# Facing complications

*Dr Patrick Treacy discusses dermal filler complications and how to deal with them*

Soft tissue augmentation with temporary dermal fillers is a fast-expanding field that has become an integral part of many aesthetic practices. According to the American Academy of Aesthetic Plastic Surgeons, 2,448,716 people received hyaluronic acid (HA) injections from plastic surgeons in 2013. These filler products are mostly safe, having a known incidence of mild and transient adverse events, but serious complications can also occur causing gross biofilm reactions or vascular embolism causing severe skin necrosis or even blindness.

At present, there's a paucity of literature regarding both the prevention and management of serious events, despite the fact that these complications are the very things that patients and physicians both continually fear. Many physicians, (including myself), feel that corporate prefer not to address these issues and they are driven underground. Over the years, many of my colleagues have referred me their more serious problems and I consequently have developed a certain experience in this area. This insight into complications of dermal filler use will serve to highlight both of these problems and try to help one manage these complications if they should ever happen to you.

**The size of the problem**

Fortunately, most adverse reactions are mild and transient. Adverse events can be grouped into expected procedure-related events, such as bruising, erythema, and tenderness; events potentially related to improper technique, such as nodule formation; and reactions to the product, such as granuloma formation.

We had a serious problem in both the UK and Ireland some years ago, with 168 different fillers being used in this marketplace. In comparison, the USA had effectively just three or four, which had been passed by the Food and Drug Administration. Things are thankfully much better in Europe now, since most of the fillers that have survived the passage of time here, are ironically the exact same ones that were passed for clinical use in the USA. This also raises the question of whether we really require a similar vetting procedure regarding the safety of filler products in this jurisdiction.

**Specific problems**

Many minor side effects, like swelling, can easily be controlled by use of oral steroids. Bruising effects are mostly due to patients taking supplements including vitamin E or omega fish oils, aspirin, Lipitor or antidepressants prior to procedure.

Moderate problems of dermal fillers are usually due to delayed onset nodules resulting in granuloma formation, inflammation and immune response. More serious adverse reactions include biofilm and vascular embolism leading to skin necrosis or blindness.

**Why are these problems occurring?**

There are many contributing factors. The first is that a corporate is driving sales, and as a consequence, the amount of product being used is increasing. The second is the continual introduction of new compounds and pharmaceuticals into our dermal fillers. The hyaluronic acid fillers started off fairly simple structures, but in recent years the addition of butanediol diglycidyl ethers (BDDEs) altered the chains and now different HA dermal fillers exist for different positions in the face, and consequently we have seen quite an increase in problems. Through the years, my experience of these higher molecular weight hyaluronic acids (SubQ, Voluma, Macrolane) is that they have tended to be problematic in terms of delayed onset nodules. I have long advised that Radiesse should not be injected in the perioral area or the vermillion border of the lips and

question it's use in the periocular area when we have other safer compounds to use.

Bio-Alcamid has long been a problem compound and I have treated many patients with the desire to remove this innocuous material from their face. It almost became the gold standard for treating patients with HIV lipodystrophy—in the days before less problematic antiretroviral drugs, when physicians had only Sculptra as an alternative. Luckily enough for most of the HIV lipoatrophy patients that I've treated, it comes out quite easily, because you can nick it, but unfortunately it does form abscesses up to ten years later. I also noticed that many patients who removed this compound tend to stay aesthetically normal because it has formed a collagen type capsulation to occupy the area where it was placed.

**Lidocaine dangers**

I'm not an advocate of lidocaine being included in every filler. This was largely done with our permission, often to aid nurses rather than doctors, and I'm certainly not being territorial about this. In my mind, lidocaine is possibly a dangerous drug to be unquestionably adding to dermal filler, because it of its potential to cause vasodilation. By using it widely and without proper recourse to its effects, we're most probably setting up the arterial facial vessels like ducks in a row to be damaged by needles used by the unwary. I don't remember seeing this level of complications prior to the widespread use of lidocaine, so whilst I can't scientifically confirm it's to blame, it's definitely something we should now be considering. If we used lidocaine and adrenaline, the vessels would get smaller and they would be less easy to hit. For an ischemic event, you certainly don't want adrenaline on board, but adrenaline only has an effect for about eight minutes, whereas lidocaine has an effect certainly in the region of two to four hours.

**Biofilm**

I have had several patients present to me with biofilm in recent years and have been able to successfully reverse these patients with hyalase, dexamethasone and in one instance 5-fluorouracil within three to four weeks.

I have my own theories on the etiology of biofilm, which I'm fairly confident will be proven to be true. Primarily, I feel a lot of these biofilms are possibly mycobacteria. We like to think that the age of mycobacteria is long gone but it's not—these organisms are present in many different environments around us, including in our water supply. We know that these specialist micro-organisms tend to form colonies, if not sub-colonies and we also know that ordinary antibiotics are largely ineffective in treating them.

We know that when we take sterile cultures from biofilms, we usually grow nothing. However, if you can test for mycobacteria, quite a number of them are positive. You need to do a special PC test for mycobacteria and in the absence of this facility you'll probably miss it. Hence, I would advocate starting the patient on rifampicin and isoniazid as well as clindamycin. Biofilm is emerging more and more with the BDDE added to the HLAs, and as a consequence, these colonies actually migrate and form problems in other areas.

