



Cerebral Embolism as a Result of Facial Filler Injections: A Literature Review

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Abstract

Background: With the growth in the popularity of facial filler injections, increased numbers of severe adverse events, such as cerebral embolism, have been reported.

Objectives: The aim of this article was to summarize the clinical manifestations and proposed mechanisms of filler-induced cerebral embolism (FICE).

Methods: A literature review was performed with the search keywords “filler injection,” “hyaluronic acid,” “fat graft,” “cerebral infarction,” “cerebral embolism,” “stroke,” “cerebrovascular infarction,” “disorders of consciousness,” and “hemiplegia.”

Results: Among the 43 cases of FICE enrolled from 35 articles, 37 patients were female, and 6 were male. Twenty-nine of these patients had received fat grafting, and 12 hyaluronic acid injection. Most FICE patients had been injected in the glabella, followed by the temporal, forehead, and nasal areas. Among 30 patients injected under local anesthesia, 43.33% presented with neurologic symptoms during the procedure. The main symptoms were consciousness disorders and hemiplegia. Most of the embolization sites were in the middle cerebral artery, followed by frontal lobe infarction and anterior cerebral artery infarction. Three patients developed cerebral hemorrhage after embolism. Twenty-six patients presented with newly acquired vision loss. The management for FICE cases included embolectomy, thrombolysis, decompressive craniectomy, antiplatelet/anticoagulant therapy, and symptomatic and nutritional treatment. Nearly half of the patients recovered or exhibited improved neurologic manifestations but not visual loss. Five patients died.

Conclusions: FICE is a severe complication following facial filler injection. Careful prevention, timely identification, and treatment are crucial to decreasing the morbidity and mortality of FICE.

Level of Evidence: 4



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Facial contouring or volumization and the treatment of facial grooves, lines, depressions, or hollows can be achieved with synthetic off-the-shelf injectable facial fillers and autologous fat injections. Although the two techniques are

conceptually and technically different, both are considered facial filler injections for the purposes of this review.

Injection of synthetic facial fillers is a widely used, minimally invasive facial cosmetic treatment that is increasing

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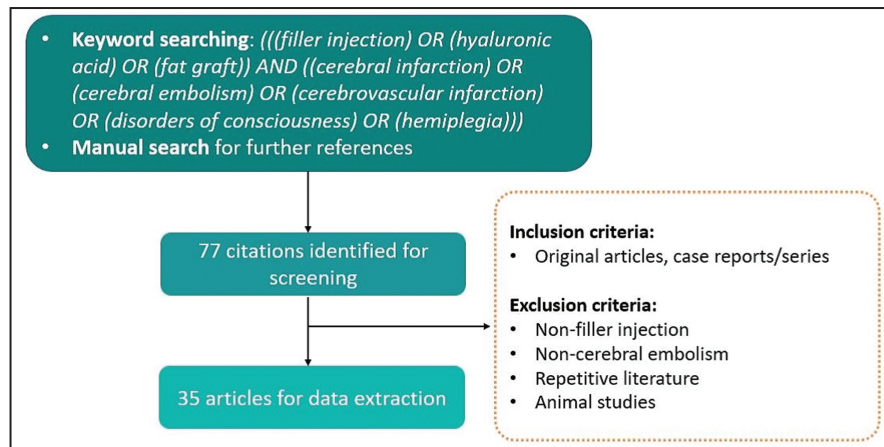


Figure 1. Flow diagram for study screening, selection, exclusion, and inclusion.

in popularity due to the ready availability, variety, ability to achieve natural outcomes, and perception of lower morbidity offered by these materials.¹ FDA-approved absorbable/temporary materials used in these fillers include hyaluronic acid (HA), collagen, calcium hydroxyapatite, poly-L-lactic acid (PLA),²⁻⁴ and polymethylmethacrylate microspheres. The widely used term “dermal fillers” is actually a misnomer because most fillers can be placed at multiple levels of the soft tissue—subdermally, intramuscularly, or even deep on the periosteum. They should more correctly be called “soft tissue fillers.”

Autologous fat harvested by liposuction is also used as a facial filler although this is more correctly classified as a tissue-grafting procedure or operation. It is frequently performed worldwide and more so in Asia where facial 3-dimensional contouring is popular. Although autologous fat can be considered a filler injection, the method of insertion for fat grafts differs from that of synthetic filler injections: the latter does not require blood supply. Collectively, these injectable facial fillers (synthetic or fat) are an option in the treatment of age-related soft tissue volume loss, depressed scars, facial sculpting and contouring, augmentation of specific anatomic sites, wound reconstruction, and atrophy or asymmetry caused by disease.⁴

Synthetic facial filler injections are considered relatively safe with short recovery times and little risk of complications. General anesthesia or sedation is not required and most patients can return to work immediately. Mild and temporary adverse events such as swelling, bruising, redness, surface deformity, and infection can occasionally occur after synthetic facial filler injections^{1,4} and are acceptable risks. However, the increased use of synthetic facial injections has also led to a rise in reports of associated severe adverse events, such as hypersensitivity, cutaneous vascular complications with skin and tissue necrosis, blindness, and cerebral embolism.^{1,5,6}

Facial fat injections, on the other hand, often require general anesthesia or intravenous sedation and are

associated with higher rates of postoperative bruising, swelling, longer recovery times, and higher rates of morbidity that can include fat embolism and cerebral embolism.

Cerebral embolism associated with filler injections, whether by synthetic facial filler or facial fat injections, is a severe complication that has not received sufficient attention due to its low incidence rate and the lack of standardized approach to diagnosis, treatment, and prevention. The mechanism by which it occurs is not entirely clear and allows for healthy discussion. Here, we reviewed cerebral embolism cases induced by facial aesthetic filler injections, whether by synthetic fillers or fat grafting. We aimed to summarize the clinical manifestations, mechanisms, assessment, treatment, and prognosis of filler-induced cerebral embolism (FICE) patients, which may help clinicians understand this dreaded complication.

