CASE REPORT

Visual loss after hip and shoulder arthroplasty, two case reports

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Abstract

Purpose: The report of two cases of ischemic optic neuropathy after hip and shoulder arthroplasty under general anesthesia. One of them is the first reported posterior ischemic optic neuropathy after shoulder surgery up to our knowledge.

Methods: Case reports and review of the literature reporting also data of the anesthesia period.

Results: The first case is a 74-year-old male patient with postoperative visual loss after awakening from hip arthroplasty. He had bilateral visual loss due to an anterior ischemic optic neuropathy with no vascular risk factors associated, probably due to intraoperative blood loss and short periods of drop of his blood pressure. The second case is a 65-year-old man who developed postoperative visual loss because of posterior ischemic optic neuropathy in one single eye after shoulder arthroplasty. To the best of our knowledge there are only three cases reported of ischemic optic neuropathy after shoulder surgery and none of them due to posterior ischemia. This patient had history of vascular risk factors, such as hypertension and diabetes. The repeated tests during the follow up of the patients revealed no significant improvement of their visual function.

Conclusions: Perioperative visual loss after hip and shoulder surgery is a very rare but fatal complication that is difficult to prevent with a poor visual prognosis. Both anesthetist and surgeon should be aware of this problem.

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Pérdida de visión después de artroplastia de cadera y hombro. Dos ensayos clínicos

Resumen

Objetivo: Presentar dos casos de neuropatía óptica isquémica posterior a artroplastia de cadera y hombro bajo anestesia general. Por lo que sabemos, uno de ellos es el primer caso registrado de neuropatía óptica posterior isquémica dentro de una intervención en el hombro.

Métodos: Ensayos clínicos y revisión de la literatura, con registro asimismo de los datos durante el período de anestesia.

Resultados: El primer caso es un varón de 74 años de edad con pérdida de visión postoperatoria al despertarse de una artroplastia de cadera. La pérdida de visión fue bilateral debido a una neuropatía óptica isquémica anterior sin factores de riesgo vascular asociados, probablemente ocasionada por la hemorragia intraoperatoria y por breves intervalos de bajada de la tensión arterial. El segundo caso consiste en un varón de 65 años de edad que manifestó pérdida visual postoperatoria debido a una neuropatía óptica isquémica posterior en un ojo después de una artroplastia de hombro. Hasta donde sabemos, existen únicamente tres casos tipificados de neuropatía óptica isquémica después de una intervención en el hombro, y ninguno de esos fue ocasionado por una isquemia posterior. Este paciente tenía antecedentes de factores de riesgo vascular, como hipertensión y diabetes. La repetición de las pruebas durante las revisiones de los pacientes no mostraron ninguna mejora significativa de su función visual.

Conclusiones: La pérdida de visión peroperatoria después de operación de cadera y hombro es una complicación poco frecuente pero grave que resulta difícil de prever, y que cuenta con un mal pronóstico visual. Tanto los anestesistas como el cirujano deben ser conscientes de este problema.

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Introduction

Postoperative visual loss (POVL) after nonocular surgery is an unexpected and fatal complication. The causes of POVL include ischemic optic neuropathy (ION), central or branch retinal artery occlusion, cortical blindness and acute glaucoma.1 POVL appears more frequently after cardiac, spine and radical neck dissection surgery.2,3 Precipitating risk factors in the etiology of POVL are massive bleeding, decreased systemic blood pressure, increased intraocular pressure and presence of systemic vascular disease (e.g., atherosclerosis, hypertension, diabetes).

We report two cases of POVL, first was confirmed as an anterior ION (AION) after hip arthroplasty and the second case of a posterior ION (PION) after shoulder arthroplasty under general anesthesia.

Case 1

A 74-year-old man was admitted to the hospital for the revision of the right hip prosthesis. He had no history of hypertension, cardiac ischemia, diabetes, neurological or ophthalmological disease. General anesthesia was induced without complications and the patient was placed in the lateral decubitus position. During the anesthesia arterial blood pressure monitoring was performed with a noninvasive cuff placed on the right arm. Due to severe blood loss during the operation his hematocrit (Ht) fell from 51% to 27.8%, with the lowest hemoglobin level at 9.3 g/dl. During the procedure the patient had four 5-min periods when his systolic blood pressure (SBP) was below 80 mmHg with mean arterial pressure (MAP) 53 mmHg. He received four blood units and 4500 ml of crystalloids and coloids.

