Postoperative Visual Loss: A Report of One Patient With Unilateral Blindness After Orthognathic Surgery

Constance Delmotte, MD,[*†] Arnaud Depeyre, MD,*[‡§||¶#] Isabelle Barthelemy, MD, PhD,*[‡§**] and Joel Ferri, MD, PhD*[*†¶#]

Introduction: Blindness after orthognathic surgery may be the result of the surgical procedure itself or the consequence of factors induced by general anesthesia. However, the exact mechanism between is not known. The purpose of this article is to present a case of a postoperative visual loss after orthognathic surgery under general anesthesia concluding with a brief literature review about this topic.

Report of case: We report the case of a patient who suffered unilateral blindness with homolateral frontal paresthesia after orthognathic procedure in 2 steps. He presented intraoperative bradycardia with a potential undiagnosed hypertension, associated with significant blood loss and volume resuscitation by colloids and cristalloids.

Postoperative examination concluded to posterior ischemic optic neuropathy.

Discussion and Conclusion: By a systematic literature review, we discuss about surgical and anesthesic causes of postoperative visual loss, and particularly pathophysiology mechanism of posterior ischemic optic neuropathy. Some predisposition and risk factors have been identified and need to be taken into account.

Key Words: Amaurosis, general anesthesia, orthognathic surgery, posterior ischemic optic neuropathy, prolonged hypotension

(*J Craniofac Surg* 2019;30: 223–225)

Postoperative visual loss (POVL) is a rare but severe complication. Vision loss occurring after nonocular surgery under general anesthesia most frequently results from one of the following etiologies: anterior ischemic optic neuropathy, posterior ischemic optic neuropathy (PION), central retinal artery occlusion, central retinal vein occlusion, pituitary apoplexy, cortical blindness, or direct compression.[1] The most common cause is PION, characterized by sudden painless visual loss, associated with normal immediate ophthalmic evaluation, computed tomography (CT), and magnetic resonance imaging (MRI). Disc pallor will appear on the fundus examination a few weeks later.[2] It is a diagnosis of exclusion although some risk factors have been identified, such as prolonged operative time, anemia, intraoperative hypotension, diabetes, obesity, male sex, major intraoperative blood loss, microvascular pathology, and massive colloid administration during the procedure.[1,3,4]

Orthognathic surgery is recognized as a safe and predictable procedure thanks to an increasing knowledge about facial anatomy and progress in orthodontics treatment over the last 30 years.[5,6] Initially, maxillofacial orthopedic surgery was intended for the treatment of malocclusion in younger populations; however, with the development of this surgery, older populations are now receiving support for temporomandibular joint pain or preprosthetic reconstruction. This change creates an added surgical or anesthetic risk linked to the patients' age. Blindness after orthognathic surgery may be the result of the surgical procedure itself when basal skull fracture, orbital hematoma, or constraint on intraorbital structures are observed.[5,7–11] However, it may be the consequence of factors induced by general anesthesia, especially when patients have comorbidities. Fourteen cases of postoperative blindness after Le Fort I osteotomy were described in the literature.[7–15] The exact mechanism of anesthesia and surgery implication is not known. Herein, we report a case of a POVL after orthognathic surgery procedure under general anesthesia and conclude with a brief literature review about this topic aimed at describing the physiological mechanism involved.

PRESENTATION OF PATIENT

A 53-year-old male patient sought consultation in the department of oral and maxillofacial surgery of the University Hospital of Lille (France). He presented bilateral temporomandibular joint pain due to class III malocclusion with transverse maxillary discrepancy requiring an orthognathic surgery.

He weighed 71 kg and was 1.78 m tall (BMI, 22.4 kg/m^2). His medical past history was characterized by chronic headaches, diverticulitis, malaise of vasovagal origin, and right cholesteatoma. He presented with generalized periodontal disease, which was exacerbated by smoking and malocclusion. Preoperative evaluation found blood pressure at 140/80, heart and lungs examination without abnormalities, including electrocardiogram and chest radiography.