METHODS

For this study, a literature review was performed in June 2020 (H.C.W. conducted the search and N.Y. reviewed it) according to the guidance provided by Murad et al (Figure 1).⁷ The following search terms were used in PubMed (United States National Library of Medicine [NLM], Bethesda, MD) to obtain all the relevant English-language literature published up to June 2020: “filler injection,” “hyaluronic acid,” “fat graft,” “cerebral infarction,” “cerebral embolism,” “stroke,” “cerebrovascular infarction,” “disorders of consciousness,” “hemiplegia.” The relevant articles selected for this study included original articles and case reports/series that investigated or discussed the role of filler injection in cerebral infarction. The articles excluded from this study were those utilizing non-filler injection or those discussing complications other than cerebral embolism. Articles presenting cerebral embolism as posters/abstracts were excluded, and animal studies were also excluded. The following data were extracted from the articles: author(s), year of publication,

age and sex of patients, filler substance, injection site, anesthesia, symptoms and signs, onset time, diagnostic imaging results, infarction site, treatment, and prognosis. We were only able to analyze data captured in these articles, in some of which the reporting was incomplete.

RESULTS

There were 35 articles reporting 43 cases of FICE in total enrolled for data extraction according to our inclusion and exclusion criteria (Table 1).^{5,6,8-40} Information about the publication year is shown in Figure 2.

Patients' Characteristics

Among the 43 cases, 37 patients were female and 6 patients were male. The age of the patients ranged from 19 to 65 years (mean, 33.93 years). Twenty-nine (67.4%) patients received fat grafting, 12 (27.9%) patients received HA injection, 1 patient received PLA injection, and 1 patient received hydroxyapatite injection. Thirty-five (81.4%) patients had 1 injection site, 7 (16.3%) patients received injections at multiple sites, and 1 patient's site was unknown. Of the 8 patients for whom the injection site was not specified, 6 (75%) received fat grafting. Data on the filler substance and injection sites are shown in Figure 3.

With regard to the clinical background of the patients, 30 (69.8%) patients were healthy, 3 patients had a history of eye conditions with visual loss due to cancer resection or trauma, 1 patient had hypertension, and the histories of 9 patients were unknown. Most of these cases were from East Asia.

Neurologic Manifestations

Regarding the neurologic manifestations (Figure 4), the main presenting symptoms were disorders of consciousness ($n = 18$) and hemiplegia ($n = 16$). Thirty (69.8%) patients received injections under local anesthesia, 6 (14.0%) patients received injections under general anesthesia, 2 patients received injections under intravenous sedation, and 5 patients' anesthesia information was unknown. Among the 30 patients receiving local anesthesia, 43.33% presented with neurologic symptoms during the procedure, and 16.67% presented with similar symptoms within 2 hours postprocedure (Figure 5).

Cerebral Embolism

To confirm a diagnosis of cerebral embolism, 26 patients underwent a magnetic resonance imaging scan, 12 patients underwent diffusion-weighted imaging, 8 patients underwent a computed tomography scan, 5 patients underwent

computed tomography angiography, and 2 cases underwent superselective cerebrovascular angiography. Most of the embolization sites were at the middle cerebral artery ($n = 29$), followed by frontal lobe infarction ($n = 14$) and anterior cerebral artery infarction ($n = 11$) (Figure 6). Notably, 3 patients developed cerebral hemorrhage after embolism.

Vision Loss

Three patients had pre-existing visual loss (1 due to cancer resection, 2 due to orbital trauma). Of the 43 patients who suffered a FICE, 26 patients (60.5%) presented with concomitant vision loss and a stroke, whereas 17 patients (39.5%) presented only with FICE without any concomitant vision loss.

Among the 26 cases with vision loss, 5 patients receiving general anesthesia presented with vision loss after awakening, along with other neurologic signs. The remaining 21 patients had their procedures performed under local anesthesia: 10 of these patients (47.6%) presented with blindness and neurologic signs synchronously during or shortly postoperation (up to 2 hours). In 8 (23.8%) patients blindness appeared quickly postoperation, but their neurologic symptoms only occurred after several hours (at 4, 5, 8, 9, 24, 24, and 24 hours, respectively, and 1 case described as after "several hours"). The situation of 5 patients was unknown.

Among the 17 patients without concomitant vision loss, 5 patients (29.4%) had injections in the temporal area, 5 patients (29.4%) had injections in the glabella, 1 patient had injections in the forehead, 1 patient had an injection in the periocular region, 1 patient had an injection in the nasolabial folds, and 1 patient's injection site was unknown.

Treatment and Prognosis

The treatment of 28 patients was described in the literature (Figure 7). Seven patients underwent embolectomy and 2 patients underwent thrombolysis. Nine patients received decompressive craniectomy. Five patients underwent antiplatelet/anticoagulant therapy. The rest received only symptomatic treatment and nutritional treatment, including steroids, nutritional neuropharmaceuticals, mannitol, and hyperbaric oxygen. Regarding the prognosis, 21 (48.8%) patients recovered or improved neurologically (Figure 8). Five patients (11.6%) remained unimproved and 5 (11.6%) patients died.

DISCUSSION

Patients' Characteristics

The patients with FICE were predominantly females. Moreover, the vast majority of these patients were from

