Chapter 5
Stem Cell Therapy for Avascular Femoral Head Necrosis: From Preclinical to Clinical Study

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Abbreviations

ADSCs	Adipose-derived stem cells
AVNFH	Avascular necrosis of the femoral head
BM	Bone marrow
BMMNCs	Bone marrow mononuclear cells
BMMSCs	Bone marrow mesenchymal stem cells
BMP	Bone morphogenetic protein
cBMMNC	Cryopreserved bone marrow mononuclear cells
CT	Computed tomography
EPCs	Endothelial progenitor cells
HGF	Hepatocyte growth factor
hIDPSC	Human immature dental pulp stem cells
HIF-1α	Hypoxia-inducible factor-1α
MRI	Magnetic resonance imaging
MSCs	Mesenchymal stem cells
PBSC	Peripheral blood stem cells
SCPP	Strontium-doped calcium polyphosphate
TCP	Tricalcium phosphate
THA	Total hip arthroplasty
UC-MSCs	Umbilical cord mesenchymal stem cells
VAS	Visual analogue scale
VEGF	Vascular endothelial growth factor

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5.1 Introduction

Avascular necrosis of the femoral head (AVNFH) is a progressive disease related to blood vessels of the femoral head. AVNFH is the result of critical ischemia of the femoral head because of blood vessel injuries. About 20 % of total hip replacements are related to AVNFH. To date, there are no standard treatments for AVNFH. Therefore, almost all AVNFH patients undergo total hip replacement. Stem cell therapy is considered to be a new therapeutic strategy for AVNFH. Initial studies showed that stem cell therapy is a promising approach for AVNFH treatment with significant improvement in hip functions as well as blood supply to necrotic tissues. This chapter focuses on certain sources of stem cells used to treat AVNFH in both animals and humans. In the final section, mechanisms of stem cells in AVNFH treatment are also explored.

As mentioned above, AVNFH is the result of a lack of blood supply to the trabecular bone in the femoral head. This condition causes local ischemia at the femoral head in which cells undergo apoptosis and necrosis, leading to articular cartilage collapse and subsequent osteoarthritis. However, the pathogenesis of AVNFH is very complicated, and there are various risk factors for AVNFH, such as trauma, hormones, intemperance, and connective tissue diseases (Table 5.1). AVNFH tends to affect patients aged 20–40 years with the average age at presentation being 38 years (Lavernia et al. 1999; Mont and Hungerford 1995).

In terms of cellular biology, AVNFH is related to the loss of equilibrium between osteoblast and osteoclast formation. The osteoclast formation process is stronger than osteoblast formation. Therefore, a part of bone can be destroyed, leading to damage at the femoral head. This disequilibrium is related to a reduction in the

Table 5.1 Risk factors for AVNFH

Traumatic/direct injury	Nontraumatic
Femoral neck/head fracture	Corticosteroid use
Hip dislocation	Alcohol abuse
Slipped capital femoral epiphysis	Idiopathic
	Sickle cell disease
	Caisson disease
	Systemic lupus erythematosus
	Cushing's disease
	Organ transplantation
	Prior radiation therapy
	Smoking
	Pregnancy
	Chronic pancreatitis
	Coagulopathy
	Chronic renal failure
	Lipid disorders