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CASE REPORT

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

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## Abstract

**Introduction:** Post-operative vision loss (POVL) following neck surgery is an uncommon and catastrophic complication. While several intraoperative and postoperative risk factors have been implicated, the exact aetiology still remains vague.

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.

**Conclusion:** 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

## Introduction

Post-operative vision loss (POVL) is an unexpected complication that is most often associated with instrumented spinal fusion and head and neck surgery [1,2]. The incidence of postoperative vision loss after non-ocular surgery is 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. The causes of postoperative visual loss include ischaemic optic neuropathy, central retinal artery thrombosis amongst others [3-5]. Ischaemic optic neuropathy (ION) is the most frequently cited cause of postoperative visual loss following general anaesthesia [5].

The preoperative risk factors include hypertension, diabetes mellitus, polycythaemia, smoking, renal failure [3]. The intraoperative risk factors include prolonged hypotension and anaemia, blood loss greater than 1 Litre, operating time greater than 6 hours [1,6]. The aetiology of postoperative vision loss remains obscure.

There are few reports of post-op visual loss in the literature, especially following thyroidectomy. We report this patient with post thyroidectomy visual loss to increase awareness of this condition among physicians, surgeons and ophthalmologists.

## Case Report

A 62-year-old woman presented at the Emergency Department with difficulty in breathing of an hour duration. She had an anterior neck swelling for 10 years which progressively increased in size and associated with dysphonia and snoring. There were no thyrotoxic symptoms. She is Para 1+0 and 15 years post-menopausal. She had been diabetic and hypertensive

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for 20 years with poor control of the hypertension. She neither smoked cigarettes nor drank alcohol. She had no known drug or food allergies.

## Clinical findings

An obese middle-aged woman, with a Body Mass Index (BMI) of 34.1 kg/m$^2$. She was in respiratory distress but not cyanotic; 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 4 mm bilaterally and reacting sluggishly to light. The power in all muscle groups was normal. She had World Health Organization (WHO) Grade III goitre non-tender, no scalp swelling or exophthalmos and trachea deviated to the right. It was multinodular with the left lobe larger than the right, 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%. There were crepitations in both lung fields, 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.

There were initial failed attempts at endotracheal intubation and tracheostomy following which she had a cardiac arrest. However, she was successfully intubated and resuscitated subsequently. She was admitted into the Intensive Care Unit (ICU) on oxygen via endotracheal tube, commenced on nasogastric tube feeding, antibiotics, analgesics (IV ceftriaxone 1g 12 hourly and IV Febramol 1g 8 hourly 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 110 mg/dL to 120 mg/dL. She had a total thyroidectomy on the 5$^{th}$ day of admission to allow time for the heart to recover from previous cardiac arrest.

The immediate preoperative blood pressure was 130/88 mmHg. The intra-operative findings were a 360g goitre, left lobe larger than the right. The endotracheal tube was left *in situ* after surgery cause of trachea kink. The estimated blood loss was 650 mls and she received 500 mls whole blood transfusion. The post-operative Packed Cell Volume was 33%. She was extubated on the 2$^{nd}$ postoperative day. However, she had respiratory distress and poor blood oxygen saturation, she was re-intubated following which she complained of bilateral visual loss.

## Diagnostic assessment

Ophthalmic evaluation 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 (0.4 Tesla) of the brain done on the 10$^{th}$ post-operative day did not reveal any abnormality.

## Therapeutic intervention

She received six doses of intravenous methylprednisolone 500 mg 12 hourly over 72 hours, followed by antioxidant (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 10$^{th}$ 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 5 mg daily, Nifedipine 20 mg BD, Natrilix 1.5 mg daily) and oral antidiabetic agent (Tabs Metformin 500 mg BD). The histology of the total thyroidectomy specimen showed papillary carcinoma of the thyroid and she was referred for radio-iodine therapy.

Her vision improved slightly to hand motion on the 15$^{th}$ 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.

## Follow-up and outcomes

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 eye drops 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 × 90° spectacle prescriptions 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.

## Discussion

Post-operative vision loss is an uncommon and distressing complication of non-ocular surgery. While ischaemic optic neuropathy (ION) is the commonest cause of post-operative vision loss, the American Society of Anaesthesiologists (ASA) reported that 81% of postoperative visual loss is diagnosed as ischaemic optic neuropathy [7].

ION can be classified into anterior and posterior subtypes. Anterior ischaemic optic neuropathy (AION) is 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 [4]. Although AION is more common than PION 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.

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 postoperative vision loss. 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, 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 [6]. 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 prone to fall in perfusion pressure or anaemia [7]. PION is usually a diagnosis of exclusion as there are often no abnormal ophthalmoscopic findings as with the index case, it's 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 is documented to occur following head and neck operations [6].

It is suggested that the pathogenesis underlying progressive nerve damage starts with compression and hypoxia leading to eventual apoptosis (irreversible nerve damage) and degree of compression may play a significant role in determining the severity 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, hypoxia and surgery lasted almost four hours. The subsequent improvement in vision following the administration of high dose Intravenous Methylprednisolone is also in keeping with non-arteritic PION [10-12]. 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 [12]. 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 Methylprednisolone 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 fluoresce in 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 hemodynamic 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% [4]. Nevertheless, this case suggests that visual recovery in PION may be very slow and may continue for months after initial steroid therapy.

## 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 has been reported to highlight the possibility of post-thyroidectomy visual loss in a patient with pressure symptoms, cardiac arrest and comorbidities so surgeons can be on the look-out for this rare scenario.

## Patient Perspective

The patient was satisfied with recovery attained.

## Informed Consent

Informed consent was obtained.

There were no conflicts of interest.

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