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Mini Review

Histopathology of Intraarticular Loose Body in View of the Joint-Related Vasculature

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- Articular cartilage

Abstract

Intraarticular loose bodies (ILBs) are not uncommon orthopedic disease. ILBs are caused by various osteoarthritic or joint diseases, some of which are associated with the joint-related vasculature. However, non-specific additional changes can occur in ILBs associated with or without the vasculature. The former represents revascularized enchondral ossification due to re-attachment of ILBs to the synovium, and the latter includes gullying of cartilaginous matrix, secondary synovial chondromatosis, and fatty metaplasia. Histopathological examination of ILBs in considering such relationship with the vasculature can be useful for assessment of the source and/or etiology of ILBs.

ABBREVIATIONS

ILBs: Intraarticular Loose Bodies

INTRODUCTION

Intraarticular loose bodies are not uncommon disease [1-4]. In this minireview, we herein describe histopathology of ILBs related or unrelated to the vasculature.

TEXT

Intraarticular loose bodies (ILBs) are relatively common orthopedic conditions frequently involving knee joint and sometimes producing pain, locking, and joint effusion [1-4]. ILBs are caused by traumatic osteochondral fracture, osteochondritis dissecans, primary osteonecrosis, osteoarthrosis, torn meniscus, fibrinous synovitis, osteoarthrosis, and primary synovial chondromatosis [1-7]. Some of these etiologies are related to the vasculature of bone and joint and its nutritious condition [1-7]. Bone and bone marrow tissues receive blood supply [2,4,8]. Therefore, vascular insufficiency due to fractures or corticosteroid administration induces primary osteonecrosis or avascular necrosis [2,8]. Main inner or central part of meniscus is mostly avascular whereas the peripheral or outer part only attached to the joint capsule contains vessels [1]. The former avascular part is vulnerable to trauma and poorly repaired, and can produce ILBs [1,2]. Synovium is rich in vessels, and ILBs re-attached to the synovium can be re-vascularized [1,2,6,7].

ILBs are histologically classified into fibrinous bodies, fibrocartilaginous bodies, and osteochondral or cartilaginous bodies [1,2]. Fibrinous ILBs and fibrocartilaginous ILBs are chiefly associated with fibrinous exudates of synovitis and torn menisci, respectively [1,2]. Lobulated or clustered growth of hyaline cartilage in ILBs indicates a relationship with primary synovial chondromatosis [1-5]. Homogenous hyaline cartilage in osteocartilaginous ILBs suggest that these ILBs are originated from detached articular cartilage and subchondral bone, rather than ossified synovial chondromatosis [2-4]. The presence of tidemark within these osteochondral ILBs also supports this etiology [2,4,6]. Central situated necrotic bone and/or bone marrow within osteochondral loose bodies raise two possibilities: 1) detached fragments derived from primary osteonecrosis; and 2) detachment of previously dead bone and/or bone marrows [2,3,6,7]. In addition, proliferative, metaplastic, and degenerative changes can occur in long-standing loose bodies, leading to difficulty in assessing the source of loose bodies [2,4,6,7,9,10]. Particularly, articular hyaline cartilages, unlike bone and bone marrow tissues, are directly nourished by joint fluid, and detached articular cartilaginous components can survive, progressively develop, and modify the initial histology of ILBs [2-4,6,7,9,10]. Such additional changes in ILBs include gullying cartilaginous matrix [10], spindle cell changes and fibrocartilaginous metaplasia of detached hyaline chondrocytes [5,10], and concentric laminar arrangement of osteocartilaginous tissue, also called secondary synovial chondromatosis [2-6]. Osteocytes in such secondary synovial chondromatosis are alive [2,3], unlike detached preexisting bones, and would be vessel-independent metaplasia from detached hyaline cartilage.

Matsukuma et al. [7] identified unique vessel-independent fatty metaplasia of detached articular cartilaginous cells in 19% of true free ILBs (ILBs without re-attached to the synovium). Such fatty metaplasia in all cases contained lipomembranous fat necrosis, suggesting the presence of hypoxic or mal-nutritious conditions of intraarticular spaces [7]. Moreover, enchondral ossification is occasionally found in surgically removed ILBs of any etiology [2-4,6,7]. This phenomenon is considered to be re-vascularized condition due to re-attachment to the synovium, not true free ILBs, because enchondral ossification requires the vasculature [2-4,6,7,10]. Indeed, in such ILBs, viable small vessels containing erythrocytes are present [6]. This re-vascularization also induces absorption of detached necrotic bones [2,3].

CONCLUSION

Some of the cause and subsequent phenomena of intraarticular loose bodies are closely related to the vasculatures. Histopathological examination in considering such relationship with the vasculatures can be useful for assessment of source and/or etiology of intraarticular loose bodies.

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