# Irvine-Gass syndrome refractory to conventional clinical treatment associated with clinical decompensation of glaucoma after cataract surgery

## Síndrome de Irvine-Gass refratária ao tratamento clínico associada à descompensação clínica do glaucoma após cirurgia de catarata

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**Descritores:**
Edema macular; Pressão intraocular; Injeções intravítreas; Glaucoma; Drenagem; Dexametasona; Catarata

## ABSTRACT

We describe a case of Irvine-Gass syndrome and the clinical decompensation of glaucoma after uneventful cataract surgery with resistance to conventional clinical treatment. Drainage tube implantation and intravitreal injection of dexamethasone were performed simultaneously with the resolution of cystoid macular edema and good control of intraocular pressure, which remained consistent after 1 year of follow-up.

## RESUMO

Descrevemos um caso de síndrome de Irvine-Gass e a descompensação clínica do glaucoma após cirurgia de catarata sem intercorrências e com resistência ao tratamento clínico convencional. O implante do tubo de drenagem e a injeção intravítrea de dexametasona foram realizados simultaneamente com a resolução do edema macular cistoide e o bom controle da pressão intraocular, que permaneceu consistente após um ano de acompanhamento.

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## INTRODUCTION

According to data from the World Health Organization (WHO), cataract and glaucoma are the leading causes of blindness in the population. The correct management of these pathologies and the mastery of their complications and therapeutic failures are of fundamental importance for preserving good visual acuity for the patient.[1-4]

Some complications related to cataract surgery may favor the uncontrolled clinical development of glaucoma.[2] The treatment of some of these complications may also lead to increased intraocular pressure.[5] Irvine-Gass syndrome is a frequent complication of cataract surgery. Its management often includes corticosteroids, which may cause increased aqueous humor outflow resistance with consequent intraocular pressure elevation, imposing adjustments in clinical glaucoma therapy for disease control.[6]

The relevance of the article resides in demonstrating a chronic case of Irvine-Gass syndrome after uncomplicated cataract surgery, associated with a significant uncontrolled intraocular pressure with resistance to conventional clinical treatment.

This study was evaluated and approved by the Ethics Committee (CAAE: 80035824.0.0000.5243).

## CASE REPORT

A 61-year-old, brown, administrator man was referred from another service and attended an ophthalmology appointment in November 2020, complaining of low visual acuity after cataract surgery. He denied any systemic disease and had a previous diagnosis of glaucoma with topical use of dorzolamide and timolol maleate in both eyes, and topical corticosteroids (dexamethasone) 6/6 hours in the right eye.

He had a history of cataract surgery in the right eye 4 months ago without intraoperative complications, but developed cystoid macular edema (CME) after 4 weeks and significant uncontrolled intraocular pressure. His visual acuity was 20/100 in the right eye and 20/40 in the left eye with the best correction, biomicroscopy without alterations. The initial intraocular pressure was 18 mmHg in the right eye, and 13 mmHg in the left eye. In fundus examination, there was a macular brightness alteration, evidencing CME, which was confirmed in the optical coherence tomography exam (Figure 1), and enlarged cup disk ratio in both eyes (Figure 2).

Topical anti-inflammatory (nepafenac) was initiated 8/8 hours with no clinical improvement.

After 3 weeks, oral prednisone (30 mg/day) was initiated, which resulted in increased intraocular pressure,

