Background

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterized by low platelet count (<100 x 10^9/L) due to increased platelet destruction and decreased platelet production. ITP can be a primary condition or develop as secondary symptoms. The principal mechanisms of ITP involve autoantibodies to platelet glycoproteins, which activate complement and result in the destruction of platelets. Platelet destruction can also be mediated by complement activation and platelet desialylation.

Objectives

We hypothesize that desialylated platelets show increased binding of ITP antibodies. Increased ITP platelet binding contributes to complement activation.

Methods

Complement activation

Whole blood samples were incubated with control IgG or IgG antibodies from the blood of 5 patients for 45 minutes at 37°C. Samples were centrifuged, and the collected supernatant was either frozen at -80°C or used immediately. Complement activation was then measured using a C5a ELISA.

Results

C5a ELISA showed no difference in complement activation between samples incubated with IgG and IgG antibodies.

References


Acknowledgements

I would like to thank Dr. Arepalli and her lab members, the Duke OHSU-PSU Program, and LMU-UCOM.