The surgery was performed in 2 stages. First, a surgically assisted rapid palatal expansion was completed. Ten months later, a bimaxillary orthognathic surgery was performed with a Le Fort I osteotomy with initial advancement followed by a sagittal split osteotomy of the mandible.

For this second procedure, induction of anesthesia was uneventful. Few minutes later, the patient's blood pressure was 160/80 mm Hg,

From the *Oral and Maxillofacial Department, Roger Salengro Hospital, CHU Lille; †Lille University, Lille; ‡Oral and Maxillofacial Department, Estaing Hospital, CHU Clermont Ferrand, Clermont Ferrand; §Auvergne University, Faculty of Medicine, Clermont-Ferrand; ||Auvergne University, CROC Laboratory EA 3847, Faculty of Dental Surgery, Clermont-Ferrand; ¶Inserm, U1008, Controlled Drug Delivery Systems and Biomaterials, Lille; #International Association of Oral and Maxillofacial Medicine (IAOMM), Villeneuve d'Ascq; and **Inserm U1107 Neuro-Dol, Trigeminal Pain and Migraine, Faculty of Dental Surgery, Clermont-Ferrand, France.

Received June 3, 2018.

Accepted for publication October 14, 2018.

Address correspondence and reprint requests to Constance Delmotte, MD, Service de Chirurgie Maxillo-Faciale Et Stomatologie, Hôpital Roger Salengro, CHRU Lille, 59000 Lille, France;
E-mail: constancedelmotte@hotmail.com

The authors report no conflicts of interest.

Supplemental digital contents are available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.jcraniofacialsurgery.com).

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000005151

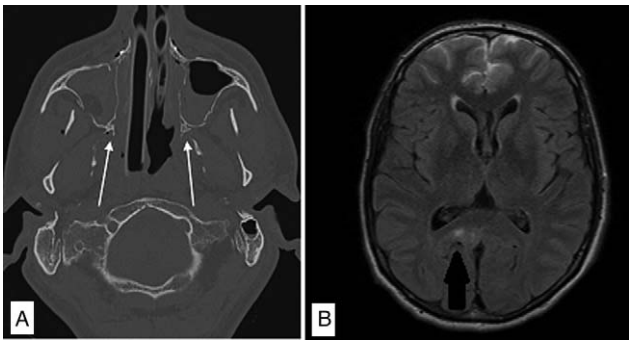

FIGURE 1. (A) Immediate postoperative computed tomography scan axial cut showing normal jaw osteotomies, with pterygomaxillary disjunction. (B) Postoperative cerebral and orbital magnetic resonance imaging axial cut showing slight flair hypersignal in the right internal occipital cortex and in the insular right parenchyma.

which then decreased to 80/50 mm Hg. Volume expansion was introduced by crystalloids (500 mL). After that and before the beginning of the procedure, the patient presented with bradycardia at 44 bpm that was resolved by an injection of atropine (0.01 mg/kg).

The procedure began with a maxillary down-fracture with pterygomaxillary disjunction using a curved Obwegeser osteotome and realized without difficulties or injuries. We noticed bleeding originating from the right maxillary artery, but hemostasis was immediately achieved. Then, we performed bilateral sagittal split mandibular osteotomies. The mean arterial pressure values ranged from 60 to 90 mm Hg during the entire surgery. The procedure was performed over 225 minutes, and the intraoperative blood loss was estimated at 1000 mL. No intraoperative incidents were registered. A perfusion solution of 500 ml of hydroxyethylamidon was administered intravenously with 2 injections of an antifibrinolytic drug, tranexamic acid (0.014 g/kg for 1 h followed by 0.014 g/kg for 8 h). Postoperative hemoglobin was evaluated at 13.7 g/dL.

