Research Article

Transplanted Human Umbilical Cord Mesenchymal Stem Cells Facilitate Lesion Repair in B6.Fas Mice

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Background. Systemic lupus erythematosus (SLE) is a multisystem disease that is characterized by the appearance of serum autoantibodies. No effective treatment for SLE currently exists. Methods. We used human umbilical cord mesenchymal stem cell (H-UC-MSC) transplantation to treat B6.Fas mice. Results. After four rounds of cell transplantation, we observed a statistically significant decrease in the levels of mouse anti-nuclear, anti-histone, and anti-double-stranded DNA antibodies in transplanted mice compared with controls. The percentage of CD4+CD25+Foxp3+ T cells in mouse peripheral blood significantly increased after H-UC-MSC transplantation. Conclusions. The results showed that H-UC-MSCs could repair lesions in B6.Fas mice such that all of the relevant disease indicators in B6.Fas mice were restored to the levels observed in normal C57BL/6 mice.

1. Introduction

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease. SLE was generally fatal before the advent of immunosuppressive medications. However, despite these advances in immunosuppressive medical therapies, SLE remains potentially fatal in some patients, particularly in those patients with treatment-refractory disease.

SLE is characterized by antibodies associated with injuries to multiple organs of the renal, cardiovascular, neural, musculoskeletal, and cutaneous systems [1]. The pathology of SLE involves the destruction of targeted organ tissues and the accumulation of autoreactive lymphocytes and immune complexes. Although disease severity and organ involvement vary significantly among SLE patients, T and B lymphocyte abnormalities are universal [2–3]. Moreover, SLE manifests multifaceted immune modulation that induces both deficiency and hyperactivity of the immune system. A deeper understanding of the underlying pathology is crucial for developing optimal therapies to restore immune homeostasis without compromising the protective immune response to pathogens [4].

In addition to conventional medical therapies, such as cyclophosphamide (CTX) and mycophenolate mofetil, several new strategies have been developed that target specific pathways relevant to SLE pathogenesis [1, 5]. For example, B cell depletion therapies using the monoclonal antibodies rituximab and ocrelizumab have benefited a specific subpopulation of SLE patients [6]. Recently, hematopoietic stem cell transplantation has been reported to improve disease severity in treatment-refractory SLE patients [7] and to reverse organ dysfunction in several animal models [8]. Despite improved supportive care, aggressive immunosuppressive medical therapies, and new therapeutic interventions, certain SLE patients continue to suffer significant morbidity and mortality resulting from active disease with visceral organ involvement. Therefore, the development of more effective therapies for SLE, particularly for treatment-refractory patients, is urgently needed.