

ERRATUM

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The complete abstract is given below.

The publisher and organizers sincerely apologize for the errors and regret the inconvenience they may have caused.

EL1-2

Basic knowledge of joint structures and disease types with joint destruction

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Type of joint is mainly classified as "diarthrodial" and "synarthrodial". Diarthrodial joint is also called as "synovial joint". The structure is composed of articular surface of hyaline cartilage located both end of adjacent bones, which is covered with capsular tissues, often with ligamentous structure. Inner layer of capsular tissues is outlined by synovial connective tissue. Thus, the structure has joint cavity, in which synovial fluid is present. Many joints in extremities belong to diarthrodial type (synovial joint). Diarthrodial joints can be classified into 'plane', 'hinge', 'ball and socket', 'ellipsoidal', 'condyloid', 'pivot' and 'saddle'. Synarthrodial joints lack joint capsule, joint cavity and synovial membrane. Synarthrodial joints connect adjacent bones by the connective tissues of symphysis, synchondrosis, syndesmosis and/or synostosis. The mobility of the joint is permitted by firm ligamentous structure, but limited when compared to diarthrodial joint, or absent. Intervertebral disks of vertebral bodies and pubic symphysis are included in this category.

Mode of joint destruction is depending on type of diseases and characteristics of each joint. Rheumatoid arthritis (RA) is one of the representative autoimmune-based inflammatory disorders accompanying joint destruction. Uncontrolled disease activity of RA induces cartilage destruction and bone erosion. Osteoporosis and cystic changes of periarticular bones are observed. Contracture and laxity, as well as musculo-tendinous malalignments, accelerate joint instability and deformity. These pathologic conditions deeply contribute to progress of joint destruction. Ankylosis of affected joints is also seen. Symmetrical and peripheral arthritis is one of important features of RA, but not only hand and foot, also other all the synovial joints, such as elbow, shoulder, hip, knee, and spine, are possibly destroyed with some specific feature and manner depending on each joint characteristics. Juvenile idiopathic arthritis, adult onset Still's disease, psoriatic arthritis and reactive arthritis also show autoimmune-based inflammatory synovitis, thus leading to joint destruction. Occasional minor joint destruction is seen in connective tissue disorder caused by progressive systemic sclerosis and systemic lupus erythematosus. Septic arthritis is troublesome, because rapid and progressive joint destruction is induced when local host response to the microorganism is not controlled well.

Osteoarthritis (OA) is one of most common joint diseases with joint destruction, although the etiology of OA is broad and various. Diffuse degeneration and wearing of hyaline articular cartilage cause joint space narrowing, which is one of the characteristics of OA. However, mechanical loading and stress distribution, combined with each specific morphologic feature, often seen in such as knee and hip joints, can induce localized degeneration and wearing

of articular cartilage in the process of OA change. Along with progression of OA, sclerosis of subchondral bone and cyst formation, as well as osteophyte formation, are observed. OA change accompanying joint destruction is also seen in inflammatory OA, crystal-induced arthritis, hemochromatosis, ochronosis, Wilson disease and post-traumatic status, depending on each cause of degenerative change. Hematologic and vascular disorders often induce joint destruction. Hemophilia and avascular necrosis (idiopathic / steroid, trauma, embolic status) are well-known. Osteochondrosis / apophyseopathy, such as Perthes, Kienböck and Freiberg diseases, can induce osteoarthritic change, often leading to joint destruction. Neurogenic osteoarthopathy (Charcot joint), endocrine disorder (i.e., hyperparathyroidism, acromegaly), skeletal dysplasia (i.e., multiple epiphyseal dysplasia, spondyloepiphyseal dysplasia), amyloidosis and tumor allied condition (i.e., pigmented villonodular synovitis, osteochondromatosis) has potential to develop OA with joint destruction. Biomaterial-derived foreign body reaction can also cause joint destruction. Rapid destructive coxarthropaty is often seen in senile.

Spinal column is composed by combination of diarthrodial and synarthrodial joints. Destruction of spinal column elements are known as results of, such as, spondylosis deformans and ochronosis by degenerative changes, infectious spondylitis by micro-organisms, RA and seronegative spondylarthropaties (ankylosing spondylitis, psoriatic arthritis and reactive arthritis) by autoimmune-based inflammatory disorders, diffuse idiopathic skeletal hyperostosis, dialysis-associated spondylarthopathy and post-traumatic status.

It is important to know and bring to mind the basic structures and characteristics of each joint in the musculoskeletal system, as well as to understand pathologic mechanism responsible for joint destruction caused by various disease types and structural features, for accurate diagnosis and appropriate treatment.