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Rehabilitation of Common Rheumatological Disorders

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SUMMARY PEARLS

- Rheumatic diseases include immune-related and nonimmune-related musculoskeletal disorders. Physiatrists should learn evaluation and treatment of rheumatic diseases.
- Pain, limited morning stiffness, reduced function, crepitus, restricted movement, and bony hypertrophy are the central features of osteoarthritis (OA).
- Treatment of OA includes nonpharmacological intervention, pharmacological treatment, and surgical options, and recently prolotherapy with intraosseous injection and transarterial microembolism has shown promising results.
- Rheumatoid arthritis (RA) is a chronic, systemic, inflammatory disease of unknown etiology that primarily involves the joint, especially small joints.
- Pharmacological treatment of RA comprises nonsteroidal antiinflammatory drugs, low-dose oral corticosteroid, and disease-modifying antirheumatic drugs, including conventional synthetic, targeted synthetic, and biological agents.
- Rehabilitative management of rheumatic diseases should be started with detailed evaluation and include patient education, exercise (strengthening, stretch, aerobic, and recreational), orthoses, and physical modalities (heat, cold, low-power laser, and electric therapy).

Introduction to Rheumatic Diseases

Rheumatology is a branch of medicine devoted to the evaluation, diagnosis, and treatment of immune-related and nonimmune-related musculoskeletal disorders or other connective tissue disorders, including different types of arthritis, lesions of soft tissues (such as muscle, tendon, cartilage, bursa, and fascia), vasculitis, and hereditary connective tissue disorders. Arthritis may occur because of immunological dysfunction (e.g., rheumatoid arthritis [RA], ankylosing spondylitis [AS], and psoriatic arthritis); degeneration (e.g., degenerative arthritis or osteoarthritis [OA]); metabolic problems (e.g., gout and other crystal-related arthritis); and bacterial or microbial infection. Arthritis and soft tissue disorders may cause pain, impairment, physical disability, dependence on others for activities of daily living (ADLs), occupational disability, and psychosocial problems, leading to the loss of wages and socioeconomic burdens.

Since 2000, a burst of biological agents and synthetic antirheumatic drugs has changed the treatment and clinical outcomes of rheumatic diseases; however, pain and physical disability persist in patients with rheumatic diseases. Although physiatrists are not much concerned regarding immunology and new biological and synthetic drugs, they pay considerable attention to the biomechanical problems of rheumatic diseases and act as leaders in the rehabilitation team for most musculoskeletal disorders. Because soft tissue lesions are discussed in detail in other chapters, this chapter focuses mainly on arthritis and related disorders.

A diarthrodial or synovial joint is composed of the ends of bones, synovium, cartilage, and a joint capsule, which encloses the joint. The joint capsule is surrounded by ligaments and other periarticular structures such as bursae, tendons, and muscles. The joint capsule contains a joint cavity, which is filled with a lubricating synovial fluid. Diarthrodial joints allow a large range of movement and are the most common joints in the extremities, such as the shoulders, elbows, wrists, hips, knees, and ankles. Arthritis is the inflammation of a joint or joints and is clinically characterized by pain, tenderness, swelling, warmth, and redness. In the chronic stage, joint damage may occur, and deformity, crepitus, reduction in the range of motion (ROM) of the joint, or abnormal movement pattern may develop. Reduction in the joint ROM may be caused by joint inflammation (synovial hypertrophy and possible

effusion), contracture of soft tissues, or irreversible damage to the articular cartilage or subchondral bone.

Clinical examination of patients with rheumatic diseases should involve observation of the entire person, including movement and posture, and then examination of each region. The procedure of local regional examination includes look, feel, move, and strength. For "look," pay attention to swelling, deformity, skin change, and muscle atrophy. For "feel," one first evaluates warmth by using the backs of the fingers, palpates tenderness, and determines the precise location of swelling. For detection of joint effusion, techniques such as the bulge sign (Video 32.1) and patellar ballottement (Video 32.2) may be used. For "move," both active and passive movements are assessed. Active movement is observed mainly to screen for possible lesion location. Pain or limited ROM are criteria for abnormality.

