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Post-Thyroidectomy Visual Loss in a Patient with Comorbidities – Case Report

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Abstract:- Background: Post-operative vision loss (POVL) following neck surgery is an uncommon and devastating complication. While several intraoperative and postoperative risk factors have been implicated, the exact aetiology still remains unclear. We report a case of bilateral POVL in a 62-year- old female patient with multiple systemic comorbidities who had total thyroidectomy following acute respiratory obstruction from a huge compressive multinodular goitre. She had a difficult intubation complicated by cardiac arrest in the perioperative period. She noticed acute bilateral severe vision loss on the second day post-thyroidectomy. She was diagnosed with posterior ischaemic optic neuropathy, and received a short course of intravenous methylprednisolone. Thereafter, she slowly recovered her vision over a period of 10 months. This case is presented to highlight the complication that can occur after total thyroidectomy in a patient with comorbidities.

Keywords:- *Bilateral Visual Loss, Post Thyroidectomy, Comorbidities, Conservative Management, Case Report.*

I. INTRODUCTION

Post-operative vision loss (POVL) is an uncommon and unexpected complication that is most often associated with cardiothoracic surgery, instrumented spinal fusion and head and neck surgery (1,2). The incidence of postoperative vision loss after non-ocular surgery varies between 0.01 to 1% depending on the type of surgery(3). Disability from POVL can range from transient blurring or loss of vision to permanent bilateral blindness. Surgical procedures posing the highest risk are cardiac (incidence, 0.09%) and spinal surgeries with incidence as high as 0.2%(3). The recognized causes of postoperative visual loss include ischaemic optic neuropathy, central retinal artery thrombosis, cortical blindness and corneal abrasions (3-5). Ischaemic optic neuropathy (ION) is, however, the most frequently cited cause of postoperative visual loss following general anaesthesia(4).

The recognized preoperative risk factors include hypertension, diabetes mellitus, polycythaemia, smoking, renal failure, narrow angle glaucoma, atherosclerotic vascular disease and collagen vascular disorders(3). The recognized intraoperative risk factors for developing ischaemic optic neuropathy include prolonged hypotension and anaemia, estimated blood loss greater than 1 Litre, operating time greater than 6 hours, and the prone position (1,7). The aetiology of postoperative vision loss remains incompletely understood. There are few reports of post-surgical visual loss in the literature, especially following thyroidectomy. Ischaemic optic neuropathy following thyroidectomy is more uncommon in Africa. The cases of ophthalmopathy post thyroidectomy in the literature are few overall with little knowledge about the pathophysiology, though some risk factors have been identified. We, therefore, report this patient with post thyroidectomy visual loss to increase awareness of this condition among physicians, surgeons and ophthalmologists practicing in the region.

II. CASE REPORT

A 62-year old woman presented at the Emergency Department with difficulty with breathing of an hour's duration. She had had an anterior neck swelling for 10 years. The anterior neck swelling had progressively increased in size and was associated with dysphonia and snoring. There were no thyrotoxic symptoms. She was Para 1+0 and 15 years post-menopausal. She had been diabetic and hypertensive for 20 years with poor control of the hypertension. She neither smoked cigarettes nor drank alcohol. She had no known drug or food allergies. Examination revealed an obese middle-aged woman, with a Body Mass Index (BMI) of 34.1 kg/m^2. She was in respiratory distress with flaring alar nasi, but not cyanotic; she was well hydrated, not pale, anicteric, and had no pedal oedema. Her Glasgow Coma Scale Score was 7 (E-2, V-1, M-4) and the pupils were 4mm bilaterally and reacting sluggishly to light. The power in all muscle groups was normal. She had a World Health Organization (WHO) Grade III goitre without scalp swelling or exophthalmos, and the trachea was deviated to the right. The goitre was non-tender with normal overlying skin. It was multinodular with the left lobe larger than the right. There was no retrosternal extension, or cervical lymphadenopathy. She was tachypnoeic and dyspnoeic with a respiratory rate of 36/min, SPO_2 ranged between 80 and 84% on the pulse oximeter. There were crepitations in both lung fields. The pulse was 128/min, the blood pressure was 240/70 mmHg and the heart sounds were normal. The abdomen was normal. A clinical diagnosis of acute upper airway obstruction secondary to a WHO Grade III simple multinodular goitre was made.

