

review

Transarterial embolization of the external carotid artery in the treatment of life-threatening haemorrhage following blunt maxillofacial trauma

Crt Langel¹, Dimitrij Lovric¹, Ursa Zabret¹, Tomislav Mirkovic², Primoz Gradisek², Anita Mrvar-Brecko², Katarina Surlan Popovic¹

¹ Institute of Radiology, University Medical Centre Ljubljana, Ljubljana, Slovenia

² Department of Anaesthesiology and Surgical Intensive Care, Division of Surgery, University Medical Centre Ljubljana, Ljubljana, Slovenia

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Correspondence to: Assoc. Prof. Katarina Šurlan Popović, M.D., Ph.D., Clinical Institute of Radiology, University Medical Centre Ljubljana, Zaloška c 7, 1000 Ljubljana, Slovenia. E-mail: katarina.surlan-popovic@mf.uni-lj.si.

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Background. Severe bleeding after blunt maxillofacial trauma is a rare but life-threatening event. Non-responders to conventional treatment options with surgically inaccessible bleeding points can be treated by transarterial embolization (TAE) of the external carotid artery (ECA) or its branches. Case series on such embolizations are small; considering the relatively high incidence of maxillofacial trauma, the ECA TAE procedure has been hypothesized either underused or underreported. In addition, the literature on the ECA TAE using novel non-adhesive liquid embolization agents is remarkably scarce.

Patients and methods. PubMed review was performed to identify the ECA TAE literature in the context of blunt maxillofacial trauma. If available, the location of the ECA injury, the location of embolization, the chosen embolization agent, and efficacy and safety of the TAE were noted for each case. Survival prognostic factors were also reviewed. Additionally, we present an illustrative TAE case using a precipitating hydrophobic injectable liquid (PHIL) to safely and effectively control a massive bleeding originating bilaterally in the ECA territories.

Results and conclusions. Based on a review of 205 cases, the efficacy of TAE was 79.4–100%, while the rate of major complications was about 2–4%. Successful TAE haemostasis, Glasgow Coma Scale score ≥ 8 at presentation, injury severity score ≤ 32 , shock index ≤ 1.1 before TAE and ≤ 0.8 after TAE were significantly correlated with higher survival rate. PHIL allowed for fast yet punctilious application, thus saving invaluable time in life-threatening situations while simultaneously diminishing the possibility of inadvertent injection into the ECA-internal carotid artery (ICA) anastomoses.

Key words: blunt maxillofacial trauma; external carotid artery injury; intractable bleeding; non-adhesive liquid embolization agent; precipitating hydrophobic injectable liquid, neurointervention

Introduction

Maxillofacial trauma comprises roughly 10% of all trauma cases.¹ It is associated with a wide range of problems, including airway compromise, cervical spine injuries and bleeding.² Life-threatening haemorrhage secondary to blunt maxillofacial trauma is considered rare, occurring in 1.2%–4.5%

of trauma-related maxillofacial fracture cases.²⁻⁵ The most common origins of haemorrhage in maxillofacial trauma are the internal maxillary artery (IMA), the IMA's distal branches, and the main trunk of the external carotid artery (ECA).⁶

A diverse range of imaging manifestations can present in the setting of blunt carotid artery trauma, including various dissection subtypes (mini-