Table 1. The Data of the Enrolled Cases

Year	Author	Age/sex	History	Anesthesia	Filler	Site	Neurologic signs (onset time)	Vision loss	Infarction site	Treatment	Prognosis (follow-up time)
1993	Bitar et al ³¹	47/F	Healthy	Local	Fat	Glabella	Agnosia, facial paralysis, hemiplegia (during the operation)	Right eye	Frontal lobe, parietal lobe, MCA	Not known	Improved (3 weeks)
1996	Lee et al ³²	42/F	Not known	Local	Fat	Nasolabial fold	Headache, consciousness disorder (during the operation)	Left eye	Caudate lobe, thalamus, left cerebral hemisphere cortex	Hyperbaric oxygen therapy	Improved (3 months)
1998	Feinendegen et al ³³	47/F	Healthy	General	Fat	Nasolabial folds, lip, chin	Hemiplegia, aphasia, consciousness disorder (7 hours postoperation)	No	Frontal lobe, temporal lobe, MCA	Not known	Improved (4 months)
2001	Danesh et al ³⁴	43/M	Not known	Local	Fat	Nose, nasolabial fold	Headache, aphasia, hemiplegia (10 minutes postoperation)	Left eye	MCA	Not known	Not known
2003	Yoon et al ³⁵	39/F	Healthy	Local	Fat	Glabella	Aphasia, hemiplegia, consciousness disorder (1 minute postoperation)	Left eye	left hemisphere, ICA	Mechanical ventilation, steroids	Death
2004	Thaunat et al ³⁶	39/M	Left eye cancer	Local	Fat	Temporal, eyelids, glabella	Consciousness disorder (during the operation)	NA	ACA	Not known	Improved (1 year)
2011	Lee et al ³⁷	44/F	Not known	Intravenous	Fat	Periocular area	Dysarthria (2 hours postoperation)	Left eye	Insula, MCA	Mannitol, hyperbaric oxygen therapy	No improvement
2010	Lee et al ³⁸	24/F	Not known	Intravenous	Fat	Forehead	Motor disturbance, paresthesias (1 day postoperation)	Left eye	MCA	Methylprednisolone	Improved (5 months)
2010	Toledano et al ³⁹	33/F	Eye wound	General	Fat	Left orbit	Hemiplegia (when awaking)	NA	MCA	Not known	Not known
2011	Hu et al ⁴⁰	28/F	Healthy	General	Fat	Temporal	Consciousness disorder, aphasia, hemiplegia (postoperation)	No	temporal lobe, parietal lobe, MCA	Mannitol, hydrocortisone, hyperbaric oxygen therapy, antiplatelet therapy	Improved (6 weeks)
2012	Park et al ⁴¹	24/F	Healthy	Local	Fat	Glabella	Not known	Left eye	MCA	Not known	Not known
		26/F	Healthy	Local	Fat	Glabella	Not known	Left eye	MCA, ACA	Not known	Not known
2013	He et al ⁴²	52/F	Not known	Local	HA	Glabella	Headache (a few minutes postoperation)	Right eye	frontal lobe, occipital lobe, parietal lobe, ACA, MCA, PCA	Timolol maleate, acetazolamide, aspirin	Not known
2014	Hong et al ¹⁰	27/F	Healthy	Local	Fat	Glabella, forehead, cheeks	Short-term memory disturbance, naming difficulty (several hours postoperation)	Left eye	frontal lobe	Not known	Improved mildly (1 year)
		50/F	Healthy	Local	HA	Glabella, cheeks	Dysarthria, hemiplegia, facial paralysis (24 hours postoperation)	Left eye	ACA, MCA, followed with cerebral hemorrhage at 2-week follow-up	Not known	Improved (6 months)
2014	Kim et al ²³	23/M	Not known	Local	HA	Nose	Right facial paralysis, left limb paralysis (during the operation)	Right eye	MCA, frontal, temporal and parietal lobes, followed by cerebral and subarachnoid hemorrhage by thrombolysis	Thrombolysis (plasminogen activator), decompressive craniectomy	No improvement (3 months)

Table 1. Continued

Year	Author	Age/sex	History	Anesthesia	Filler	Site	Neurologic signs (onset time)	Vision loss	Infarction site	Treatment	Prognosis (follow-up time)
2014	Kim et al ⁴³	Not known/F	Healthy	Local	HA	Nose	Not known	Right eye	Frontal lobe	Corticosteroids	Not known
2014	Hong et al ⁴⁴	31/F	Not known	General	Fat	Glabella	Arm weakness (24 h postoperation)	Right eye	MCA, frontal lobe, parietal lobe, temporal lobe, occipital lobe	Not known	Recover (5 months)
2014	Wang et al ⁴⁵	22/F	Healthy	General	Fat	Forehead, temporal	Hemiplegia, Babinski sign (+), aphasia (5 hours postoperation)	Left eye	ACA, MCA, ICA, ECA	Decompressive craniectomy	Improved (2 months)
2015	Roshandel et al ⁴⁶	65/F	Hypertension	Not known	Fat	Forehead	Hemiplegia (several hours postoperation)	Right eye	frontal lobe, parietal lobe, occipital lobe, MCA	Not known	Not known
2015	Lin et al ⁴⁷	25/F	Healthy	Local	HA	Nose	Nausea, dizziness, weakness (4 hours postoperation)	Right eye	MCA	Not known	Not known
2019	Wang et al ⁵	49/F	Healthy	Local	HA	Forehead	Consciousness disorder, headache, hemiplegia (during the operation)	No	Temporal, frontal and parietal lobes, followed with cerebral and subarachnoid hemorrhage	Low-molecular-weight heparin, clopidogrel, mannitol	Death
2016	Shen et al ¹⁴	30/F	Healthy	Local	Fat	Temporal, chin	Consciousness disorder, left limb weakness, incontinence, vomiting (8 hours postoperation)	No	Right brain hemisphere, ICA, ECA, CCA, MCA, superficial temporal artery	Lowering intracranial pressure, antiplatelet aggregation, decompressive craniectomy	Improved (2 months)
2016	Kang et al ⁴⁸	32/F	Healthy	Local	Fat	Glabella	Consciousness disorder, aphasia, hemiplegia (during the operation)	Left eye	ACA, MCA	Thrombolytic agents	Improved (3 months)
2016	Li et al ⁴⁹	25/F	Healthy	Local	HA	Nose	Left upper limb weakness (9 hours postoperation)	Right eye	MCA	Not known	Not known
2017	Ragam et al ¹⁹	55/F	Healthy	Local	PLA	Forehead	Dizziness, weakness, consciousness disorder (during the operation)	Right eye	ACA, frontal lobe, corpus callosum	Methylprednisolone	No improvement
2018	Marumo et al ⁵⁰	26/F	Healthy	Local	Hydroxyapatite	Glabella	Nausea, diplopia, consciousness disorder (during the operation)	Left eye	ECA	Not known	Not known
2020	Zhang et al ²⁴	31/F	Not known	Local	HA	Nose	Headache, nausea and vomiting, incontinence (5 minutes postoperation)	Left eye	MCA	Steroids, hyperbaric oxygen therapy, EHATSA	Improved
		46/F	Not known	Local	HA	Palpebra superior	Emotional disorder (hyperactivity) (not known)	Right eye	Lacunar cerebral infarction	Glucocorticoids, neurotrophic drug, hyperbaric oxygen, EHATSA	No improvement
2020	Liu et al ⁵¹	35/F	Healthy	Not known	Fat	Not known	Hemiplegia (during the operation)	No	MCA, ECA	Aspirin, atorvastatin, dexamethasone	Recover (3 months)
2020	Yang et al ⁶	40/F	Healthy	Local	HA	Nose	Nausea, vomiting, headache, consciousness disorders (30 minutes postoperation)	Left eye	Frontal lobe, parietal lobe, temporal lobe, occipital lobe	Mannitol, glucocorticoid, mechanical ventilation	Death
2020	Wang et al ¹⁷	32/F	Healthy	Local	HA	Glabella	Emotional disorder (not known)	No	Frontal lobe	Antidepressant therapy	Improved

