総説

変形性膝関節症の滑膜炎症と疼痛発生のメカニズム

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要旨：本邦では内側型変形性膝関節症の頻度が最も高く、膝関節は内反変形を呈する。内反変形は必然的に内側コンパートメントの過剰な負荷をもたらし、軟骨下骨硬化、骨棘形成さらに関節軟骨変性を誘導する。軟骨変性の進行に伴い、中期から末期では内外反動揺性を認める（側方動揺）。変形性膝関節症の滑膜炎は軟骨磨耗片により生じる二次性と考えられているが、力学的負荷が、日常生活動作で絶えず滑膜に加わることを認識する必要がある。力学的負荷に呼応する組織には神経組織があり、膝関節滑膜には交感神経系ばかりでなく、体性知覚神経系も存在する。内側型変形性膝関節症では、内側滑膜で多数のサブスタンスPやCGRP陽性神経線維を認め、さらに炎症所見が他の部位に比較し最も顕著である。また症例の多くが，内側関節裂隙に一致した部位で自発痛や圧痛を愁訴することから、変形性膝関節症の疼痛と滑膜炎症過程には、軟骨磨耗片ばかりでなく神経原性炎症が関与する可能性がある．

索引用語：内側型変形性膝関節症，神経原性炎症，サブスタンスP，疼痛発生機序，滑膜炎症

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Mechanism of Pain Provocation and Synovitis　in Osteoarthritis of the Knee

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Abstract: The majority of primary osteoarthritis of the knee (OA) has medial involvement with varus deformity. Varus deformity causes excessive biomechanical stress loaded onto some focal area in the medial compartment, inducing osteosclerosis of subchondral bone, spur formation and degeneration of articular cartilage. Furthermore, OA knees in a moderate or an end stage show varus-valgus instability (lateral thrust). Synovitis in OA may be merely a secondary reaction brought about by the debris of cartilaginous destruction. However, it may be very important to notice that the medial synovium is exposed to various mechanical stresses in daily activities. Neural elements initially receive those noxious mechanical stimuli.

The synovium in a knee joint has an extensive neural network in the somatic and autonomic nervous systems. In medial compartmental osteoarthritis of the knee, neuropeptides were most abundant, with an especially large number of substance P (SP) and calcitonin gene-related peptide (CGRP) immunoreactive free nerve endings. In the medial region, the synovitis was more remarkable than in the lateral region. Patients complain pain on the antero-medial portion of the knee joint when walking or standing. Therefore, these findings suggest that neurogenic factors may be involved in the development and persistence of inflammatory synovitis and the pain pathway in OA knees.

Key words: Medial compartmental osteoarthritis of the knee, Neurogenic inflammation, Substance P, Pain provocation, Synovitis

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