Osteoarthritis Across Multiple Joints (Wear-and-Tear Arthritis)

Articular Cartilage Breakdown, Subchondral Sclerosis, Multi-Joint Distribution Patterns, Etiology, and Therapeutic Management

Osteoarthritis (OA), commonly referred to as wear-and-tear arthritis, is the most prevalent form of joint disease globally. It is characterized by the progressive deterioration of articular cartilage coupled with changes in the underlying subchondral bone, marginal osteophyte formation (bone spurs), and low-grade synovial inflammation. While it can occur in a single joint following trauma, generalized osteoarthritis affects multiple joint groups throughout the body.

> **Core Clinical Principle:** Osteoarthritis is no longer viewed as purely mechanical wear-and-tear; it is recognized as an active whole-joint disease involving cartilage degradation, bone remodeling, and inflammatory changes affecting synovial tissues.

1. Multi-Joint Distribution Patterns & Clinical Features

Generalized osteoarthritis typically targets specific weight-bearing and high-use joints symmetrically or across multiple regions.

Affected Joint Group	Pathological Findings & Characteristics	Clinical Presentation & Functional Impact
Knees (Tibiofemoral / Patellofemoral)	Cartilage loss, joint space narrowing, subchondral cysts, and osteophytes.	Pain with weight-bearing (walking, stairs), crepitus, stiffness after inactivity, and joint effusion.
Hips (Acetabulofemoral)	Degeneration of the ball-and-socket joint, superior or axial cartilage loss.	Groin or lateral hip pain, reduced internal rotation, and antalgic gait.
Spine (Cervical & Lumbar)	Facet joint arthritis and vertebral body osteophyte (spur) formation.	Neck or lower back stiffness, radiating discomfort, and occasional nerve root compression.
Hands (DIP, PIP & Thumb CMC)	Heberden and Bouchard nodes in fingers; degeneration at the thumb base.	Finger stiffness, bony enlargement, and weakened pinch and grip strength.

2. Pathophysiology & Underlying Etiology

The progression of osteoarthritis involves a complex interplay of mechanical, cellular, and biochemical factors.

- **Cartilage Matrix Breakdown:** Chondrocytes attempt to repair damaged cartilage, but catabolic enzymes (such as matrix metalloproteinases) outpace synthesis, leading to loss of type II collagen and proteoglycans.
- **Subchondral Bone Remodeling:** As protective cartilage cushions wear away, underlying bone hardens (sclerosis), forms microfractures, and develops marginal bony outgrowths (osteophytes).
- **Etiology & Risk Factors:** Driven by advanced age, systemic genetic predisposition, obesity (which overloads weight-bearing joints), joint misalignment, and prior acute trauma or repetitive occupational stress.

3. Clinical Presentation & Management Principles

Comprehensive management targets pain control, functional preservation, and minimizing joint loading.

- **Clinical Presentation:** Joint pain that worsens with activity and improves with rest, morning stiffness lasting under 30 minutes, decreased range of motion, and joint enlargement.
- **Therapeutic Strategy:** Combines non-pharmacological interventions (structured physical therapy, aerobic exercise, weight management, assistive devices) with pharmacologic therapy (topical or oral NSAIDs) and surgical options (joint replacement/arthroplasty) for advanced disease.