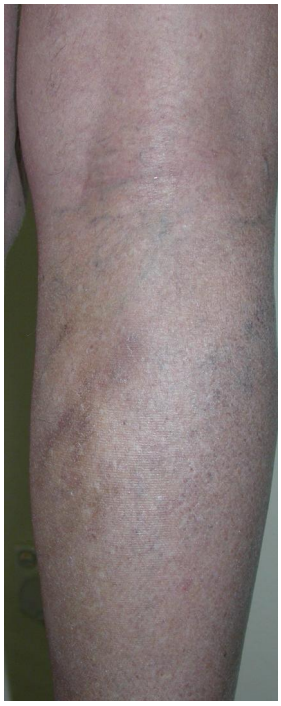
The experienced Phlebologist should be able to promptly identify sclerotherapy complications and commence appropriate treatment immediately. Most sclerotherapy complications may be classed as minor and readily manageable without significant sequelae. Though a catastrophic event is unlikely, all treatments should be performed in a clinic properly equipped with acute resuscitative equipment. See the college for guidelines on clinic set-up.

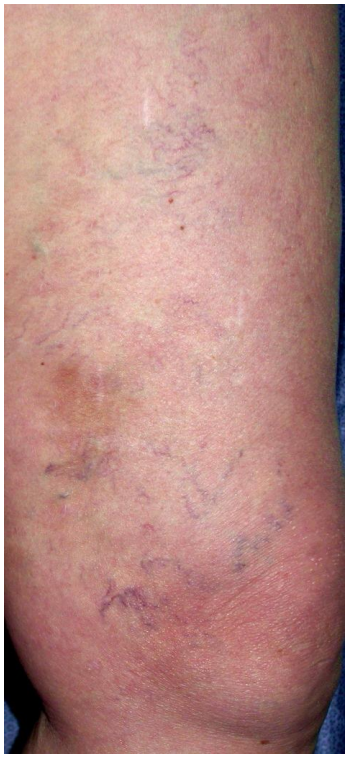
Important references:
(Guex, Allaert, Gillet, & Chleir, 2005)

Transient Dermal Pigmentation



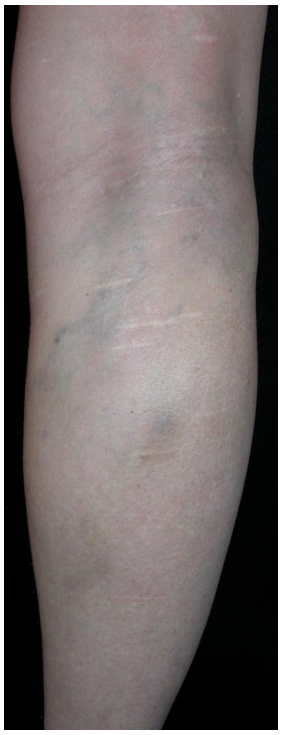
- Incidence: 1 in 4-20 (Weiss & Weiss, 1990).
- Grade/Severity: minor but may persist for up to 12 months after a treatment session.
- Aetiology/Predisposing Factors:
    - Operator Dependent: more likely if high sclerosant concentrations in superficial and dermal veins.
    - Patient Dependant: more likely if patient has high FP classifications (FPC III to V)
    - Drug Dependant: HT Saline then STS more likely to produce pigmentation than Aethoxysclerol.
    - Unknown/Undiscovered: ???
- Prevention: eliminate predisposing factors and treat proximal disease first.
- Diagnosis:
    - History: gradual onset following treatment session. Peak usually at 4-6 weeks.
    - Physical Examination: non-blanching, non-tender, non-raised brown discolouration.
    - Further Investigations: CW doppler for inadequate treatment of incompetent reticular veins.
    - Exclude: Trapped blood, heamosiderrin staining, tetracycline induced.
- Mechanism:
    - Pathophysiology: inflammatory mediated, increased melanin production by melanocytes. Heamosiderrin and RBC degradation products produced by sclerosed vessels.
    - Anatomy:
    - Histology: Increased melanin granules within dermis and persistent heamosiderrin with in the dermis and hypodermis.
    - Hydrodynamics:
- Treatment:
    - Acute: NIL
    - Long Term Management: Avoid extreme UV exposure which may cause exacerbation or permanence.
- Prognosis: Good

