MYSTERY CASE
Louisiana ACP 2020

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History of Present Illness

• 28 year old female with a history of alcohol abuse, paroxysmal supraventricular tachycardia, gastritis, multiple admissions for acute pancreatitis and a Mallory-Weiss tear s/p banding presented w/ **emesis, shortness of breath, and abdominal pain** for one day. Emesis was described as bilious and non-bloody w/ multiple episodes throughout the day. Abdominal pain was epigastric in nature w/ radiation to the back.
Review of Systems

- Constitutional: Chills and Fever
- Respiratory: Shortness of breath secondary to pain
- Cardiovascular: Chest Tightness
- Gastrointestinal: Abdominal pain, nausea, vomiting
- MSK: Back pain
- Endocrine: Polydipsia
Questions ???
Past History

PMHx:
• Alcohol abuse
• Paroxysmal supraventricular tachycardia
• Gastritis
• Acute pancreatitis
• Mallory-Weiss tear

Surgical Hx:
• Dilation and Curettage
• Mallory-Weiss tear: Treated with clips

Home Medications:
• Atenolol 50mg daily
Past History (con’t)

Family Hx:
• Mother: Fibromyalgia

Health Maintenance:
• Vaccines were NOT up to date
• UTD on pap smear
Past History (con’t)

Allergies:
- Penicillin

Social History:
- Former tobacco use
- Current alcohol use: 2-3 mixed drinks with vodka a week
- Previously heavy drinker: 1 handle Jack Daniels nightly
- Previous cocaine and THC use; none currently
Physical Exam - Vitals

- Blood Pressure: 111/79 mmHg
- Pulse: 126 bpm
- Temperature: 97.9°F
- Respiratory Rate: 26
- SaO2: 99% on room air
- Ht.: 5’6”
- Wt.: 65.9 kg
- BMI: 23.4 kg/m²
Physical Exam (cont’d)

• General: **Mild Distress**. Alert and Cooperative
• Eyes: PERRL, conjunctiva clear, EOMI
• Lungs: **Tachypnea**, unlabored breathing, **coarse breath sounds in bilateral lung bases**
• Chest wall: No deformity or tenderness
• Heart: RRR, S1 and S2 normal; no murmurs, rubs, gallops
• Abdomen: **Diffusely tender abdomen that was worse in the epigastric region. Hypoactive bowel sounds.**
  – No rebound tenderness, guarding, or rigidity
Thoughts from the Panel
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<th><strong>Laboratory Data</strong></th>
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- **Neutrophils**: 89% (32-64%)
- **Lymphocytes**: 3% (25-48%)
- **Monocytes**: 8% (4-6%)
Laboratory Data

- **Na⁺**: 136 (3.5-4.9)
- **K⁺**: 3.1 (3.5-4.9)
- **Cl⁻**: 91 (96-108)
- **CO₂**: 20 (21-30)
- **BUN**: 14
- **Cr**: 0.9
- **Glu**: 152 (78-105)
- **Mg**: 1.6
- **Total Protein**: 8.8 (5.9-8.4)
- **Albumin**: 4.9
- **Total bilirubin**: 1.8 (0.2-1.0)
- **Direct bilirubin**: 0.4 (0.0-0.2)
- **AST**: 351 (10-40)
- **Alk. phos.**: 133 (45-122)
- **ALT**: 179 (10-36)
- **Lipase**: 903 (16-63)
- **EtOH**: 33
- **TSH**: 1.01
Diagnostic Tests

- Urinalysis: yellow, slightly cloudy, >1000 mg/dL glucose, 30mg/dL protein, >150mg/dL Ketones, negative nitrites, negative blood, 500 uL leukocyte esterase, 6-10 WBC’s, 3-5 RBC’s, and mod squamous epithelial cells

Abdominal U/S: peripancreatic edema correlating with acute pancreatitis
(Subsequent findings: Hepatomegaly, Hepatic Steatosis)
Thoughts from the Panel
Admit Diagnosis

