A 31-year-old healthy female presented four weeks post cesarean delivery with weakness and shortness of breath, after being found hypotensive and tachycardic at a routine visit.

Vitals: Temp 37.0 C, HR 148 /min, RR 22 /min, BP: 80/50, O2 Sat: 97% on RA

Physical exam was significant for mottled bilateral lower extremities, consistent with livedo reticularis

Diagnostic tests: DIC, APS, aHUS, TTP

Table 1. Biomarker changes in various TMAs. DIC disseminated intravascular coagulation, APS antiphospholipid syndrome, aHUS atypical hemolytic uremic syndrome, TTP thrombotic thrombocytopenic purpura, aPTT activated partial thromboplastin time

Hb/g/L 7.9 ± 2.6 % ↓ ↓
INR 1.3 
Platelet count 17 K/uL ↓
Creatinine 2.5 mg/dL ↑

All normal during delivery 1 month ago

Case Presentation

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Diagnostic tests:

<table>
<thead>
<tr>
<th>Primary cause and target of coagulopathy</th>
<th>Platelet count</th>
<th>D-Dimer</th>
<th>PT / aPTT Fibrinogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>DIC</td>
<td>Macrophage, endothelial cell</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>APS</td>
<td>Antiphospholipid antibody</td>
<td>↓</td>
<td>PT →</td>
</tr>
<tr>
<td>aHUS</td>
<td>Complement system</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>TTP</td>
<td>ADAMTS 13</td>
<td>↓</td>
<td>↓</td>
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</table>

On the day of admission, tightly regulated anticoagulation and plasmapheresis were started with suspected diagnosis of TTP and atypical HUS.

After forty-eight hours of plasma exchange, she showed rapid improvement.

Workup for autoimmune hemolytic anemia, systemic lupus erythematosus, catastrophic antiphospholipid syndrome, disseminated intravascular coagulation, typical HUS, heparin-induced thrombocytopenia, drug-induced TMA, and malignancy returned negative.

Blood cultures were positive for Group B streptococcus (prior to delivery GBS testing negative) and TEE showed a large mobile tricuspid vegetation without PFO; antibiotics were initiated. Once stabilized, the patient was referred for percutaneous vegetation debulking and encouraged to pursue genetic testing for complement protein mutations.

Hospital Course

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Conclusion

This is an unusual presentation of C-TMA provoked by SBE. Had this patient had hereditary C-TMA, her course would have been refractory to plasma exchange and eculizumab would have been initiated.

GBS endocarditis of the tricuspid valve, particularly in patients without a history of IVDA or other predisposing risk factors is a rare entity. Only 24 different cases have been reported thus far in the literature, of which 9 are pregnancy-related and only 2 post c-section cases.

Early recognition and rapid communication with an interdisciplinary multispecialty team resulted in immediate intervention with plasmapheresis as rescue therapy until the provoking endocarditis was treated.

References