In the recovery room, the patient complained of complete visual loss of the left eye. Eye examination revealed totally impaired vision associated with complete abolition of direct light reflex, while the fundus was normal. The CT scan only showed normal jaw osteotomies (Fig. 1A), and no irradiated split on the skull base. As left central retinal artery occlusion was suspected, a treatment by intravenous solumedrol 0.014 g/kg (once daily for 3 days), aspirin 250 mg, and enoxaparin sodium 40 mg was introduced. Two hours after the administration of medication, he complained of left frontal paresthesia. Cerebral and orbital MRI showed a slight flair hypersignal in the right internal occipital cortex and in the insular right parenchyma (Fig. 1B). No signal abnormality of the optic nerve was visualized. Antihypertensive treatment with nicardipine was introduced to decrease the elevated blood pressure during the first postoperative night.

The next day, a complete ophthalmologic examination was conducted. On the left side, the light perception and direct light reflex were missing, but the consensual light reflex was maintained. The fundus examination highlighted a clear optic nerve. Visual-evoked potentials showed absence of left eye reactivity. The diagnosis was reviewed and PION of the left eye was retained.

Polygon CT angiography showed normal permeability of the ophthalmic arteries. The carotid Doppler showed normal results. Fundal angiography was also normal. After 6 months, fundal examination revealed disc pallor on the left eye. At the final follow-up, the visual acuity of the left eye was 2/10.

DISCUSSION

Orthognathic surgery is now recognized as a safe procedure[5,6] with several minor complications: infection, excessive bleeding, bad split, tooth, and inferior alveolar nerve injury.[5,10] Severe complications such as condylar resorption, osteonecrosis, cerebrovascular accident, and carotid-cavernous sinus fistula were reported in the literature.[5] Blindness after Le Fort I osteotomy is an uncommon but serious complication, which can be isolated or associated with others nerve injuries.[5,10,13] One of the most common causes is PION. The few causes that have been described in the literature are summarized in the supplemental table, Literature Review of POVL After Orthognathic Surgery (see Table, Supplemental Digital Content 1, http://links.lww.com/SCS/A395). The exact mechanism of amaurosis is not clearly identified so that its prevention remains difficult.

In the above-mentioned case, the patient had no cardiovascular disease detected in the preoperative evaluation; however, as he presented with high arterial pressure before and after the surgery, undiagnosed hypertension could be suspected. Also, he presented with intense bradycardia after induction and a high variability in blood pressure. He immediately complained about left amaurosis associated with left ophthalmic nerve (V_1) hypoesthesia after bimaxillary orthognathic surgery. CT scan, MRI, and initial fundus examination did not present abnormalities. The 6-month examination revealed disc pallor. The recovery of the visual defect and the nerve injury were partial. During the surgery, the patient suffered from significant blood loss despite prolonged hypotension and received a significant quantity of fluids (colloids and crystalloids). These different factors may induce PION involving decreased visual acuity.

Causes of Postoperative Visual Loss and Factors Risk of ION

Classical surgical causes of POVL in orthognathic surgery are direct injuries of the visual system by irradiated fractures of the skull base or by bone fragment displacement close to the optic nerve.[7,9–11] Irradiated fractures of the skull base could be explained by incomplete pterygomaxillary disjunction or primary Le Fort I osteotomy with the presence of secondary bone callus.[7] Indirect trauma of the optic nerve can also cause POVL and can be explained by transmitted forces during the pterygomaxillary disjunction or the maxillary down fracture.[8,10]

However, ION has been reported after several other types of surgical procedures.[7,15] Some risk factors associated with these procedures include: pre-existing patient-related comorbid conditions (hypertension, diabetes, hyperlipidemia, tobacco use, obesity, anemia, sleep-apnea syndrome, or hypercoaguability), risk factors of arteriopathy, prolonged period of increased cerebral venous pressure (prone spine surgeries, surgeries in steep Trendelenburg position, or bilateral head and neck surgical procedures), exceeding procedure duration (an average duration of 6.5 h [range: 2–12 h]), amount of blood loss (mean loss, 44.7% [range: 10–200%] of estimated blood volume), and the need for intraoperative blood pressure support with vasoactive agents.[3,4,16] Another accepted risk factor is intraoperative prolonged hypotension. The 2006 report from the ASAs' POVL registry did not find any definitive causal link between hypotension and ION, although many patients experienced intraoperative hypotension. However, ASA approved the induction of intentional prolonged hypotension in patients without preoperative chronic hypertension.[3,4,15] This relative hypoperfusion can be increased by perioperative hemorrhage and blood replacement by colloids or crystalloid solutions to maintain intravascular volume. These factors are responsible for decreased oxygen delivery to the optic nerve.[16] The percentage of colloid used for nonblood replacement was inversely related to the odds ratio of ION

