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Clinical dilemma of management: Cardiac arrest after microsclerotherapy for lower limb telangiectasia with liquid 0.3% aethoxysklerol or idiopathic cardiac arrest?

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Abstract

A 48-year-old woman attended to discuss a dilemma. She had suffered a cardiac arrest immediately following microsclerotherapy of leg telangiectasia with 0.3% aethoxysklerol. She had successful defibrillation and been transferred to hospital. In hospital, despite normal cardiac tests, she was diagnosed as having idiopathic cardiac arrest. The exposure to aethoxysklerol was discounted by her cardiologists as a cause of her arrest. Following the hospital protocol, she was strongly advised to have an implantable defibrillator. Cardiac arrest and myocardial infarction are documented after aethoxysklerol injection with proposed mechanisms being anaphylaxis, direct cardiotoxicity or endothelin-1 release. Before consenting to an implantable defibrillator, which may have its own complications in the long term, doctors and the patient need to be certain that this arrest was not due to a reaction to aethoxysklerol.

Keywords

Cardiac arrest, sclerotherapy, aethoxysklerol, anaphylaxis

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Introduction

Aethoxysklerol is one of two widely used detergent sclerosants by doctors treating telangiectasia and varicose veins. Injected directly into the target vein, its mechanism of action is to insert itself into the predominantly phospholipid cell membrane of the endothelial cells lining the venous wall.[1]

As concentration increases, the cell wall disrupts, killing the endothelial cell. This stimulates cellular inflammation and apoptosis in the adjacent media cells, resulting in immediate inflammation and then healing by fibrosis and occlusion.[2] Serious complications are rare.

Implantable cardiac devices (implantable defibrillators) can be lifesaving. However, like any medical device, they can malfunction, and as with any implantable foreign body, they can be associated with complications. Such malfunctions and complications occur in 4%–11% of patients in the early post-implantation period (i.e. haematoma, device malfunction and lead problems) and in the longer term, 10 per 100 patient years have been reported in a registry of patients over 65 years old followed for a median of 2.7 years.[3] Such longer-term problems include failure to shock, inappropriate shocks and infections. Hence, although risks are relatively low and are acceptable if the indication for an implantable defibrillator is strong, they become more of a concern if there is only a very weak case for recommending one in the first place.

This case has been reported as it leads to a dilemma of future management, depending on whether the cardiac arrest was due to a reaction to aethoxysklerol or was an unrelated factor.

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Case report

A 48-year-old female presented seeking advice, having had a cardiac arrest immediately following microsclerotherapy for lower limb telangiectasia performed elsewhere. The patient had undergone microsclerotherapy treatment of her lower limb telangiectasia (CEAP C1), on the lower leg front and back and behind her knee, with 0.3% aethoxysklerol, made up by diluting 2% lauromacrogol. She had not had a duplex ultrasound as this was not the practice of her aesthetic doctor at the time.

She had suffered from an upper respiratory tract infection for the week preceding the microsclerotherapy but was otherwise well. Her only known allergy was to sodium lauryl sulphate. She had never had any previous cardiac history. The patient had had the same treatment annually for approximately the last decade. Subsequently, she reported that after previous treatments she had often felt that she had a 'heavy feeling' in her chest and blackspots before her eyes, sometimes accompanied by a headache.

On this occasion, she felt strange approximately 15 min after the start of the injections. She was prone with chin resting on her hands. After standing for 1–3 min, she felt dizzy, and a feeling of 'heat rushing up her body from her legs to her chest and neck'. She felt faint and collapsed. She was found to be in cardiac arrest and cardiopulmonary resuscitation (CPR) was commenced. She underwent immediate defibrillation (one shock) with an automatic defibrillation device that was within the clinic.

An ambulance attended within 3 min and the crew confirmed ventricular fibrillation. They performed one defibrillation putting the patient into atrial fibrillation and then one more shock, into sinus rhythm. They then took her to hospital. On arrival at the hospital, photographs taken by her friend showed that her face was swollen, her eyelids were puffy and a red rash extended down her neck. The swelling resolved quite quickly in hospital, but the rash remained for several hours. She was given morphine which made her vomit violently.

She was admitted under the cardiologists and underwent cardiac magnetic resonance imaging (MRI), coronary angiography, chest x-ray, 24 h electrocardiogram (ECG), cardiac stress and ajmaline tests. All were normal, and no patent foramen ovale found. No cardiac cause was found for her cardiac arrest.

