Retinal vascular occlusions after nonocular surgeries

Oclusões vasculares retinianas após cirurgias não oculares

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ABSTRACT

Ophthalmologic complications of nonocular surgeries are rare events, but can lead to irreversible conditions of low visual acuity. They are often associated with spine, heart and neck surgery, however they can occur after procedures on other systems. The main local causes are ischemic optic neuropathies, vascular occlusions, cortical lesions, and acute angle-closure glaucoma. We report two cases of sudden low visual acuity secondary to vascular occlusions after gastrointestinal procedures. In the first case, a 57-year-old patient electively admitted for colon reconstruction after Hartmann's colostomy, progressed with intra- and postoperative complications and required subsequent complementary surgeries. Once month later he presented with sudden bilateral low visual acuity, painless and non-altitudinal, and was diagnosed as papillophlebitis, which resolved spontaneously with the progression of the condition. The second case, a 69-year-old patient with no comorbidities underwent rectal resection due to suspected malignant tumor, and progressed on the third postoperative day, with pain and bilateral low visual acuity secondary to acute angle-closure glaucoma, and branch retinal artery occlusion in right eye; treated with iridotomy and ocular hypotensive eye drops, with only slight recovery of vision. The article aims to discuss the etiological mechanisms of the reported conditions and present a literature review.

RESUMO

Complicações oftalmológicas de cirurgias não oculares são raras, mas podem levar a condições irreversíveis de baixa acuidade visual. Em geral são associadas à cirurgia de coluna, coração ou pescoço, mas podem ocorrer após procedimentos em outros sistemas. As principais causas são neuropatias ópticas isquêmicas, oclusões vasculares, lesões corticais, e glaucoma agudo de ângulo fechado. Relatamos dois casos de baixa acuidade visual súbita, secundária a oclusões vasculares, após procedimentos cirúrgicos gastrointestinais. No primeiro caso, um paciente de 57 anos foi internado de forma eletiva para reconstrução do cólon após colostomia de Hartmann. Evoluiu com complicações nos períodos intra- e pós-operatório, e necessitou de outras cirurgias complementares. Um mês depois apresentou baixa acuidade visual bilateral súbita, indolor e não altitudinal, e foi diagnosticado como papiloflebite, com resolução espontânea na evolução. O segundo caso, uma paciente de 69 anos, sem comorbidades, foi submetida à ressecção do reto por suspeita de tumor maligno e, no terceiro dia de pós-operatório, evoluiu com dor e baixa acuidade visual bilateral, secundária a glaucoma agudo de ângulo fechado, e oclusão de ramo da artéria retiniana no olho direito; tratada com iridotomia e colírios hipotensores, com recuperação parcial da visão. O objetivo do artigo é discutir os mecanismos etiológicos das doenças relatadas, e apresentar uma revisão da literatura.

INTRODUCTION

Visual loss after nonocular surgeries is a catastrophic and rare event. Its incidence is 0.013%, and it varies according to the procedure performed. It occurs more often after spine and cardiac surgeries, especially when there are some risk factors, such as hypotension, anemia, blood loss, infusion of large volumes of crystalloids, use of vasoconstrictors, low head position, ocular compression and elevation of intraocular pressure (IOP). Individual susceptibility contributes for the event.[1-3]

The main ophthalmologic causes for low visual acuity (LVA) after nonocular surgeries are corneal abrasions during anesthesia, ischemic optic neuropathies, retinal vascular occlusions, acute angle closure crisis and cortical infarctions. The pathophysiology is not well understood yet, but can involve changes in blood viscosity, in ocular perfusion pressure (OPP) and the cerebral perfusion pressure (CPP).[4,5]

Retinal venous occlusions are the second major cause of vascular disease of the retina, after diabetic retinopathy. The incidence increases with age and presence of cardiovascular comorbidities. They may be central vein occlusions (CVO) and branch vein occlusions (BVO), and may or not have secondary ischemic manifestations, which directly affect the prognosis.[6] Up to one third of patients with BVO improve spontaneously, reaching visual acuity (VA) better than 20/40. Central vein occlusions have a benign course, are non-ischemic and self-limited and also known as papillophlebitis.[7,8]

Arterial occlusions are more common than venous ones after nonocular surgeries.[9] They may be central retinal artery occlusion (CRAO), branch retinal artery occlusion (BRAO) and ciliary artery occlusion. The pathophysiology may involve embolism, intramural thrombosis, vasculitis, arterial spasms, hemodynamic changes, and hypertensive necrosis. The symptoms depend on the injured vessel. In BRAO, up to 89% of patients manifest VA equal or better than 20/40 with disease progression, even with no treatment.[10-12]

The cases reported are from two patients with LVA and retinal occlusions after gastrointestinal surgeries and their ocular and systemic complications.