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There were initial failed attempts at endotracheal intubation and tracheostomy probably due to inexperience of the attending, following which she had a cardiac arrest. However, she was successfully intubated and resuscitated subsequently. She was admitted into the Intensive Care Unit (ICU) and was placed on oxygen via endotracheal tube, commenced on nasogastric tube feeding, antibiotics, analgesics (IV ceftriaxone 1g 12hourly and IV Febramol 1g 8hourly respectively), Ramipril and subcutaneous Clexane. The chest radiograph showed accentuated lung markings, enlarged heart (Cardio-thoracic Ratio: 0.58) with a left ventricular preponderance. There were no focal lung lesions. The electrocardiograph, serum Troponin I and T were normal. The echocardiography was normal with a ventricular ejection fraction of 77%. The blood glucose levels ranged from 110mg /dL to 120mg/dL

She had a total thyroidectomy on the 5th day of admission, surgery delayed to allow some time for the heart to recover from earlier cardiac arrest. The immediate preoperative blood pressure was 130/88mmHg. The intra-operative findings were a 360g goitre with the left lobe larger than the right. The trachea was slightly kinked at the lower end. The endotracheal tube was therefore left in situ after surgery. The estimated blood loss was 750 mls and she received 500 mls whole blood transfusion. The post-operative Packed Cell Volume was 33%. She was extubated on the 2nd postoperative day. However, she had respiratory distress and poor blood oxygen saturation and was re-intubated.

While she was fully conscious on the second day post-operation, she complained of bilateral visual loss. Ophthalmic assessment showed that visual acuity was light perception in both eyes, the pupils were regular, round, reactive to light and fundoscopy was normal. Assessment of visual field by confrontation or any other method was obviated by the severity of vision loss. Following a neuro-ophthalmological examination, a diagnosis of bilateral posterior ischaemic optic neuropathy was made. Magnetic Resonance Imaging(0.4Tesla) of the brain done on the 10th post-operative day did not reveal any abnormality. She received six doses of intravenous methylprednisolone 500mg 12hourly over 72 hours, followed by antioxidant (containing bilberry extract) for neuro-protection. She remained in the Intensive Care Unit with a turbulent post-operative course on endotracheal intubation and supplemental oxygen for 12 days. She had fluctuating hypertension, though her diabetes was controlled (with SC soluble Insulin). She had a single episode of left sided seizure which later became generalised, with confusion and irrational behaviour on the 10th post-operative day, and these were controlled with Intravenous midazolam.

She was thereafter commenced on replacement *l*-thyroxine tablets of 100 micrograms daily, antihypertensive medications (Ramipril 5mg daily, Nifedipine 20mgBD, Natrilix 1.5mg daily) and oral antidiabetic agent (Tabs Metformin 500mgBD). The histology of the total thyroidectomy specimen showed papillary carcinoma of the thyroid and she was, therefore, referred for radio-iodine therapy.

Her vision improved slightly to hand motion on the 15th post-operative day (five days after completing intravenous methylprednisolone) and vision remained at this level till she was discharged home after 20 days of admission. She was followed up in the neuro-ophthalmology clinic four weeks post-operation and her vision remained at the same level of hand motion bilaterally. Pupils were sluggish but reacting bilaterally and she had bilateral temporal disc pallor in a cup-disc ratio of 0.4. She was placed on brimonidine eyedrops and continued on oral antioxidant therapy(bilberry). She also had refraction, with improvement in her vision to 6/60 in the right eye and 6/36 in the left eye with a +2.00 DS-1.00 DC x 90º spectacle prescription in each eye. She was referred for low-vision assessment. However, she did not keep that clinic appointment. The last evaluation was at 10 months post-operation, with unaided vision of 6/36 in the right eye and 6/36+1 in the left eye, which improved to 6/9-1 in both eyes, with her glasses. She had sluggish pupils bilaterally with temporal disc pallor and cup-disc ratio of 0.5 bilaterally. The intraocular pressures were normal throughout the period of vision loss.

III. DISCUSSION

Post-operative vision loss is an uncommon and distressing complication of non-ocular surgery. While ischaemic optic neuropathy is the commonest cause of post-operative vision loss, the American Society of Anesthesiologists (ASA) closed claims database reports that 81% of postoperative visual loss is diagnosed as ischaemic optic neuropathy.

Other causes include corneal exposure and abrasion, central retinal artery occlusion and cortical blindness from occipital stroke(3).