In the post-anesthesia care unit patient complained of bilateral painless loss of vision. An initial examination found a corrected visual acuity (VA) of light perception in his right eye (RE) and 4/10 in his left eye (LE). Afferent pupillary defect was present in both eyes. The rest of the anterior segment slit lamp examination was normal. Posterior segment showed swelling of both optic discs and no macular or vascular abnormalities. Intraocular pressure (IOP) did not show high levels (11/12 mmHg, respectively). Orbital and cranial magnetic resonance was normal. The Visual Field (VF) showed an absolute scotoma in RE and a concentric defect in the LE that only left free the central ten degrees of the field. Pattern Visual Evoked Potentials were abnormal, compatible with bilateral optic neuropathy. VF tests have been repeated during the follow up with no significant improvement. Three years after the surgery the patient needs the help of second person to make his daily routine possible. The fundus examination shows pale and atrophic discs.

Case 2

A 65-year-old man with the history of hypertension, ischemic heart disease and diabetes was admitted for the revision of the left shoulder prosthesis. He also had right eye prosthesis as a result of a complicated retinal detachment in the past. The general anesthesia was induced without complications.
and the noninvasive arterial blood pressure was being monitored. The patient was placed in the beach chair position for the operation. During procedure the patient developed prolonged hypotension with his MAP reduced by almost 51% (from 103 mmHg to 51 mmHg) for 95 min. The blood loss was insignificant and postoperative level of hemoglobin was 12.3 g/dl.

Upon awakening after the surgery the patient complained about loss of vision in his single LE. The VA in his LE was “hand movement” and 24 h later the vision had improved to counting fingers at 1 m. The fundus examination revealed a normal anterior segment with an incipient cataract, a slightly pale papilla, no swelling of the optic disc, no damage on the macula and signs of vascular sclerosis. IOP was 19 mmHg. Six days after surgery his VA was 5/10 with a normal macular optic coherence tomography (OCT). Cranial and orbital scan was normal. He was initially diagnosed to have a posterior ischemic optic neuropathy (PION). In the postoperative period he was treated with acetylsalicylic acid and low molecular weight heparin. Fundus examinations, visual fields tests and OCT were repeated during the follow up. Angiographic study showed no anomalies apart from the typical findings in a myopic eye and the vascular sclerosis changes previously mentioned. One month after the orthopedic surgery the VA was 6/10. Two months later the fundoscopic examination revealed a pale optic disc and a normal macula. In the three and half years follow up the clinical situation has not changed.

Discussion

The term ischemic optic neuropathy (ION) is used as a general term and includes anterior and posterior optic neuropathy based on the different pattern of blood supply to the optic nerve. Etiologically ION is due to transient nonperfusion or hypoperfusion of the optic nerve or to embolic lesions of the vessels feeding the nerve. Based on its blood supply, the optic nerve can be divided into two distinct regions. The anterior part (the optic nerve head) is almost entirely supplied by the posterior ciliary arteries (PCAs) arising from the ophthalmic artery. The posterior portion of the optic nerve is supplied by the pial vascular plexus which receives blood supply from multiple other sources.4

Anterior ION (AION) typically presents with sudden onset painless vision loss and it is diagnosed on fundoscopy by diffuse or segmental disc edema. Posterior ION (PION) also manifests as sudden onset loss of vision but, in contrast, the fundoscopic examination initially reveals a completely normal fundus. The optic nerve atrophy occurs after 4–6 weeks.

Each type of ION is further classified either in arteritic, caused by giant cell arteritis, or in nonarteritic form, which is responsible for the POVL. The differential diagnosis is crucial to start the rapid and appropriate treatment, especially in the arteritic form.