Table 1. Continued

Year	Author	Age/sex	History	Anesthesia	Filler	Site	Neurologic signs (onset time)	Vision loss	Infarction site	Treatment	Prognosis (follow-up time)
2019	Zhou et al ²⁶	22/F	Healthy	Local	Fat	Temporal	Hemiplegia, consciousness disorder (4 hours postoperation)	No	ICA, MCA	Mechanical thrombectomy + thrombus aspiration technique	Improved (3 months)
2019	Ansari ⁹	20/F	Healthy	Local	HA	Glabella	None	Right eye	parietal lobe, circle of Willis	Aspirin, prednisone	NA
2019	Liu et al ¹⁷	42/F	Healthy	Local	Fat	Temporal	Lethargy, aphasia, hemiplegia (during the operation)	No	ICA, ACA, MCA, frontal, temporal and parietal lobes, superficial temporal artery	Decompressive craniectomy	No improvement (2 years)
2019	Renard et al ⁵²	50/M	Eye wound	General	Fat	Right orbit	Hemiplegia (when awaking)	NA	ACA, MCA	Not known	Not known
2018	Huo et al ⁸	33/F	Healthy	Local	Fat	Glabella	Motor disturbance, consciousness disorder (during the operation)	No	ICA, MCA	Embolectomy + decompressive craniectomy	Improved
		25/F	Healthy	Local	Fat	Glabella	Motor disturbance, consciousness disorder (during the operation)	No	MCA, ACA, frontal, and temporal lobes	Embolectomy + decompressive craniectomy	Improved
		24/F	Healthy	Not known	Fat	Periocular	Seizure, consciousness disorder, motor disturbance (2 hours postoperation)	No	ACA, CCA, ICA, MCA, PCA	Embolectomy	Death
		19/M	Healthy	Not known	Fat	Glabella	Hemiplegia, consciousness disorder (1 hour postoperation)	No	MCA	Embolectomy + decompressive craniectomy	Improved
		28/M	Healthy	Not known	Fat	Glabella	Seizure, consciousness disorder (5 hours postoperation)	No	Not known	No	Death
2018	Wang et al ¹⁶	22/F	Healthy	Local	Fat	Temporal	Consciousness disorder, limb weakness (during the operation)	Both	MCA, superficial temporal artery	Decompressive craniectomy	Improved (2 years)
		30/F	Healthy	Local	Fat	Temporal	Weakness (during the operation)	No	Right hemisphere, superficial temporal artery	Decompressive craniectomy	Not known

ACA, anterior cerebral artery; CCA, common carotid artery; ECA, external carotid artery; EHATSA, endovascular hyaluronidase application through superselective angiography; ICA, internal carotid artery; MCA, middle cerebral artery; NA, not available; PCA, posterior cerebral artery.

East Asia (only 9 out of 35 papers were from the West). This gender and ethnic bias may reflect the aesthetic aspirations of many Asian women today who wish to achieve an oval or heart-shaped face with smooth, full, and convex forehead contours and an absence of temporal hollows.⁴¹ This aesthetic trend has led to an increase in the use of autologous fat or synthetic fillers to create the desired convex shape. The forehead, brow, and temples are well-established danger zones where numerous arterial connections between the internal and external carotid artery systems exist as well as terminal arterial branches of the ophthalmic artery. These can

be inadvertently punctured, leading to a filler embolus entering the internal carotid artery system, and may explain why so many patients with FICE are in fact Asian and women. Males, whether Asian or Caucasian, are less interested in shaping their foreheads and temporal regions, which may explain their lower risk for this severe adverse event.

Clinical Manifestations

The clinical manifestations of FICE were mainly neurologic symptoms and signs related to the location and degree

of the obstruction, including hemiplegia and consciousness disorders. In the literature, the symptoms of some patients with mild obstruction were less recognizable, and were identified as obstruction by further imaging examinations. These patients sometimes presented with neuropsychiatric symptoms only, such as emotional or mood changes, memory disturbance, or asymptomatic lacunar infarction, which was seen only on imagings.^{8,13,37,39} Therefore, we speculate that the incidence of FICE may be much higher than that reported in the literature. We also noticed that the recognition of the onset of cerebral embolism symptoms was affected or delayed by the type of anesthesia used.

Generally, neurologic symptoms occurred during the operation or within 1 hour postoperation for the FICE patients receiving filler injection under local anesthesia.

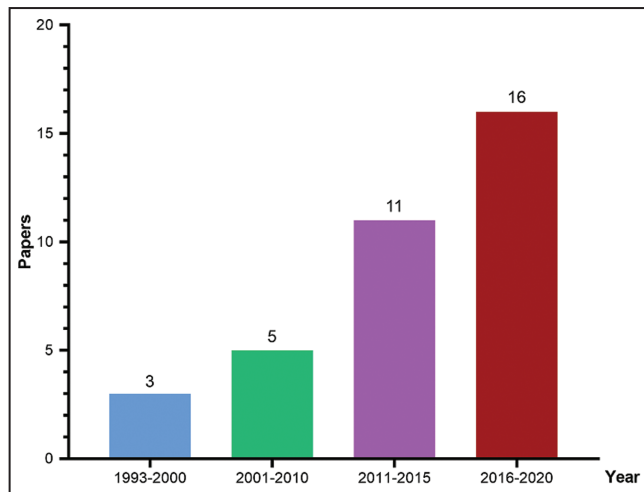


Figure 2. Publications reporting filler-induced cerebral embolism have increased over time.

However, it took more time for the patients under general/intravenous anesthesia to complain about neurologic symptoms because they needed time to wake up from anesthesia. This may delay the timely diagnosis of cerebral embolism. Therefore, to detect cerebral embolism in time, patients recovering from general/intravenous anesthesia should be closely examined for neurologic signs, including muscle strength, muscle tension, pupillary light reflection, and pathologic reflexes.