Permanent Dermal Pigmentation



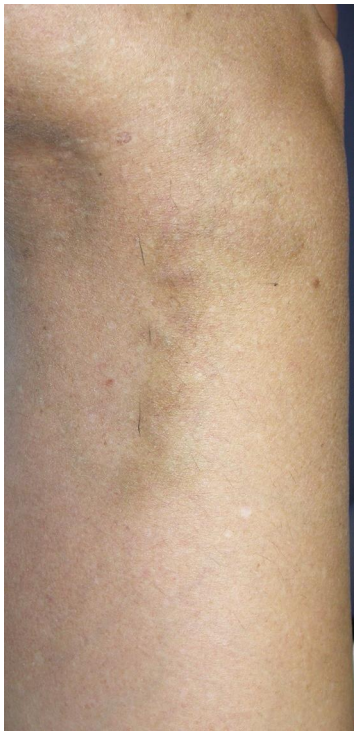
- Incidence: 1 in 80-120 (Georgiev, 1993; Tafazzoli, Rostan, & Goldman, 2000; Thibault & Wlodarczyk, 1992; Weiss & Weiss, 1990)
- Grade/Severity: moderate
- Aetiology/Predisposing Factors:
    - Operator Dependent:
    - Drug Dependant: ?HT Saline.
    - Patient Dependent: Iron Supplementation, Oral Tetracycline
    - Unknown/Undiscovered: ???
- Prevention: Avoid predisposing factors.
- Diagnosis:
    - History: pigmentation >12 months duration.
    - Physical Examination: as with transient dermal pigmentation.
    - Further Investigations: Skin biopsy
- Mechanism:
    - Pathophysiology: inability of to resorb heamosiderrin and iron from dermis.
    - Anatomy
    - Histology: granules in dermis.
    - Hydrodynamics
- Treatment:
    - Acute:
    - Long Term Management: Q switched ruby laser
- Prognosis: Poor

Telangiectatic Matting



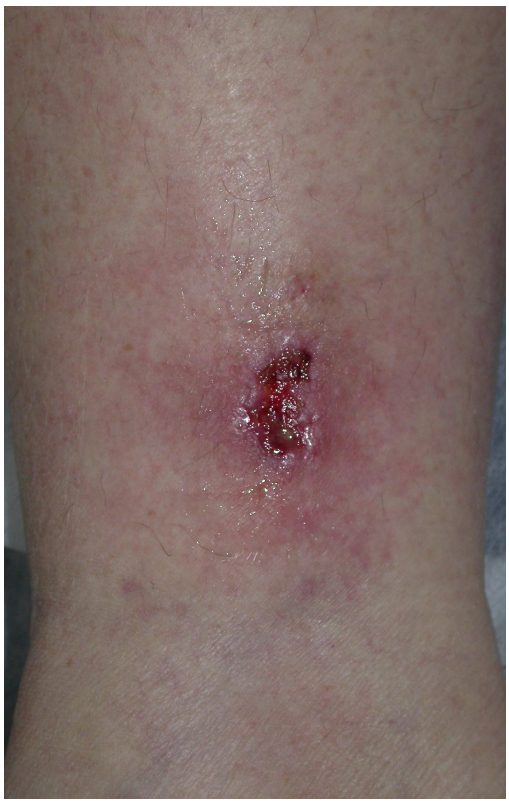
- Incidence: 1 in 10-30 (Davis & Duffy, 1990)
- Grade/Severity: mild-moderate
- Aetiology/Predisposing Factors:
    - Operator Dependent: high concentration, high volume, high hydrostatic injection pressure
    - Drug Dependant: related to drug sclerosing power.
    - Patient Dependent: Transparent skin, females and high oestrogen more likely, untreated proximal disease – persistent reflux.
    - Unknown/Undiscovered: ???
- Prevention: avoid predisposing factors.
- Diagnosis:
    - History: patchy pigmentation, onset 4-6 weeks post treatment.
    - Physical Examination: Blanching, visible telangiectatic matting.
    - Further Investigations: CW to identify or exclude underlying incompetent reticular veins.
- Mechanism:
    - Pathophysiology: Chemical induced angioneogenisis.
    - Anatomy
    - Histology: Diffuse thin walled vessels at the with in the superficial most layers of the dermis.
    - Hydrodynamics
- Treatment:
    - Acute: Nil
    - Long Term Management: identify and treat proximal disease, treat remaining telangiectatic matting with sclerotherapy.
- Prognosis: Good if retreated.