incidence.[3,4] Obviously, several ION risk factors were combined in our case: intense bradycardia with preoperative hypertension, massive blood loss and fluid replacement.

Theoretical Pathophysiology

Posterior ischemic optic neuropathy is characterized by ischemic optic nerve injury often on the posterior part of the optic nerve (several millimeters anterior to the optic canal). Compared with the anterior optic nerve, this area is vulnerable to hypoperfusion because of the minimal overlap of blood supply in these watershed areas. However, it is unclear if the optic nerve ischemia is caused by edema formation and compression of the small vessels, high interstitial pressure causing direct injury or venous hemorrhage or infarction.

Hypotension, hypovolemia, and anemia decrease oxygen delivery to the optic nerve. Lee et al studied the lone and combined implication of these 3 factors on the blood flow of optic nerve in pig models. They showed that isolated hypotension, anemia, or hypovolemia did not significantly modify the blood flow, but the combination of hypovolemia and hypotension resulted in significant reduction in optic nerve blood flow.[17] Anemia may only be a surrogate marker for low oncotic pressure. Higher blood loss is also frequently associated with intermittent reduction in cardiac output and perfusion.[16]

Excessive volume resuscitation resulting in hypervolemia may alter the venous outflow orbital parameters, resulting in orbital compartment syndrome.[3,4] Fluid may accumulate in the lamina cribrosa compressing the axons of this region. Replacement with colloids causes less reduction in oncotic pressure and theoretically reduces the risk of edema.

In the case presented here, the patient was suffering from intense bradycardia secondary to the anesthetic induction. Bradycardia can be explained by the action of propofol, which may induce vasomotor paralysis resulting in hypotension and bradycardia. This is particularly true in cases of preexisting uncontrolled hypertension. This intense bradycardia may be a predictive factor of a bad response to prolonged hypotension. The association of preoperative arterial hypertension, intraoperative prolonged hypotension, massive bleeding, and fluid replacement by crystalloids presented a combination of factors that led to a decreasing optic nerve blood flow resulting in left PION.

This way to explain the PION etiology is more attractive than the possibility of vascular thrombosis or surgical complication. However, because the patient was a smoking 50-year-old man, the risk that he suffered from carotid artery disease and that he embolized an atheromatous carotid plaque during surgery has to be considered. This plaque embolization could occlude vaso vasorum and induce the PION. The preoperative clinical check-up and the orbital MRI did not find any arguments for this as well as the CT examination for the PION surgical origin option. This, despite the fact that the patient underwent 2 procedures on the maxilla and that a bone callus may also explain elevated forces transmitted during the maxillary down fracture potentially participating in the optic nerve injury.

Management of Posterior Ischemic Optic Neuropathy

No treatment has been found effective to recover or to improve visual loss after orthognathic surgery.[18] Awareness of risk factors and appropriate precautions are important for prevention. Particularly for frail patients with preoperative high blood pressure, a careful consideration should be given to avoid prolonged arterial hypotension, excessive fluid replacement by colloids or crystalloids and massive hemodilution. The Practice Advisory on Peri-operative Blindness of the ASA recommends the use of colloids during prolonged surgery, and perioperative blood transfusion is generally not required for hemoglobin values >8.0 g/dL.[3,4]

Many studies have shown the benefit of therapies that aim to decrease intraorbital inflammation or swelling; including optic-sheath fenestration, systemic corticosteroids, or attempts to restore blood flow by modulating the coagulation cascade (anticoagulants, thrombolytic agents, and antiplatelet agents) and various pharmacologic agents such as acetazolamide and diuretics.[3,4,16]

CONCLUSION

POVL after orthognathic surgery is a severe complication. Surgical and anesthetic causes may have a role. However, although not all mechanisms are known, some predisposing risk factors have been identified and need to be taken into account. Patients in whom prolonged procedures, substantial blood loss, or both are anticipated need to be informed that there is a possible risk of perioperative visual loss.

