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Chronic Graft-Versus-Host Disease of the Kidney after Allogeneic Hematopoietic Stem Cell Transplantation

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Abstract

Chronic graft-versus-host disease of the kidney is rare. Its usual symptom is nephrotic syndrome but the pathogenesis and optimal therapy are unclear. Steroids and calcineurin inhibitors are the most effective drugs. And monitoring proteinuria in these patients may is necessary.

Keywords: Chronic graft-versus-host disease; Kidney; Allogeneic hematopoietic stem cell transplantation

Introduction

Rapid advances in allogeneic hematopoietic stem cell transplantation (allo-HSCT) have increased the possibility of successful treatment in patients with several hematological diseases [1,2]. Chronic graft-versus-host disease (cGVHD) is one of the most frequent and serious complications following allo-HSCT [3]. Since cGVHD of the kidney is rare and there are very few described cases, the pathogenesis and optimal therapy are unclear [4]. Here we reviewed the previous reports and discussed the clinical and biological characteristics of kidney involvement secondary to cGVHD.

The cGVHD of kidney is a rare, but severe, complication after allo-HSCT. Its incidence is 1.03% [5]. It occurs in synchronization with manifestation of cGVHD after the cessation of immune suppressive therapy and usually manifests itself as a nephrotic syndrome (NS). The most common pathology of NS secondary to cGVHD is membranous nephropathy (MN), followed by minimal change disease (MCD) [6]. They may manifest as slowly increasing creatinine, often accompanied by proteinuria, anemia and hypertension.

The mechanism underlying the development of cGVHD of kidney after allo-HSCT remains unclear. GVHD is a T-cell–mediated disease, in which donor T lymphocytes recognize disparate minor or major histo compatibility complex (MHC) antigens in the host [7]. And donor B-cell antibodies augment cutaneous cGVHD in part by damaging the thymus and increasing tissue infiltration of pathogenic T helper 17 cell (Th17) cells [8]. Murine models have demonstrated the roles of nephrin and its autoantibody in the development of glomerular lesion [9].

Patients who develop cGVHD of the kidney after HSCT are at an increased risk of morbidity and mortality [10]. Steroids and calcineurin inhibitors are the most effective drugs. They can reduce the synthesis of cytokines and induce a remission of proteinuria [11]. And other immunosuppressant drugs, such as cyclophosphamide, and rituximab are commonly used [12,13]. Besides, monitoring renal function through proteinuria, serum blood urea nitrogen and serum creatinine levels are necessary [5].

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