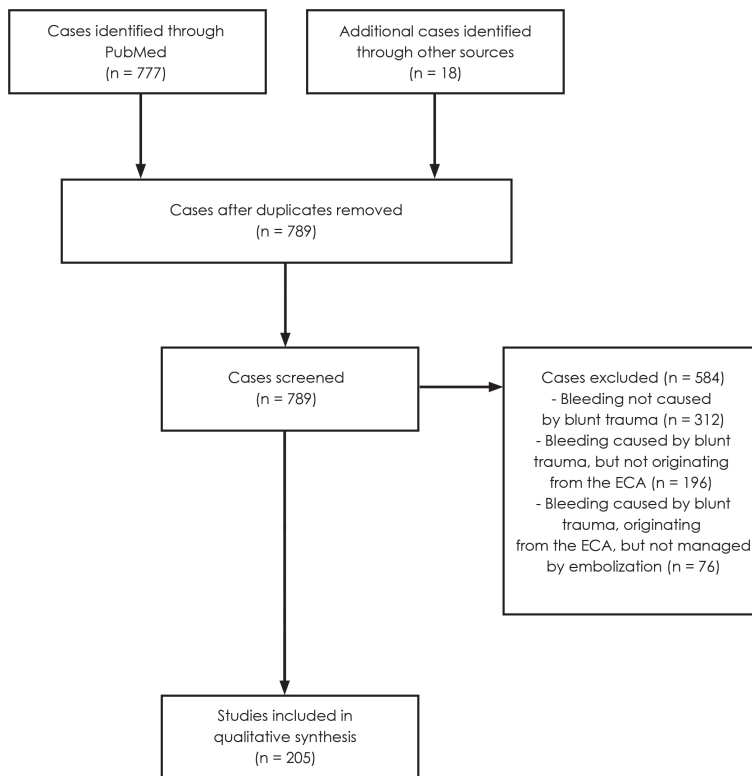


FIGURE 1. A flow diagram based on simplified Preferred Reporting Items for Systematic Reviews and MetaAnalyses (PRISMA) guidelines depicting the number of cases identified, included and excluded. The reasons for the exclusions are also noted.¹³

mal intimal injury, raised intimal flap, dissection with an intramural hematoma, occlusion), pseudoaneurysm, transection with an active haemorrhage, and arteriovenous fistula.^{7,8}

In treating severe maxillofacial injuries, airway, breathing and circulation management precede all other procedures. Special care is aimed towards the protection of the airway as the tongue and the soft tissues of the lower face can move backward and obstruct the pharynx due to the decreased level of consciousness, bilateral mandibular fractures, soft tissue swelling and expanding hematomas.² Further treatment includes manual compression, nasal packing, coagulopathy correction, cauterization, reduction of the fractures and local vascular control to stop the bleeding from the intra-osseous branches near the fracture lines.⁹ Open surgical ligation or transarterial embolization (TAE) of the ECA are available as the most definite options. Advantages of TAE over ligation include rapid access, not necessarily requiring general anaesthesia, superior haemorrhage origin localization using angiography, the ability to control multiple

local bleeding points, the ability to perform super-selective therapeutic vessel occlusion by cannulating the smaller vessel branches not amenable to open surgical repair, and short procedure time.¹⁰ In addition, ECA ligation oftentimes requires repeat ligation or subsequent TAE to effectively stop the bleeding, whereas TAE is usually efficacious in a single session.^{2,11} Furthermore, TAE offers the option to embolize the bleeding origins of a possible concomitant abdominal or other internal haemorrhage in the same session.¹² In certain guidelines, surgical ECA ligation has been completely replaced by TAE for maxillofacial bleeding control.²

The initial search data for the review part of this manuscript was processed following a simplified variant of the Preferred Reporting Items for Systematic Reviews and MetaAnalyses (PRISMA) guidelines showing the number of cases identified, included and excluded, plus the reasons for exclusions (Figure 1).¹³ The included cases were then analysed in order to obtain the data regarding the use of embolization agents, and the efficacy and safety of the TAE procedure in the context of blunt maxillofacial trauma with bleeding originating from the ECA.

Transarterial embolization of the ECA

Embolization therapy aims at controlling an active bleeding by occluding the feeding artery with an embolization agent (EA).¹⁴ The TAE technique was first suggested by Brooks *et al.* in the 1930s. In the early 1970s, Rosch and Dotter used it for the first time to treat a traumatic vascular injury.¹⁵ It also became an accepted treatment option for a variety of vascular lesions unrelated to trauma, including arteriovenous malformations, glomus tumours, juvenile angiofibromas, and intracranial meningiomas.¹⁶ A pioneer case describing a successful IMA TAE in the treatment of intractable epistaxis was reported by Sokoloff *et al.* in 1974.¹⁷

Decades of innovation brought about major advances in embolization materials, however, to this day, the principles of the TAE procedure remain largely unaltered.¹⁸ Initially, a vascular access is established by a standard transfemoral approach using the Seldinger technique.¹⁹ Alternatively, brachial or axillary arterial access might be required in case of severe bilateral lower extremity injury.²⁰ Diagnostic angiography of the whole circulation at risk is then performed, including bilateral common carotid arteries (CCAs), internal carotid arteries (ICAs), vertebral arteries (VAs), and ECAs. If the

preliminary computed tomography angiography (CTA) or clinical findings suggest an injury to a specific smaller vessel, e.g. the IMA, the lingual artery or the superficial temporal artery, further microcatheter angiography of these territories is carried out. The purpose is to obtain a general overview of the complete vasculature and to exclude a possible concomitant injury to the CCA, ICA or VA.²¹ The vessel that is the source of an active haemorrhage is then therapeutically occluded. The aim of the embolization is to stop the active bleeding, prevent any subsequent rebleeding events, and preserve as much perfusion to the nearby structures as possible, thereby reducing the chance of unnecessary tissue damage. This means embolization is attempted as close to the lesion as possible to avoid occluding the vessels branching off proximally to the embolization site. Circumstances permitting, the culprit vessel should ideally be embolized both distally and proximally to the lesion in order to prevent rebleeding via the collateral circulation.²¹ The delivery of the EA is performed under fluoroscopic visualization to the point of contrast medium stasis within the embolized vessel.

