Successful Treatment of Thrombocytopenia with Staphylococcal Protein A (PRTX-100) in a Murine Model of Immune Thrombocytopenia (ITP)

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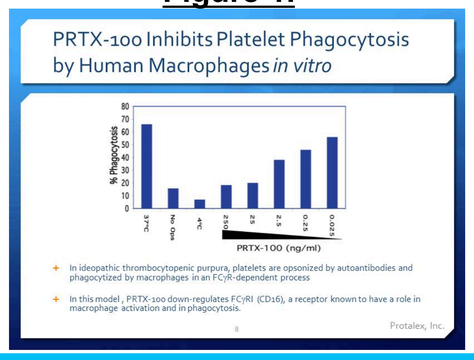
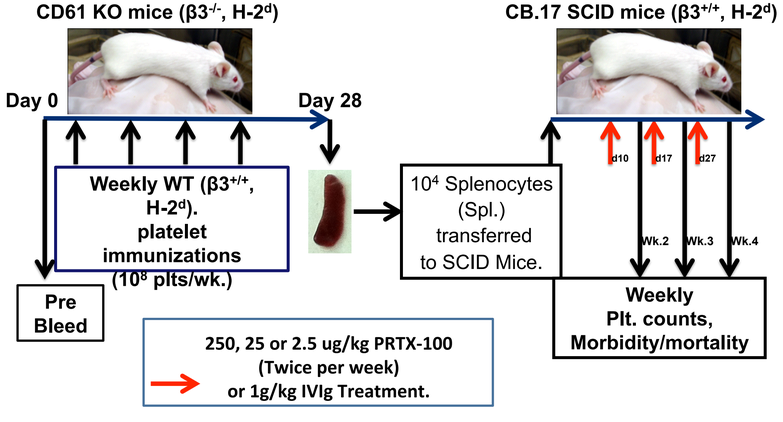
Introduction

Immune thrombocytopenia (ITP) is an autoimmune bleeding disorder in which autoantibodies and autoreactive T cells target the destruction of platelets and megakaryocytes in the spleen and bone marrow. Several therapeutic options e.g. steroids, IVIg, Rituximab and thrombopoietin mimetics, are available for patients but inadequate efficacy, side effects and/or expense can make them undesirable. PRTX-100 is a highly purified formulation of Staphylococcal Protein A that has been evaluated in clinical trials with rheumatoid arthritis patients. Previous work has shown that PRTX-100 can efficiently inhibit FcR-mediated phagocytosis of opsonized platelets in vitro (Figure 1). To determine if PRTX-100 also has the ability to increase platelet counts in vivo, we analyzed its efficacy in a well-established murine model of ITP (1). Our results show that compared with control thrombocytopenic SCID mice, mice treated with either PRTX-100 or IVIg had platelet counts that increased to within normal levels within 1-2 weeks after treatment. These results demonstrate that PRTX-100 was effective in elevating platelet counts in a murine model of human ITP and support the proof of principle that PRTX-100 may be beneficial in patients with ITP.

Figure 1.



Experimental Design



Results

Figure 2. The effect of PRTX-100 on platelet counts In ITP mice.

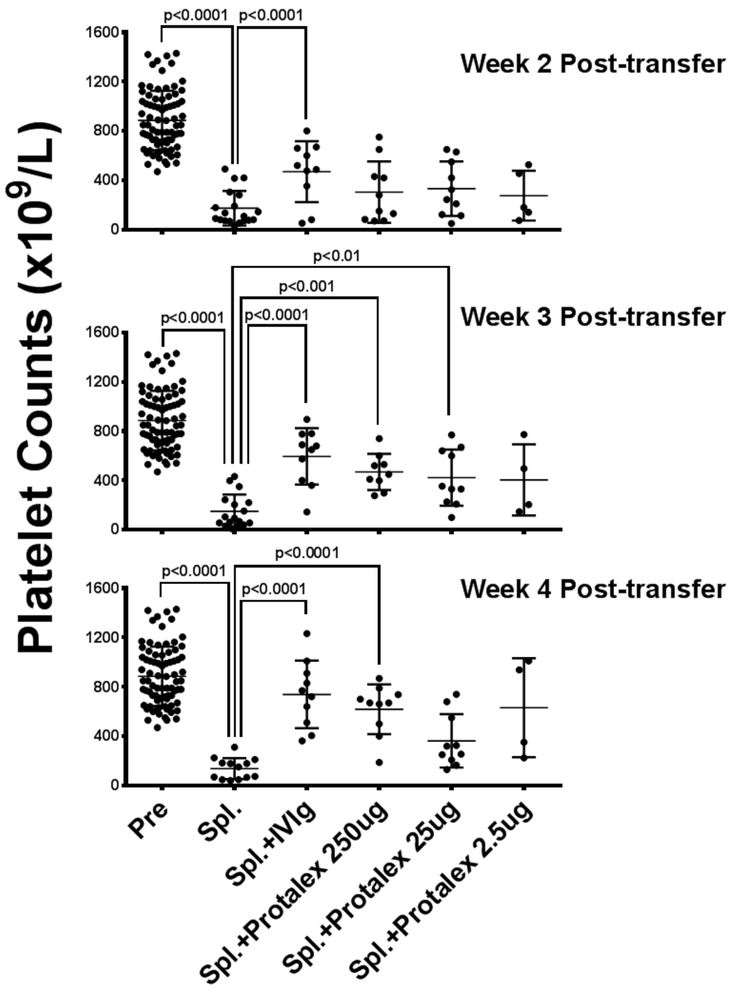
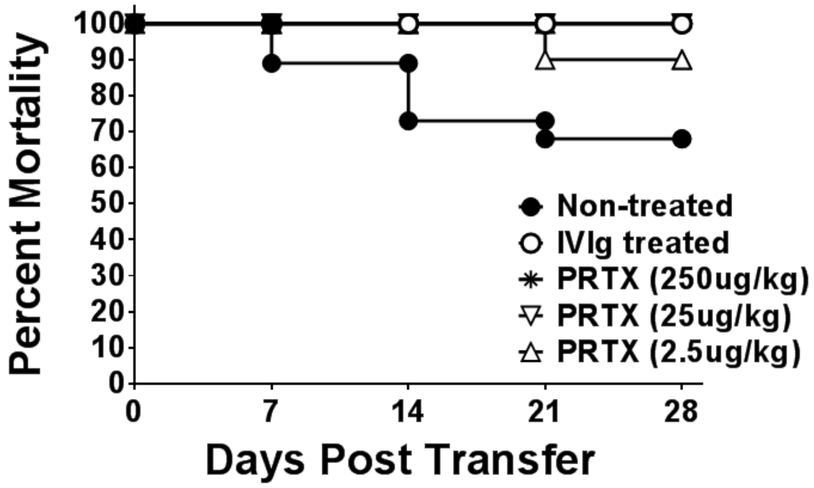


Figure 2. SCID mice were transferred with splenocytes from immune CD61 KO mice and platelet counts were measured weekly. Results are expressed as individual platelet counts in the indicated groups of mice. Mean +SD bars are shown. Statistical comparisons of interest are shown.

Figure 3. The effect of PRTX-100 on mortality in ITP mice.



Conclusions

PRTX-100 treatment of ITP mice significantly increased platelet counts and prevented mortality in a dose dependent manner. The treatment caused no side effects in the mice.

References

1. Chow L, et al. Blood. 2010;115:1247-53.

Acknowledgements

Supported by a grant from Protalex Inc.

Disclosures:
There are no relevant conflicts of interest to disclose.