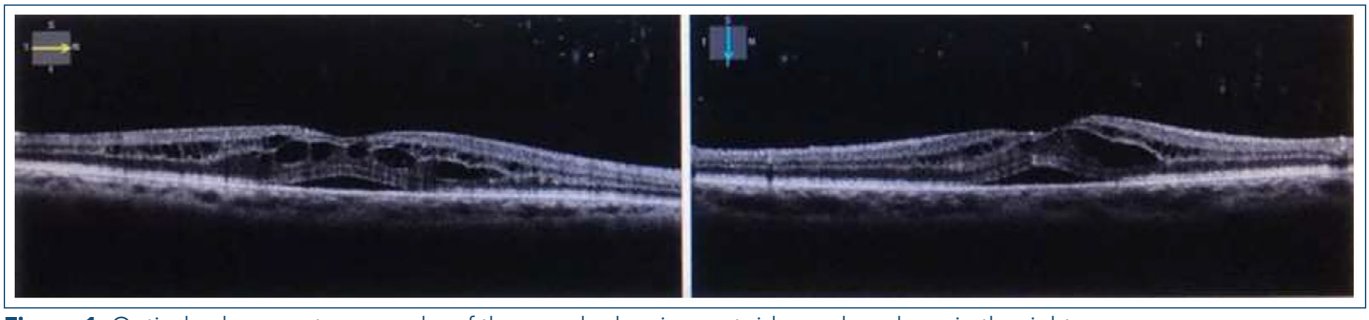

**Figure 1.** Optical coherence tomography of the macula showing cystoid macular edema in the right eye.

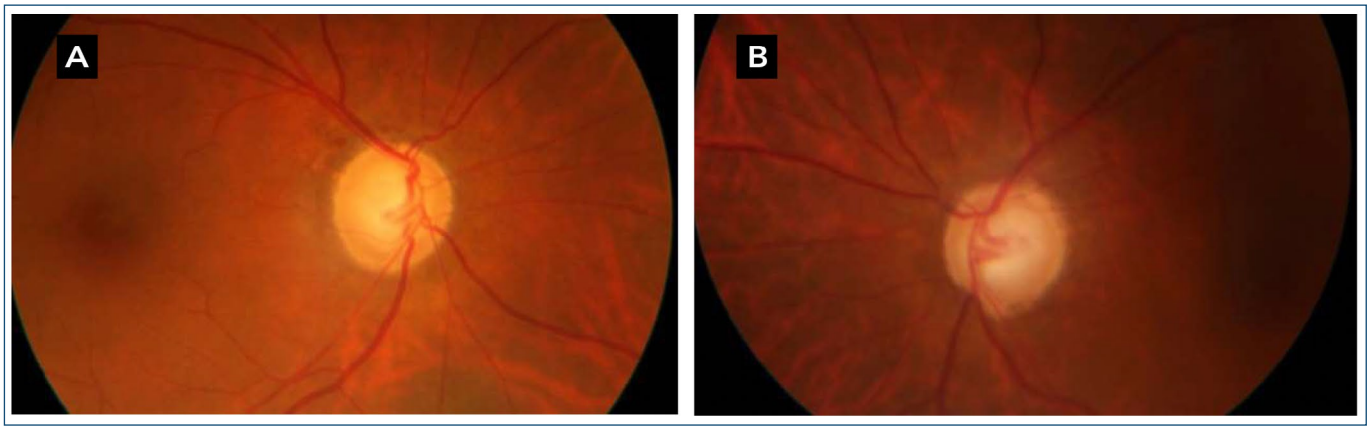


**Figure 2.** Photograph of the optic nerve of the right (A) and left eye (B).

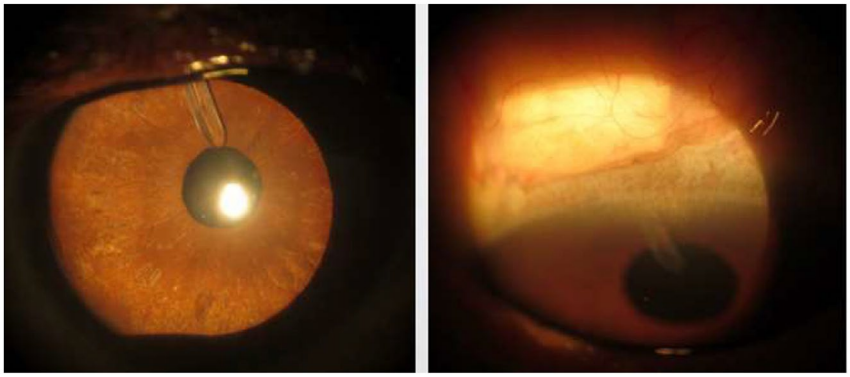

**Figure 3.** Antiglaucomatous surgery with Ahmed tube implantation showing the tube well positioned in the anterior chamber.

with levels of 28 mmHg in the right eye and 20 mmHg in the left eye, and partial improvement of the CME. Hypotensive eye drops of brimonidine tartrate 12/12 hours and acetazolamide (250 mg, one tablet, 6/6 hours orally) were added with little response in reducing intraocular pressure. When corticosteroid reduction was started, the patient had a decrease in intraocular pressure, but the CME was reactivated.

