Case Report

# Endocapsular Hematoma: A Rare Form of Ocular Hemorrhage after Thrombolysis with Streptokinase

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## Abstract

**Context:** Endocapsular hematoma has previously been described as a cataract surgery complication more commonly observed in eyes receiving a combined cataract and glaucoma surgery. However, it can also be a rare form of ocular bleed following thrombolysis with streptokinase. **Case Report:** A 65-year-old diabetic male presented to us with complain of sudden painless diminution of vision in his left eye, which he developed while he was being thrombolysed with streptokinase administered intravenously for an episode of acute myocardial infarct. On examination, left eye was pseudophakic with collection of blood in the capsular bag (Endocapsular hematoma). **Conclusion:** Endocapsular hematoma can be a rare form of ocular bleeding complicated by thrombolysis. Reporting this case becomes more relevant in the present era with increasing number of patients suffering from ischemic heart disease and likely to undergo thrombolysis.

**Keywords:** Endocapsular hematoma, Streptokinase, Thrombolysis

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## Introduction

Endocapsular Hematoma (ECH) in pseudophakic eyes is often attributed to as a late post-cataract surgery complication, more so if the cataract surgery is combined with a trabeculectomy or with pre-existing neovascularisation of iris or capsule.[1-3] Thrombolysis can rarely cause ocular hemorrhage, the risk of which is increased in patients with diabetes. Therefore, diabetic retinopathy has been identified as a contraindication to thrombolytic therapy.[4] We describe for the first time endocapsular hematoma in pseudophakic eye of a diabetic case following administration of streptokinase for an acute myocardiac infarct.

## Case Presentation

A 65-year-old male presented to us with sudden painless loss of vision in the left eye of 3 weeks duration when he was thrombolised with streptokinase for an acute inferior wall myocardial infarct. There was no history of ocular trauma. The patient was on oral aspirin in the dose of 150 mg daily for an episode of ischemic heart disease since over 1 year and on high dose aspirin in the dose 600 mg daily since 4 days after the heart attack. The patient is a known case of diabetes mellitus since past 10 years and is receiving oral anti-diabetic medication. The patient does not have a documented status of blood sugar levels over past 10 years. The patient underwent uneventful cataract surgery with intraocular lens implant in both eyes elsewhere, 4 months back in the right and 2 years back in left eye. The right eye was diagnosed to have proliferative diabetic retinopathy 2 years back and was treated with panretinal photocoagulation. His best corrected visual acuity was 20/40 in his right eye and light perception with accurate projection of rays in the left eye. Right eye was pseudophakic with normal anterior segment and gonioscopy findings. Intraocular pressure (IOP) was 12 mmHg. Examination of fundus revealed findings of stable lasered proliferative diabetic retinopathy. The left eye had a posterior chamber intraocular lens (PCIOL) in the bag. There was a thick hematoma behind PCIOL and anterior to the posterior capsule in the capsular bag [Figures 1 and 2]. IOP was 12 mmHg. Fundus was not visible due to thick and total hematoma. No iris new vessels were seen. B Scan

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showed anchogenic vitreous cavity ruling out any vitreous hemorrhage [Figure 3]. There were no angle vessels on gonioscopy and angles were open. Routine hematological profile including coagulation screen was normal.

We had treatment options of either clearing visual axis with neodymium (Nd)-YAG Laser capsulotomy primarily aiming to restore vision or surgical removal of the clot by a limbal route. The later was preferred in view of the need to evaluate the fundus and perform a Fundus Fluoroscein Angiography (FFA) later in order to rule out a proliferative diabetic retinopathy in the left eye. Patient had a cleared clot, an intact posterior capsule, with restoration of vision of 20/40 on first post operative day [Figure 4]. Postoperative fundus was normal with no evidence of neovascularization on retina [Figure 5]. FFA done on first week follow up revealed subtle neovascularisation on disc [Figure 6], for which pan retinal photocoagulation was later performed.

