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SHORT COMMUNICATION

Case report

Intraocular hemorrhage after systemic thrombolytic therapy in a patient with exudative macular degeneration

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PURPOSE. *To report a hemorrhagic complication from thrombolytic therapy in a patient with exudative macular degeneration*

CASE REPORT. *A 75 year old patient with exudative macular degeneration developed pain and loss of vision in the left eye shortly after receiving tissue plasminogen activator (t-PA) for a myocardial infarction. Examination revealed the patient to be in angle closure. A CT scan revealed the etiology of the angle closure to be a dense vitreous hemorrhage pushing the iris-lens diaphragm forward. Intraocular pressure was treated successfully, but the final visual acuity was only light perception*

CONCLUSIONS. *Thrombolytic therapy can lead to devastating intraocular hemorrhages. The presence of exudative macular degeneration may potentially increase the risk of developing such complications. (Eur J Ophthalmol 2003; 13: 96-8)*

KEY WORDS. *Intraocular, Hemorrhage Thrombolytic, Complication, Macular degeneration*

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INTRODUCTION

Thrombolytic therapy is widely used as a treatment of acute thromboembolic disorders, in particular, myocardial infarctions. Hemorrhagic complications of thrombolytic agents are the main reason for their associated morbidity and mortality. Both intraocular and orbital hemorrhages have been reported with the systemic use of these agents (1-4). As expected, the risk of ocular or peri-orbital hemorrhage is significantly higher in patients who have undergone ocular or orbital surgery shortly before receiving thrombolytics. However, patients with a remote history or no prior surgery are also susceptible. Reports have included bilateral vitreous hemorrhage (1), suprachoroidal hemorrhage with secondary angle closure (2-3), and choroidal hemorrhage (4) all in patients with no significant past ocular history. The visual results can be devastating, particularly if not recognized and treated early. In the three cases reported by Chorish et al (4), two patients suffered significant visual loss (light perception or worse) due to glaucoma or compressive optic neuropathy. Herein we describe a patient with age-related macular degeneration who developed acute angle closure resulting from intraocular hemorrhage following systemic administration of t-PA.

Case report

A 75 year old female complained of loss of vision in her left eye approximately 12 hours after receiving t-PA for an anterior myocardial infarction. The patient had a history of exudative macular degeneration in

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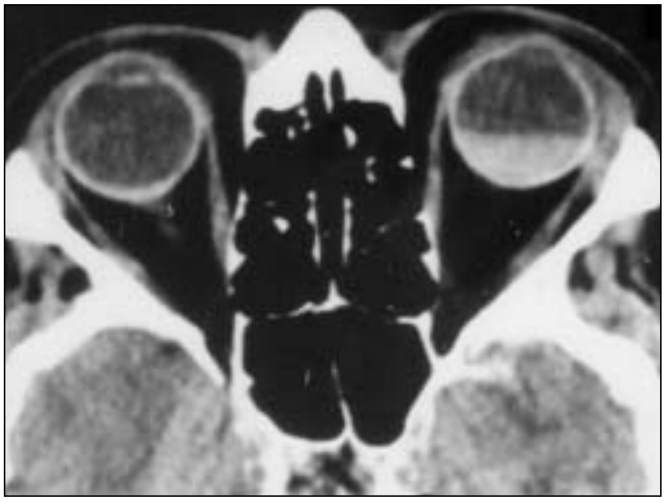

Fig. 1 - *A head CT scan of the patient after receiving systemic t-PA revealed a dense vitreous hemorrhage in the left eye. Note the shallow anterior chamber in the left eye compared to the normal right eye.*

both eyes and both had been treated with laser photocoagulation multiple times in the last few years. The left eye also had a stage 3 macular hole. Both eyes had undergone cataract extraction with placement of posterior chamber lenses, two years earlier. The baseline visual acuity was 8/200 in each eye.

Upon examination in the coronary care unit, the patient's visual acuity was HM OD and light perception OS. The intraocular pressure was 13 mmHg OD and 41 mmHg OS. The left pupil was fixed and mid-dilated with a relative afferent defect. The left cornea was edematous and the anterior chamber was shallow. There was no view of the left fundus. A CT scan which had been obtained to rule out an intracranial bleed, revealed a dense intraocular hemorrhage which was also pushing the lens and iris forward resulting in angle closure configuration (Fig. 1). The intraocular pressure was treated aggressively with topical medications. The next day, the intraocular pressure had normalized and the patient had a hypopyon presumably due to the inflammatory reaction to the angle closure. B-scan ultrasonography revealed the presence of a vitreous hemorrhage with no choroidal detachment. The ocular inflammation was successfully treated with topical steroids, but the eye became hypotonous. At 3 months, there was persistent vitreous blood and the eye remained hypotonous. Her final visual acuity was light perception.

DISCUSSION

Systemic thrombolytic agents can occasionally lead to hemorrhagic ocular complications (1-4). Overall, the risk of ocular bleeding after systemic thrombolytic therapy appears to be quite low. Among 40,899 patients enrolled in the GUSTO trial (Global Utilization of Streptokinase and t-PA for Occluded coronary arteries), only 12 patients (0.03%) developed an ocular hemorrhage. Intraocular hemorrhage was confirmed in only one patient (5). Traditionally, the risk factors for ocular hemorrhage have included prior ocular surgery and proliferative diabetic retinopathy. However, Mahaffey et al (5) reviewed the patients in the GUSTO trial and found only one case of ocular hemorrhage (eyelid hematoma after a fall) in the 6,011 patients (15%) with a history of diabetes. They concluded that diabetic retinopathy should not be considered a contraindication to thrombolysis.

Massive subretinal or vitreous hemorrhage has previously been reported as a complication of age-related macular degeneration (5-8). A review of 15 cases by el Baba revealed that 19% were taking warfarin or aspirin at the time of their hemorrhage (7). Pathologically, in that study the source of hemorrahge was demonstrated to be a large choroidal artery emerging from breaks in Bruch's membrane (7). In our case, we could not determine the source of hemorrhage and can only suspect the choroidal neovascular membrane. However, it could have also been from a suprachoroidal hemorrhage which in turn could have also contributed to the subsequent development of hypotony. Either way, exudative macular degeneration should not be considered a contraindication to potentially lifesaving thrombolytic or anticoagulation therapy.

The mechanism of the elevated intraocular pressure with such massive hemorrhages can either be secondary angle closure or the sudden increase in the volume of the posterior cavity. Wood et al (8) reported 6 eyes that developed acute angle closure due to a total hemorrhagic retinal detachment. In our case, the etiology of acute angle closure was interestingly determined by CT scan. Clinically, the patient was difficult to examine in the coronary care unit and having the CT scan allowed us to differentiate angle closure secondary to the mass effect of the hemorrhage from other causes such as pupillary

block or aqueous misdirection. This differential is important since the treatment is quite different. Acute angle closure due to a posterior vitreous mass is treated with cycloplegics and aqueous suppressants while pupillary block is treated with laser iridotomy.

To our knowledge this is the first case report of intraocular hemorrhage after systemic thrombolytic therapy in a patient with exudative macular degeneration. While the risk appears to be low, patient counseling may be warranted prior to administration of thrombolytics.

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