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Editorial

Repairing, reconstructing, and recovering after COVID-19

As the global socio-economic recovery from COVID-19 continues, surgical repair and reconstruction are urgent needs for people suffering from musculoskeletal disorders. Over the past few years, we have reported on the impact of the pandemic crisis on orthopaedic practice and the treatment of complications associated with COVID-19 [1–3]. In this issue, Tsui KHM et al. conducted a cohort study on COVID-19 hip fracture patients in the Chinese population in Hong Kong [4]. The study found that patients with both COVID-19 and hip fractures are at high risk of mortality and complications. Therefore, early operative treatment is recommended as soon as possible. However, Ct values and D-dimer levels have no prognostic roles for hip fracture outcomes.

Repairing large bone defects is a major challenge in orthopaedic clinics. Critical-sized defects are defined as those that cannot heal without surgical intervention, and the growth of fibrous tissue usually hinders the bone healing process. Gu et al. conducted a study on a rabbit radius critical-sized bone defect model to test the effect of silicone rubber (SR) sealed channels in inducing bone repair in vivo [5]. The results showed that the SR sealed channel could prevent fibrous tissue from entering the fracture end, thereby promoting self-healing of long tubular bone through endochondral bone formation. It was believed that the hematoma tissue formed in the early stages of healing contained osteogenesis and angiogenesis-related proteins, which contributed to the vascularization and endochondral osteogenesis in bone defects. These findings indicate that the self-healing potential of bone needs to be protected and mobilized during the repair process of bone defects.

Reconstruction of tissue function can be achieved by cell-based tissue engineering strategies. In this issue, Donderwinkel et al. prepared a hydrogel made of poly(ethylene glycol)/gelatin to encapsulate human bone marrow-derived MSCs (hBMSCs). They then investigated the impact of static and intermittent cyclic uniaxial strain mechanical stimulation, in combination with transforming growth factor-β3 (TGF-β3) supplements, on the tenogenic differentiation of hBMSCs [6]. The findings showed that TGF-β3 promoted the expression of key tenogenic genes, and intermittent cyclic uniaxial strain aided matrix deposition in the engineered tissue. These results demonstrated the significance of growth factors and mechanical stimulation in an improved protocol for tendon tissue engineering. In another study led by Huang et al. it was found that melatonin, a regulator molecule for circadian rhythm, can significantly reduce the apoptosis of nucleus pulposus mesenchymal stem cells by activating the PI3K/Akt pathway in vitro. Further, in vivo experiments demonstrated that melatonin administration partially reduced the degree of intervertebral disc degeneration, as shown by the X-ray, MRI, and histological analyses [7]. These results provide a promising strategy to target endogenous stem cells for alleviating intervertebral disc degeneration.

There are two review papers in this issue. One paper, authored by Sheng et al. focuses on the muscle-bone crosstalk and summarizes the role of myokines and osteokines in the development osteo-sarcopenia. The authors suggest that myostatin, irisin, RANKL, and SOST may be potential targets for treating fracture patients with osteo-sarcopenia [8]. This topic has been previously discussed in several publications in JOT, where the role of mitochondrial function in the pathogenesis of sarcopenia has been emphasized [9,10]. Huang et al. presented an overview in another paper on different animal models for osteomyelitis, which included rodent, rabbit, avian/chicken, porcine, minipig, canine, sheep, and goat models. The corresponding clinical scenarios for each animal model were elaborated, and the importance of choosing the right animal model to facilitate the development of novel management strategies for osteomyelitis was highlighted [11].

Functional recovery of patients with musculoskeletal disorders is dependent on the efficient repair and reconstruction of damaged tissue structure. The new findings, reported in this issue, represent the ongoing efforts of the orthopaedic scientific community to improve the quality of life after COVID-19. As we approach the year 2024, I would like to take this opportunity to wish all authors and readers a very prosperous New Year!

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