Embolization agents

Depending on the vessel calibre, type of vessel injury and other factors, a variety of EAs with differing inherent properties and behaviour may be used. Historically, the first EA was autologous tissue (including blood clots, subcutaneous tissue and muscle) followed by silk threads.²² Their use gradually declined with the advances in newer EAs, imaging and (micro)catheter technologies.^{14,23}

Data gathered in Table 1 indicates coils and gelatine foam (Gelfoam) are the EAs most frequently used for TAE in the context of blunt maxillofacial trauma. There have also been numerous instances of polyvinyl alcohol (PVA), microspheres, and N-butyl-2 cyanoacrylate (NBCA) use. One case of silastic spheres, two cases of Onyx, and a single case of precipitating hydrophobic injectable liquid (PHIL) use have been reported.

Coils are made from platinum or steel, measure 0.2–1.3 mm in diameter and can be supplied in a variety of lengths, shapes and levels of stiffness. They may be bare or fibered with materials such as wool, silk, nylon fibres, polyester, Dacron or PVA.¹⁸ Coils embolize a vessel by physically slowing down the local blood flow, by providing a thrombogenic locus, and by damaging the vessel wall, thus inducing the release of thrombogenic factors. Time to oc-

clusion is typically 5 minutes or less after coil insertion, depending on the type of coil used, the rate of blood flow through the target vessel and the blood's coagulation properties.¹⁸ Larger diameter platinum coils offer good radiopacity, while smaller diameter coils (microcoils) provide for more targeted distal deployment. The possible complications of TAE using coils are non-target vessel occlusion, vessel injury, coil migration, and infection.^{18,24} Coils prohibit any future endovascular access distal to the occlusion point, which is particularly relevant in rebleeding events following collateralization.²¹

Gelatine foam (Gelfoam) (Pfizer, Kalamazoo, MI, USA) is a porous material with haemostatic properties prepared from purified porcine skin gelatine. It usually supplied as a block of sponge that needs to be cut into smaller cube- or torpedo-shaped particles prior to embolization. Gelfoam induces foreign body reaction and necrotizing arteritis, resulting in the formation of a thrombus.²⁵ Gelfoam as a standalone EA provides a temporary vessel occlusion; recanalization typically occurs within 3 weeks to 3 months, but the exact time and the extent cannot reliably be predicted.²³ A combined coils-Gelfoam embolization may be particularly well suited for coagulopathic patients as such vessel occlusion is precise, fast, and permanent.²³ The most significant disadvantage of Gelfoam-only embolization is the reliance on manual preparation of the particles, limiting the reproducibility and predictability of the exact embolization site. Furthermore, air bubbles typically form in the Gelfoam-contrast mixture, presenting a potential risk for an aerobic infection.¹⁸

Polyvinyl alcohol (PVA) particles (Boston Scientific, Cork, Ireland; Cordis J&J Endovascular, Miami, FL, USA) are irregularly-shaped permanent embolic agents ranging from 100 to 1100 μm in size. The PVA's mechanism of action includes adherence to the vessel wall, induction of an inflammatory reaction, focal angioneclerosis and the resulting vessel fibrosis. There is a considerable variability in particle size because fragments smaller than the stated size range are allowed to enter the particulate mixture during production. This in turn increases the risk of distal, non-target embolization as particles tend to lodge in the smallest vessel they can fit in. On the other hand, the PVA particles are also prone to aggregation; this can lead to more proximal vessel occlusion than expected based on the stated size range of the particles.¹⁸

Microspheres (Embosphere and EmboGold, Merit Medical Systems, South Jordan, UT, USA; Contour SE, Boston Scientific, Natick, MA, USA;

