Pressure Induced Stromal Keratopathy After Phacoemulsification: Case Report

Queratopatia Estromal Induzida por Pressão Após Facoemulsificação: Relato de Caso

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ABSTRACT

INTRODUCTION: To report a case of pressure induced stromal keratopathy (PISK) after phacoemulsification and intraocular lens (IOL) implantation in a patient with ectasia after laser *in situ* keratomileusis (LASIK) previously treated with femtosecond-assisted intracorneal ring segment (ICRS).

CASE REPORT: A 53-year-old man with post-LASIK ectasia in his right (OD), underwent phacoemulsification and intraocular lens implantation two months after ICRS implantation in OD. On the first operative day, the patient complained of decreased visual acuity. His best corrected visual acuity (BCVA) was 20/80, intraocular pressure was 55 mmHg and biomicroscopy revealed diffuse interface haze and corneal edema. Anterior segment optical coherence tomography (AS-OCT) showed interface fluid accumulation. Steroid drops were discontinued and antihypertensive drops were added. One week after surgery, his BCVA was 20/40, the cornea was totally clear, and AS-OCT showed complete fluid reabsorption.

CONCLUSION: Although PISK usually develops early after LASIK, it can also occur different setting associated with high IOP. This report raises concerns regarding postoperative intraocular hypertension prophylaxis in eyes with previous corneal interface creation, in particular the use of corticosteroids, which are known to trigger PISK development.

KEYWORDS: Cataract Extraction; Intraocular Pressure; Keratomileusis, Laser In Situ; Phacoemulsification/adverse effects.

RESUMO

INTRODUÇÃO: Descrição de um caso clínico respeitante a queratopatia estromal induzida pelo aumento da pressão intraocular (PISK) após facoemulsificação num doente previamente submetido a implante de anel intra-estromal por ectasia pós cirurgia refrativa.

DESCRIÇÃO DO CASO: Doente do sexo masculino de 53 anos com história de ectasia pós

LASIK no olho direito, foi submetido a facoemulsificação e implante de lente intraocular dois meses após implante de anel intraestromal. No primeiro dia pós-operatório, apresentava queixas de diminuição da acuidade visual. A melhor acuidade visual corrigida (MAVC) para longe era 20/80, a pressão intraocular (PIO) era 55 mmHg e a biomicroscopia revelou edema de córnea e a presença de haze difuso na interface. A tomografia de coerência ótica do segmento anterior (OCT-SA) confirmou a presença de líquido na interface. Os corticoesteróides foram suspensos e foi introduzida medicação anti-hipertensora. Uma semana após a cirurgia, a MAVC melhorou para 20/40, a córnea apresentava-se transparente e o OCT-SA revelou uma completa reabsorção do fluido.

CONCLUSÃO: Embora a PISK normalmente se apresente no pós-operatório imediato de LASIK, pode ocorrer noutras situações clínicas associadas a elevação da PIO. O presente caso clínico realça a importância da profilaxia dos picos hipertensivos pósoperatórios em olhos com história de cirurgia que envolva a criação de uma interface na córnea, particularmente no que respeita ao uso de corticosteróides que parecem potenciar o desenvolvimento de PISK.

PALAVRAS-CHAVE: Ceratomileuse Assistida por Excimer Laser In Situ/efeitos adversos; Extração de Catarata; Facoemulsificação; Pressão Intraocular.

INTRODUCTION

Pressure-induced stromal keratopathy (PISK), also known as interface fluid syndrome, is an uncommon complication after laser in situ keratomileusis (LASIK). It was first described by Lyle and Jin in 1999 and is characterized by fluid collection in the flap interface.[1] Gab-Alla reported an incidence of 2.9% in an Egyptian population, but the literature lacks information on its real incidence.[2]

PISK results from increased intraocular pressure (IOP) which occurs mainly due to postoperative corticosteroids use. It usually presents from the first week to the sixth postoperative month, when intensive corticosteroids are used.[3–5] However, late PISK cases have been also reported in eyes with LASIK history experiencing high IOP, either surgical induced or associated with intraocular inflammation.[6–8] Moreover, PISK has been also reported after small incision lenticle extraction (SMILE) and after descemet stripping endothelial keratoplasty (DSAEK) as these techniques also produce a potential space into the cornea.[9,10] PISK has a favorable prognosis but, if not recognized and treated promptly, could lead to irreversible visual impairment either from corneal haze or optic nerve damage.[5,11]

PISK can mimic diffuse lamellar keratitis (DLK) which is another LASIK complication.[11] Both should be differentiated because PISK do not respond or may be even aggravated by corticosteroids which are the treatment mainstay of DLK.[11] Despite hypertensive pathophysiology of PISK, applanation intraocular pressure could be falsely lowered by fluid accumulation in lamellar interface.[1,5] Anterior segment optical coherence (AS-OCT) tomography shows interface fluid accumulation and confirms the diagnosis.

