I. **Case History**

Demographics

59-year-old white male

Chief complaint

Metamorphopsia OS following a fall from a 6ft ladder resulting in coma, multiple skull fractures, and a subarachnoid hemorrhage.

Ocular history

1. Non-visually significant cataract OD, pseudophakia OS
2. Acute posterior vitreous detachment OS two years prior

Medical history

Recent head trauma from falling off a 6ft ladder, occurred two months prior to the eye exam, and resulted in coma, subdural hematoma, subarachnoid hemorrhage, and multiple skull bone fractures

Hypertension

Hyperlipidemia

Sleep Apnea

Coronary Artery Disease

Aortic valve stenosis

Medications

Hydrochlorothiazide/Lisinopril

Clonidine HCL

Levetiracetam

Aspirin

II. **Pertinent findings**

Clinical

Best corrected acuity: OD: 20/25; OS: 20/150, PHNI

Preliminary Testing: Amsler grid OD: normal

OS: metamorphopsia nasal and temporal

Pupils, extraocular muscles, and confrontation visual fields were all normal

Anterior Segment Exam: unremarkable OU

Tonometry (GAT mmHg): 12 OD, 13 OS

Dilated Fundus Exam:

OD: mild sectoral optic nerve edema with small splinter hemorrhages on nerve margin. Few scattered nerve fiber layer and intraretinal hemorrhages throughout posterior pole.

OS: sectoral optic nerve edema. Dense intraretinal hemorrhage adjacent to macula. Scattered large sub-retinal hemorrhages along vascular arcades.

Imaging studies:
- CT scan of brain without contrast showing:
  - Multiple skull fractures including
    - Left occipital fracture with extension to the temporal bone
    - Right zygomatic process fracture
    - Right lateral orbit wall fracture
  - Multiple hemorrhages including:
    - Subdural hematoma
    - Right periorbital hematoma
    - Right lateral extracranial hematoma
    - Subarachnoid hemorrhage
    - Hemorrhage into right lateral rectus muscle
  - Pneumocephalus

- Longitudinal studies over three months with optical coherence tomography (OCT) of retina and optic nerve with accompanying fundus photos
- Initial and follow-up threshold visual fields

### III. Differential diagnosis

**Leading**

- Terson’s Syndrome
- Purtscher’s retinopathy

**Others**

- Valsalva retinopathy
- Commotio retinae
- Hypertensive retinopathy

### IV. Diagnosis & Discussion

Terson’s syndrome (TS) refers to any form of intraocular hemorrhage, either retinal or vitreal, following a subarachnoid hemorrhage (SAH), traumatic brain injury (TBI) or intracerebral hemorrhage. Its incidence is difficult to determine due to the high mortality rate associated with intracranial hemorrhage, though it has been reported to occur in 8-46% of patients with SAH. It is an important diagnosis to make because it is associated with a low Glasgow coma scale, high Hunt and Hess grade, high Fisher scale, and a high mortality rate of up to 90%. Historically, it was believed that TS occurred due to an aneurysmal rupture that transmitted through the optic nerve sheath and deposited into the retina. Recent studies have disproved this mechanism, though, as TS has been reported in cases in which no SAH has occurred. It is now thought to occur due to a rapid increase in intracranial pressure (ICP) transmitted along the optic nerve sheath resulting in venous hypertension and the rupture of retinal vessels.

Common visual complaints from TS include blurred vision and floaters. A vitreous hemorrhage is the most common presentation, but the macula and various layers of the retina are frequently involved as well. Considering the accepted mechanism of TS is increased intracranial pressure,
one would expect papilledema to be a more common finding. However, given the severity of SAH, most studies examined post-mortem eyes or eyes that had the initial intracranial hemorrhage several months prior to the fundus exam and were unable to be examined sooner due to coma or prolonged hospitalization. Therefore, although few studies report papilledema, the true incidence is likely underreported. Presentation is often bilateral, though asymmetric, and signs typically appear within hours to days of the precipitating event. An intraocular hemorrhage may be apparent on CT scan, though definitive diagnosis should be made with fundoscopy. TS has a good visual prognosis with most cases showing spontaneous resolution over a course of weeks, although vitreous hemorrhages may take up to 10-12 months to resolve. Close monitoring for the first three to six months is indicated as complications can occur that may require surgical intervention including epiretinal membrane, retinal folds, proliferative vitreo-retinopathy, retinal detachment and macular holes. In these events early vitrectomy may improve visual prognosis, especially in cases of non-clearing vitreous hemorrhages.

V. Treatment & Management
The patient was initially managed for the intracranial hemorrhages and coma at the ER. He did not need surgical intervention to relieve intracranial pressure or repair the skull bone fractures. He is currently co-managed by his primary care physician and neurologist. The patient will continue to be monitored in the eye clinic monthly for at least six months. As of his five month follow-up, the patient has shown a three-line improvement in visual acuity and improvement of retinal hemorrhages and optic nerve edema.

VI. Conclusion
Terson’s Syndrome is a rare and likely underreported condition, yet its diagnosis is significant as it is associated with high rate of coma and mortality. Intraocular hemorrhages often resolve spontaneously, but they may progress to further complications. Therefore close monitoring with fundus exams is indicated so that prompt surgical intervention can be performed if necessary to improve visual outcome.

VII. Bibliography