After several attempts of clinical management, it was decided to perform a combined antiglaucomatous surgery with placement of an Ahmed drainage implant (Figure 3) associated with the application of an intravitreal injection of a 0.7mg dexamethasone biodegradable implant (Ozurdex®).

Three weeks after the procedure, the patient progressed to CME resolution (Figure 4), significant improvement in visual acuity (20/30 with correction), and improved intraocular pressures: 14 mmHg with timolol maleate eye drop monotherapy. After 1 year of follow-up, his condition remains stable.

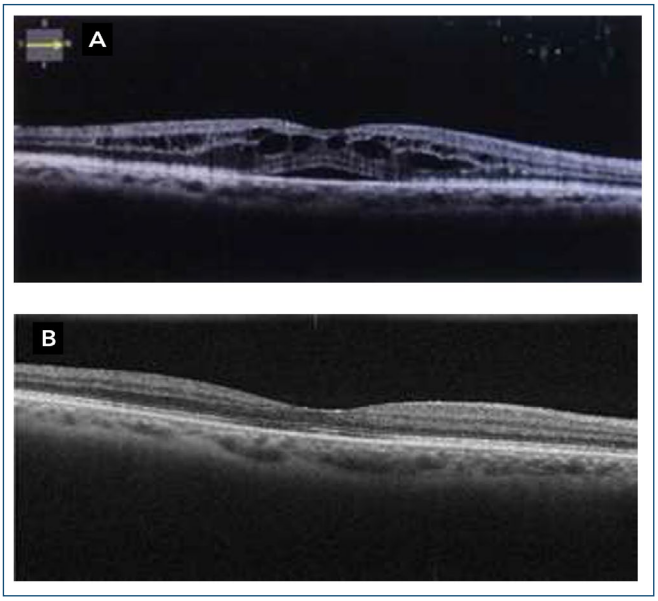


**Figure 4.** Comparison of macular anatomy before (A) and after (B) the treatment performed in the right eye.

## DISCUSSION

The correct management of CME or Irvine-Gass syndrome after cataract surgery in glaucomatous patients is of fundamental importance for non-progression of the disease and good visual prognosis.[2]

Irvine-Gass syndrome has multifactorial pathogenesis associated with inflammatory conditions, posterior capsule rupture, ocular hypotonia, and vitreomacular traction, among others.[1,7-9] In glaucoma patients, prostaglandin analog eye drops and those containing benzalkonium chloride may also be associated with the development or perpetuation of macular edema after surgery, which is an additional challenge in clinical management and patient follow-up.[2]

Irvine-Gass syndrome treatment includes topical anti-inflammatory drugs and systemic and intravitreal corticosteroids.[1] The use of corticoids in the treatment, regardless of the route, may trigger elevations in intraocular pressure by causing structural changes in the trabecular meshwork, reducing the phagocytic activity of its cells and causing increased resistance to the aqueous humor drainage.[3,5] The patient addressed in this report exhibited Irvine-Gass syndrome, was unresponsive to initial clinical therapies, and presented significant uncontrolled intraocular pressure with the use of corticosteroid. Surgical therapy was indicated after clinical attempts to control glaucoma were exhausted. We opted for the surgery with an Ahmed drainage implant due to the inflammatory character that Irvine-Gass syndrome presents in its pathogenesis, associated with the elevation of intraocular pressure caused by corticoids. This was the option with the highest probability of success for guaranteeing

the drainage of the aqueous humor, without further progression of the disease, and was carried out in conjunction with a simultaneous intravitreal dexamethasone injection, aiming at a different approach from the ones already used to resolve the macular edema.[10]

Although the two treatments are antagonistic in their side effects, the joint approach allowed the resolution of the complications, significantly improving the final visual acuity and achieving good clinical control of intraocular pressure. This approach may be considered for non-responsive cases of Irvine-Gass syndrome associated with uncontrolled intraocular pressure.

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