

Visual loss following head and neck surgery

A J B CROCKETT¹, A TRINIDADE¹, P KOTHARI¹, J BARNES²

Departments of ¹ENT and ²Ophthalmology, Luton and Dunstable Hospital, Luton, UK

Abstract

Introduction: Non-arteritic ischaemic optic neuritis is a known post-operative complication of ophthalmological and maxillofacial surgery, but has not been widely described as a potential consequence of head and neck surgery.

Aim: To highlight non-arteritic ischaemic optic neuritis as a potential risk in patients undergoing head and neck surgery.

Subject and method: Case report of a 60-year-old man undergoing total laryngectomy and bilateral neck dissection for laryngeal squamous cell carcinoma.

Result: On day 14 post-operatively, the patient suffered substantial oral bleeding secondary to an internal jugulo-neopharyngeal fistula. Following emergency haemostatic measures, the patient was immediately aware of visual disturbances. The ophthalmologists concluded that these were due to non-arteritic ischaemic optic neuritis, caused by acute hypoxaemia secondary to substantial blood loss.

Conclusion: The prognosis of non-arteritic ischaemic optic neuritis is poor. Thus, it is crucial that otolaryngologists are aware of this complication of head and neck surgery, as immediate diagnosis and treatment can help prevent worsening visual loss.

Key words: Neck Dissection; Optic Neuritis; Blindness; Complications

Introduction

Non-arteritic ischaemic optic neuritis normally presents on waking as a painless visual disturbance in one or both eyes. On investigation, these patients are found to have reduced visual acuity and visual field loss. Predisposing factors include systemic vasculopathies such as hypertension, diabetes and atherosclerosis. The precise aetiology is still currently unclear; however, theories include hypotension, blood loss and hypertension of the ophthalmic artery, anaemia, long procedural duration, surgical positioning, and facial swelling. Post-operative non-arteritic ischaemic optic neuritis usually results in severe, irreversible, bilateral visual loss.

Case report

A 60-year-old man presented to our department with ongoing stridor, dysphagia and hoarseness of voice. He had previously been well, with a past medical history only of arthritis, for which he had undergone a left knee replacement. The patient had smoked 40 cigarettes a day for the past 40 years, and had regularly drunk six to 10 units of alcohol a day.

Nebulised adrenaline and intravenous dexamethasone (8 mg twice daily) improved his stridor.

Panendoscopy revealed a left-sided vocal fold mass, which histological analysis identified as a squamous cell carcinoma. A staging computed tomography (CT) scan of the neck and chest revealed a 5 cm, transglottic mass crossing the midline, with destruction of the left superior cricoid, left arytenoid and inferior thyroid lamina, and extending to

and involving the epiglottis and aryepiglottic folds, with effacement of the pyriform fossae. There was also a left-sided, 8 mm, level IV node, giving a tumour-node-metastasis staging of T₄ N₁ M₀ laryngeal squamous cell carcinoma.

A multidisciplinary team decision was made to undertake a total laryngectomy and bilateral selective neck dissection, which were performed without incident.

Post-operatively, the patient was electively admitted to the intensive therapy unit, and was transferred to the ENT ward two days later. The patient made a good immediate recovery, with no visual disturbance.

However, on day 14 post-operatively the patient developed sudden, substantial oral bleeding. He was taken immediately to the operating theatre for exploration, where it was found that the right internal jugular vein had broken down in three places, and that a 4 cm right internal jugulo-neopharyngeal fistula had formed. Haemostasis was achieved, the internal jugular vein was repaired by oversewing, and the fistula was primarily closed. The patient was once again transferred to the intensive therapy unit.

On waking from the second operation, the patient was immediately aware of visual disturbance of his left eye. Investigation was undertaken by the ophthalmologists, who found reduced visual acuity bilaterally (right eye acuity = 6/5; left eye acuity = 6/12) and reduced left eye colour vision. There was a left-sided relative afferent pupillary defect with bilateral peripheral visual loss (Figures 1 and 2). There were no clinical signs or symptoms of vasculitis.

