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Posterior Ischemic Optic Neuropathy After Radical Neck Disection

Radikal Boyun Diseksiyonu Sonrasında Gelişen Posterior Optik Nöropati

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ABSTRACT

Blindness is a very rare and unexpected complication of neck dissection. We presented a case of a posterior ischemic optic neuropathy (PION) after a staged radical neck dissection two years apart from each other. There is no effective treatment for surgical PION has been described. Therefore, taking preventive measures is the most important step.

Keywords
Radical neck dissection;
posterior ischemic optic neuropathy

ÖZET

Körlük, boyun diseksiyonu sonrasında gelişen çok nadir ve beklenmeyen bir komplikasyondur. Çalışmamızda iki yıl ara ile aşamalı boyun diseksiyonu sonrasında posterior iskemik optic nöropati (PION) gelişen bir hasta sunulmuştur. Cerrahi sonrasında gelişen PION'nun bilinen efektif bir tedavisi yoktur. Bu yüzden koruyucu önlemlerin alınması çok önemlidir.

Anahtar Sözcükler
Radikal boyun diseksiyonu;
posterior iskemik optic nöropati

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INTRODUCTION

Blindness is a very rare and unexpected complication of neck dissection. To our knowledge, there are 15 published cases in the literature.[1-16] It was first reported by Milner in 1960 after a staged bilateral neck dissection performed 2 months apart.[1] Blindness occured after radical neck dissection in all reported cases except the one published by Öğretmenoğlu et al. in which the internal juguler vein was preserved.[12]

Distinct parts of the optic tract are affected in various cases. There are different theories about the etiology of the complication. Predominating theories are anterior (AION) and posterior (PION) ischemic optic neuropathies.[6,7,13] Other theories include central retinal artery occlusion with an emboli, optic atrophy, and blindness due to massive hemorrhage.[1-3] In most of the cases the probable etiologic factors responsible for blindness are hypotension, anemia and hemodilution. Herein we present a case report of a posterior ischemic optic neuropathy after a staged radical neck dissection which was performed two years later.

CASE REPORT

Our patient was a 58-years-old Turkish man, who was being followed-up with palatal malign melanoma. He was diagnosed 4 years ago and had brachytherapy. Left radical neck dissection had been performed two years ago due to neck metastasis and adjuncted with interferon treatment. During his routine follow up, a 10x5 cm mass developed on the right side of neck therefore contralateral radical neck dissection was planned. His medical history revealed previous diagnosis of hypertension (HT), diabetes mellitus (DM) and a right facial paralysis due to which he had a facial decompression surgery. The preoperative hemoglobin and hematocrit levels were 13.4 g/dl and 38.8% respectively. All other laboratory investigations and electrocardiogram were normal. His preoperative blood pressure was approximately 140/90 mmHg and his mean pulse rate was 85/min.

Right radical neck dissection, which lasted 8 hours was performed. The patient received 5400 ml cristalloidal, 1000 ml colloidal solutions and 300 ml red blood cell suspension intraoperatively. His average blood glucose level was 220 mg/dl during the surgery with infusion of ¼ neutralized 5% Dextrose solutions. During the operation average blood pressure was 100/60 mmHg and total urine output was 5350 ml. The final hemoglobin and hematocrit levels were 7.7 gr/dl and 21.6% respectively.

At the end of the surgery he had mild facial edema with ocular chemosis. These signs improved in 2 days with diuresis and head elevation. Bilateral visual loss was noticed on the first postoperative day. His ophthalmological examination revealed only light perception with absence of light reflex in the left eye; however he was able to see hand movements from a 30 cm distance with a weak light reflex in the right eye. The posterior segment examinations of both eyes were normal. Rest of his neurological examination was normal. There was no pathological finding on cranial MRI. Visual evoked potential test revealed conduction defect in both visual pathways. The right visual defect recovered spontaneously during his 2-weeks clinical follow-up. The patient was able to count fingers from a 3 meter-distance at the time of discharge. Afterwards, interferon therapy was planned due to the presence of eight metastatic lymph nodes.

DISCUSSION

Bilateral radical neck dissections were initially performed succesfully in 1949. The first reported case of blindness was reported in 1960 by Milner.[1] Blindness is a very rare complication of neck dissection with only 15 cases reported in the literature.[1-16] The affected parts of the optic tract in various case reports are also different: anterior or posterior ischemic optic neuropathies or occipital infarctions. In our clinic, we had another experience in which the patient had developed a right-sided occipital lobe infarction following modified radical neck dissection with preservation of the internal jugular vein. His postoperative angiography revealed occlusion of bilateral common carotid arteries.[12] Surprisingly, there were no findings suggesting either occipital infarction or anterior ischemic optic neuropathy in this case. All other neurological and ophthalmoscopic examinations and cranial MRI were normal. Excluding other etiological factors, we concluded that the cause of blindness in this case was PION.

