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SHORT REPORT

Reversible Neurological Deficit after Foam Sclerotherapy

K. Hartmann [a,c,*], **L. Harms** [b], **M. Simon** [c]

[a] *Hartmann & Partners Phlebology Practice, Zähringerstr. 14, 79108 Freiburg, Germany*
[b] *Charité Centre for Neurology, Neurosurgery and Psychiatry, 10117 Berlin, Germany*
[c] *M. Simon Dermatology Practice, Hauptstr. 131, 10827 Berlin, Germany*

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Abstract A 37-year-old male was treated with 9 ml of 3% polidocanol foam, and he immediately reported photopsiae lasting a few minutes, though without migraine. Two hours after sclerotherapy, the patient developed speech disturbance for a few minutes. A pathological examination revealed nothing except a patent foramen ovale (PFO). Given the contrast between the high prevalence of PFO in general population and the extremely low incidence of neurological deficits after foam sclerotherapy, these deficits may only arise due to as-yet-unknown aetiology.

Case Report

We performed a duplex ultrasound-guided foam sclerotherapy with 3% polidocanol and 9 ml foam volume in a 37-year-old male patient with recurrent varicosity of the left great saphenous vein and a truncal varicosity of the left small saphenous vein (both in stage C4EpAsPr). The foam was produced using DSS (double syringe system) method with a polidocanol-to-air ratio of 1:4. The foam was injected within 10 s into the varicosity of the left leg.

During injection, the patient was held supine on an electrically operated tilt table. Spreading of foam in the thigh and calf was guided by duplex sonography, and on reaching the saphenofemoral junction, the patient's position was changed for 2 min to 20° head-down position. Hereafter, the patient was again held for 3 min in a horizontal resting position.

Immediately following foam injection the patient reported photopsiae for a few minutes. Thereafter, he recovered and was able to follow the recommended movement programme. Class II thigh compression stocking was recommended throughout the day for a period of 2 weeks.

* Corresponding author. K. Hartmann, Member of the Advisory Board of the German Society of Phlebology (DGP) and the Consortium Sclerotherapy of the DGP, Hartmann & Partners Phlebology Practice, Zähringerstr. 14, 79108 Freiburg, Germany.
E-mail address: kahartmann@web.de (K. Hartmann).
URL: http://www.venenzentrum-freiburg.de

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Two hours after sclerotherapy the patient developed speech disturbance for 5–7 min. There were no other signs or symptoms. He had no history of other significant illness.

Neurological examination, cranial magnetic resonance (MR scan), electrocardiography (ECG) and thrombophilia findings were unremarkable.

Multiplanar trans-oesophageal echocardiography (TEE) revealed a persistent foramen ovale.

Intravenous administration of a contrast agent that does not pass the pulmonary circulation resulted in four and 10 HITS (high-intensity transient signals) without and with the Valsalva manoeuvre, respectively. This indicates the presence of a slight right-to-left shunt during the Valsalva manoeuvre.

Discussion

The report by Forlee et al. on the occurrence of an ischaemic stroke following foam sclerotherapy[1] led to renewed debate about the safety of foam sclerotherapy. Neurological complications, including visual disturbances and migraine attacks, have been reported after foam sclerotherapy but are rare, occurring in 1% or less of patients.[2] The visual disturbances were described as ocular migraine and are said to be due to a vasospastic reaction to polidocanol rather than to the presence of air bubbles in the sclerosant, since they occur with both liquid and foam sclerosants.[3] It seems unlikely that an episode of vasospasm would cause a brief period of hypoperfusion of the speech centre only after considerable delay. The visual disturbances after foam sclerotherapy could, however, be a warning sign of potential neurological deficits. If a patient reports these disturbances, he/she should be asked to remain at the clinic for a while or the treating doctor should be contactable by the patient at all times. Hospitalisation is unnecessary as this condition occurs very rarely.

Air bubble embolism can also cause transient symptoms, as has been observed in divers after surfacing.[4] Because the pressure gradient between the right and left atrium is small in most patients with PFO, the air bubbles can pass into the left atrium only when the pressure in the right atrium is increased (e.g., as a result of a Valsalva manoeuvre). The actual effects of these air bubbles are not yet known. The delayed onset of neurological symptoms in our patient could be because, over a period of 2 h, the air bubbles might have enhanced the vasospasm induced by polidocanol and thereby caused a brief period of reduced arterial perfusion, resulting in the phonemic paraphasia. The air bubbles associated with foam sclerotherapy would thus be acting as a source of paradoxical embolism.

Further, a wash-out of coagulation products from the lower leg after sclerotherapy cannot be excluded, as Van der Plas states in his ''Letter to the editor'' in 1994, indicating that cellular debris or sludge following sclerotherapy could cause neurological deficits.[5] However, more questions arise: Does sclerotherapy result in the release of serotonin from lysed platelets causing a migraine-like episode? Could plasminogen activator inhibitor-1 (PAI-1) be involved?

Given the contrast between the high prevalence of PFO in general population (up to 30%)[6] and the extremely low incidence of neurological deficits[2] after thousands of foam sclerotherapy treatments daily worldwide, these deficits might arise only if another, yet unknown, factor is present.

In summary, transient visual disturbances and other neurological deficits such as phonemic paraphasia can occur after foam sclerotherapy. The reported deficits have disappeared relatively rapidly. Patients should be advised about the occurrences of such transient events. Awareness of these facts could obviate the need for expensive investigations at a later stage and spare the patient from unnecessary anticoagulant therapy. As reported by Bush et al.,[7] hyperbaric oxygen therapy, as used in diving accidents, may be beneficial in the event of a prolonged neurological deficit occurring after foam sclerotherapy. No definite cause of these very rare neurological deficits has been established to date; however, PFO should be regarded as a risk factor.

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