The patient was treated with oral corticosteroids for 10 days. On ophthalmological review, there was no change in visual acuity, but there was an improvement in the

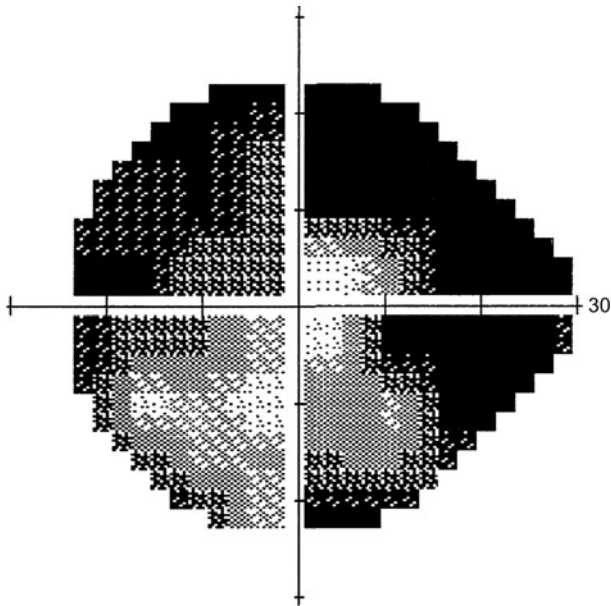


FIG. 1

Visual field tests showing left-sided relative afferent pupillary defect and peripheral visual loss.

left-sided visual field. However, on the right there was also a sharp altitudinal defect affecting the inferior nasal field. Both optic discs were swollen, with ‘flame’ haemorrhages adjacent to the right optic disc margin (Figures 3 and 4).

A CT scan of the orbits showed no pathology. Nonetheless, the ophthalmologists concluded from the clinical picture that the patient had suffered non-arteritic ischaemic optic neuritis secondary to acute hypoxia caused by substantial blood loss as a result of his jugulo-neopharyngeal fistula.

Follow up at six months revealed no further improvement in the patient’s vision.

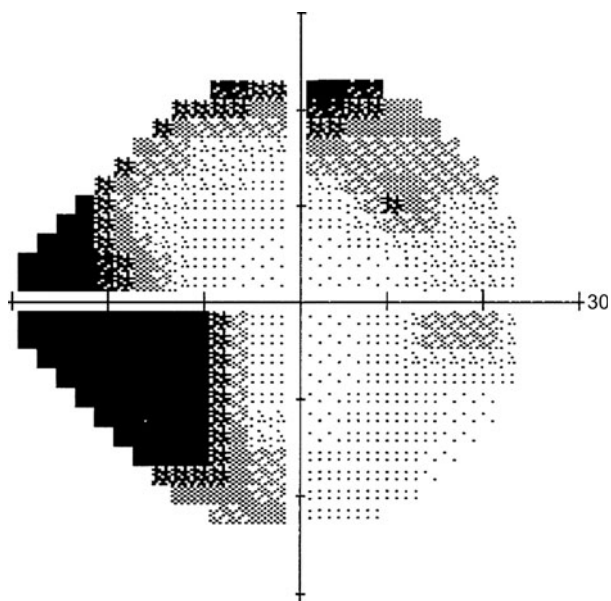


FIG. 2

Visual field tests showing right peripheral visual loss.

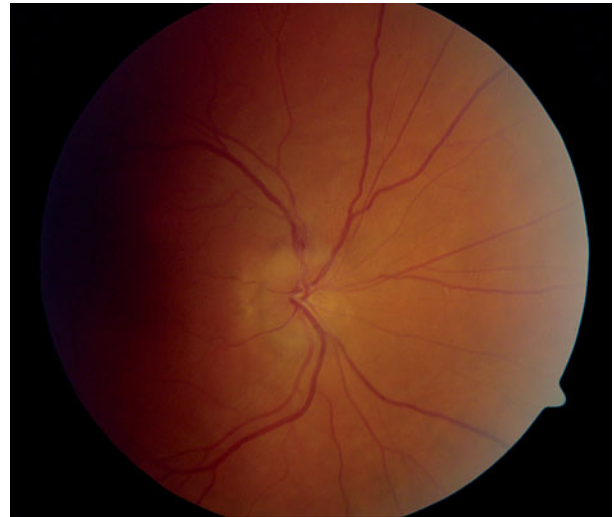


FIG. 3

Fundoscopy photograph of left fundus showing a swollen optic disc.

Discussion

Post-operative non-arteritic ischaemic optic neuritis has been mainly reported in ophthalmology, anaesthetic and maxillo-facial journals.¹ Anecdotally, discussion within the ENT community has revealed that most ENT head and neck consultants are unaware of blindness secondary to non-arteritic ischaemic optic neuritis as a potential risk of surgery.

In the majority of reported cases of post-operative non-arteritic ischaemic optic neuritis following head and neck surgery, patients underwent bilateral neck dissections which involved increased venous congestion peri-operatively, usually due to jugular vein ligation.^{2,3} In our case, both internal jugular veins were preserved; however, the right jugular vein was later found to have broken down.

