Aplastic anemia secondary to SARS-Co-V-2

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Background

- Aplastic anemia with incidence of an estimated two per million in Western countries.
- Rare hematological disease characterized by pancytopenia and a profoundly hypocellular bone marrow.
- Exact pathophysiology unknown but activation of cytotoxic T-cells which inappropriately target antigens on hematopoietic stem cells thought to play a role.
- Known to be induced by viral infections in particular HIV and the hepatitis viruses.
- Second known adult case of aplastic anemia following infection with the SARS-Co-V-2 virus.

Clinical Case

- 40-year-old Hispanic female with BMI 32, pre-diabetes and history of papillary thyroid cancer s/p left hemithyroidectomy.
- Presented with lower limb petechiae 10 days following resolution of symptoms from confirmed SARS-Co-V-2 infection.
- Initial laboratory workup revealed pancytopenia.
- ITP first suspected and treatment with dexamethasone and IVIG failed to produce an improvement in the pancytopenia.
- Subsequent bone marrow was performed, confirming diagnosis of severe aplastic anemia.

Workup/Results

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>10.3 g/dL</td>
</tr>
<tr>
<td>Platelets</td>
<td>5 x 10^9/L</td>
</tr>
<tr>
<td>WCC</td>
<td>2 x 10^9/L</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>0.7 x 10^9/L</td>
</tr>
<tr>
<td>Reticulocyte count</td>
<td>2.7 x 10^9/L</td>
</tr>
<tr>
<td>Reticulocyte index</td>
<td>0.03</td>
</tr>
<tr>
<td>Coagulation studies</td>
<td>Normal</td>
</tr>
<tr>
<td>HIV ½ Ab and P24 Ag</td>
<td>Non-reactive</td>
</tr>
<tr>
<td>Hepatitis B Surface Ag</td>
<td>Non-reactive</td>
</tr>
<tr>
<td>Hepatitis C Ab</td>
<td>Non-reactive</td>
</tr>
</tbody>
</table>

Peripheral smear: Pancytopenia with morphologically normal platelets, leukocytes, and red blood cells. No atypical lymphoid cells, overtly dysplastic granulocytes, immature monocytes or blasts are seen.

Flow cytometry on BM aspirate: phenotypically normal T-cells, not immunophenotypically in keeping with non-Hodgkin lymphoma.

Immunostaining negative for parvovirus-19

Flow cytometry negative for paroxysmal nocturnal hemoglobinuria (PNH).

Treatment

Failed to respond to immunosuppressive therapy remaining pancytopenic with multiple admission with febrile neutropenia.

Suffered severe hyperbilirubinemia secondary to cyclosporine, requiring dose reduction.

Thrombopoietin agonist, eltrombopag started at 150mg daily.

Eventually had response with HLA matched platelet transfusion.

Most recently had Hgb 7.5g/dL, platelets of 14 x10^9/L, WCC 2.6 x 10^9/L, neutrophils of 1.5 x 10^9/L

Progress

Patient age precluded her from allogenic stem cell transplant as first line treatment.

Started on immunosuppressive therapy with anti-thymocyte globulin (ATG), cyclosporine (CSA), and prednisone.

Initial therapy with the thrombopoietin agonist, eltrombopag, was not possible due to lack of insurance and immigration status.

Conclusion

- Adds to the growing literature of the side effects of SARS-Co-V-2 infection.
- Illustrates that the bone marrow can also be targeted as a result of the cytokine storms associated with the morbidity and mortality of SARS-Co-V-2.
- Adds to our understanding of the array of viruses that can be implicated in the development of aplastic anemia.
- Highlights the complexities of providing evidenced based care for patients without health insurance and legal documentation during a global pandemic, which further widens the inequities in outcomes following infection with this novel virus.

References