In addition, due to the fact that a considerable number of patients with cerebral embolism were associated with immediate blindness, or even delayed blindness, which in turn was related to the mechanism of emboli entering the internal carotid or ophthalmic blood vessels at different times, those patients who complain only of ocular symptoms should also be carefully evaluated for signs and symptoms of cerebral embolism as well. We have noted 8 cases where the neurologic signs developed between 4 and 24 hours postoperatively.

Mechanism

In the literature the most frequent injection site associated with FICE was the glabella, followed by the temporal, forehead, and nasal areas. At present, it is thought that FICE and blindness is a direct result of accidental injection of filler material into a facial vessel that is a terminal branch of the ophthalmic artery (internal carotid artery system) or into a branch of the facial artery (external carotid artery system) that, in turn, anastomoses with branches of the internal carotid artery system. This leads to subsequent retrograde embolism and obstruction of certain cerebral vessels or the ophthalmic artery branches.

By analyzing the imaging findings of the FICE patients reported in the literature, we summarized the possible

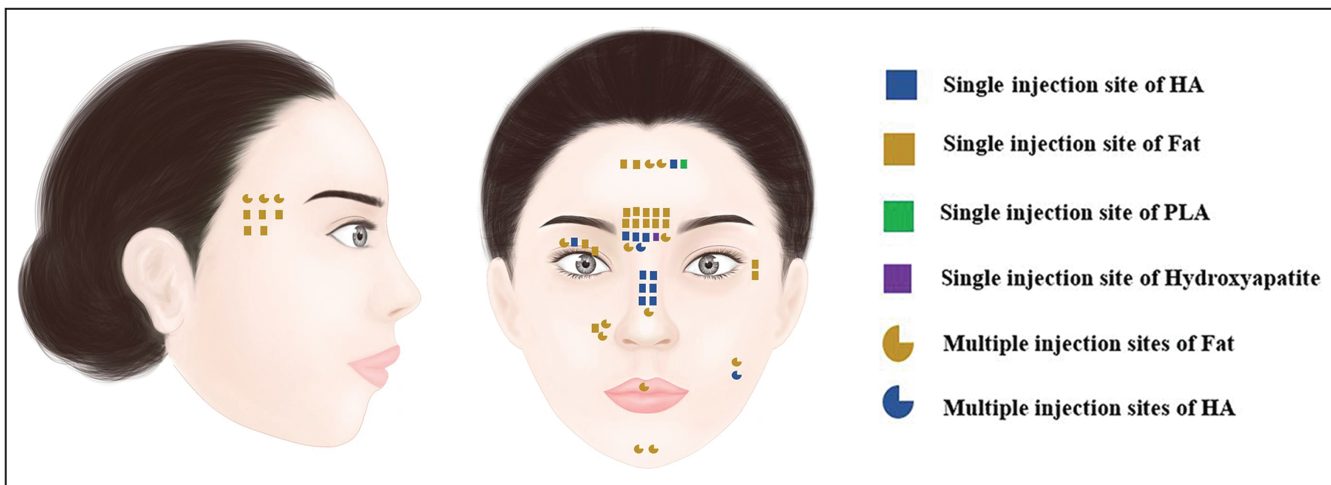


Figure 3. Filler substances and injection sites. HA, hyaluronic acid; PLA, poly-L-lactic acid. Artwork created by author H. C. Wang, reproduced with permission from the author.

ways of the filler leading to intracranial vascular embolism into 3 types, which are described in Table 2.

One possible mechanism is that during the injection procedure, the filler material was inadvertently injected under pressure into one of the extracranial terminal branches of the ophthalmic artery (supratrochlear, supra-orbital, dorsal nasal, anterior ethmoidal, and lacrimal arteries) or injected into any of the anastomotic branches between the internal carotid and external carotid artery systems (Figure 9). Filler emboli may then reach the ophthalmic artery and/or the internal carotid artery. Blindness occurred when the central retinal artery (CRA), or posterior ciliary artery (PCA) was obstructed, whereas cerebral embolism occurred when the terminal intracranial branches of the internal carotid artery were obstructed (Figure 10).

It is a known anatomic fact that many anastomotic branches exist between the external and the internal carotid artery systems, and although there is significant

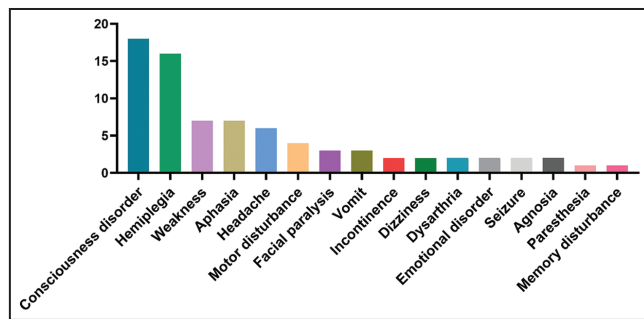


Figure 4. Neurologic manifestations.

human variation, 4 main groups of these anastomotic vessels are described:⁴²

- (1) The frontal (anterior) branch of the superficial temporal artery anastomoses with the lacrimal, palpebral, or supra-orbital branches of the ophthalmic artery.
- (2) The angular artery is a terminal branch of the facial artery, which is in turn derived from the external maxillary artery (external carotid artery system). The angular artery can anastomose directly with the inferior palpebral artery, the dorsal nasal artery, or the supratrochlear artery, all of which are terminal branches of the ophthalmic artery (internal carotid artery system). The supratrochlear or anterior ethmoidal arteries may continue down the dorsum of the nose as the dorsal nasal artery, which often anastomoses with the alar and sidewall branches of the facial artery.⁴³
- (3) The orbital branches of the middle meningeal pass through the superior orbital fissure, and anastomose with the lacrimal or other branches of the ophthalmic artery.
- (4) The infraorbital branch of the internal maxillary artery may anastomose with the dorsal nasal branch of the ophthalmic artery.

It must be appreciated that there are many arterial and anastomotic variations, with some individuals having one and not the other, or even having multiple of these connections.