Non-arteritic ischaemic optic neuritis is still poorly understood. The clinical presentation is usually a unilateral, painless, sudden loss of vision and visual fields, affecting



FIG. 4

Fundoscopy photograph of right fundus, showing a swollen optic disc with ‘flame’ haemorrhages adjacent to the right optic disc margin.

patients over the age of 50 years and with a higher incidence in Caucasians. Loss of vision is usually noticed on waking. The exact mechanism of action is still unclear, but it is thought to be an ischaemic process affecting short posterior ciliary arteries that supply the optic nerve at its exit from the eye, leading to infarction of the laminar or retrolaminar portion of the optic nerve.^{4,5} At this point, it is thought that axonal swelling can occur, causing a compartment syndrome which results in axonal degeneration.⁶

- **Non-arteritic ischaemic optic neuritis is a known complication of ophthalmological and maxillofacial surgery**
- **It is not widely known as a complication of head and neck surgery**
- **It can be caused by acute hypoxaemia due to massive blood loss**
- **Permanent post-operative visual loss can occur**
- **The prognosis is poor; immediate recognition and treatment are critical to reduce severity**

Fundoscopy normally reveals a pale, swollen optic disc which can sometimes be associated with flame haemorrhages and 'cotton-wool' exudates. Visual loss is usually sudden, but has been reported to occur over a few days at most, and is usually permanent. Recovery is rare, but can sometimes occur within the first few weeks or months. Optic atrophy often ensues within the next few weeks. Initial CT imaging is classically unrevealing.

A number of risk factors have been proposed, including atherosclerosis, diabetes, hyperlipidaemia, hypertension, hypotension, haemoconcentration, haemodilution, hypercoagulable states, and small or crowded optic discs.⁷

Currently, there is no proven effective treatment for non-arteritic ischaemic optic neuritis. However, both medical and surgical interventions are being investigated, as is the pathophysiology of the condition.^{8,9}

Conclusion

Non-arteritic ischaemic optic neuritis is a rare complication of head and neck surgery, and in particular of neck dissection. It should be borne in mind in patients complaining of visual loss following neck dissection, and when managing any arising complications. Currently, the prognosis of

non-arteritic ischaemic optic neuritis is poor, and no proven effective treatment is available. However, it is critical that ENT teams be aware of this complication of head and neck surgery, as prompt diagnosis and treatment may to some degree help prevent further visual loss and ameliorate existing damage.

References

- 1 Schobel GA, Schmidbauer M, Millesi W, Undt G. Posterior ischemic optic neuropathy following bilateral radical neck dissection. *Int J Oral Maxillofac Surg* 1995;**24**:283–7
- 2 Aydin O, Memisoglu I, Ozturk M, Altintas O. Anterior ischemic optic neuropathy after unilateral radical neck dissection: case report and review. *Auris Nasus Larynx* 2008;**35**:308–12
- 3 Wilson JF, Freeman SB, Breene DP. Anterior ischemic optic neuropathy causing blindness in the head and neck surgery patient. *Arch Otolaryngol Head Neck Surg* 1991;**117**:1304–6
- 4 Nawa Y, Jaques JD, Miller NR, Palermo RA, Green WR. Bilateral posterior optic neuropathy after bilateral radical neck dissection and hypotension. *Graefes Arch Clin Exp Ophthalmol* 1992;**30**:301–8
- 5 Holy SE, Tsai JH, McAllister RK, Smith KH. Perioperative ischemic optic neuropathy: a case control analysis of 126,666 surgical procedures at a single institution. *Anesthesiology* 2009;**110**:246–53
- 6 Kerr NM, Chew SS, Danesh-Meyer HV. Non-arteritic anterior ischemic optic neuropathy: a review and update. *J Clin Neurosci* 2009;**16**:994–1000
- 7 Torossian A, Schmidt J, Schaffartzik W, Wulf H. Loss of vision after non-ophthalmic surgery: systematic review of the literature on incidence, pathogenesis, treatment and prevention [in German]. *Anaesthesist* 2006;**55**:457–64
- 8 Buono LM, Foroozan R. Perioperative posterior ischemic optic neuropathy: review of the literature. *Surv Ophthalmol* 2005;**50**:15–26
- 9 Obuchowska I, Mariak Z. Perioperative posterior ischemic optic neuropathy – pathogenesis and clinical characteristics. *Klin Oczna* 2009;**111**:375–7

Address for correspondence:

Mr Aaron Trinidad,
Department of ENT,
Luton and Dunstable Hospital,
Lewsey Road,
Luton LU4 0DZ, UK

Fax: +44 (0)1582 718 281

E-mail: aarontrinidad@gmail.com

Mr A Trinidad takes responsibility for the integrity of the content of the paper
Competing interests: None declared