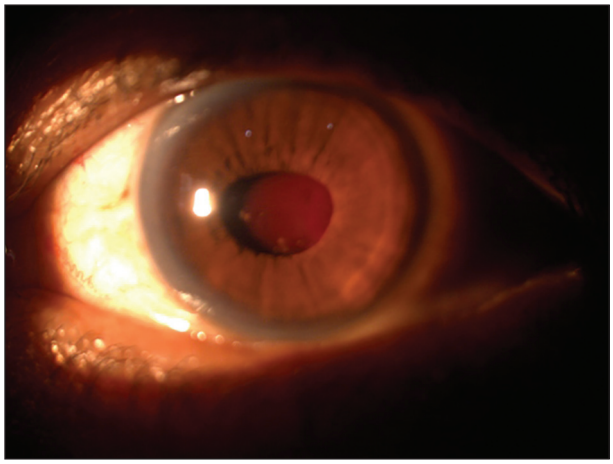

**Figure 1:** Slit lamp picture of the left eye in diffuse illumination showing endo capsular hematoma

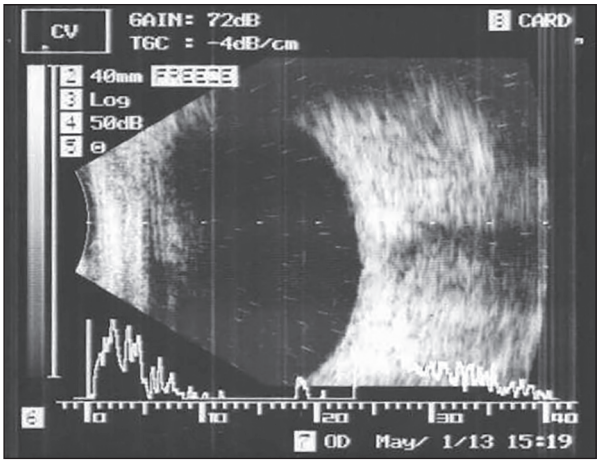


**Figure 3:** B-scan of the left eye showing an echogenic vitreous

## Discussion

First issue is to rule out ECH in this case as a post cataract surgery complication. Cataract surgery was done in the left eye 2 years back and postoperative course over this period was uneventful. It was not a combined cataract and glaucoma surgery. However, ECH as a cataract surgery complication is reported to be seen within a few weeks after cataract surgery combined with a trabeculectomy.[1,2]

Second issue is the role of diabetic status of the patient in as a possible etiology and or contributing factor. Unlike previous reported cases of ECH in diabetic subjects, where neovascularisation of iris or capsule was responsible for the bleed, our case did not have any rubeosis iridis or capsulare as a source of bleed.[3] Hence, we believe that thrombolysis with streptokinase caused this bleed probably from friable iris vessels, since

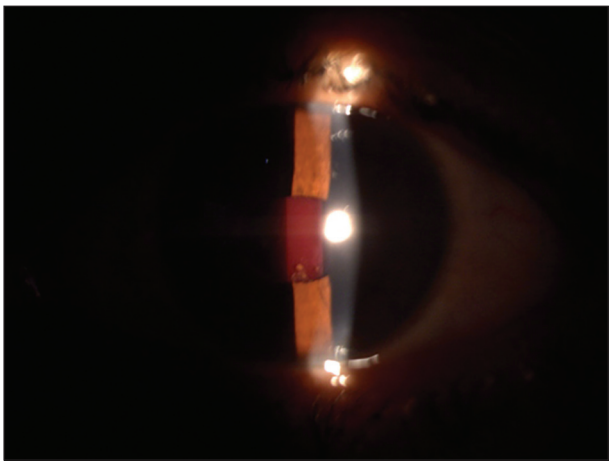

**Figure 2:** Slit lamp picture of the left eye in slit beam illumination showing posterior capsular intraocular lens and endo capsular hematoma

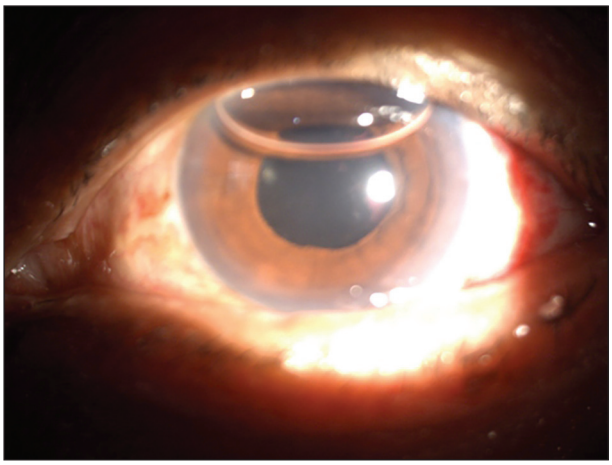

**Figure 4:** Slit lamp photo of the left eye on post-operative day 1 showing a cleared axis with posterior capsular intraocular lens *in situ* and a superior air bubble