When dealing with a colony of biofilm, three things need to be dealt with: The HLA in the middle; the collagen capsule around it; and the bacterial colonisation. I suggest that as many as maybe 10-20% of them are mycobacterium. I advocate using Hyalase (Hyaluronidase) to deal with the hyaluronic acid, dexamethasone to deal with the collagen, and antibiotics to deal with the infective commensals colonising the structure. One can't be done without the other and if you give intra-lesional steroids alone, you're probably creating a problem because you're reducing the patient's local immunity, and the infection will remain.

**What is the exact mechanism of vascular problems?**

While it is widely accepted that serious vascular compromise is caused by embolisation of filler material into part of the facial

artery, I have dealt with some cases where the patient showed no clinical evidence of vascular compromise during injection, but presented some hours later with marked reticulation. I surmise that these patients may have sustained some venous compression due to hyaluronic acid swelling and considers the use of intravenous steroids in these patients.

Vein illumination with AccuVein vein visualisation technology is a new technology that may reduce vascular embolic events by simply holding the device above the skin, and the vessels below are displayed on the surface of the skin. It is easy to use, and these vein finders may save valuable time for practitioners less familiar with facial anatomy.

**Hose-pipe theory**

The hose-pipe theory hypothesises that all vascular problems with dermal fillers resulting in skin necrosis or blindness are due to direct embolisation of the facial or temporal arteries or their collateral branches. Many physicians state the venous part of the system is not involved, because if you place a finger on the arterial system it will keep pulsing and supplying tissues.

**Roundabout theory**

I advocate a slightly different vascular understanding, which for the sake of simplicity I shall call the roundabout theory. In my experience many patients present the next day, meaning they're possibly presenting as a consequence of venous compression rather than embolism. Imagine the capillaries going into a small pressure system and the veins exiting the other side—if you block the veins, then nothing can go in on the arterial side.

If we look at a small filler bolus, it's usually carried downstream by blood flow, and that's the classic embolism. That's the sort of thing that we see for example in the labial artery. There's no collateral circulation and if you damage the artery on the way in, then you're going to cause a problem.

**Reversal protocol for retinal vascular occlusion**

Whenever a large filler bolus embolises a vessel, the consequence is retrograde flow to the blood's normal direction, in a distal segment whenever the pressure is taken off the syringe. If you inject the temporal area, you can end up with an obvious area of blanching, or at worse retinal vascular occlusion, and as a consequence unilateral blindness occurring within a short time. Are you confident you know what to do if this happens to you?

First, don't panic. You have to obviously discontinue the injection if it's happening in front of your eyes. The first evidence of necrosis is pain and this happens even before blanching. Next massage the area and use heat packs on the area both as a means of dispersal and vasodilation. It's most important to understand exactly why it has happened and in my mind to establish whether it's arterial or venous. Is it reversible or not? If it's hyaluronic acid, you have to use Hyalase (Hyaluronidase).

I do this slightly differently to other physicians and use much higher doses than noted in the literature. I mix Hyalase (Hyaluronidase) with lidocaine because it burns like an acid. If you add lidocaine—*and notice I'm saying lidocaine, not lidocaine-adrenaline*—lidocaine will widen the embolised vessels. With the Hyalase—there are 1,500 units in the ampoule and I add 1ml of bacteriostatic saline, because it's buffered so it's not going to hurt the patient. I then draw out 0.2 ml—that's 300 units of Hyalase (Hyaluronidase). I then mix the same amount with 2% lidocaine, and each 0.1ml of that is a nice controllable 75 units. Use as much as you have to, to save the patient's face and inject in many different areas along the area of reticulation. Use up to 750 units twice daily or 375 units four times daily for smaller problems. I would even advocate the use of retro-orbital or even intra-orbital use of Hyalase (Hyaluronidase) in the case of impending blindness.

**Venous problems**

If it is considered venous, my way of treating the problem is also very different. In a case presenting the next day, I give 10 mg

of dexamethasone IV stat as a bolus. This will be unproblematic because it will reduce the gross inflammatory response secondary to necrosis, even if it's arterial. Note also that hyaluronic acid, absorbs water when it goes into the skin, and consequently increases in size by three to four times, so you're going to reduce that that oedema also.

**Using vasodilatories**

Everybody says to use topical nitrates, but in my experience there are issues with this. Firstly, you can't use them during the night time because the patient's asleep and it requires hourly application; plus they're very messy. I advocate the use of other vasodilatory nitrates which are more long lasting and easier to find, especially Viagra, Cialis and Levitra. This may sound unconventional but I consider any sort of drugs that we use for erectile dysfunction are perfect for this. They're going to cause vasodilation exactly the same as the other nitrates by increasing GMP, smooth muscle action. As a consequence, we get vasodilation.

**Summary**

To summarise the reversal protocol: discontinue immediately, massage the affected area, apply warm packs, bolus the dexamethasone, mix the Hyalase (Hyaluronidase)—don't be afraid to use it—and consider the use of topical nitrates or Viagra. However, care should be taken when using the two together, as an older patient may be a cardiopath with pathology on board. They can also cause a syncopal episode, and I certainly know one patient that was admitted to hospital some years ago as a consequence of getting topical nitropaste given liberally during a vascular event.

If you're a doctor who uses fillers, I would consider it essential to not only carry Hyalase (Hyaluronidase) in your fridge and be very familiar with your reversal protocol, but to be aware of which of your colleagues you could approach for assistance, should the need arise. It could save your patient's face.

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