TABLE 1. Data from studies, case series and case reports pertaining to TAE of the ECA or its branches in the treatment of haemorrhage caused by blunt maxillofacial trauma

| First author | Year Published | Case number | Vessel injured | Vessel embolized | Embolization agents used | efficacy (complete cessation of bleeding following TAE of the ECA or its branches) | complications |
|------------------------|----------------|-------------|--|---|--------------------------|--|---|
| Bynoe ² | 2003 | 1 | none identified | RL ECA | | 100% | partial tongue necrosis |
| | | 2 | R IMA | R IMA | | | none |
| | | 3 | R IMA | RL IMA | | | none |
| | | 4 | none identified | RL ECA above LA | | | none |
| | | 5 | none identified | RL ECA above LA | C GF | | groin hematoma |
| | | 6 | none identified | RL ECA above LA | PVA | | none |
| | | 7 | L IMA | L IMA | | | none |
| | | 8 | L IMA | L IMA | | | groin hematoma |
| | | 9 | L IMA | R ECA above LA, L IMA | | | none |
| | | 10 | none identified | R ECA above LA | | | none |
| Chen ¹² | 2009 | 11 | R IMA, R STA | not specified | C GF | 100% | could not be assessed |
| | | 12 | R IMA | not specified | C GF | | none |
| | | 13 | L IMA | not specified | GF | | could not be assessed |
| | | 14 | R IMA | not specified | GF | | none |
| | | 15 | L IMA | not specified | GF | | none |
| | | 16 | RL IMA | not specified | GF | | could not be assessed |
| | | 17 | L ECA | not specified | C GF | | none |
| | | 18 | L IMA | not specified | GF | | none |
| Cogbill ⁴⁸ | 2008 | 19–39 | not discernible due to the merging of blunt and penetrating trauma patients' data | not discernible due to the merging of blunt and penetrating trauma patients' data | C GF | 85% | none |
| Kim ⁴⁹ | 2011 | 40 | not specified | L IMA | PVA | success | none |
| Komiyama ⁵⁰ | 1998 | 41 | not specified | R SPA | | 100% | none |
| | | 42 | not specified | RL SPA, IOA, FA | | | none |
| | | 43 | not specified | L SPA | | | none |
| | | 44 | not specified | RL SPA, LPA, ADTA ⁶ , IAA ⁷ , FA, LA | | | none |
| | | 45 | not specified | RL SPA, FA | C GF PVA | | none |
| | | 46 | not specified | BL SPA, AAA ⁸ | | | could not be assessed |
| | | 47 | not specified | R STA, FA | | | none |
| | | 48 | not specified | RL GPA ⁹ , EA | | | none |
| | | 49 | not specified | RL SPA, SPAA ¹⁰ | | | none |
| Kuan ⁴⁷ | 2015 | 50–76 | 12 x IMA 6 x FA 6 x LA 5 x MMA ¹¹ 3 x ECA 1 x APA ¹² 5x other vessels Note: this statistics also includes one penetrating trauma. | not specified | C GF PVA NBCA | 92.3%; data includes one penetrating injury | no serious systemic or neurologic complications |

| First author | Year Published | Case number | Vessel injured | Vessel embolized | Embolization agents used | efficacy (complete cessation of bleeding following TAE of the ECA or its branches) | complications |
|--------------------------|----------------|-------------|---|---|----------------------------------|--|---|
| Langel | 2020 | 77 | L IMA + R SPA | L ECA + R SPA | C PHIL 25 | success | none |
| Liao ⁴⁰ | 2007 | 78–112 | 25 x IMA 5 x MMA 4 x ECA 4 x SPA 4 x STA 2 x APA 1 x FA 1 x SALA 1 x IALA 1 x DPA 5 x other (observed contrast pooling) | not specified | not specified | 79.4% | none reported |
| Liu ³ | 2008 | 113 | L STA + L IMA | not specified | GF | success | none reported |
| Maiorello ⁵¹ | 2011 | 114 | L FA | L FA | Onyx 18 | success | none |
| Mauldin ⁵² | 1989 | 115 | R ECA | R ECA | C | success | none reported |
| Mehringner ⁵² | 1982 | 116–194 | not discernible due to the merging of blunt and penetrating trauma patients' data | not discernible due to the merging of blunt and penetrating trauma patients' data | GF PVA silastic spheres | 100% | 1x cerebral infarction 2x transient oculomotor nerve palsy |
| Mehrotra ⁶ | 1984 | 195–196 | R IMA L IMA | R IMA L IMA | GF PVA GF | success success | none reported none reported |
| Noy ³⁸ | 2007 | 197 | L IMA, RL FA | L IMA, RL FA | MS NBCA C | success | none |
| Remonda ⁵⁴ | 2000 | 198 | RL IMA | not specified | PVA NBCA | success | Transient trismus |
| Thiex ³³ | 2011 | 199 | L FA | not specified | Onyx | success | none |
| | | 200 | L STA | not specified | C | | none |
| | | 201 | L FA | not specified | C | | none |
| Wang ⁵⁵ | 2015 | 202 | R STA | not specified | C | 100% | none |
| | | 203 | R STA | not specified | C | | none |
| | | 204 | L STA | not specified | C | | none |
| Wong ⁵⁶ | 2013 | 205 | R IMA | R IMA | NBCA | success | none |