Acute Pancreatitis
Hospital Course – Day 1

• Admitted to the general medicine floor w/ the diagnosis of acute alcoholic pancreatitis.
• Received IVF and pain control along w/ PRN Zofran for nausea.
• Started on multivitamin supplementation w/ Thiamine.
• Restarted home Atenolol for Paroxysmal SVT.
Hospital Course – Day 1 (con’t)

- Patient’s Abdominal pain was improving
- Now able to tolerate a Full Liquid Diet
- Experienced Visual disturbances upon awakening from a nap
- Described as blurred vision w/ floaters which was Not improved with application of home Corrective lens
Thoughts from the Panel
Hospital Course – Day 2

• Following Morning Patient woke up with:
• Worsening blurry vision
• Continued visualization of Floaters
• Right eye worse than Left
• Morning Bedside Physical Exam completed with the Use of the Snellen Chart
• Exam Findings: Patient with grossly evident decrease in Visual Acuity
Hospital Course – Day 2 (con’t)

- Bedside eye exam revealed: Multiple cotton wool spots bilaterally.
- Ophthalmology Evaluation:
  - Macular Edema
  - Neurosensory detachment of macula bilaterally
  - Loss central field vision bilaterally
  - Mild Preservation of peripheral vision
  - Visual acuity 20/200 bilaterally with no improvement on pinhole
20/20  vs  20/200
Flourescein Angiogram
Advanced Fundus Photography
Optical Coherence Tomography
Management

• Graded Adjustment in Corrective Lenses with Intraocular steroid injections weekly with close follow up

• Exam at 8 weeks: Improvement to visual acuity ~20/30 bilaterally
  – Patient was initially legally blind and now able to see with corrective lens
  – Denies any worsening symptoms of retinopathy
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Final Diagnosis

Purtscher’s Retinopathy secondary to Etoh induced Acute Pancreatitis
Thoughts/Discussion
Discussion
Purtscher’s retinopathy is a rare condition first described in 1910 following intracranial trauma.

This occlusive microvasculopathy is characterized by sudden visual loss with multiple areas of retinal whitening in the posterior pole of the eye.

Retinopathy can present secondary to non-traumatic systemic diseases like acute pancreatitis.

First described this way in 1975 by Dr. Inkeles and Dr. Walsh.

The development of these visual changes is independent to the severity of pancreatitis and presents with a wide range of manifestations.

The diagnosis can be made clinically with sudden loss of visual acuity with typical fundus appearance in the context of systemic disease.
Pathophysiology

Non-Disease Specific

• Non-Traumatic Purtschers: Complement-activating systemic diseases cause a systemic cascade leading to coagulation and leukoembolization at the level of the retinal precapillary arterioles.

• Once these emboli become lodged in the arterioles they cause the clinical appearance of Purtscher’s retinopathy: **Optic nerve edema**, **Impaired visual acuity** and **visual field loss**, along with retinal findings such as cotton-wool spots, retinal hemorrhage, and Purtscher flecken.

Disease Specific

• **Pancreatitis**: Proteases (i.e. Trypsin) and Fat Emboli released during inflammation/injury activate the complement system cascade.
Pathophysiology

• Purtscher Flecken are pathognomonic for this disease
• They are variable, polygonal regions of retinal whitening with a clear zone between the affected retina and the adjacent arteriole.
• The treatment for the ocular complications of Purtscher’s retinopathy secondary to systemic disease have not been proven and prognosis depends on the areas of the retina that are affected.
• Isolated case reports have shown rapid improvement in vision with high dose IV steroids. Steroids may help preserve the neuronal membrane and suppress granulocyte aggregation caused by complement activation.
• Although rare, patient’s complaining of changes in vision following an episode of acute flares/phases of a Systemic Disease should undergo a comprehensive Eye examination

• Which begins at the BEDSIDE

• Counseling with meticulous Follow-up is a must and should be performed in order to avoid severe complications.
Thank You
References