CASE 1

A 57-year-old male, with no previous cardiovascular or ophthalmologic comorbidities, underwent an intestinal transit reconstruction for a Hartmann's colectomy performed 3 years before, due to acute diverticulitis. He progressed with intraoperative complications, suture dehiscence, and abdominal sepsis in the immediate postoperative period, which required an exploratory laparotomy and small intestine enterorrhaphy. Extensive fluid replacement and vasoactive drug infusion were needed due to hemodynamic instability; peritoneal lavage and segmental enterectomy were also performed. Twenty days after the last surgery, he presented bilateral painless LVA, sudden and non-altitudinal. On examination he had on both eyes (OU): VA of hands movement, slowed pupillary light reflex, optic disc hemorrhage and edema, discrete venous tortuosity, and perivascular hemorrhages along superior and inferior arcades. The diagnosis was CVO. One month later, in the first ophthalmologic follow-up evaluation, the patient had recovered well from the abdominal process, and had substantial visual improvement. On examination on OU: VA of 20/40, biomicroscopy revealing normal and symmetrical pupillary-light reflex, expressive reabsorption of the perivascular hemorrhages, and complete resolution of the optic disc edema (Figure 1). Fluorescein angiography (FA) indicated minor areas of hypofluorescence caused by blockage, corresponding to hemorrhage areas and absence of abnormal hyperfluorescence of the optic disc (Figure 2). Optical coherence tomography angiography (OCT-A; Cirrus HD OCT, Zeiss Angioplex) structural B-scan of the macula showed no central edema, and angiography scan revealed no abnormal vascular perfusion. Automated perimetry demonstrated diffuse non-altitudinal field defects, sparing the central area. Based on these new exams and clinical progression, the diagnosis of papillophlebitis was made.

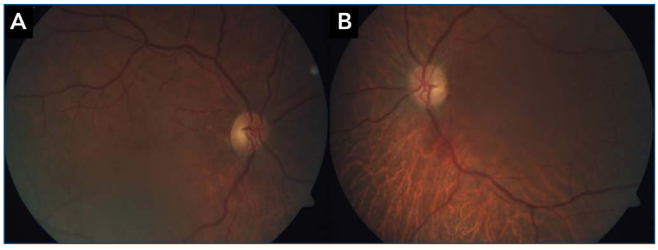

Figure 1. Case 1 – Fundus examination (1A – right eye; 1B – left eye) revealing discrete areas of hemorrhages along the vascular arcades and absence of optic disc edema.

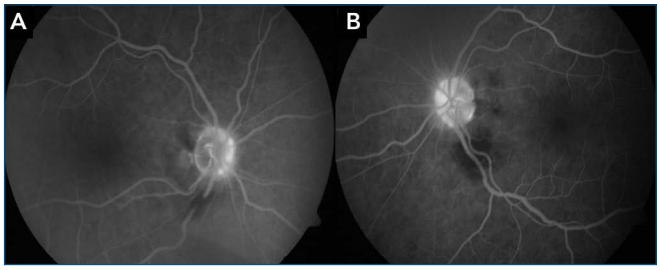

Figure 2. Case 1 - Fluorescein angiography (2A – right eye; 2B – left eye) showing areas of hypofluorescence caused by blockade along the vascular arcades, which coincide with areas of hemorrhage on fundus examination, and absence of optic disc edema and vascular non-perfusion areas.

CASE 2

A 69-year-old female patient, with no ocular or systemic comorbidities, complained of pain, hyperemia and progressive LVA in right eye (OD) for 15 days. She reported symptoms initiated 3 days after a rectal resection of a neoplastic lesion. Blood pressure at the postoperative period was 90x70 mmHg. On examination of the OD: VA of 20/80, with no improvement on visual potential tests, semi-mydriatic pupil with little reaction to light.

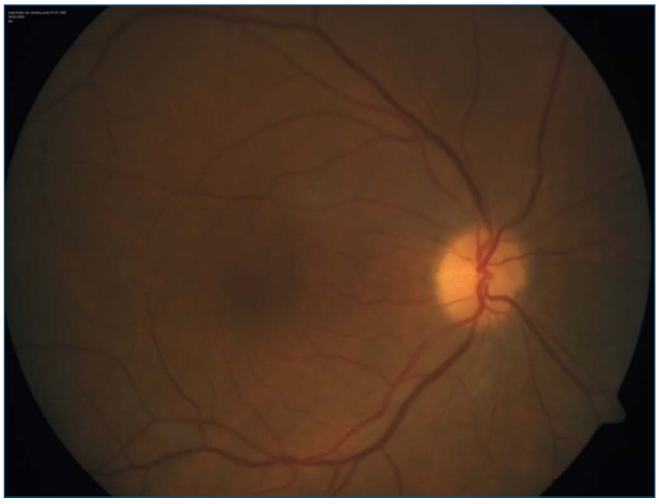

Figure 3. Case 2 - Fundus examination with segmental arterial attenuation on the temporal inferior arcade associated with a perivascular whitish lesion, suggestive of ischemic edema of the nerve fiber layer (cotton wool spot).