Anatomical abbreviations: AAA = anterior auricular artery; ADTA = anterior deep temporal artery; APA = ascending pharyngeal artery; DPA = descending palatine artery; ECA = external carotid artery; FA = facial artery; GPA = greater palatine artery; IAA = inferior alveolar artery; IALA = inferior alveolar artery; IMA = internal maxillary artery; IOA = infraorbital artery; L = left; LPA = lesser palatine artery; MMA = middle meningeal artery; R = right; SALA = superior alveolar artery; SPA = sphenopalatine artery; SPAA = superior posterior alveolar artery; STA = superficial temporal artery

Embolization agent abbreviations: C = coils; GF = Gelfoam; MS = microspheres; PHIL = precipitating hydrophobic injectable liquid; PVA = polyvinyl alcohol particles

Embozene, Celenova, San Antonio, TX, USA; Quadrasphere, Merit Medical Systems, South Jordan, UT, USA; Bead Block and LC Bead, Biocompatibles, Farnham, UK) are smooth globular structures made from an acrylic polymer matrix

impregnated with porcine gelatine. They are hydrophilic, non-resorbable, non-aggregating, non-fragmenting spheres sized 40 - 1200 µm. Sizing inside a particular stated size range follows the Gaussian distribution and is thus more predictable

than is the case with PVA particles. After lodging in vessels, microspheres induce a histological reaction similar to PVA particles. The most notable downside of using microspheres is the necessity to intermittently agitate the particle-saline suspension prior to application in order to prevent sedimentation.^{18,25,26} Also of note is the fact that microspheres of different manufacturers vary in elasticity and, as a consequence, particles of identical size range but different composition occlude vessels at different levels of the vascular tree.^{25,27}

NBCA (TruFill, Cordis, Miami Lakes, FL; Histoacryl, B. Braun Aesculap, Tokyo, Japan; Glubran 2, Gem, Viareggio, Lucca, Italy) is a synthetic adhesive liquid EA (glue) that is accompanied by a separately packed tantalum powder, acting as a radiographic opacifier, and ethiodized oil, functioning as a polymerization retardant. All three components are mixed just before deployment. Once the solution is exposed to anionic environment such as blood, polymerization takes place at a rate dependent on the NBCA concentration. NBCA forms a permanent cast obstructing the vessel lumen. This occurs independently of the endogenous coagulation system - an important characteristic when dealing with exsanguinating trauma cases.¹⁸ NBCA glue also induces vessel wall inflammation reaction resulting in fibrosis. The downside of NBCA use is that the catheter may become glued to the vessel wall if not pulled back quickly enough following glue injection. Glue polymerisation can also occur both distally or proximally to the intended occlusion location. NBCA deployment thus requires a skilled operator.²⁸

Silastic spheres are the oldest non-absorbable particulate EA, introduced in 1964, and have since been replaced by more modern EAs.²⁹

Onyx (Medtronic, Dublin, Ireland) is an ethylene vinyl alcohol (EVOH) copolymer-based non-adhesive liquid EA dissolved in dimethyl sulfoxide (DMSO) with added radiopaque tantalum powder. It was introduced in 1990. As is the case with other DMSO-based non-adhesive liquid EAs, once injected, the DMSO component dissolves into the blood and the copolymer component starts gradually precipitating in a centripetal fashion. The non-adhesive nature allows for long injection times and mid-injection control angiography, if needed. Onyx's final solidification occurs in 5 minutes.³⁰ There are several drawbacks to this EA, including long pre-injection preparation time (20 minutes of mixer shaking are required to achieve homogenization), a self-hiding effect when used in larger amounts due to high radiopacity, plenty of

artefacts in a postinterventional imaging, and the potential to combust or produce sparks during monopolar surgical cauterization.^{31,32} In addition, Onyx's dark colour may result in a black discoloration of the skin after superficial embolization or subcutaneous extravasation of the EA.^{33,34}

Precipitating hydrophobic injectable liquid (PHIL) (MicroVenton, Tustin, CA, USA) is a non-EVOH copolymer-based non-adhesive liquid EA suspended in DMSO with iodine covalently bonded to copolymer to provide radiopacity. It was introduced in 2015. In comparison to Onyx, PHIL is supplied ready-to-use (no shaking is necessary), requires lower volume to achieve the same extent of embolization, is faster to fully precipitate (3 minutes), does not suffer from the self-hiding effect, produces fewer artefacts in postinterventional imaging, is not hazardous to surgical cauterization, and is not dark coloured which diminishes the possibility of skin discoloration.^{35,36} PHIL is also more homogenous on fluoroscopy during prolonged injections, but less radiopaque than Onyx once injected.²³