Trapped Blood



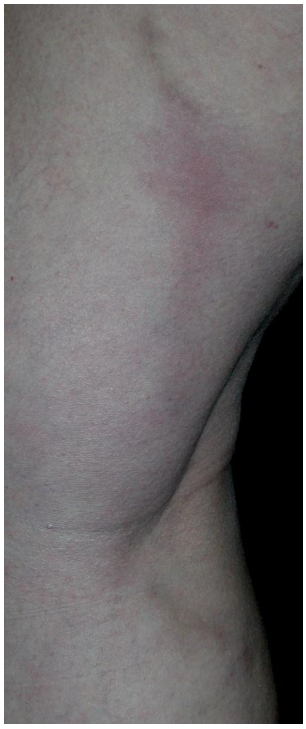
- Incidence: 1 in 3-5 (Kern, 2002)
- Grade/Severity: mild
- Aetiology/Predisposing Factors:
    - Operator Dependent: ?
    - Drug Dependant: ?
    - Patient Dependent: ?
    - Unknown/Undiscovered: ?
- Prevention: ?
- Diagnosis:
    - History: tender lump, onset 1 to 6 weeks post-sclerotherapy.
    - Physical Examination: tender, non-blanching, brown discolouration.
    - Further Investigations: CW Doppler used to confirm abolition of reflux, evacuation of liquefied thrombus.
- Mechanism:
    - Pathophysiology: liquefied thrombus trapped between two treated ends of vessel. Intralesional pressure increases due to increased volume of blood or reduced volume of untreated segment of vessel.
    - Anatomy: image of treated ends.
    - Histology:
    - Hydrodynamics:
- Treatment:
    - Acute: evacuation using 19G needle.
    - Long Term Management: repeat evacuation up to 6 months after treatment.
- Prognosis: Excellent.

Dermal Necrosis/Ulceration



- Incidence: 1 in 100-500 (Bihari & Magyar, 2001; , "Consensus paper on venous leg ulcer. The Alexander House Group", 1992; Kiehlmann & Lechner, 1999; Masuda et al., 2006)
- Grade/Severity: mild to catastrophic.
- Aetiology/Predisposing Factors:
    - Operator Dependent: inadvertent intra-arterial or arteriolar injection (microsclerotherapy). Excessive injection pressure into the superficial veins may cause retrograde flow of sclerosant into the arterial capillary vasculature
    - Drug Dependant:
    - Patient Dependent: more likely in smokers, vasculitis (Henoch-Schonlein, Erythema nodosum, Polyarteritis nodosa, Temporal (giant cell) arteritis, Takayasu's arteritis, Wegener's granulomatosis.)
    - Unknown/Undiscovered: ???
- Prevention: Exclude risk factors such as vasculitis and cease smoking.
- Diagnosis:
    - History: onset of intense pain with in 6 to 24 hours after the ischaemic event. Skin is pale and discoloured.
    - Physical Examination: initially dermis is pale with blue-grey dusky appearance. Dermal sloughing occurs within 24 to 72 hours after the ischaemic event. Moderately tender ulcer crater develops quickly.
    - Further Investigations: Check for underlying vasculitis, duplex doppler to check for arterial wall thickness and abnormalities.
- Mechanism:
    - Pathophysiology: Ischaemic necrosis resulting from complete occlusion of arterial component of capillary vascular bed. Unrecognised arterio-venous shunts allow sclerosant to enter the arterial circulation. Compare this to occlusion of non end-artery or end-arteriole. Sclerosant may also enter the arterial capillary vasculature via retrograde flow due to excessive injection pressure.
    - Anatomy: diagram of anastomosis.
    - Histology:
    - Hydrodynamics:
- Treatment:
    - Acute: Supportive, compression, occlusive dressings
    - Long Term Management: dermal scars may need further treatment.
- Prognosis: Very good when extent of necrosis is minimal.