Whichever of these direct or anastomotic routes is traversed, it is reasonable to assume that a column of filler material must first find its way into either the dorsonasal,

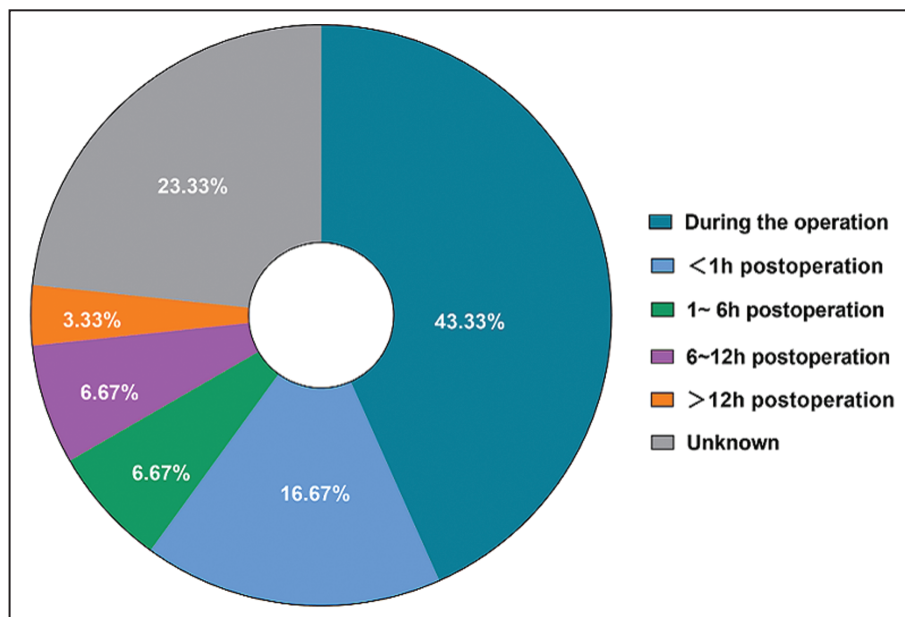


Figure 5. The onset of neurologic manifestations in 30 patients receiving filler injections under local anesthesia.

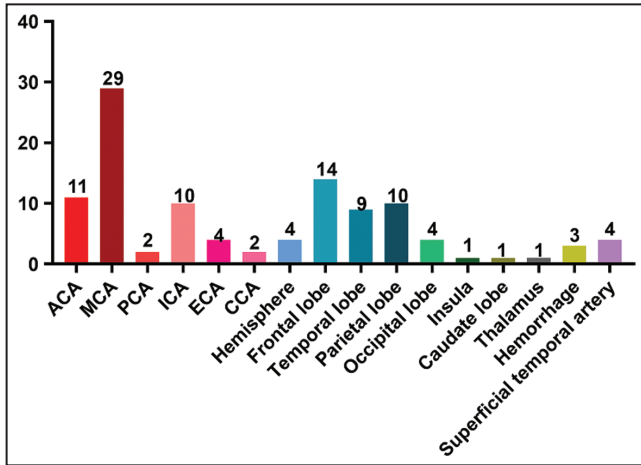


Figure 6. Embolization sites. ACA, anterior cerebral artery; CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery.

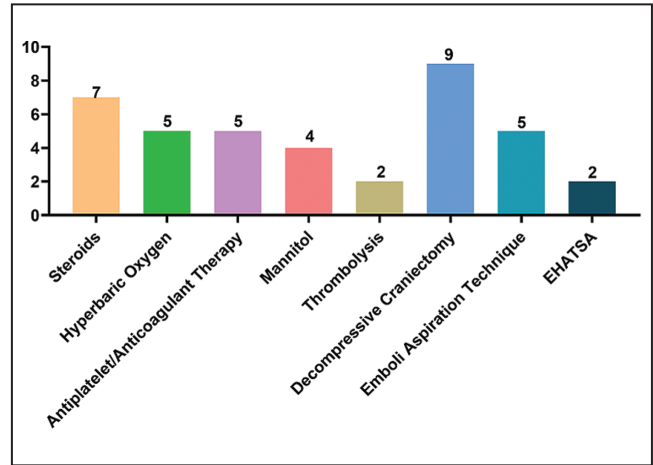


Figure 7. Filler-induced cerebral embolism treatment. EHATSA, endovascular hyaluronidase application through superselective angiography.

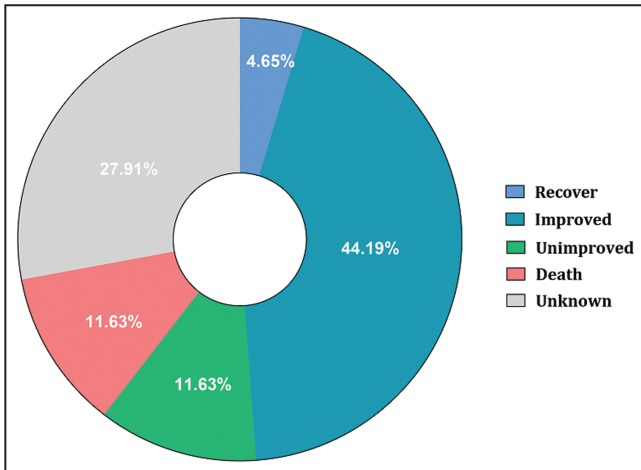


Figure 8. The prognosis of the filler-induced cerebral embolism patients.

supratrochlear, or supraorbital arteries (all being externalized terminal branches of the ophthalmic artery) before it is forced retrogradely into the ophthalmic artery itself, past the origins of the central retinal and posterior ciliary arteries⁴⁴ and subsequently past the origin of the ophthalmic artery from the internal carotid artery.

If the column of filler material is sufficient to go past where the CRA and PCA originate from (Figure 10) but not into the internal carotid artery, then that embolus may be carried anterogradely down the CRA or PCA and give rise to visual disturbances. A cerebral embolism would not occur in this situation.

If, however, the filler material traverses the entire length of the ophthalmic artery and enters the internal carotid

artery, it can then potentially be carried anterogradely downstream until it reaches the middle cerebral artery or other intracranial vessels that derive from the internal carotid artery, and then eventually obstructs a terminal branch in the brain.

In Table 2, this is referred to as the Type I mechanism for cerebral embolism. This type of vascular embolism can cause immediate cerebral embolism and/or blindness. It has been shown in experimental and anatomic models that as little as 0.08 mL of HA filler can cause blindness.⁴⁴ It is not known how much filler is required to induce a cerebral embolism.

