Therapeutic Apheresis in Hypertriglyceridemia Induced Hemorrhagic Pancreatitis: Case Discussion

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History

- Age: 39, Sex: F
- BMI: 31.1
- Endo PMHx: DM2 (poorly controlled), dyslipidemia (severe HTG), obesity, HTN
- Other Hx: chronic pancreatitis, chronic pain syndrome, DVT, GERD, anxiety
History

- Surgical Hx: Distal pancreatectomy (2010, 30% pancreas removed), splenectomy, cholecystectomy, C-section
- Family Hx:
  - Paternal Grandfather: diet controlled DM2
- Medications: Fenofibrate micronized 200mg QD, Icosapent ethyl (fish oil, 1g TID), Niacin XR 500mg QD, Pravastatin 80mg QD, losartan 25mg QD, Creon TID
- DM2 Regimen:
  - Levemir 90 units BID
  - Novolog 14-16 units with meals
History: DM2

- DM2: found to have GDM at age 17 (1993), treated with lifestyle modification
- Diagnosed with T2DM in 2003, originally on oral regimen, transitioned to insulin 2009
- First seen by Endocrinology in 10/2013
  - Escalating insulin/dyslipidemia regimens without success
  - Hx suggestive of nonadherence to regimens
- HbA1c: 9.3-11.7 since seeing Endocrinology
### Lipid Panels (2011-Present)

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Range</th>
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<tbody>
<tr>
<td>HDL</td>
<td>18 - 36 (&gt;45 nml)</td>
</tr>
<tr>
<td>LDL</td>
<td>Incalculable</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>178-563</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>562 - 7255 (1000-4000 outpt)</td>
</tr>
</tbody>
</table>
Last Clinic Visit

- Complaining of hot flashes since starting niacin (recently switched to XR formulation)
- Was only taking 90 units levemir BID, ran out of novolog x 3 weeks
- Triglyceride level: 7255
- Denied abdominal pain, n/v
- Vital signs wnl
- Orlistat discussed
- Admission discussed, patient declined
One week later in ED...

- Patient went home after work with an “upset stomach” but was still able to tolerate dinner
- Woke up in the middle of the night with severe “20/10” abdominal pain
- Severe nausea/vomiting, retching every 20-30 minutes
- Stated she was adherent with medications
- +headaches, fevers, chills
- Plan: admit for acute on chronic pancreatitis
ED Visit: Data

- Na: 131 (l)
- K: 3.3 (l)
- Cl: 86 (l)
- BiC: 9 (l)
- BUN: 12
- Cr: 0.7
- Glu: 278
- Lipase: 637 (h)
- TG: 13,667 (h)
- Ca: 6.9 (l)
- Alb: 3.3 (l)
- Lact: 2.9 (h)
- pH: 7.26
- BHBA: 24.2 (h)
- WBC: 19.4 (h)
- H/H: wnl

Vitals
- BP: 130/89
- HR: 120-150
- T: 36.9
- RR: 18
- SpO2: 96% on 3L
ED Course

- ED diagnosed DKA from acute pancreatitis
- Started on insulin GTT (no bolus given) at 0.1U/kg/hr
- 2L IVF given
- Admitted to medicine service, but patient remained in ED for >10 hours
- Endocrinology consulted by IM team
Endocrinology Consult

- Patient clinically appeared worse than initial assessment by ED, IM teams
  - Patient hunched over w/ guarding, in severe distress
  - Minimally engaged in interview
  - Endorsed that this was worst episode of pancreatitis she has ever had
Based on labs (severe HTG, serum lipase >3x upper limit of normal, hypocalcemia, lactic acidosis), apheresis considered, discussed with IM attending

Recommended stat CT abdomen/pelvis, continued supportive care

Repeat labs difficult to obtain consistently 2/2 degree of lipemia
CT Abdomen/Pelvis

- Advanced changes of acute pancreatitis with new extensive peripancreatic fluid extending into the lesser sac, along the retroperitoneal fascia bilaterally, and tracking along the intrahepatic IVC. Enhancement of the pancreatic parenchyma without frank necrosis is seen but there is a small focus of decreased perfusion at the pancreatic head. No pseudocyst or organized fluid collection is present
Hospital Course

- After discussion with radiologist (possible necrotizing pancreatitis), patient was admitted to ICU for close monitoring
- Repeat labs showed worsening hypocalcemia (6.1 → 5.6)
- Decision made to pursue urgent therapeutic apheresis
- Received 2 rounds of apheresis with continued supportive care (insulin GTT, analgesia, broad spectrum abx, IVF)
# Hospital Course: Triglycerides

<table>
<thead>
<tr>
<th>Admission (Day 0)</th>
<th>Day 4</th>
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<tbody>
<tr>
<td>13,667</td>
<td>11,580</td>
</tr>
<tr>
<td>1,810</td>
<td>1102</td>
</tr>
<tr>
<td>467</td>
<td>423</td>
</tr>
<tr>
<td>395</td>
<td>348</td>
</tr>
<tr>
<td>281</td>
<td>222</td>
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</table>

- Apheresis #1
- Apheresis #2
Hospital Course

- Patient slowly improved
- Eventually started on clear liquids 5 days after admission
- MRI Abdomen:
  Imaging findings of acute on chronic pancreatitis with multiple hemorrhagic pseudocysts in pancreatic bed as described. No pancreatic tissue necrosis is noted. Pancreatic duct is not dilated.
- Surgery teams consulted who recommended conservative course
HTG Induced Pancreatitis

- Serum TG >1000 increases risk of AP [1]
- 5% risk w/ TG >1000 [2]
- 10-20% risk >2000 [2]
- Mechanism: breakdown of TG into toxic FFA by pancreatic lipases induces AP [3]
- Primary HTG: genetic disorders
  - Familial Chylomicronemia (type 1 HLD, LPL def.)
- Secondary HTG:
  - DM1/2 (DKA can trigger HTGP) [4]
  - Pregnancy (56% of AP in pregnancy, 1/6790 pregnancies) [5]
Apheresis Indications

- Therapeutic Plasma Exchange is modality of choice (removal of plasma and replacement with colloid solution) [6]
- Labs [7]
  - Severe HTG (>1000)
  - Lipase >3x upper limit of normal
  - Hypocalcemia
  - Lactic acidosis
  - worsening organ dysfunction (renal, hepatic function)
- Mainly case reports, series
References