During passive movement, pain, reduced ROM, and abnormal "end feel" (sensation of the examiner's hand at end ROM) should be recorded. "Strength" is usually examined for the contractile tissues (i.e., muscle and tendon). It is usually tested with maximal isometric contraction at the neutral position. If pain and/or weakness during or after the test is reported, a lesion of contractile tissues, including the muscle, tendon, tendon sheath, musculotendinous junction, and tenoperiosteal junction (enthesis), is likely (Video 32.3). The exact location of contractile tissue lesions may be determined through palpation or accessory tests (see later). An accessory test is sometimes required for lesion localization (e.g., passive shoulder horizontal adduction to detect an acromioclavicular lesion and passive forearm pronation to provoke pain and thus to detect biceps brachii tendinopathy at the distal insertion [radial tuberosity]) (Video 32.4). To provoke ligament pain, stretching can be used; to provoke bursa pain, compression or pinching may be used. In addition, joint stability should be evaluated by stressing a joint.

If indicated, imaging studies and laboratory tests should be conducted. In the recent decade, musculoskeletal ultrasound has become popular. The benefits of ultrasound examination are that it is radiation free and is useful for the detection and grading of synovitis, joint effusion, and bony erosion. Laboratory tests include screening for erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), complete blood count, uric acid, rheumatoid factor (RF), anticitrullinated protein antibody (ACPA), or anticyclic citrullinated peptide (anti-CCP), antinuclear antibody, human leukocyte antigen-B27 (HLA-B27), and synovial fluid analysis. A combination of history taking, physical examination, imaging studies, and laboratory tests is usually used to arrive at the correct diagnosis or disease classification. For rehabilitation evaluation of rheumatic diseases, the International Classification of Functioning, Disability and Health (ICF) model is suggested.

Osteoarthritis

OA is the most common type of arthritis, typically affecting the hands, spine, hips, knees, and feet. It is characterized by cartilage degradation, changes in the subchondral bone, meniscal degeneration, synovial inflammation, and a periarticular bone response. OA is caused by multiple factors, including genetic predisposition, joint integrity, mechanical forces, and cellular biochemical processes.[26]

Knee Osteoarthritis

Clinically, knee OA is characterized by joint pain that worsens with activity and restricts movement. It typically shows focal cartilage loss, new bone formation, and affects all joint tissues, as reflected in radiographic findings. Approximately 12.5% of people suffer from knee OA, with a higher prevalence among females and older adults, increasing with age.[32]

Risk Factors

Identifying risk factors for knee OA is essential for early detection and prompt, effective treatment to prevent worsening of the condition. Major risk factors include:

Age: Aging is the most important risk factor. The risk increases with age, as wear and tear on the joints accumulates over the years.

Sex: Females are more likely than males to develop knee OA, especially after menopause.

Overweight: Obesity is a significant modifiable risk factor. The lifetime risk for knee OA increases as body mass index (BMI) increases. Individuals with a BMI over 30 are three times more likely to develop early OA compared with those of normal weight. Excess weight stresses knee joints not only through mechanical load but also through obesity-related inflammatory markers that may exacerbate OA.

Prior joint injuries: Injuries to the knee, such as those occurring during sports or accidents, can increase the risk of OA. Even old injuries that seemed to have healed can increase the risk.

Repetitive stress: Occupations or activities involving repetitive knee stress, such as squatting, kneeling, or heavy lifting, further increase the likelihood of developing OA.