According to the literature, no cases of blunt trauma-related ECA-territory embolization using other modern non-adhesive liquid EAs, such as Squid (Emboflu, Gland, Switzerland), have so far been reported. In the future, other cutting-edge EAs, such as homogenous microparticles or biodegradable drug-bearing microspheres produced by droplet microfluidics technology, are expected to see regular clinical use.³⁷

Efficacy and safety

Various studies and case reports have shown the ECA TAE to be a safe and efficacious method in maxillofacial blunt-trauma related haemorrhage control, although direct comparison of the reviewed literature is rendered difficult by the variations in reporting. Two studies authored by Noy *et al.* and by Hayes *et al.* investigating intractable maxillofacial bleeding of various aetiologies, including but not limited to trauma, enrolling 74 patients in total, found TAE to be efficacious in 89.1 - 90.0%.^{38,39} A study by Liao *et al.* focusing exclusively on trauma-related oronasal bleeding enrolling 34 patients discovered TAE to be efficacious in 79.4%.⁴⁰ The data collected in Table 1 show that the efficacy of TAE ECA ranges from 79.4% to 100%, with the largest series attaining the perfect success rate comprising 10 cases. These results are similar to the efficacy of non-trauma-related TAE procedures involving the ECA (80%–97%).⁴¹⁻⁴³

The complications among the 205 cases reviewed in Table 1 include groin hematoma (2 cases), cerebral infarction (1 case), partial tongue necrosis (1 case), transient oculomotor nerve palsy (1 case), and transient trismus (1 case), indicating an overall complication rate of 3%. However, there is high variability in reporting styles regarding the complications. For example, certain authors limited the reporting only to serious neurologic or systemic complications without further expounding on the exact criterion that delimited the serious complications from the minor ones. Furthermore, complications that could not be assessed might have occurred, e.g. due to patient dying or entering a vegetative state. In addition, it was not possible to determine the risk of publication bias or selective reporting. It is thus safe to assume the overall complication rate to be higher than directly indicated by Table 1 data. Duncan *et al.* found the rate of major complications (comprising cerebral vascular insult only) to be 2% but also reported a rate of minor complications (comprising headache, transient facial pain, paraesthesia, and local groin complications) of 25% in their series of 57 embolizations, of these 3 trauma-related.⁴⁴ Cullen and Tami reviewed the literature of 264 cases of IMA embolizations for the treatment of posterior epistaxis and found the rate of major complications (hemiplegia, facial nerve paralysis, cheek necrosis, ICA intimal injury, catheter stuck in a vessel, myocardial infarction) to be 4%, and the rate of minor complications (IMA spasm, hypotension, hematoma, groin bleed, oedema, trismus, paraesthesia, persistent pain, skin slough, palate ulceration, aspiration pneumonia, hepatitis) to be 10%.⁴⁵

The relatively low rate of major complications might be in part due to the rich collateral flow between the ipsilateral and contralateral ECA branches distal to the lingual artery that ensure adequate tissue perfusion in case of one-sided ECA embolization.^{46,2} Duncan *et al.* discovered that the complication rate tends to drop with the use of microcatheter techniques. They have also observed a decrease in complication rate in the more recent studies that could be ascribed to factors such as improvement in catheter and guidewire design and increased operator experience.⁴⁴

Survival prognostic factors

Liao *et al.* examined a series of 34 cases of craniofacial trauma requiring TAE and discovered that there was a significant contribution of successful

TAE haemostasis to patient survival ($p = 0.001$). Glasgow Coma Scale score (GCS) ≥ 8 at presentation, injury severity score (ISS) ≤ 32 , and shock index (SI; heart rate divided by systolic blood pressure) ≤ 1.1 before TAE and ≤ 0.8 after TAE were also significantly correlated with the patients' higher survival rate ($p < 0.05$). The need to treat a secondary abdominal bleeding origin by laparotomy significantly decreased the rate of survival ($p = 0.023$). The patients' age, the need to perform craniotomy, the bilateral distribution of the bleeding vessels, and the number of the haemorrhaging vessels per patient were not correlated with the patient survival ($p > 0.05$).⁴⁰

Kuan *et al.* confirmed some of the findings by Liao *et al.* and further discovered that patients with initial haemoglobin level lower than 10 g/dL and patients with brain midline shift observed by computed tomography (CT) had statistically higher odds ratios predicting mortality than their counterparts as estimated by univariate logistic regression.⁴⁷