In the Type II mechanism of cerebral embolism, the superficial temporal artery is injured during temporal injections. After entering the superficial temporal artery, the filler emboli may be pushed into the external carotid artery or even the common carotid artery under excessive injection pressure, which then flows into the internal carotid artery and the intracranial blood vessels, resulting in cerebral embolism and/or blindness.^{25,32,36,45} This kind of embolization can be preliminarily confirmed by loss of pulsation of the superficial temporal artery on palpation.¹⁶

The Type III mechanism occurs when the veins of the face are damaged during the injection process. The filler then embolizes to the heart first, after which it follows the systemic circulation, before it reaches the terminal branches and causes vascular complications, such as pulmonary embolism, cerebral embolism, or blindness.^{29,46-48} The manifestations of this type of vascular embolism may present hours after the initial injection;^{29,46} multiple factors, such as overinjection, injection displacement, muscle activity, and other factors, may also play a role in vascular complications of filler injection.

Table 2. Three Possible Ways for Filler to Lead to Intracranial Vascular Embolism

Type	Mechanism	Route by which emboli enter the brain	Onset of neurologic signs	Other possible manifestations
I	The filler emboli enter the extracranial branches of the ophthalmic artery injured during the injection, or the anastomotic branches of the internal carotid artery and the external carotid artery injured during the injection	Extracranial branches of ophthalmic artery/anastomotic branches of ICA and ECA → ophthalmic artery → MCA	Immediate	Blindness
II	The filler emboli enter the superficial temporal artery, the filler emboli may be pushed into the external carotid artery or even the common carotid artery under excessive injection pressure	Superficial temporal artery → ECA → CCA → ICA → MCA	Immediate	Blindness
III	The filler emboli enter veins damaged during the injection	Veins on face → anterograde venous system → heart → brain/lung/eye	Delay	Delayed blindness, pulmonary embolism

CCA, common carotid artery; ECA, external cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery.

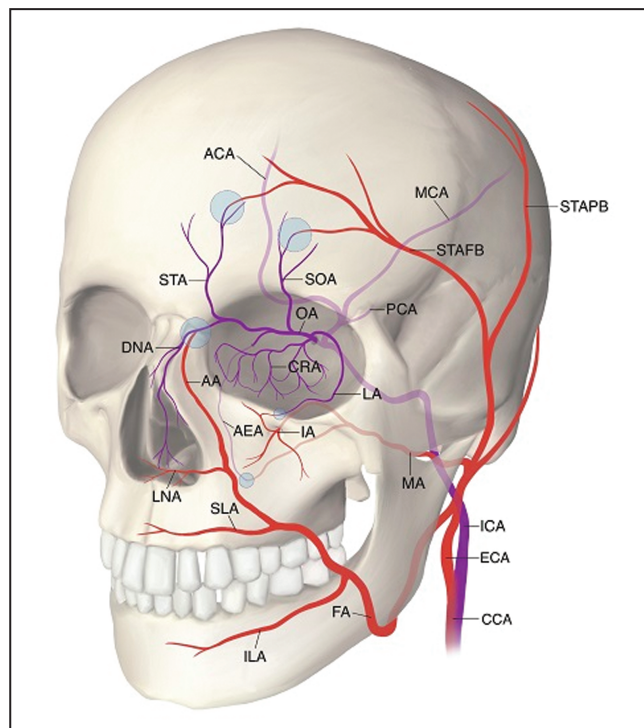


Figure 9. Schematic diagram showing the extracranial branches of the ophthalmic artery and the anastomotic branches (purple circle) of the internal carotid artery (blue) and the external carotid artery (red). AA, angular artery; ACA, anterior cerebral artery; AEA, anterior ethmoidal artery; CCA, common carotid artery; CRA, central retinal artery; DNA, dorsal nasal artery; ECA, external carotid artery; FA, facial artery; IA, infraorbital artery; ICA, internal carotid artery; ILA, inferior labial artery; LA, lacrimal artery; LNA, lateral nasal artery; MA, maxillary artery; MCA, middle cerebral artery; OA, ophthalmic artery; PCA, posterior cerebral artery; SLA, superior labial artery; SOA, supraorbital artery; STA, supratrochlear artery; STAFB, superficial temporal artery frontal branch; STAPB, superficial temporal artery parietal branch. Artwork created by author R. Dong, reproduced with permission from the author.

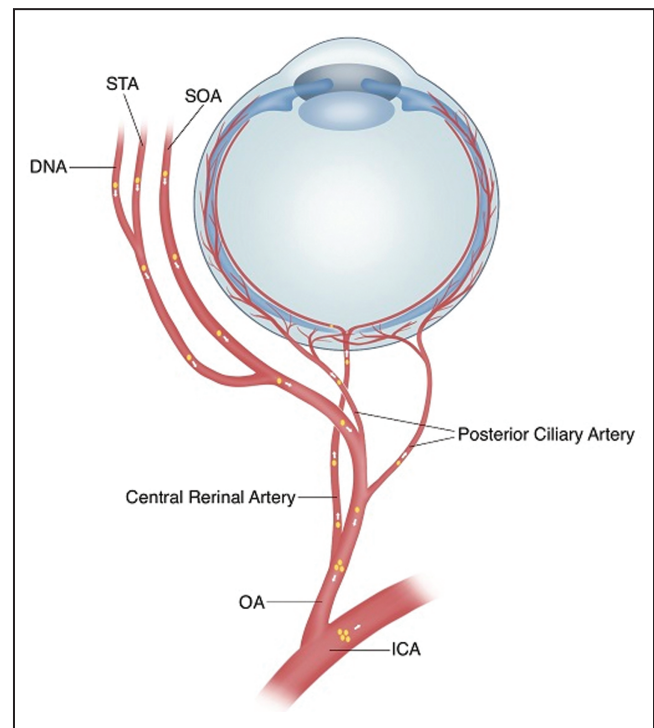


Figure 10. Schematic diagram showing the relation between blindness and cerebral embolism. CRA, central retinal artery; DNA, dorsal nasal artery; ICA, internal carotid artery; OA, ophthalmic artery; SOA, supraorbital artery; STA, supratrochlear artery. Artwork created by author R. Dong, reproduced with permission from the author.