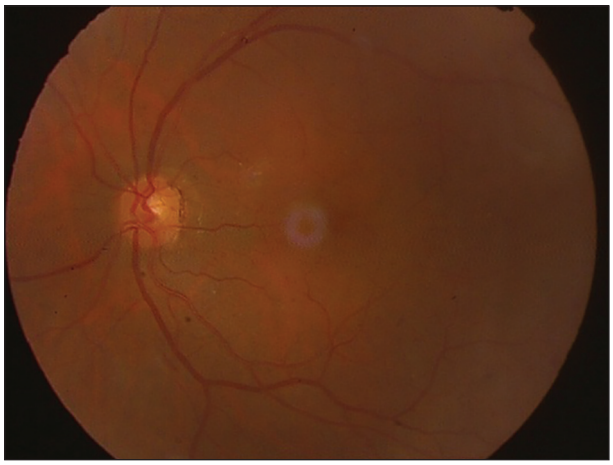

**Figure 5:** Fundus photograph of the left eye showing no obvious neo-vascularization on the retina

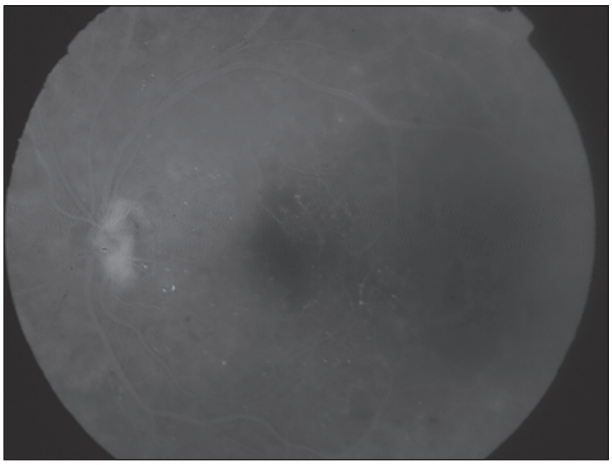

**Figure 6:** Fundus Fluoroscein Angiography of the left eye revealing a subtle neo-vascularization fo the disc

this eye already had a proliferative diabetic retinopathy. This could explain ECH in our report but findings of neovascularisation were not present in this case to qualify as rubeosis capsular.

Ocular hemorrhage, and more importantly, intraocular hemorrhage after thrombolytic therapy for acute myocardial infarction is extremely uncommon. The incidence of intraocular hemorrhage in patients with diabetes is 0.05%. Most common type of hemorrhage reported was sub-conjunctival hemorrhage reported, followed by periorbital hematoma and retinal hematoma.[4] ECH complicating a thrombolytic therapy is not yet been reported. However, one report describes a case of total hyphema in a patient who underwent cataract surgery 8 days before, after the intravenous administration of 750000 units of streptokinase for massive anterior wall myocardial infarction.[5]

Regarding the role of treatment for ECH, most of the times ECH is minimal and absorbs on its own over a few weeks or months, sometimes it may require Nd-YAG laser capsulotomy to drain the endocapsular blood into the vitreous cavity.[1,2,6] However, in the present case, it was a total ECH failing to absorb on its own till 10 days of onset and the blood was thick enough to warrant a surgical evacuation, which in turn made post-surgical fundus evaluation and FFA possible.

Clinicians should be aware of the risk of visual loss after thrombolysis in high-risk patients. In patients with prior history of ocular disease, the risk of hemorrhage and subsequent irreversible vision loss needs to be discussed with the patient and family. Ophthalmologists should consider history of thrombolytic therapy as a cause while dealing with a rare endocapsular hematoma or any other ocular hemorrhage for that matter.

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