

Corticosteroid-induced severe glaucoma and posterior subcapsular cataract: a case report

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Abstract

• Corticosteroid-induced glaucoma (CIG) is a form of open angle glaucoma associated with both topical and systemic administration of corticosteroids. Here we described the clinical findings in a patient with severe glaucoma and posterior subcapsular cataract (PSC) after topical administration of corticosteroid eye drops. We concluded corticosteroid eye drop was an effective medicine for inflammation of the eyes; however, prolonged use could cause severe vision loss as a result of intractable corticosteroid-induced glaucoma and cataract.

• **KEYWORDS:** corticosteroid; glaucoma; cataract

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INTRODUCTION

Corticosteroid-induced glaucoma (CIG) is a form of open angle glaucoma associated with both topical and systemic administration of corticosteroids^[1]. CIG cases have been reported occasionally, but the case that patients suffered from severe CIG and posterior subcapsular cataract (PSC) at the same time has been reported rarely. In most cases, topically administered medication, such as corticosteroid eye drops or ointment, is the primary cause leading to glaucoma and cataract. However, due to various conditions, some CIG patients will not see doctors until severe visual disorder is present. In this article, we will report a case of severe CIG and PSC in a young Chinese woman.

CASE REPORT

A 29-year-old young woman was in good health. One year previously, because of itching in both eyes, she began using the eye drop Diquedang (Neomycin + Dexamethasone, dexamethasone 1g/L). This eye drop eased her discomfort, so she used it whenever her eyes bothered her. This continued for over a year, until she began to experience dull pain and

reduced eyesight. She went to a rural clinic and was diagnosed with glaucoma. Unfortunately, although she was managed with topical pressure-lowering medication, 5g/L timolol, Diquedang eye drops had not been discontinued at once. As a result, the increased intraocular pressure (IOP) was not reduced. Three months later, this patient suffered from severe visual disorder.

Ocular examination revealed the following: uncorrected visual acuity (UCVA) was 8/400 in the right eye and 30/400 in the left eye. The corneas were clear, with deep and clear anterior chambers. The color of each iris was normal, without dark pigmentation and new vessels. The sizes of the pupils differed; right eye was 6mm and left was 5mm, with slow direct and indirect light reflexes. Both crystalline lenses were opaque with PSC (Figure 1). Glaucomatous disks and optic atrophy were found in both eyes. Value of cup/disc was 1.0 in right and 0.8-0.9 in left eye (Figure 2). The anterior chamber angles of both eyes were open. IOP was RE: 44mmHg and LE: 25mmHg. Severe peripheral visual field loss was found in both eyes using Humphrey visual field analysis (Figure 3). Family history for glaucoma was negative. Clinical diagnosis: ① CIG (OU); ② Complicated cataract (PSC, OU).

The corticosteroid eye drop was ceased and the patient was treated with three topical pressure-lowering medications OD and one OS. Within one week, the IOP in the right eye normalized to 16mmHg and in the left eye to 14mmHg. Two months later, the patient underwent surgery for cataracts and intraocular lens implant. After four months, the corrected visual acuity was 20/200 OD and 20/50 OS. The IOP was 18mmHg OD and 16mmHg OS without any pressure-lowering agents.

DISCUSSION

Corticosteroid-induced ocular hypertension was first reported in 1950, with the observation of glaucoma in association with the systemic administration of adrenocorticotrophic hormone (ACTH)^[2]. Since then, CIG has been studied intensively. The exact mechanism of CIG has not been fully explained, but it has been proven that corticosteroids can increase aqueous outflow resistance. The morphological examinations revealed accumulations of basement membrane-like and fine fibrillar-like materials in the outer trabecular meshwork (TM) of CIG specimen. Type IV collagen, anti-heparan sulfate proteoglycan (HSPG) and fibronectin antibodies in these specimens showed a greater degree of staining in the outer TM in comparison to the primary open angle glaucoma (POAG)

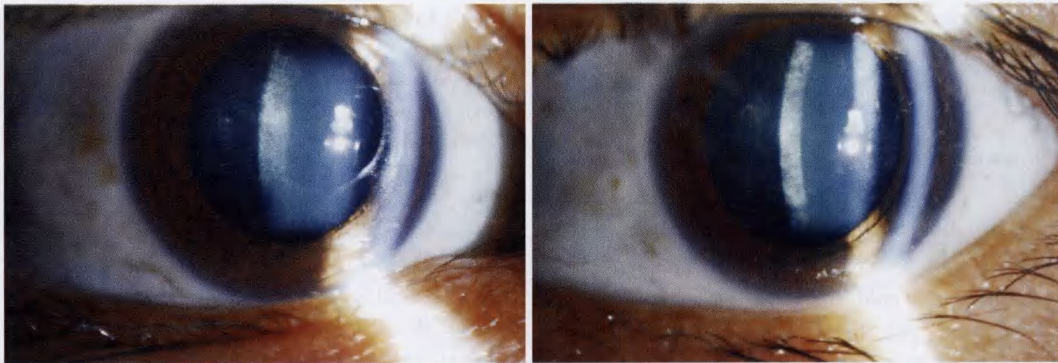


Figure 1 Slit-lamp microscope photographs illustrating posterior subcapsular cataract in both eyes