Cannula vs Sharp Needle

The debate over which one of these modalities is safer continues. It was initially thought that a blunt cannula would be a safer instrument than a sharp needle for facial filler injections. However, both cannulas and sharp needles have been seen to penetrate blood vessels and cause filler emboli.

A more important consideration would be at which level of soft tissue (subdermal, superficial fat, fascia, muscle, deep fat, or periosteum) these fillers were injected and whether there are major blood vessels in the vicinity that could potentially be penetrated by either of these instruments.

The 35 articles surveyed for this systematic review are silent on the use of cannulas but it can safely be assumed that in all the patients who received facial fat injections (29 out of 43, 67.4%) the injections would have been performed with a cannula, as is the usual practice. Of the 12 patients who received HA filler injections (27.9%), we can also extrapolate that at least 50% of these would have been administered by a cannula. This implies that up to 82% of all FICE cases may have been associated with the use of a cannula.

This alarming statistic could be due to the very nature of facial fat injections as they require the fat to be delivered in multiple passes and evenly distributed throughout the middle lamellar (muscle) of the soft tissue so as to allow a blood supply to develop. In the face, the arteries lie mainly in or just deep to the middle lamella of facial muscles, and therefore multiple passes of the cannula in this layer increase the risk of vascular damage and penetration.

With regard to the depth of injection, detailed knowledge of facial anatomy would suggest that the subdermal and/or supraperiosteal layers are safer for injection because there are few major arteries running in these 2 layers. In our opinion, it makes sense to inject immediately subdermally in the subcutaneous fat or deep on the bone (avoiding the vascular bony foramina such as the infraorbital or supraorbital foramen) to reduce inadvertent penetration of the blood vessels (such as the facial or superficial temporal arteries) which are more intimately associated with the layer of facial muscles (the middle lamella). Injecting into either the subdermal or preperiosteal layers is possibly easier to achieve with a sharp needle than a cannula. A cannula may provide the injector with a false sense of security, leading to more vascular trauma.

Treatment

The current treatment of FICE includes general symptomatic and nutritional therapies such as hyperbaric oxygen, neuropharmaceuticals, mannitol, and steroids.

Thrombolytic therapy or antiplatelet/anticoagulant therapy is not only ineffective for patients with cerebral embolism induced by filler injection (which is a nonthrombotic embolism), it may also cause drug-induced cerebral hemorrhage, aggravating the patient's condition.^{5,19} For vascular embolism caused by HA, some researchers have tried to achieve recanalization of the occluded vessel by administering intra-arterial thrombolysis therapy with the injection of hyaluronidase and/or urokinase. In a study of 24 patients with vision loss caused by HA injection, 42% of the patients ultimately showed improvements in visual acuity following intra-arterial thrombolysis therapy even when the recommended window for optimal thrombolytic treatment had passed.⁴⁹ In addition, the authors found that hyaluronidase combined with urokinase was a more effective therapy than hyaluronidase alone. In cases of filler-induced blindness, we know that extravascular or retrobulbar hyaluronidase plays little or no role in resolving visual loss.^{44,50} For embolization due to fat, some researchers have used an emboli aspiration technique to partially recanalize the occluded vessel; however, this technique carries the potential risk of triggering distant cerebrovascular embolism.^{16,40}

The prognosis of FICE is related to which vessels are occluded. Unlike the obstruction of small blood vessels leading to mild symptoms with a good prognosis, occlusion of large blood vessels can lead to a large-area cerebral infarction with secondary exacerbation of the edema, which was associated with high morbidity and high mortality.¹⁸ Under such circumstances, decompression craniectomy is strongly recommended to reduce mortality.¹⁶ It is crucial for FICE patients to be diagnosed and treated in time. Delayed treatment may also lead to poor prognosis.^{16,40}

Prevention

With treatment options for FICE being somewhat limited, and to an extent ineffective, it would be prudent for practitioners around the world to be acutely aware of this complication and take the necessary steps to reduce its occurrence. From the papers sourced for this review, we notice a worrying trend in the occurrence of FICE. Between 1993 and 2010 (17 years) 8 cases of FICE were reported. This jumped to 11 cases between 2011 and 2015 (4 years) and a further jump to 16 cases in the period 2016 to 2020 (4 years). This means that in the last 8 years the total number of FICE cases has risen to 27, more than 3.5 times that in the period 1993 to 2010 (Figure 2).

Most of these cases were from East Asia and the majority were associated with facial fat injections with cannulas. This alarming increase in the frequency of FICE occurrence may reflect the current situation where there is a proliferation of underground illegal cosmetic clinics in

this region. Here, untrained operators without a semblance of understanding of anatomy are more likely to damage blood vessels when injecting filler.

For medical practitioners performing these procedures, some measures have been widely recommended to reduce the incidence of vascular complications during the injection process. These include: (1) aspirating and maintaining negative pressure upon entry all the way to the designated site; (2) injecting small volumes slowly while closely observing the injection site and the patient's reaction—physicians should avoid using too many fillers at one time; (3) injecting filler with blunt cannulas rather than needles. This last point is arguable because it flies in the face of the evidence we have—over 80% of FICE patients in this review were likely treated with cannula injections. This therefore contradicts the dictum that “cannulas are safer.” In fact, delivering filler directly to the bone with a sharp needle may provide a safer alternative than cannulas which may give the injector a false sense of security.

We submit that, for injecting fillers, the critical skill a medical practitioner should possess is a sound knowledge of facial anatomy—so that they know the course and changing levels of the major vessels, have an understanding of the sites of anastomosis between internal and carotid artery systems, and are familiar with the different soft tissue layers and their relations with the arteries of the face.⁵¹

Even then, these measures may still fail to completely avoid the occurrence of filler-associated vascular complications, due to multiple factors such as anatomic variations and operator skill. The development of ultrasound-assisted injection may allow practitioners to clearly recognize the location of blood vessels in relation to the different anatomic layers⁵²⁻⁵⁴ of soft tissue. The use of ultrasound-assisted injections, although requiring training, is not that complicated to master. This may be the trend for filler injection methods in the future. We also recommend performing filler injections under local anesthesia as much as possible in order to identify and treat FICE in a timely manner.

CONCLUSIONS

FICE is a severe complication following facial filler injection with limited treatment options. Careful prevention and timely identification and treatment are crucial to decrease the morbidity and mortality of FICE.

Disclosures

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