An illustrative case report

A 20-year-old, previously healthy male was brought to the emergency department in 2019 after an accidental 20 m (65 ft), head-first fall to the ground. He had been cleaning windows of his 7th floor dorm room when he lost balance and fell. Eyewitnesses reported the patient had been lying on his stomach after impacting the ground but later managed to roll on his back by himself. A physician-led emergency medical service arrived on the scene in 10 minutes, finding the patient verbally responsive and making an effort to get up. Initial Glasgow Coma Scale (GCS) was an estimated 13. Severe facial trauma compromising the airway and, within a few minutes, cessation of spontaneous respiration necessitated rapid sequence intubation which proved challenging with several failed attempts. Asystole was observed on ECG prompting resuscitation efforts that resulted in the return of spontaneous circulation and sinus rhythm 20 minutes later. Upon arrival at a Level I trauma centre, the patient presented with GCS 5, blood pressure 70/40 mmHg, heart rate 100/min., a multifragment facial fracture (Figure 2A), a ruptured right eye, massive bleeding from the nose and the left ear, a fractured right 4th rib, a fractured left radius, a displaced left femoral fracture and a fractured left tibia. Adhering to our institution's standard trauma protocol, the possible abdominal and thoracic sources

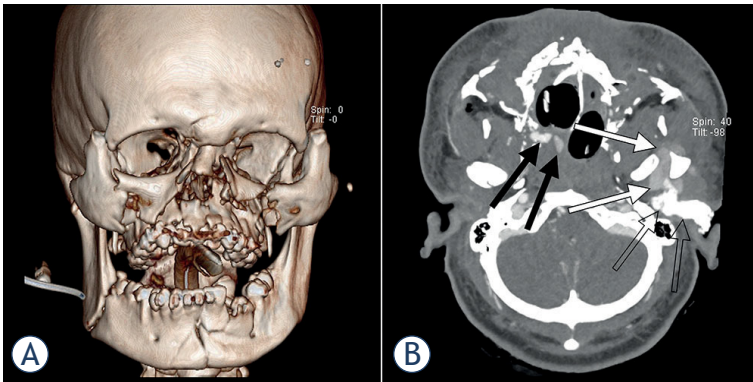


FIGURE 2. (A) A 3D CT reconstruction showing multiple maxillo-facial fractures. (B) An aortocervical CTA showing two small hematomas in the region of the right pterygopalatine fossa and nasal cavity (black arrows) and a cm 3 × 4 cm hematoma in the region of the left masticatory space and deep parotid space (white arrows). Also visible is a hyperdense material used in left ear tamponade (empty arrows).

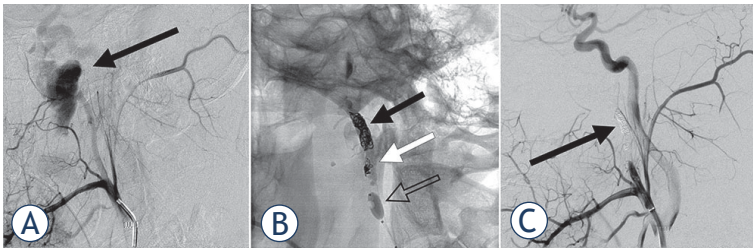


FIGURE 3. (A) A lateral left ECA angiogram showing ECA laceration with 3 × 4 cm pseudoaneurysm continuing into the proximal part of the left IMA. Contrast extravasation can be observed in the vicinity of the pseudoaneurysm. (B) A fluoroscopic view showing left ECA embolization using coils (black arrow) and PHIL 25 liquid embolization agent (white arrow) under balloon flow control (empty arrow). Also of note area small number of stray coils anchored in the vessel in the region of PHIL application (white arrow). (C) A post-embolization lateral left ECA angiogram showing complete exclusion of the ECA distally to the facial artery (black arrow).

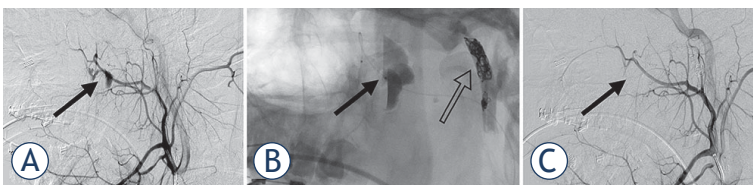


FIGURE 4. (A) A lateral right ECA angiogram showing two pseudoaneurysms in the region of the right pterygopalatine fossa and nasal cavity (black arrows). (B) Fluoroscopy showing microcatheter proximally to the hematoma in the right pterygopalatine fossa (black arrow) prior to PHIL 25 application. Also visible is the embolized contralateral ECA (empty arrow). (C) A post-embolization right ECA angiogram showing complete sphenopalatine artery occlusion (black arrow). Also visible are the patent vessels proximally to the embolization.