Hayreh classifies PION into three types according to their etiologies: (a) arteritic PION due to giant cell arteritis (GCA), (b) nonarteritic PION related to causes other than GCA, and (c) surgical PION attributable to a surgical procedure.[17] He pointed out in his paper that,

the diagnosis of PION should be made after all other possibilities are carefully ruled out. The term 'surgical PION' was used for the entity and explained the pathology as being multifactorial, including severe and prolonged arterial hypotension, hemodilution, chemosis, anemia and direct orbital compression.

In the literature, there are some surgical PION cases, almost invariably associated with prolonged systemic surgical procedures, including spinal and other orthopaedic surgical procedures, radical neck dissections, venous grafting in extremities, coronary artery bypass, hip surgery, nasal surgery, thoracotomy for hemothorax, penetrating thoracoabdominal surgery, cataract surgery and strabismus surgery.[5,8,11,13,17-19] The exact incidence of perioperative PION is not known, although it appears to be very low. Balm et al reported that out of 1200 neck dissections, only one patient (0.08%) had visual loss resulting from PION.[7] In their review of 83 perioperative PION cases, Buono and Foroozan found that spinal surgery and radical neck dissections are the procedures after which most of the PION cases are seen.[18] In many other otolaryngologic surgeries, like endoscopic sinus surgeries and otologic surgeries, hypotensive anesthesia is employed in order to reduce bleeding. However there are no reports of PION following these operations. This gives rise to the thought that, venous congestion is the primary factor in the pathophysiology of the PION and hypotension may be a secondary etiologic factor in deteriorating the already disturbed blood perfusion.

The pathogenesis of surgical PION is multifactorial.[13,17,18] The leading factors causing optic nerve ischemia are anemia and hypotension pre or perioperatively. Internal jugular vein ligation, often performed as a part of radical neck dissection usually increases orbital venous pressure which may lead to a decrease in arterial perfusion pressure causing ischemia. To prevent such a complication, bilateral radical neck dissection should be performed sequentially, but in the literature there is a case in which surgical PION is seen although the dissections were done 9 years apart.[13] Contralateral eye was predominantly affected in our patient. This might be due to more dependant position of the left eye or constriction of the contralateral neck vessels due to head rotation. Patients with an atherosclerotic disease who experience decreased perfusion pressure may also have a greater risk for developing PION.[18] In our case, the duration of the surgery was long and during this period our patient was hypotensive. Anemia, as a result of blood loss and hemodilution, might be another possible factor in our patient. Either internal jugular vein ligation or DM are other possible risk factors for our patient. Direct orbital compression can cause perfusion problems as well.[17] Clearly hemodynamic derangements play a role in the pathogenesis of PION but not all individuals are equally susceptible to these effects; otherwise, perioperative PION would be more common. The facts that the intraorbital optic nerve is selectively infarcted and that the remaining central nervous system is spared in most cases strongly suggest that the optic nerve is more susceptible to the effects of hemodynamic derangements in some patients. The nature of this susceptibility is uncertain.[21]

The management amounts to prophylactic measures because once the visual loss is established, it is usually bilateral, severe, and irreversible.[17] Correction of the hemodynamic derangements was thought to be responsible for the recovery of at least visual acuity in two patients in the literature; however the natural history of PION already includes spontaneous visual recovery.[18,19] Prophylactic measures during surgery may include avoidance of arterial hypotension, excessive fluid replacement and hemodilution, pressure on the eyeball and orbit, and the dependent position of the head, as well as shortening the duration of surgery to the minimum as much as possible. Mean blood pressure level of 60 mmHg is usually accepted as the limit of the hypotension in healthy individuals however, in hypertensive patients lowering the blood pressure may lead to hypoperfusion at higher levels. Since the systemic cardiovascular risk factors predispose surgical PION, it may be advisable to consider those factors prior to surgery.[17]

CONCLUSION

Blindness is a very rare complication of radical neck dissection. PION is one of the causes of blindness during this surgery. In the literature, hypotension and anemia are the leading risk factors of PION. Up to date, no effective treatment for surgical PION has been described. Therefore, preventive measures are crucially important in order to avoid the mentioned complication.

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