Superficial Thrombophlebitis



- Incidence: 1 in 30 to 90 (Coleridge Smith, 2005; Sadick, 1991)
- Grade/Severity: mild to moderate
- Aetiology/Predisposing Factors:
    - Operator Dependent: high sclerosant concentration, high injection pressure and increased contact time.
    - Drug Dependant: relates to sclerosant power.
    - Patient Dependent:
    - Unknown/Undiscovered: ???
- Prevention: Post-Sclerotherapy compression therapy.
- Diagnosis:
    - History: gradual onset of tender red track approximately 2-6 weeks after sclerotherapy.
    - Physical Examination: tender, indurated, erythematous longitudinal lesion which follows the course of the saphenous trunk.
    - Further Investigations: Duplex doppler examination is essential to establish the diagnosis and to exclude a concomitant or subsequent DVT.
- Mechanism:
    - Pathophysiology: Chemical injury causing an inflammatory reaction within the segment of saphenous vein that has been treated. Intraluminal thrombus and causes complete occlusion of the vessel but may also demonstrate antegrade progression. There is perivascular oedema and this can be readily identified on B-mode ultrasound.
    - Anatomy:
    - Histology: see diagram
    - Hydrodynamics
- Treatment:
    - Acute: compression, ambulation, anti-inflammatory medication
    - Long Term Management:
- Prognosis: Excellent

Deep Vein Thrombosis

- Incidence: 1 in 500-1000 treatments
- Grade/Severity: mild to severe
- Aetiology/Predisposing Factors:
    - Operator Dependent:
    - Procedure Dependent: prolonged procedure time, perforator vein treatment, high injection volumes, treatment of distal leg varices.
    - Drug Dependant: Aethoxysclerol may produce greater effect at distant sites, sinus thrombosis risk with periocular inections.
    - Patient Dependent: Gene mutations, thrombophilia, previous history of DVT, immobilisation, smoking, hyperhomosysteinaemia, long haul flights, dehydration.
    - Unknown/Undiscovered: ???
- Prevention: graded compression, exercise, anticoagulation.
- Diagnosis:
    - History: sudden onset of calf pain and/or ankle swelling, pain when walking.
    - Physical Examination: tenderness to palpation, positive Homan's sign.
    - Further Investigations: Duplex ultrasound examination is essential to establish the diagnosis and assess progression of the disease.
- Mechanism:
    - Pathophysiology: Virchow's triad
    - Anatomy: Axial veins and crural veins
    - Histology:
    - Hydrodynamics:
- Treatment:
    - Acute: Assessment, compression, anticoagulation
    - Long Term Management: CVI, CVH, recurrence, prophyllaxis
- Prognosis: Good to very poor

Other Dermal Complications

- **Hirsutism**
- **Contusion**

Deep Venous Injury

- **Deep Venous Thrombosis**

Lymphatic Injury

- **Lymphocoele – Phlebectomy Only**

Pulmonary Injury

- **Embolism- Air/Thrombotic**
- **Sclerosis**

Arterial Injury

- **Ischaemia**

Allergic (Anaphylaxis)

CNS

- **Migraine (Hanisch, Muller, Krivokuca, & Winterholler, 2004)**
- **Thrombotic/Embolic (PFO)**
- **Hypersensitivity**

Peripheral Nerve Injury

- **Inflammation**
- **Scar**

APPENDIX:

GUEX ET AL: COMPLICATIONS OF SCLEROTHERAPY (pp126)
Dermatol Surg, 31:2:February 2005