Intraocular pressure of 36 mmHg; gonioscopy showing camerular sinus up to Schwalbe's line visible on 360º, with no synechiae. Fundus examination indicated optic disc was pink, with sharp margins, cup/disc ratio of 0.3 (Figure 3), attenuation of a segment of temporal inferior artery, with a perivascular cotton wool spot, and visible pulsation of the artery on this topography; anterior chamber and corneal OCTA (Cirrus HD OCT, Zeiss Angioplex) confirmed angular narrowing OU. Left eye (OS) did not show any other alteration on examination. Ocular hypotensive eye drops (timolol 0.5% and brimonidine 0.2% 12/12 hours) and acetazolamide 250 mg *p.o.* 12/12 hours were prescribed, and new ancilalry exams were ordered. After ten days, she referred improvement of symptoms and had VA in right eye of 20/50 and IOP of 15 mmHg. FA showed minute perfusion deficits along the temporal inferior arcade of OD and normal exam in OS. The optical coherence tomography (OCT) scans revealed absence of macular edema and diffuse thickness reduction of the ganglion cell layer in OD (Figure 4); fundus examination showed no more arterial pulsation, but there was persistence of the cotton wool spot and arteriolar attenuation. Based on the ophthalmologic findings and clinical course, diagnosis of acute angle closure and BRAO was made in OD. Nd:YAG laser iridotomy was performed on both eyes and IOP follow-up was made in the glaucoma sector.

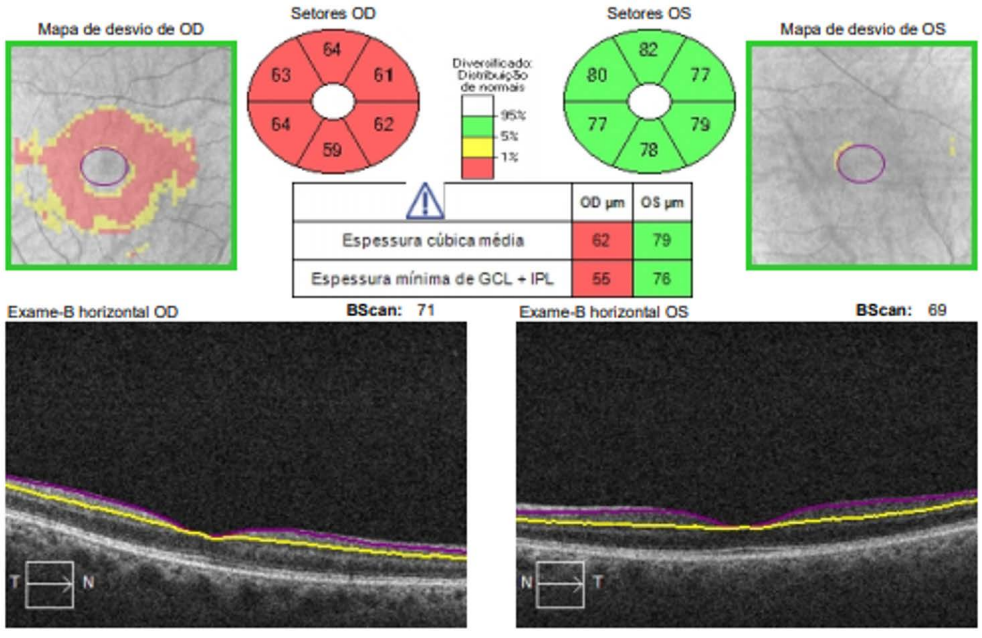

Figure 4. Optical coherence tomography revealing diffuse thickness reduction of the ganglion cell layer in right eye and normal exam in left eye.

