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Could donor multipotent mesenchymal stromal cells prevent or delay the onset of diabetic retinopathy?

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Abstract

Diabetes mellitus is a complex metabolic disease that has become a global epidemic with more than 285 million cases worldwide. Major medical advances over the past decades have substantially improved its management, extending patients' survival. The latter is accompanied by an increased risk of developing chronic macro- and microvascular complications. Amongst them, diabetic retinopathy (DR) is the most common and frightening. Furthermore, during the past two decades, it has become the leading cause of visual loss. Irrespective of the type of diabetes, DR follows a well-known clinical and temporal course characterized by pericytes and neuronal cell loss, formation of acellular-occluded capillaries, occasional microaneurysms, increased leucostasis and thickening of the vascular basement membrane. These alterations progressively affect the integrity of retinal microvessels, leading to the breakdown of the blood-retinal barrier, widespread haemorrhage and neovascularization. Finally, tractional retinal detachment occurs leading to blindness. Nowadays, there is growing evidence that local inflammation and oxidative stress play pivotal roles in the pathogenesis of DR. Both processes have been associated with pericytes and neuronal degeneration observed early during DR progression. They may also be linked to sustained retinal vasculature damage that results in abnormal neovascularization. Currently, DR therapeutic options depend on highly invasive surgical procedures performed only at advanced stages of the disease, and which have proved to be ineffective to restore visual acuity. Therefore, the availability of less invasive and more effective strategies aimed to prevent or delay the onset of DR is highly desirable. Multipotent mesenchymal stromal cells, also referred to as mesenchymal stem cells (MSCs), are promising healing agents as they contribute to tissue regeneration by pleiotropic mechanisms, with no evidence of significant adverse events. Here, we revise the pathophysiology of DR to identify therapeutic targets for donor MSCs. Also, we discuss whether an MSC-based therapy could prevent or delay the onset of DR.