the diagnosis. Indeed, there are no standard assays for such antibodies, no standardized criteria for distinguishing positive from negative results, and no data on sensitivity and specificity of these assays based on clinical criteria for a causal relationship.\(^1,3\)

Although causality assessment methods in pharmacology remain a matter of debate, in our patient acetazolamide caused pure and severe thrombocytopenia with “certain” evidence according to the World Health Organization system of causation of a drug reaction,\(^6\) with “very likely” evidence according to the French standardized methodology,\(^7\) and with the highest level of evidence (“definite”) according to standardized criteria recently developed by Rizvi et al\(^2\) (database available at http://moon.ouhsc.edu /jgeorge). Such high levels of evidence for the causal relationship of acetazolamide to thrombocytopenia have never been reported until now, to our knowledge.

Discovery of isolated thrombocytopenia in a patient who is taking several medications also presents a challenging clinical problem. The principal interest of the level of evidence is to help clinical decision making about which drugs may more likely be implicated as a cause of thrombocytopenia and therefore should be discontinued as quickly as possible.

Acetazolamide should be considered a definite thrombocytopenia-inducing agent. Potential consequences of thrombocytopenia seem to be limited when the drug is prescribed for a few days, whereas it appears different with much longer treatment. In that case, regular complete blood cell count, especially in the presence of bleeding, should be recommended.\(^5\)

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**Choroidal Neovascularization After Globe Penetration by Peribulbar Anesthesia**

Iatrogenic choroidal neovascularization is a rare complication of ocular surgery. It is usually a result of laser photocoagulation, retinal cryotherapy, or subretinal fluid drainage. It was believed to be induced by damaging the Bruch membrane and/or retinal pigment epithelium, from which the reparative processes trigger the release of angiogenic factors.\(^1\)

We report a case of global penetration-induced choroidal neovascularization following peribulbar anesthesia for cataract surgery.

**Report of a Case.** A 75-year-old woman originally scheduled for phacoemulsification with an intraocular lens implant in the right eye had a procedure complicated by global penetration during peribulbar anesthesia. Dilated fundus examination revealed a suspected penetration site; preretinal and subretinal hemorrhages were also found in the right posterior pole. The operation subsequently proceeded because the intraocular pressure was not soft after penetration. It was then complicated by a posterior capsular tear, and an anterior vitrectomy was done to complete the surgery. Two months after surgery, the patient’s best-corrected visual acuity was 20/100 OD with preretinal and subretinal hemorrhages around the macula on ophthalmoscopic examination.

Preoperatively, the patient was a hypermetrope. Subjective refraction was +1.5 diopters (D) in the right eye and +1.0 D in the left eye. Best-corrected visual acuity was 20/60 OU. The axial length was 23.04 mm in right eye and 23.21 mm in left eye.

**Figure 2.** Timeline of platelet count after reintroduction of acetazolamide. Vertical lines indicate period of acetazolamide administration.
Examination of cornea, anterior chamber, pupil, and intraocular pressure readings were unremarkable except nuclear sclerosis of bilateral lens. There were no signs of macular degeneration in each eye.

The visual acuity was stable until 8 months after the surgery. The patient visited the clinic complaining of a gradual blurring of her vision in the right eye. Dilated fundus examination revealed residual preretinal hemorrhage and a choroidal retinal lesion, corresponding to the previously suspected penetrating site (Figure, A). In the early phase of a fluorescein angiography, there was an oval lesion with hypofluorescence and a clear margin surrounded by a ring of hyperfluorescence (Figure, B). The adjacent area became progressively hyperfluorescent during the transit phase with leakage in the late phase (Figure, C and D). The patient was diagnosed with iatrogenic choroidal neovascularization resulting from global penetration while administering peribulbar anesthesia.

Comment. Peribulbar anesthesia, during which local anesthetic is injected outside the muscle cone, has been cited by proponents as having the advantages of greater ease of performance and a lower rate of globe perforation. However, ocular penetrations (single entry) and perforations (entry wound and exit wound) have been reported occasionally, especially in patients with long axial length.

In clinical situations, the detection of preretinal, subretinal, or vitreous hemorrhage either immediately after surgery or on postoperative visits should remind the physician of the possibilities of global penetration. Most of the penetrating site becomes a chorioretinal scar rather than a choroidal neovascularization in the end.

The penetrating site, which was very close to the macula, developed into choroidal neovascularization that resulted in visual loss in this patient. This represents an unusual complication of global penetration by peribulbar anesthesia. To our knowledge, there are no prior reported cases of choroidal neovascularization developed in the penetrating site by peribulbar anesthesia. Ophthalmologists should be aware of this complication, which might lead to loss of vision.

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Acute Severe Vision Decrease Immediately After Photodynamic Therapy

Ocular photodynamic therapy (PDT) with verteporfin has been shown to be an effective treatment for occult subfoveal choroidal new vessels (CNV) in age-related macular degeneration, but is associated with acute severe vision decrease (ASVD) in 4.4% in patients who received the treatment.1

In this case the patient complained of decreased vision within hours of treatment, affording the opportunity to examine and assess the mechanism of vision loss 4 hours after therapy.

Report of a Case. An 82-year-old female patient had a sudden decrease in visual acuity. On examination, her acuity had decreased from 20/30 to 20/40 OU and her fundus showed a mild mottling of the pigment epithelium and no evidence of hemorrhage or exudative abnormalities. Fluorescein angiography showed an occult subfoveal CNV of 2.5 disc areas in size (Figure 1). On the basis of recent disease progression2 and small lesion size she was treated with PDT. Following infusion of verteporfin (6 mg/m²), laser light at 698 nm was applied using a 3.5-mm spot, with an intensity of 600 mW/cm² for 83 seconds.

Two to 3 hours later, the patient reported a dramatic decrease in her central vision with increased distortion and was examined 4 hours after PDT. Her Snellen visual acuity was 20/200 OU. Stereoscopic fluorescein and indocyanine green angiography revealed a gross central serous retinal detachment and outlined the CNV within the choroid (Figure 2). There was an intense spot of hyperfluorescence on the superotemporal margin of the CNV indicating a focal area of hyperpermeability. This was confirmed in the midphase showing a pool of indocyanine green collecting under the pigment epithelium (Figure 3). Over the next few days, the patient reported gradual disappearance of the “gray shadow” obscuring her vision and a return of her ability to read. After 4 days, the visual acuity returned to 20/40 OU and angiography confirmed complete closure of the occult CNV, cessation of hyperpermeability and leakage, and resolution of the retinal detachment in parallel with a return to pretreatment visual acuity. The visual acuity improved and remained stable at 20/30 OU on examination at 3, 6, and 9 months without further treatment.

Comment. The 4-hour findings in our case were consistent with the preclinical studies which showed that shortly after PDT, the O₂-radical–mediated damage to the cytoskeleton causes rounding and contraction of the endothelial cells,3 interruption of the interendothelial cell tight junctions, and exposure of the subendothelial basement membrane. Histamine is released from the damaged endothelium, and activated polymorphonuclear leukocytes aggregate to the vessel wall, leading to an increase in vascular permeability and a propensity for exudation and edema.4 The 4-day findings were consistent with the studies showing that PDT dam-

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