Intracranial Hypertension with Papilledema secondary to Venous Sinus Thrombosis

Abstract (word limit 35 words):
Venous sinus thrombosis accounts for secondary elevation of intracranial pressure which can lead to irreversible vision loss. The following report describes a case of chronic papilledema subsequently developing visual symptoms.

I. Case History
• **Patient demographics:** 59 year old BF
• **Chief complaint:** sudden postural change in vision that goes blurry and sometimes dark
  o Location: right eye
  o Onset/Duration/Timing: recent onset; episodes occur with motion and reoccurs four or more times a day
  o Associated factors: occasional dizziness; no pain/headaches
  o Relieving factors/current treatment: none
• **Ocular history:** Optic nerve edema OU x 7 years; diagnosed by an outside VA provider
• **Medical history:** Breast cancer, Essential hypertension, Hyperlipidemia, Menopause, Tobacco use
• **Medications:** Atenolol 25 mg, HCTZ 25/Triamterene 37.5 mg, Simvastatin 20 mg, Aspirin 81 mg, Calcium 600 mg/Vitamin D 200 unit
• **Other salient information:** Previously diagnosed with optic nerve elevation OU secondary to buried drusen OU, which was confirmed with a B-scan. The patient was asymptomatic, for six years, from the initial diagnosis of bilateral disc edema.

II. Pertinent findings
• **Clinical:**
  o **Best corrected vision:** OD: 20/25+1; OS: 20/25+2
  o **Color vision (Isihara plates):** OD: 14/14 OS: 14/14
  o **EOMs:** full and smooth; (-)diplopia/pain
  o **Pupils:** PERRL (-)APD
  o **Confrontation VF:** full to finger counting OD/OS
  o **Goldmann Tonometry:** OD/OS: 14/14 mmHg
  o **Anterior segment: Lens:** gr 1+ NSC OU; tr CS OU
  o **Posterior segment: C/D:** elevated discs with indistinct margins OD>OS; peripapillary disc hemorrhage OD
  o **Optical coherence tomography:** OD average RNFL thickness 166 um; OD average RNFL thickness 137 um
  o **Humphrey visual field testing 24-2:** mildly enlarged blind spot OU
• **Physical:**
  o Blood pressure: 133/83 mmHg
  o Pulse: 81 bpm
• **Laboratory studies:** normal C-reactive protein, mildly elevated Sedimentation rate

• **Radiology studies:** CT of head and orbit showed no intracranial mass or nerve drusen with 1.5 mm thin cuts; normal carotid duplex; MRI with and without contrast showed no acute intracranial or orbital abnormality. MRV showed venous sinus thrombosis, possibly chronic, located at the left transverse sinus.

• **Others:** Lumbar puncture opening pressure 260 mmH20 (Normal range in adults 80-210 mmH20, obesity can increase it up to 250 mmH20; abnormal > 250 mmH20)

III. Differential diagnosis

• **Primary/leading:** Papilledema secondary to venous sinus thrombosis OU (Secondary intracranial hypertension)

• **Others:** Idiopathic Intracranial Hypertension OU

IV. Diagnosis and discussion

• **Elaborate on the condition:** Secondary intracranial hypertension is a rare form of stroke that accounts for 0.5% of 1% of all strokes. Clinical presentation is the same as idiopathic intracranial hypertension (IIH) which includes headache, nausea, vomiting, transient visual obscuration, bilateral disc edema, pulsatile tinnitus, diplopia, and enlarged blindspot on visual field testing. However, by definition secondary intracranial hypertension has an underlying cause such as prothrombic conditions, pregnancy, oral contraceptive use, medications, ear infection, cancer, dural venous fistulas, or venous sinus thrombosis. Laboratory evaluation including complete blood cell count as well as imaging studies such as computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance venography (MRV), and lumbar puncture are crucial in establishing timely diagnosis and preventing irreversible vision loss.

• **Expound on unique features:** An elevation in opening pressure via lumbar puncture is present in about 80% of patients with cerebral venous thrombosis. Elevated protein levels is seen in 35% and cell count is elevated about 50% of the time. However, there is no specific cerebrospinal fluid abnormality.

V. Treatment, management

• **Treatment and response to treatment:**
  
  o Diamox 500 mg BID increased to 500 mg qam and 750 mg in the evening for unresolving disc edema
  
  o Warfarin 15 mg qd for venous sinus thrombosis

• **Refer to research where appropriate:**
  
  o Recommended treatment for IIH is weight loss, Diamox or Furosemide use. Rare surgical therapy includes optic nerve sheath fenestration seen with progressive visual loss or CSF shunting procedure seen with failed medical therapy or intractable headache.
  
  o In the secondary forms, treating the underlying condition, for example treating the venous thrombosis with anticoagulation/fibrinolytic therapy, or
discontinuing the causative medication, is indicated. If visual loss progresses despite optimal medical therapy (usually acetazolamide, methazolamide, or furosemide), consideration of optic nerve sheath fenestration or lumbar peritoneal shunt is warranted. A delay in diagnosis, inadequate treatment or delayed treatment could result in permanent vision loss.

**Bibliography, literature review encouraged:**


**VI. Conclusion**

- **Clinical pearls, take away points if indicated:**
  - Pseudotumor cerebri is a diagnosis of exclusion.
  - It is important to monitor patients who are on Diamox, especially with increased dosing. Once the causative condition is resolved, patients can develop intracranial hypotension.