Treatment of nonarteritic ION often involve high dose steroids, antiplatelet drugs, mannitol and other agents to decrease intraocular pressure but none of these approaches have been shown to be effective. Recently examined the effect of crystalloid versus colloid and the use of the topical κ-agonist Brimonidine on IOP during prone spine surgery showed significantly greater IOP in patients receiving crystalloid than those receiving colloid. Topical Brimonidine showed to reduce IOP. Ocular perfusion pressure, however, did not vary between the groups suggesting that maintenance of blood pressure may be a more important factor in determining perfusion pressure.5 As there is no proven effective treatment of the nonarteritic ION, prevention is the rule in this condition. Obesity, male sex, longer anesthetic duration, greater blood loss, Wilson frame use for head support, and decreased percent colloid administration are associated with ION after spinal fusion surgery.6

One or more risk factors are often present in a patient with POVL but, on the other hand, there are many patients with ION after spinal surgery who had been relatively healthy before the procedure.7

The prevalence of ION in the orthopedic surgery is well documented after spinal fusion surgery. The large scale retrospective review of the Nationwide Inpatient Sample (NIS) from 1996 to 2005 describes the frequency and possible risk factors of POVL among the eight most commonly performed surgical procedures with the incidence of POVL after spine surgery to be 3.09/10,000 (0.03%).8 The same study revealed in the hip replacement/femur treatment operations 226 cases of POVL with 43 cases caused by ION with estimated prevalence of POVL 1.86/10,000 (0.0186%). The incidence of POVL after shoulder surgery is unknown.

Our first patient who had irreversible, bilateral anterior ION after hip arthroplasty had no history of atherosclerosis, hypertension or diabetes. The most probable cause of the ION was intraoperative blood loss and short periods of drop of his blood pressure. Our patient reached value of Ht of 27.8% (which means a fall of 45.5% of initial level) that added to a long surgical time, almost 4 h, induced probably a remarkable hypoperfusion at the optic nerve head. Despite there were no vascular risk factors; age, operative trauma and significant blood loss could have precipitated the development of the ischemic event. We found in the literature some case reports of POVL due to ION after hip surgery.8,9 All patients presented a significant perioperative/postoperative anemia. Some of them had one or more vascular risk factors for ION.

In contrast, our second patient did not have a significant blood loss, but presented vascular risk factors such as coronary atherosclerosis, hypertension and diabetes. In this case, the prolonged drop of blood pressure could have probably been the precipitating factor to develop PION as well as a defective vascular autoregulation in a patient with atherosclerotic disease that could have increased the susceptibility to ischemia of the intraorbital optic nerve.7 Another possible explication could be the anatomic variation of intraorbital blood supply.9

In the literature, there are very few published cases of POVL after this type of surgery. We found three case reports of POVL after shoulder surgery.9 In one of them the cause was a central retinal artery occlusion; the second was due to an AION; and in the third case, the cause was not available. To our knowledge, we present the first case of a POVL due to a PION after shoulder surgery. Since the diagnosis of PION is usually hard to make with certainty and it is a diagnosis of exclusion, we ruled out other possibilities of POVL as we explained in the case report. We always have to take into account the impact of the patient’s position to venous pressure and IOP.10 The prone position for spinal
fusion surgery, especially when the head position is lower than the heart, is one of the risk factor of POVL. The elevation of venous pressure, especially in obese patients when their abdomen is compressed, and development of interstitial edema can cause damage to the optic nerve. In our cases the position of the first patients head was neutral (lateral decubitus with the head at the heart level) and ION was diagnosed in both eyes. The beach chair position of the second patient in normal conditions diminishes the venous pressure. In this case we believe the venous pressure should not have been elevated, but hypoperfusion of the optic nerve could have been the most important factor to developed ION.

In conclusion, we would like to point out that ION may occur after severe intraoperative blood loss even if the patient was previously healthy as well as in a patient with risk factors without significant intraoperative blood loss but with prolonged hypotension. The anesthetists and the surgeons have to be aware of the occurrence of ION as a fatal and unexpected complication in the perioperative period which prognosis for visual recovery is very poor.

Conflict of interest

The authors declare no conflict of interest.

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References