Immune Thrombocytopenic Purpura Flare as a Presenting Sign of COVID-19 Infection

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Background

- Immune Thrombocytopenic Purpura (ITP) is an immune condition characterized by a decrease in the number of platelets in the blood.
- Thrombocytopenia has been associated with a threefold enhanced risk of severe respiratory disease in COVID-19 infection.
- The mechanism of thrombocytopenia in patients with COVID-19 infection is likely multifactorial.
- It is possible that the novel Coronavirus causes an auto-immune response against hematopoietic stem cells, megakaryocytes, and platelets inducing their growth inhibition and apoptosis.
- Platelet consumption in damaged lungs is a suggested alternative mechanism.
- We present a case of a patient with pre-existing ITP in whom acute thrombocytopenia was the only sign or symptom of infection for the first two weeks of known infection.

Case Presentation

88-year-old Italian male with past medical history significant for ITP, dementia (alert only to self), type 2 diabetes, chronic kidney disease and class III obesity was found to have severe thrombocytopenia. Routine lab work obtained at the patient’s skilled nursing facility revealed a platelet count of 5,000. COVID-19 was tested on admission given hospital policy to test all nursing home patients during the pandemic.

Initial Physical Exam

Vitals: T 36.9 C, BP 103/71, HR 87, RR 18, O2 sat 97% on room air
General: Awake and alert to self, no acute distress
Cardiac: S1/S2, regular, no murmurs
Respiratory: Clear to auscultation bilaterally, symmetrical chest expansion
Abdomen: Nondistended abdomen, nontender to light or deep palpation
Skin: Purpura on chest and bilateral upper and lower extremities

Case Presentation

- The patient received a total of 2 doses of IVIG during hospitalization. His home eltrombopag (bone marrow stimulant) medication was increased to 50 mg daily.
- During the course of hospitalization the patient did not have any cough, fevers, loss of taste or smell, or respiratory symptoms of COVID-19.
- His platelet count increased to 41,000, and he was discharged safely on prednisone and eltrombopag to his skilled nursing facility.
- Two weeks following discharge, the patient returned with epistaxis. He had an admission review event leading to acute hypoxic respiratory failure.
- During that admission, the patient’s health-care representative decided to make the patient “comfort measures only.” The patient passed away.

Discussion/Conclusion

- There is still much to learn about COVID-19, including its effect on individuals with underlying immune conditions.
- This case report suggests that COVID-19 was a trigger for a flare in ITP.
- Patients with underlying ITP, found to be COVID-19 positive, have been reported to have more severe respiratory illness.
- Low platelet count has been noted to be a biomarker associated with disease severity and risk of mortality.
- In this case, our patient had severe thrombocytopenia, but did not have typical symptoms associated with COVID-19. This brings into question findings from previous meta-analysis which postulate thrombocytopenia as a biomarker of disease severity.
- Additionally, in this patient with underlying ITP, the proposed mechanism of platelet consumption in damaged lungs does not appear to apply.

Possible Mechanisms of Thrombocytopenia

- Directly attacks cells via ACE2
- Inducing auto-antibodies and immune complexes
- Damage of lung
- Hematopoietic stem/progenitor cells
- Activation, aggregation and encapsulation of platelets
- Reduce the effective capillary bed
- Apoptosis/growth inhibition
- Increased platelet consumption
- Dysfunction of megakaryopoiesis

References

- Mo Yang, Margaret HL Ng & Chi Kong Li (2005) Thrombocytopenia in patients with severe acute respiratory syndrome (review), Hematology, 10:2, 101-105, DOI: 10.1080/10245330400265170
- Immune Thrombocytopenic Purpura in a Patient with Covid-19. April, 2020 DOI: 10.1056/NEJMc2010472