

Spontaneous Suprachoroidal Hemorrhage as a Complication of Tissue Plasminogen Activator Use

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Abstract

Suprachoroidal hemorrhage (SCH) occurs most commonly as a result of trauma or intraocular surgeries. In this report, we present a rare case of spontaneous SCH in an 82-year-old woman with no known ophthalmic disorder after receiving intravenous recombinant tissue plasminogen activator for treatment of acute ischemic stroke. Spontaneous, nontraumatic SCH is a potential sight-threatening complication of thrombolytic therapy in susceptible individuals which should be discussed with patients.

Keywords: Spontaneous Suprachoroidal Hemorrhage; Subchoroidal Hemorrhage; Recombinant Tissue Plasminogen Activator; Thrombolytic Therapy; Ischemic Stroke

Abbreviations

SCH: Suprachoroidal Hemorrhage; r-tPA: Recombinant Tissue Plasminogen Activator; VH: Vitreous Hemorrhage; IOP: Intraocular Pressure; COPD: Chronic Obstructive Pulmonary Disease; CAD: Coronary Artery Disease; CHF: Congestive Heart Failure; AMD: Age-related Macular Degeneration; AMI: Acute Myocardial Infarction

Introduction

Spontaneous, nontraumatic suprachoroidal hemorrhage (SCH) is extremely rare and has been described mostly in patients with advanced age, severe myopia, hypertension, underlying blood dyscrasias, use of systemic anticoagulation, and age-related macular degeneration (AMD) [1]. We report a case of spontaneous SCH after use of intravenous recombinant tissue plasminogen activator (r-tPA) for the treatment of acute ischemic stroke.

Case Presentation

An 82-year-old woman with past medical history of hypertension, hyperlipidemia, chronic obstructive pulmonary disease (COPD), coronary artery disease (CAD), congestive heart failure (CHF), and stroke presented emergently with acute pain and sudden loss of vision in her right eye within a day of treatment with intravenous r-tPA for presumed acute ischemic stroke. Visual acuity was hand motion and 20/20 in her right and left eye, respectively. Anterior segment examination revealed clear cornea, shallow anterior chamber with few red blood cells but no layered hyphema or rubeosis iridis, and a 3-piece posterior chamber intraocular lens within an intact capsule. Dense vitreous hemorrhage (VH) was noted on dilated examination with no view of the optic nerve and retina. B-scan ultrasonography demon-

strated diffuse VH with hemorrhagic choroidal detachment. Fundus examination of the left eye was unremarkable except for a posterior vitreous detachment and rare drusen in the macula. Her right intraocular pressure (IOP) was 68 mmHg for which she was treated with brimonidine, dorzolamide, latanoprost, and oral acetazolamide. Her IOP normalized to 8 mmHg over 2 days but the vision worsened to light perception. The patient underwent combined pars plana vitrectomy and partial drainage of choroidal hemorrhage for persistent VH and hemorrhagic choroidal detachment. The patient's vision worsened to no light perception over the next few days after which she developed endophthalmitis and underwent primary enucleation.

Histologic examination of the right eye reveals suprachoroidal organizing tissue with hemosiderin-laden macrophages, fibroblasts, and lymphocytes (Figure 1A and 1B). Prussian blue stain highlights hemosiderin-laden macrophages (Figure 1C). These findings are consistent with the organizing stage of a suprachoroidal hemorrhage.

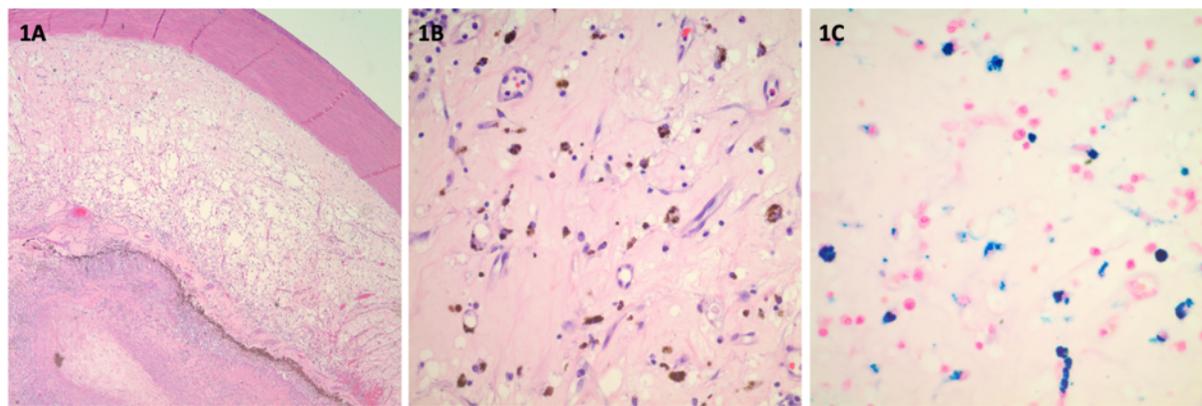


Figure 1: Figure 1A: Low-power histopathology section of patient's right globe demonstrates suprachoroidal organizing hemorrhage. Figure 1B: High-power view of suprachoroidal organizing hemorrhage reveals hemosiderin-laden macrophages, fibroblasts, and lymphocytes. Figure 1C: Histopathology section stained with Prussian blue highlights hemosiderin-laden macrophages.

Discussion

Thrombolytic therapy with intravenous r-tPA is the mainstay treatment of acute ischemic stroke. Initiation of r-tPA within 4.5 hours of stroke onset improves in-hospital mortality rate and neurologic recovery [2]. Other uses of r-tPA include treatment of acute myocardial infarction (AMI) and pulmonary embolism. Bleeding complications of r-tPA such as symptomatic intracranial hemorrhage and major systemic hemorrhage occur in approximately 6% and 2% of patients, respectively [3]. In contrast, ophthalmic complications are rare; the incidence of intraocular hemorrhages following thrombolytic therapy for AMI is estimated to be 0.002% [4]. Extensive review of the literature revealed only a handful of case reports of spontaneous SCH after thrombolytic therapy, mostly for the treatment of AMI, and usually in patients with diabetic retinopathy or exudative AMD [5-10]. There is only one report of SCH after r-tPA for an acute ischemic stroke in a patient with no known history of exudative AMD [11].

Our case of spontaneous hemorrhagic choroidal detachment occurred within a day of intravenous administration of r-tPA for an acute ischemic stroke in a patient with advanced age, CAD, CHF, and COPD and no known ophthalmic disorder. While a direct causal relationship cannot be definitively established, the timing of acute visual loss strongly suggests such a relationship.

Conclusion

In summary, sight-threatening complications of SCH, while rare, can occur after intravenous thrombolytic therapy. Internists, cardiologists and neurologists should be aware of this potential ocular complication in high risk patients with advanced age, severe myopia, hypertension, and AMD, and should counsel patients accordingly.

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