REFERENCES

1. Epstein NE. Perioperative visual loss following prone spinal surgery: a review. *Surg Neurol Int* 2016;7(suppl 13):S347–S360
2. Hayreh SS. Ischemic optic neuropathies: where are we now? *Graefes Arch Clin Exp* 2013;251:1873–1884
3. American Society of Anesthesiologists. Task Force on Perioperative Blindness: practice advisory for perioperative visual loss associated with spine surgery: a report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. *Anesthesiology* 2006;104:1319–1328
4. American Society of Anesthesiologists. Task Force on Perioperative Visual Loss: practice advisory for perioperative visual loss associated with spine surgery: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. *Anesthesiology* 2012;116:274–285
5. Steel BJ, Cope MR. Unusual and rare complications of orthognathic surgery: a literature review. *J Oral Maxillofac Surg* 2012;70:1678–1691
6. Robl MT, Farrell BB, Tucker MR. Complications in orthognathic surgery: a report of 1,000 cases. *Oral Maxillofac Surg Clin N Am* 2014;26:599–609
7. Lanigan DT, Romanchuk K, Olson CK. Ophthalmic complications associated with orthognathic surgery. *J Oral Maxillofac Surg* 1993;51:480–494
8. Rodríguez-Navarro Á, Gonzalez-Valverde FM. Unilateral blindness after orthognathic surgery: hypotensive anaesthesia is not the primary cause. *Int J Oral Maxillofac Surg* 2018;47:79–82
9. Cruz AAV, dos Santos AC. Blindness after Le Fort I osteotomy: a possible complication associated with pterygomaxillary separation. *J Craniomaxillofac Surg* 2006;34:210–216
10. Wilson MW, Maheshwari P, Stokes K, et al. Secondary fractures of Le Fort I osteotomy. *Ophthal Plast Reconstr Surg* 2000;16:258–270
11. Bendor-Samuel R, Chen YR, Chen PK. Unusual complications of the Le Fort I osteotomy. *Plast Reconstr Surg* 1995;96:1289–1296
12. Girotto JA, Davidson J, Wheatly M, et al. Blindness as a complication of Le Fort osteotomies: role of atypical fracture patterns and distortion of the optic canal. *Plast Reconstr Surg* 1998;102:1409–1421
13. Lo L-J, Hung K-F, Chen Y-R. Blindness as a complication of Le Fort I osteotomy for maxillary distraction. *Plast Reconstr Surg* 2002;109:688–698
14. Cheng H-C, Chi L-H, Wu J-Y, et al. Blindness and basal ganglia hypoxia as a complication of Le Fort I osteotomy attributable to hypoplasia of the internal carotid artery: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007;104:e27–e33
15. Mathew P, Adenwalla HS, Narayanan PV, et al. A report of 2 patients with transient blindness following Le Fort I osteotomy and a review of past reported cases. *Indian J Plast Surg* 2015;48:297–300
16. Roth S. Perioperative visual loss: what do we know, what can we do? *Br J Anaesth* 2009;103(suppl 1):i31–i40
17. Lee LA, Deem S, Glenny RW, et al. Effects of anemia and hypotension on porcine optic nerve blood flow and oxygen delivery. *Anesthesiology* 2008;108:864–872
18. Lee LA, Newman NJ, Wagner TA, et al. Postoperative ischemic optic neuropathy. *Spine (Phila Pa 1976)* 2010;35(9 suppl):S105–S116