Intravenous Thrombolysis for Acute Ischemic Stroke After Vitrectomy for Retinal Detachment

To the Editor:

Once the benefits of intravenous tissue plasminogen activator (tPA) in the treatment of acute ischemic stroke have been proved, it should be interesting to widen its use. We discuss the fact that intravenous thrombolysis with tPA may not be a contraindication to treat an acute ischemic stroke occurring soon after surgical repair of retinal detachment (RD) by means of vitrectomy.

A 65-year-old woman with a history of atrial fibrillation underwent vitrectomy for surgical repair of RD in her right eye. Vitrectomy required 3 incisions into the posterior chamber of the eye using a vitreous cutter of 25 G, an infuser of 20 G, and a light source of 20 G. Thirty-two hours later, and while in the hospital, she experienced an embolic ischemic stroke in the left middle cerebral artery territory. Immediately after the stroke onset, the NIHSS was 13. Despite the recent surgical procedure and considering the degree of neurologic deficit, we decided a tPA intravenous thrombolysis according to the SITS-MOST protocol. Both the tolerance and response of the patient were satisfactory and her NIHSS score at completion of tPA intravenous thrombolysis was 0. No complications were observed.

Vitrectomy is the most common surgical procedure used to repair RD, combined, in most cases, with scleral buckling. In this procedure the vitreous is removed and replaced with saline solution, intraocular gas, or silicone oil to repair the retinal defect. Suprachoroidal hemorrhage (SCH), which usually causes secondary acute glaucoma, is a serious but exceptional complication. Most commonly, a vitreous hemorrhage (VH) may occur affecting visual prognosis depending on its severity. No prospective studies analyzing the incidence of both complications in relation to the surgical repair of RD have been reported. Falkner et al. reported a 4.3% VH incidence in a large retrospective sample of patients undergoing vitrectomy, whereas Sharma et al. observed a 0.17% incidence of SCH in another retrospective sample. Risk factors associated with the occurrence of SCH during vitrectomy include old age, severe myopia, history of surgical repair of RD, rhegmatogenous RD, the use of cryotherapy, intraoperative systemic hypertension, and, as a technical factor, scleral buckling combined with vitrectomy.3,4

Regarding the use of thrombolytic therapy, 3 cases of spontaneous SCH secondary to the systemic administration of intravenous tPA for acute myocardial infarction5–7 have been reported. There are, however, no reports of secondary VH.

The only formal contraindication for the administration of intravenous tPA in our patient would have been the recent puncture at a noncompressible site, which might cause VH. In this sense, we must remark that the instruments used in the surgical procedure were not thick, ranging from 20 to 25 G, and that their risk of causing VH seemed low according to the evidence found in the literature. However, it is well-known that VH and SCH occur immediately after surgery, that the risk decreases with time, and that the development of severe glaucoma is a sign of hemorrhage. In our patient these complications went unnoticed; 32 hours had elapsed from surgery, she presented a severe stroke, and the therapeutic window was very short, so after balancing risks and benefits we decided to treat her with intravenous tPA. Fortunately, the clinical result was excellent and the patient recovered completely at the completion of the treatment.

In view of this, in our opinion surgical repair of RD by means of vitrectomy in combination or not with scleral buckling does not pose an absolute contraindication for the administration of intravenous tPA for acute ischemic cerebral stroke.

Disclosures

None.

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