Ischaemic optic neuropathy can be classified into anterior and posterior subtypes. Anterior ischaemic optic neuropathy (AION) is related to the disruption of the optic nerve blood supply through the posterior ciliary arteries while posterior ischaemic optic neuropathy(PION) results from disturbance to the pial blood vessel supply to the posterior portions of the optic nerve(6). PION is far less common than AION and the underlying mechanisms and pathogenesis of PION are still not fully understood. We considered the post-operative vision loss in this patient to be due to bilateral PION because of her background comorbidities, which present significant risks for impaired microvascular circulation, as well as the lack of pallor and swelling of the optic nerve head at the onset of vision loss.

The patient developed severe bilateral simultaneous vision loss upon recovery from general anaesthesia, following thyroidectomy, which had been preceded by difficult intubation and cardiac arrest in the preoperative period and complicated by respiratory distress following post-operative extubation. She was managed in the acute stages of her vision loss with intravenous methylprednisolone, proprietary antioxidants tablets and topical brimonidine (for neuroprotection).

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We report this case because post-operative vision loss following thyroidectomy is rare, while visual recuperation following such post-operative vision loss is even rarer. While the susceptibility of this patient may have been linked to the numerous comorbidities and cardiac arrest, it is likely that she suffered ischaemia to the posterior portions of the optic nerves, either as a result of hypoxia during the period of cardiac arrest or from fluctuations in her haemodynamic state intraoperatively. However, male gender, prone position during surgery, hypotension, prolonged procedures and anaesthesia duration, and decreased use of colloids are associated with Post-operative vision loss. Modifiable risk factors for microvascular ischaemia include obesity, cardiovascular disease, hyperlipidaemia, diabetes mellitus, and smoking. Therefore, the potential risk factors in this patient were obesity, poorly controlled hypertension, diabetes mellitus, hypoxia and peri-operative cardiac arrest.

In AION, there is swelling of the optic disc, whereas in PION or retrobulbar type, the optic disc initially appears normal. However, in both types, the optic disc becomes pale over time if there is irreversible damage to the nerve. It may be unilateral or bilateral(7). The posterior part of the optic nerve is farthest from its arterial supply and is most commonly implicated in postoperative visual loss associated with haemorrhagic hypotension. The pial vessels supplying the posterior portion of the optic nerve are incapable of autoregulatory control and therefore that portion is particularly vulnerable to a fall in perfusion pressure or anaemia. (13) Posterior ischaemic optic neuropathy (PION) is usually a diagnosis of exclusion as there are often no abnormal ophthalmoscopic findings. Posterior ION is most commonly associated with operations performed in the prone position and of longer duration, and typically presents as painless loss of vision when the patient recovers from anaesthesia. (8) PION has been well reported following cardiac surgery requiring cardiopulmonary bypass, but it may also occur after head and neck operations and after spine surgery.(7)

It is suggested that the pathogenesis underlying progressive nerve damage starts with compression and hypoxia, then axon depolarization, disruption of the cytoskeleton, demyelination, and finally axonal degeneration and/or apoptosis (irreversible nerve damage). The degree of the compression and the rapidity with which it occurs may play a significant role in determining the amount of nerve damage and its potential recovery(9).

In the index patient, several factors could have contributed to the postoperative vision loss. The most common risk factors are intraoperative hypotension, anaemia and hypoxia. Surgery lasted almost four hours. The subsequent improvement in vision following the administration of high dose intravenous methylprednisolone is also more in keeping with non-arteritic PION(6,10,11). Moreover, the patient had numerous microvascular risk factors for non-arteritic PION.

Although the time of onset presupposes a post-surgical mechanism, literature shows that the significant visual recovery is uncharacteristic of surgical PION(10). On the other hand, non-arteritic PION seems more likely because of the microvascular comorbidities, especially the poorly controlled hypertension, obesity and diabetes, complicated by hypoxia resulting from acute respiratory distress and cardiac arrest. Furthermore, it has been reported that early administration of intravenous methyl prednisolone is associated with significant visual recovery specifically in cases of non-arteritic PION like in this patient. While steroids are important in preventing further visual loss in arteritic PION, they do not result in visual recovery.