This paper aims to report a case of PISK after uneventful phacoemulsification in a patient with history of LASIK more than 20 years ago. Consent has been obtained to publish this case (including publication of images).

CASE REPORT

A 53-year-old healthy man with 22 years history of bilateral LASIK for myopia correction presented with decreased visual acuity in his right eye (OD). His best corrected visual acuity (BCVA) was 20/100 in OD and 20/30 in his left eye (OS). Corneal endothelium evaluation was unremarkable. Tomographic corneal examination revealed post laser vision correction (LVC) corneal ectasia in OD (Fig. 1) and the patient underwent corneal

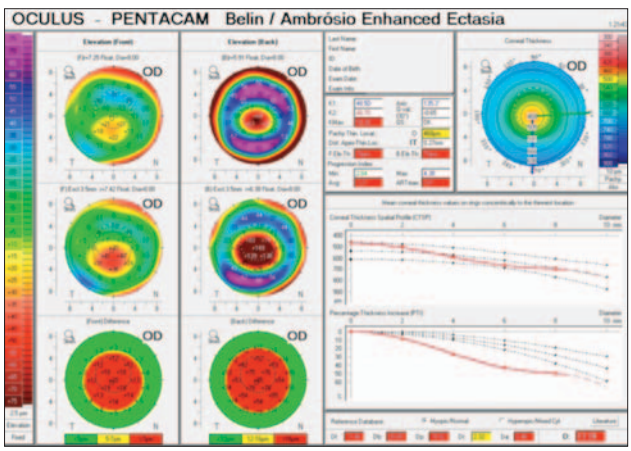

Fig. 1. Tomography of OD revealing post laser vision correction ectasia.

cross-linking (Athens Protocol) plus femtosecond laser-assisted asymmetric intracorneal ring segment (ICRS) implantation (Ferrara ring 320º, 90/340 µm). BCVA improved to 20/70. Two months later, the patient underwent uneventful phacoemulsification for nuclear cataract and +13.00 D Alcon AcrySoft IQ SN-60WF intraocular (IOL) implantation in OD. He was ordered to instill topical 5 mg/mL moxifloxacin plus 1mg/mL dexamethasone (Vigadexa®) and 0.4% ceterolac 4 times daily.

On the first postoperative day, the patient complained about decreased visual acuity. Slit lamp biomicroscopy revealed diffuse interface haze and corneal edema. The BCVA was 20/80 and IOP was 55 mmHg measured by Corvis® ST

(Oculus; Wetzlar, Germany) non-contact tonometry. Anterior segment optical coherence tomography (AS-OCT) showed fluid accumulation in the interface and PISK diagnosis was established (Fig. 2).

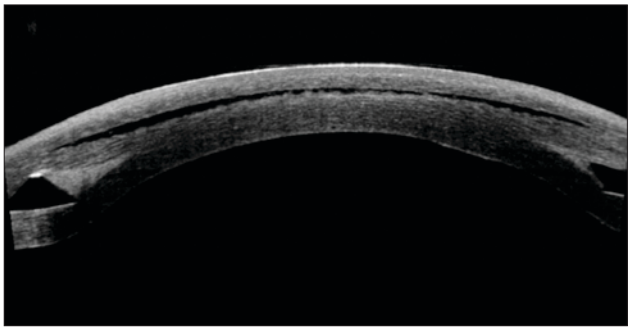

Fig. 2. First-operative day anterior segment OCT revealing interface fluid accumulation.

Steroid drops were discontinued. Oral acetazolamide (250 mg) and topical antiglaucoma drops (brimonidine-timolol 0.2%/0.5% association) were given and IOP decreased to 18 mmHg. An improvement of fluid accumulation, corneal edema and BCVA were registered at the same day (Fig. 3). Antiglaucoma fixed-combination drops

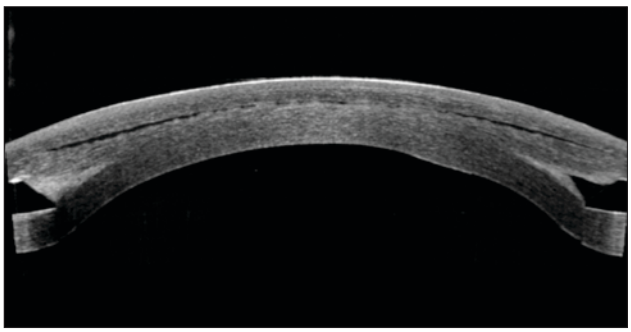

Fig. 3. Interface fluid improvement resolution 6 hours after treatment of oral acetazolamide and topical ant glaucoma drops.

were maintained twice daily for one week as well as topical moxifloxacin. One month after surgery, his BCVA was 20/40. The cornea was totally clear, and AS-OCT showed complete resolution (Fig. 4) of fluid accumulation which has maintained at his final visit at one year.