Number of Complications according to Sclerosant Form

	Liquid	Foam	Both
Immediate			
Anaphylactic shock	0	0	0
Intra-arterial injections	0	0	0
Vasovagal fainting alone	4	6	0
Headaches alone	0	0	0
Paresthesias alone	2	1	0
Nausea and vomiting alone	1	0	0
Visual disturbances alone	4	8	0
Visual disturbance associated with 1 or more of headache, nausea, vasovagal fainting	0	8	0
Others	1	5	0
Delayed			
Deep venous thrombosis	0	1	0
Muscular venous thrombosis	0	1	0
Muscular venous extension	0	1	0
Perforating venous thrombosis	0	3	0
Intense superficial thrombophlebitis	0	3	0
Skin necrosis	0	0	0
Total	**12**	**37**	**0**

Number of Sessions, Sclerosing Agent, and Type of Injected Varicose Vein

Type of Varicose Vein	Number of Sessions with Liquid	Number of Sessions with Foam	Number of Sessions with Both
Reticular and spider veins	3,631	2,293	40
Great saphenous vein trunk or junction	261	1,533	130
Small saphenous vein trunk or junction	109	492	4
Main tributaries	422	714	34
Small varices or nonsaphenous	717	332	37
Perforating veins	77	199	2
Postsurgical recurrences	217	832	97
Total	**5,434**	**6,395**	**344**

Australian TGA GUIDELINES

- Aethoxysklerol maximum dosage: 2mg/kg body weight per day or 5 mls of 3% solutions (equals 3gm per 100ml)
- Sodium Tetradecyl Sulphate maximum dosage: 4 mls of 3% or 10mls of 1%

Inflammation in Vascular system	Symptoms	Vasculitis
Small veins	Purpura - large purple circles on the skin.	Henoch-Schonlein.
Deep layers	Deep, painful red bumps on the arms and legs.	Erythema nodosum.
Medium - sized arteries	Kidney failure, heart complications, gastrointestinal problems and high blood pressure.	Polyarteritis nodosa.
Inflammation of arteries in the brain and head.	Severe headaches, blindness, and stroke	Temporal (giant cell) arteritis.
Inflammation of the large arteries around the heart.	Fever and night sweats, heart attack.	Takayasu's arteritis.
Blood vessels of respiratory tract.	Coughing, shortness of breath, nose bleeds, and ear infections	Wegener's granulomatosis.

RABE ET AL.: GUIDELINES FOR SCLEROTHERAPY OF VARICOSE VEINS. Dermatol Surg. 30:5:May 2004, pp688

Complications and Risks

If performed properly, sclerotherapy is an efficient treatment method with a low incidence of complications. Nevertheless, a series of adverse events may occur in the context of the therapy. These are, in particular:[14–17]

_ Allergic reaction;[18–20]
_ Skin necroses;[21]
_ Excessive sclerosing reaction (thrombophlebitis);
_ Pigmentation;[4,22–25]
_ Matting;[24]
_ Nerve damage;[16,26]
_ Scintillating scotomas;
_ Orthostatic collapse; and
_ Thromboembolism.[15]

6 Risk Management

6.1 ACP/NZCAM requires written informed patient consent before UGS.

6.2 The ACP/NZCAM approved sclerosants covered by this standard are

- Sodium tetradecylsulphate (STS)
- Polidocanol supplied as either Sclerovein™ or as Aethoxysclerol™

6.3 Duplex/Doppler Ultrasound equipment used in delivering this standard must include a high frequency linear array probe with colour flow and Doppler capabilities.

6.4 Under this standard, liquid and foam sclerosant formulations are approved within the maximum allowable limits (Refer **6.5, 6.6,** and **6.7** below).

6.5 Under the NZ Medicines Act, Foam is an unapproved use of an approved/or unapproved medicine (STS is the only approved sclerosant) but permitted when administered by a Registered Medical Practitioner provided full written informed consent has been given by the patient.

6.6 This standard restricts the use of STS in concentrations of 3% to 4 mls liquid per day (≡ .12g per patient per day).

6.7 Use of polidocanol must not exceed 2mg/kg body weight per treatment day.

6.8 The minimum resuscitation equipment required is:
Oxygen, intravenous fluids, adrenalin, blood pressure and cardiac monitor, pulse oximetry, defibrillator, and suction.

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