of major blood loss were excluded. Astonishingly, US, XR, CT and CTA imaging indicated no significant damage to the neurocranium, parenchymal organs or major thoracic or abdominal vessels. Aorto-cervical CTA was then performed, revealing contrast extravasation from the left IMA and the right sphenopalatine artery (SPA) (Figure 2B). This was consistent with the clinical presentation of severe antero-posterior epistaxis and pulsatile bleeding from the left ear. Nasal packing using balloon catheter inserted through the nares into the nasopharynx was performed by an ear, nose and throat (ENT) specialist to successfully control the nose bleeding. Tamponade of the left ear, however, proved to be inadequate with profound bleeding still persisting. Surgical treatment to control the haemorrhage by ligating the left ECA was decided against due to lesion inaccessibility caused by extensive soft tissue damage and swelling. Blood pressure remained low (60/40 mmHg) despite having hitherto administered a total of 5 litres of fluids, including blood transfusion. Tranexamic acid and vasopressors were also applied, to little avail. In these life-threatening circumstances, TAE of the bleeding origins was considered the only remaining option.

The patient was transferred to the neurointerventional suite and a standard right transfemoral vascular access was established. Digital subtraction angiography (DSA) showed a laceration of the left ECA with an ensuing 3 × 4 cm pseudoaneurysm continuing into the proximal part of the left IMA (Figure 3A). DSA also showed a right SPA laceration with two small accompanying pseudoaneurysms (Figure 4A). The two culprit arteries were then superselectively catheterized and embolized. ECA embolization was performed using platinum coils and PHIL 25, while the SPA was embolized with PHIL 25 only (Figures 3B and 4B). In the case of the ECA embolization, coils created a mesh scaffold acting as a thrombogenic locus, and PHIL was then added to form a coagulopathy-independent lumen-obliterating cast. PHIL was chosen over other available liquid EAs for its ready-to-use characteristics, saving precious time in an emergency setting. In addition, its lava-like polymerisation properties ensured a well-controlled application, helping prevent any inadvertent injection into the dangerous ECA-internal carotid artery and ECA-vertebral artery anastomoses.

Effort was made to embolize at or just proximal to the laceration point in order to preserve proximal arterial territories. Embolization was continued to the point of arterial stasis. Due to the well-

developed left ECA and the resulting high blood flow to the pseudoaneurysm, the ECA embolization was performed under flow control provided by temporary proximal balloon occlusion. No flow control was needed for the SPA embolization. The embolizations of both lesions were immediately followed by complete cessation of the ear bleeding.

Postembolization imaging showed total exclusion of the lacerated vessels (Figures 3C and 4C), complete patency of all proximal vessels, no collateral pathways to the pseudoaneurysm and no other origins of bleeding. There were no procedure-related complications.

To our knowledge, this case is the very first published report of PHIL 25 use for a safe and efficacious management of a massive bleeding originating in the ECA territory. Informed consent was obtained from the patient included in this case report as well as the consent for publication of the individual person's data.

Conclusions

Severe bleeding secondary to blunt maxillofacial trauma is a rare but life-threatening occurrence. Conventional treatment options include manual compression, nasal packing, coagulopathy correction, cauterization, fracture reduction and local vascular control. Open surgical ligation or TAE of the ECA are available as the most definite approaches. Both are similarly efficacious and safe, but TAE offers many advantages, including shorter procedure time, more precise haemorrhage localization, vessel occlusion superselectivity, and the ability to embolize a possible concomitant abdominal or other bleeding during the same session. Major complications of TAE are rare owing to rich collateral blood supply in the ECA territory, with the exception of the lingual artery. The diversion of embolization material into the ICA territories via the ECA-ICA anastomoses is a potentially hazardous complication warranting thorough pre-embolization angiographic overview of the whole vasculature at risk. There are several known factors positively affecting the survival of the maxillofacial trauma patients undergoing ECA TAE, most notably a successful TAE haemostasis, higher GCS, lower ISS, lower SI and higher haemoglobin level on arrival.

Novel non-adhesive liquid EAs such as PHIL allow for faster, more punctilious and coagulopathy-independent application, thus saving invaluable time in life-threatening situations while simultane-

ously diminishing the possibility of inadvertent injection into the ECA-ICA anastomoses. Prospective randomized clinical studies comparing EAs are warranted to further evaluate their efficacy, safety and feasibility.

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