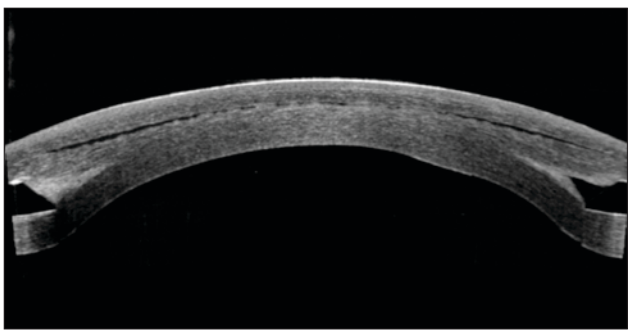

Fig. 4. Interface fluid resolution one week after surgery.

DISCUSSION

This report illustrates a very uncommon possibility of late PISK after cataract surgery with ectasia after laser *in situ* keratomileusis (LASIK) previously treated with femtosecond-assisted intracorneal ring segment (ICRS). PISK is a rare flap-related complication after LASIK, which usually occurs within the first postoperative month.[10] PISK is associated with fluid accumulation in the interface due to increased intraocular pressure.[1]

PISK can mimic diffuse lamellar keratitis (DLK), which is another complication of LASIK.[11,12] It is extremely important to distinguish PISK from DLK as the former is aggravated by steroids which are the treatment mainstay of DLK.[11] Slit lamp examination does not differentiate both entities and usually reveals diffuse interface haze.[11] Optical coherence tomography is mandatory to show fluid accumulation in the interface and thus, confirming PISK diagnosis.

Despite PISK is associated with increased IOP, obtaining the real IOP in corneas with LASIK history is challenging and PISK often presents with falsely low or normal IOP.[1] The IOP measured by Goldmann applanation tonometry is usually lower after LASIK.[13,14] Moreover, the amount of interface fluid accumulation in PISK influences the IOP measuring.[5] Peripheral measurements, outside the edge of corneal flap, are more precise but could, indeed, overestimate the real IOP as the peripheral cornea is thicker.[8] Dynamic contour tonometry may be more accurate as it is relatively immune to changes in corneal biomechanics and pachymetry after LASIK.[15]

One of the most accepted theories associates PISK with increased intraocular pressure due to corticosteroids use, either for ocular inflammation or after surgery. IOP increasing occurs in up to 30% of steroid users and usually manifests 2 to 6 weeks after its use.[4,16] Moreover, a great percentage of patients with myopia greater than 5.00 D have IOP increasing with steroid use.[17] However, our patient has previously been submitted to ICRS implantation and medicated with topical steroids and PISK did not occur. Here, we postulate that surgically induced hypertension, which is more pronounced in cataract surgery, could play a more important role for PISK development rather than steroids use. On the other hand, we also postulate that weaker corneas could have higher possibilities of PISK as our patient only developed PISK after developing ectasia and being submitted to ICRS implantation.

Other theories concern about corneal endothelium status.[18] The endothelial Na^+K^+ATPase pump is primarily responsible for maintaining stromal deturgescence, especially in the postoperative period. Corneal endothelium impairment, which occurs in Fuchs' endothelial corneal dystrophy, could potentiate interface fluid accumulation and PISK development. Our patient had unremarkable endothelium evaluation, proving that endothelial disease is not necessary to PISK development.

This report raises concerns regarding PISK prophylaxis when patients with LASIK history or other surgeries which create corneal interfaces are submitted to intraocular surgeries or medicated with steroid drops. One possibility is to check the steroid responsiveness before surgery and to minimize their use in such responsive patients, but this scenario seems to be clinical unrealistic. On the other hand, patients with LASIK his-

tory could benefit of a course of hypertonic drops in such settings to reduce the possibility of PISK. As such, a large clinical trial should be designed to better identify the risk factors with for PISK development and to clarify the effect of hypertonic drops in interface fluid.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO:

TL: Conceptualization, Writing Original Draft.
SRB, NS, AVE and NC: Methodology, Investigation.
RAJr: Conceptualization, Supervision, Project Administration.

RESPONSABILIDADES ÉTICAS

Conflitos de Interesse: Dr Renato Ambrósio Jr é consultor da Oculus, Alcon, Zeiss, Essilor, Genom, Mediphacos. Os demais autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

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ETHICAL DISCLOSURES

Conflicts of Interest: Dr Renato Ambrósio Jr is a consultant for Oculus, Alcon, Zeiss, Essilor, Genom, Mediphacos. The remaining authors have no conflicts of interest to declare.

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