Isayama and colleagues proposed the following criteria for a diagnosis of idiopathic PION: (1) sudden onset of unilateral visual disturbance in older patients: (2) normal optic disc, subsequently developing simple optic atrophy; (3) hypertensive and arteriosclerotic changes in the retinal vessels; (4) varying degrees of impaired vision, variable visual field defects; (5) associated systemic disease such as hypertension, diabetes mellitus, hyperlipidemia, hypotension(6) exclusion of other demonstrable causes of optic nerve disturbances, and (7) confirmation of abnormal hemodynamics in the posterior portion of the optic nerve by carotid angiography, ophthalmodynamography, ophthalmodynamometry and fluorescein fundus angiography(11). This patient fulfilled almost all these criteria except that we were unable to perform carotid angiography, ophthalmodynamometry or fundus fluorescein angiography to demonstrate abnormal haemodynamic flow in the posterior segments of the optic nerve.

Low-resolution magnetic resonance images of the brain showed no gross abnormality although, this study did not include diffusion weighted imaging sequences.

Although our case clearly illustrates the same potential risk factors for postoperative ION that are described in literature, it fails to shed any light on the precise mechanism or mechanisms by which the vision loss occurred. Although no treatments have been proved to improve vision in postoperative ION, several groups recommend treatment with systemic corticosteroids and transfusion to a haematocrit above 30%.(5). Nevertheless, this case suggests that visual recovery in PION may be very slow and may continue for months after initial steroid therapy.

IV. CONCLUSION

In conclusion, a combination of microvascular risk factors combined with cardiac arrest, intraoperative haemodynamic instability, predisposing to further hypoxia, contributed to the post-operative visual loss in this patient. This patient is being reported to highlight the possibility of post thyroidectomy visual loss in a patient with pressure symptoms, cardiac arrest, uncontrolled hypertension and diabetes.

REFERENCES

[1]. Grover VK, Jangra K. Perioperative vision loss: A complication to watch out. J Anaesthesiol Clin Pharmacol. 2012;28(1):11–6.
[2]. Nickels TJ, Manlapaz MR, Farag E. Perioperative visual loss after spine surgery. World J Orthop. 2014;5(2):100–6.
[3]. Mendel E, Stoicea N, Rao R, Niermeyer W, Revilla S, Cluse M, et al. Revisiting Postoperative Vision Loss following Non-Ocular Surgery: A Short Review of Etiology and Legal Considerations [Internet]. Vol. 4, Frontiers in Surgery . 2017. p. 34. Available from: https://www.frontiersin.org/article/10.3389/fsurg.2017.00034
[4]. Quraishi NA, Wolinsky JP, Gokaslan ZL. Transient bilateral post-operative visual loss in spinal surgery. Eur Spine J. 2012;21 Suppl 4:498–501.
[5]. Brown RH, Schauble JF, Miller NR. Anemia and hypotension as contributors to perioperative loss of vision. Vol. 80, Anesthesiology. 1994. p. 222–6. DOI:10.1097/00000542-199401000-00033
[6]. Hayreh, Sohan Singh. "Posterior Ischemic Optic Neuropathy." Ophthalmologica 182.1 (1981): 29–41. Web. https://doi.org/10.1159/000309085
[7]. Chang, Shu-Hong MD; Miller, Neil R. MD The Incidence of Vision Loss due to Perioperative Ischemic Optic Neuropathy Associated With Spine Surgery, Spine: June 1, 2005 - Volume 30 - Issue 11 - p 1299-1302 doi: 10.1097/01.brs.0000163884.11476.25
[8]. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, et al. The American society of anesthesiologists postoperative visual loss registry: Analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology. 2006;105(4):652–9.
[9]. Devoto MH, Golnik K, Bernardini FP, Alencar VM. Improvement from no light perception after orbital decompression for graves' optic neuropathy. Ophthalmology. 2014;121(1):431–3.
[10]. Sadda SR, Nee M, Miller NR, Biousse V, Newman NJ, Kouzis A. Clinical spectrum of posterior ischemic optic neuropathy. Am J Ophthalmol. 2001;132(5):743–50.
[11]. Isayama Y, Takahashi T, Inoue M, Jimura T: Posterior Ischemic Optic Neuropathy. Ophthalmologica 1983;187:141-147. doi: 10.1159/000309314
[12]. Williams JR. The Declaration of Helsinki and public health. Bull World Health Organ. 2008;86(8):650–2.
[13]. D. Kamming, S. Clarke, Postoperative visual loss following prone spinal surgery, *BJA: British Journal of Anaesthesia*, Volume 95, Issue 2, August 2005, Pages 257–260, https://doi.org/10.1093/bja/aei173