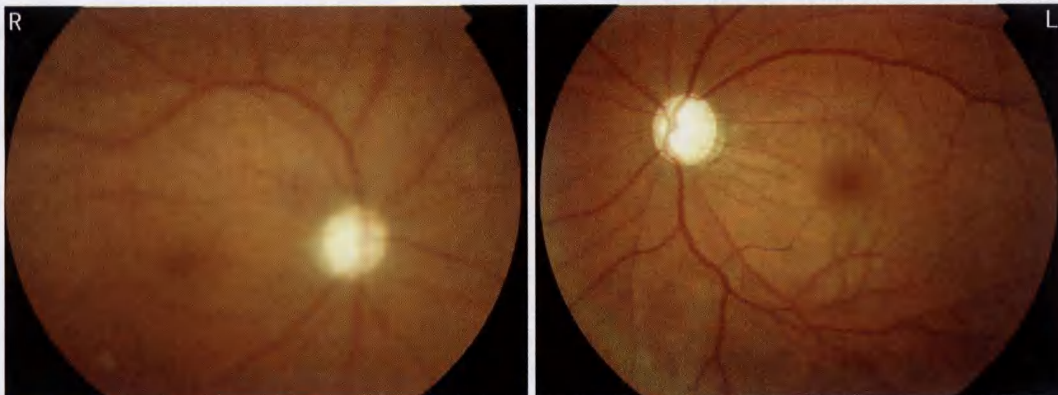


Figure 2 Bilateral fundus photographs showing glaucoma optic nerve cupping in left eye. We could not get a clear picture of right eye because of reaction after cataract and IOL implant operation

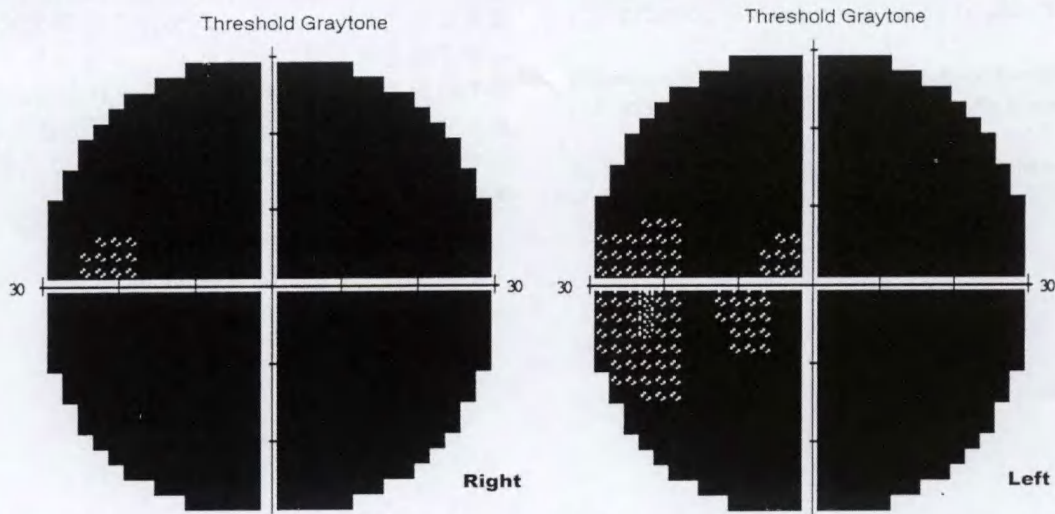


Figure 3 Severe peripheral visual field loss was found in both eyes using Humphrey visual field analysis

and non-glaucomatous specimens^[3]. In CIG there is also an increase in fine fibrillar material in the subendothelial region of sclera. These fibrils do not adhere to the sheath of the elastic fibers but are deposited underneath the inner wall endothelium^[4]. Some of these changes may be responsible for the increased outflow resistance and elevated IOP.

In addition to user of corticosteroid eye drops or ointment, high long-term risks of PSC development were found for patients of combined inhaled and oral corticosteroids^[5]. 18% patients had PSC progression in the 12 months after triamcinolone acetonide injection. The mean time to the cataract progression was 8.8 ± 3.7 months^[6]. The exact pathogenesis has not been clarified. Low doses of dexamethasone cause a moderate increase in proliferation of cultured human

lens epithelial cells (HLECs). Slightly higher but still physiologically relevant concentrations of dexamethasone result in a dose-dependent increase in apoptosis. Effects on proliferation and/or dysregulation of apoptosis in lens epithelial cells may be an important factor in human corticosteroid-induced PSC^[7]. The data indicate steroid treatment of lens epithelial cells is associated with significant changes in gene expression in several functional categories and these include transcripts related to cell proliferation^[8].

In recent years, intravitreal triamcinolone acetonide (IVTA) has been recognized as an effective therapy for uveitis, veno-occlusive disease, diabetes, and choroidal neovascularization. Moreover, with laser keratorefractive surgery popularizing, CIG caused by topical application of corticosteroid drops

increased rapidly^[9]. More and more patients suffered from corticosteroid-induced ocular hypertension and PSC. Ophthalmologists should pay more attention to this complication of IVTA. If the patient in this article could have been found and treated early, the result would be different completely.

In this case, there are two points that must be taken into consideration by doctors and patients. The first is medicine management. This situation would not have occurred if the patient in this case had not been able to acquire the Diquedang eye drop so easily. It is necessary that the government enforce stricter prescription protocols as soon as possible. The second point is the need to popularize basic medical knowledge. In developing countries, the large rural populations and struggling economies make it very difficult to popularize medical knowledge. This case report tells us that we should not forget corticosteroids' serious side effects when enjoying its benefits.

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激素诱发的晚期青光眼和后囊下性白内障 1 例

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摘要

激素性青光眼是由于局部或全身应用糖皮质激素导致的一种开角型青光眼。我们报道局部应用糖皮质激素导致的 1 例激素性青光眼并后发囊下性白内障, 从而得出结论糖皮质激素对于眼部炎症是一种很好的药物, 但长期的应用可引起严重青光眼和白内障, 导致视力严重丧失。

关键词: 糖皮质激素; 青光眼; 白内障