In the patient's family, her mother gets anaphylaxis to iodine, shellfish and paracetamol. One sister is allergic to aspartame sweetener, developed eczema after ibuprofen and is gluten-sensitive, although a biopsy was negative for coeliac disease. Another sister has Hashimoto's thyroiditis and eczema.

The dilemma for this patient is that her cardiac team has diagnosed idiopathic cardiac arrest and is advising that following their protocol, she needs an implantable defibrillator. However, the patient is understandably concerned that if her arrest is related to the injection of aethoxysklerol, she would be accepting a lifelong implant with all the risks associated with that, for no advantage.

Discussion

Sclerotherapy is a recognised treatment for unwanted leg veins. Aethoxysklerol (aka polidocanol or Lauromacrogol 400) is a detergent sclerosant. It is chemically very similar to sodium lauryl sulphate and other surfactants used in soap, shampoos and other personal cleaning products. The patient was known to have an allergy to this surfactant.

Low concentrations of liquid solution 0.25% and 0.5% aethoxysklerol are used to treat telangiectasia and small reticular veins. Larger varicose veins or incompetent truncal veins are usually treated with higher concentrations between 1% and 3%, and often made into a foam with air or gas. The foam displaces blood, allowing interaction between sclerosant and vein wall.

Aethoxysklerol has been reported to have caused cardiac arrest and myocardial ischaemia previously,[4] with anaphylaxis or direct cardiac toxicity having been suggested. Another reported the death of a 35-year-old woman following varicose veins treatment with aethoxysklerol.[5] Post-mortem showed no sign of anaphylaxis but direct action of the sclerosant on the heart muscle was suggested as the cause of death.

Reports of cardiac arrest in children where high doses of aethoxysklerol were injected into venous malformations also seem to point towards a direct cardiotoxicity of the drug,[6,7] as does a report of a 48-year-old lady who underwent cardiac arrest following the injection of '7-mL foam polidocanol injection'.[8]

Polidocanol foam sclerotherapy has also been reported to cause myocardial infarction in a 78-year-old patient,[9] but myocardial infarction with foam sclerotherapy has been reported with foam made from both aethoxysklerol and sodium tetradecyl sulphate.[10,11]

Further cases have been reported of myocardial infarction after foam sclerotherapy with aethoxysklerol or sodium tetradecylsulphate.[9–11] The difficulty of assessing these latter cases as to whether they constitute a reaction to the sclerosant or the gas bubbles in the foam is the inconsistency of reporting which detergent was used and in what concentration, what gas was used to make the foam and the total dose injected into which veins.

A further possible mechanism of cardiac toxicity is the potential release of endothelin-1 from the action of the sclerosant on the endothelium. Endothelin-1 was suggested to be a cause of visual disturbance after foam sclerotherapy[12] and a study in rats showed endothelin-1 release with aethoxysklerol injection, although only with foam and not liquid sclerotherapy.[13]

This finding was confirmed in humans[14] and endothelin-1 release after foam sclerotherapy treatment was suggested as the cause of a non-ST elevation myocardial infarction in one case, although this was sodium tetradecyl sulphate foam rather than aethoxysklerol.[15]

Conclusion

In conclusion, sclerotherapy with aethoxysklerol can cause cardiac arrest due to anaphylaxis, direct cardiotoxicity or maybe endothelin-1 release. In the presented case, a very low concentration and volume of aethoxysklerol was used. In addition, the patient had a swollen face and red rash on admission and a family history of allergy, suggesting anaphylaxis is the most likely mechanism for her cardiac arrest. The previous history of heaviness in the chest, blackspots in front of the eyes and occasional headache after previous treatments raise the possibility of sensitivity to endothelin-1 release, although the two mechanisms might not be mutually exclusive. Direct cardiotoxicity is possible but less likely.

However, with regards to the patient dilemma, there would appear to be relatively good evidence to suspect a direct causal relationship between the intravenous aethoxysklerol injections and subsequent cardiac arrest. As such, it is difficult for her to accept an implantable defibrillator, with the inherent risks of a long-term device implanted within her body, with the sole justification being that it is the protocol to do so.

Author contributions

Conception and design – M.S.W. Analysis and interpretation – not relevant. Data collection – L.K.T., J.C.K., B.E.H. Writing the manuscript – M.S.W. Critical revision of the manuscript – M.S.W., L.K.T., J.C.K., B.E.H. Statistical analysis – not relevant. Obtaining funding – not relevant.

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Guarantor

Mark S Whiteley

Ethical approval

Patient has given her written consent for reporting her case.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymised information to be published in this article.

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