DISCUSSION

The main presumptive diagnosis to be considered in patients with postoperative LVA in nonocular surgeries, especially when not involving the head and the periocular region, is ischemic optic neuropathy. Ischemic events of the optic nerve, whether anterior or posterior ischemic optic neuropathy, respectively AION or PION, are quite rare; however, they are the major agents of irreversible visual deficits in patients undergoing surgeries. Spinal surgery and extensive cardiac surgeries account for most of these events; the first involve possibly mechanisms of direct trauma to the eyeball, caused by facial support or by a face-down positioning adopted during the whole procedure, while the cardiovascular procedures are more associated with an ischemic mechanism resulting from changes in blood perfusion. Moreover, there are reports describing their occurrence after several types of surgeries, but the pathophysiology has not been fully understood yet. Systemic conditions and comorbidities of the patients and intraoperative complications have been reported as possible risk factors; nonetheless, this association has not been definitely established.[13] In a postoperative or post-traumatic context, when severe anemia and prolonged hypotension may be observed, AION has been reported associated with LVA, frequently bilateral, a few weeks after the event or abrupt blood loss. Its mechanisms have not been fully understood, despite some speculations.[14]

Retinal venous occlusions are mostly associated with local and systemic risk factors. Increased disc excavation and atherosclerosis of the retinal arteries are risk factors for their occurrence. Hypertension, diabetes mellitus, atherosclerosis, embolic and prothrombotic conditions are also involved in its pathogenesis. When they occur in young patients, with risk factors or not, prothrombotic conditions should be considered and adequately assessed.[15] Occlusive conditions have been described in pregnant women, even in the absence of any other risk factor, women using hormone-based contraceptive methods, and in patients with altered coagulation factors and tests.[16-18] Occlusion reports in a nonocular postoperative context are scarce.[19] The prothrombotic condition inherent to any major surgery, associated with prolonged restriction to bed, hemoconcentration, and blood increased viscosity may occur after intraoperative complications.

In case one, the clinical history, systemic and epidemiological conditions strongly suggested AION. However, bilaterality and visual loss pattern in a non-altitudinal situation, would rule out this diagnosis. The uncharacteristic fundus examination with absence of ischemia signs and presenting venous tortuosity and perivascular hemorrhages, suggested bilateral CVO. For both presumptive diagnoses, and for this specific patient, treatment of the systemic condition was prioritized. The confirmation of absence of altitudinal defect on perimetry, the dramatic and fast visual improvement, absence of areas of non-perfusion on FA, and absence of disk pallor three months after onset of symptoms, made diagnosis of ischemic condition less probable. In the same way, the fast resolution of fundus examination alterations and especially the almost complete visual rehabilitation suggested papillophlebitis.

Systemic and local drugs, with or without mydriatic actions, are involved in the pathophysiology of acute angular closure on patients with narrow angles and predisposition for the event.[20] The main agents implied are anticholinergic (atropine, scopolamine and muscle relaxants) and adrenergic (ephedrine and epinephrine) drugs. Likewise, non-pharmacological mechanisms also play a role, whether it is psychologic stress due to the procedure, or mydriasis induced by darkness at the postoperative period.[21-23]

Among the several mechanisms associated with retinal vascular occlusions, lower OPP stands out in the postoperative period. It is below the adequate threshold to maintain adequate homeostasis on the retinal bed. The perfusion pressure of the central retinal artery is defined as the difference between the mean arterial pressure (MAP) and the IOP. The MAP, for instance, is estimated as the weighted mean of systolic (SP) and diastolic pressures (DP), as shown in equation 1.

$$MAP = \frac{SP + 2DP}{3} \qquad \text{Equation 1}$$

Based on these measures, OPP may drop due to two main mechanisms. The first one would be after an expressive blood pressure decrease. The second, after a sharp and important rise of the IOP, as observed in acute angular closure episodes. When the IOP is equal to or exceeds the MAP of the central retinal artery, mainly in predisposed patients and with chronically decreased OPP, retinal arterial occlusions may happen, though frequently in a transient way. In these cases, FA, when performed after resolution of the causal mechanisms, may be normal or with minimal arterial perfusion deficits.

In second case, the patient was seen only 10 days after onset of symptoms, making it difficult to understand the triggering mechanisms of the condition. However, as far as our team knows, it was an acute crisis of angular

closure, possibly linked to psychological stress due to surgery, or to the drugs used in anesthesia or postoperative period. The IOP rise, associated with an episode of hypotension, may have triggered the arterial occlusion identified at the first evaluation. The fast IOP reduction following treatment and delay to perform FA may also justify the discrete angiographic changes found, despite the very characteristic fundus examination. Likewise, delay to seek care was sufficient to result in changes in the ganglion cell layer and retinal perfusion, as well as in the best corrected VA and campimetry.

Based on the reported cases, it should be noted the diagnosis of LVA after nonocular surgeries is not limited to ischemic optic neuropathies, and is not necessarily a predictor of poor visual prognosis. Understanding the pathophysiology of occlusive conditions and their risk factors helps making the correct diagnosis and management of these patients.

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