Symptoms and Signs

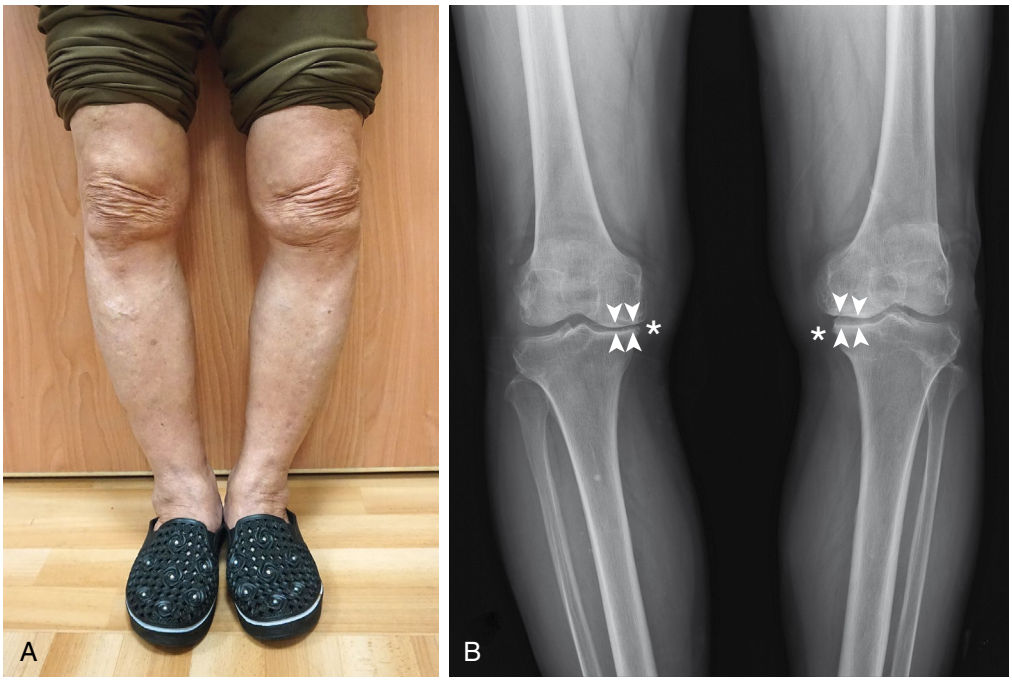
Pain at and around the knee joint is the most common symptom of knee OA, typically worsening after activity and improving with rest. Other symptoms include stiffness, swelling, deformity, crepitus, and impaired function. The nature of the pain can suggest different underlying issues: Pain during activity often points to mechanical stress or enthesopathy, pain at rest may indicate inflammation, and nocturnal pain could be caused by increased pressure within the bone. Persistent pain at rest and at night suggests advanced cases and significant joint damage. These symptoms can manifest in various combinations and primarily indicate changes in the structure and function of the knee joint. The severity and occurrence of these symptoms can vary significantly among individuals, and not everyone will experience all symptoms.

Signs indicative of knee OA include crepitus, reduced ROM, bony enlargement, and little to no effusion. Additional features may include deformity, instability, and tenderness around the joint or along the joint line. Muscle weakness and wasting may also be evident. These signs can manifest in any combination and primarily reflect structural changes in the knee joint: Crepitus indicates irregular joint surfaces, bony enlargement suggests the formation of osteophytes and bone remodeling, and restricted movement is typically caused by joint space narrowing (Fig. 32.1).

The three main symptoms of knee OA are persistent knee pain, limited morning stiffness, and reduced function. These symptoms primarily arise from mechanical limitations and inflammatory processes within the joint. The three key signs are crepitus, restricted movement, and bony enlargement. Knee pain not only results from joint damage but can also accelerate the deterioration of the joint. Persistent knee pain lasting over 1 year can result in faster cartilage loss and increases the risk of worsening changes visible on x-rays.

Imaging

Clinical OA is diagnosed based on symptoms and signs obtained from a detailed history and physical examination, while radiological



• **Fig. 32.1** (A) A 70-year-old patient with knee osteoarthritis has a varus deformity in the knee, bony enlargement, and quadriceps muscle wasting. (B) Weight-bearing views of the knee joints reveal definite osteophytes *(asterisks)* and moderate space narrowing at the medial tibiofemoral compartment *(arrowheads)*. These radiographic features reflect the altered appearance of the knee joints.

TABLE 32.1 Kellgren-Lawrence Radiographic Grading Scale for Osteoarthritis of the Tibiofemoral Joint

Grade of Osteoarthritis	Description
0	No radiographic findings of osteoarthritis
1	Doubtful narrowing of joint space and possible osteophytic lipping
2	Definite osteophytes with possible narrowed joint space
3	Definite osteophytes with moderate joint space narrowing and some sclerosis
4	Definite osteophytes with severe joint space narrowing, subchondral sclerosis, and definite deformity of bone contour

OA is identified by structural changes within the joint. The Kellgren-Lawrence grading scale (Table 32.1),[27] commonly used to define the severity of knee OA, should be evaluated alongside a clinical assessment.

Radiographs in middle-aged or elderly patients complaining of knee pain invariably show some joint space narrowing and osteophyte formation. Moreover, knee symptoms can precede any changes visible on x-ray. Therefore abnormal x-ray findings alone are not reliable for assessing knee pain. The diagnosis of symptomatic knee OA should mainly be based on clinical findings.[44]

Radiography is commonly used in clinical practice to establish a structural diagnosis of OA and to monitor disease progression. It is the most frequently used imaging modality, primarily assessing OA through the presence of osteophytes and joint space narrowing (see Fig. 32.1).

Ultrasound is safe, affordable, and effective for visualizing cartilage thickness (Fig. 32.2), synovial fluid, synovial hypertrophy, joint space narrowing, and osteophytes. It is sensitive in detecting synovitis and joint effusion but is not recommended as a first-line imaging tool for diagnosing OA.

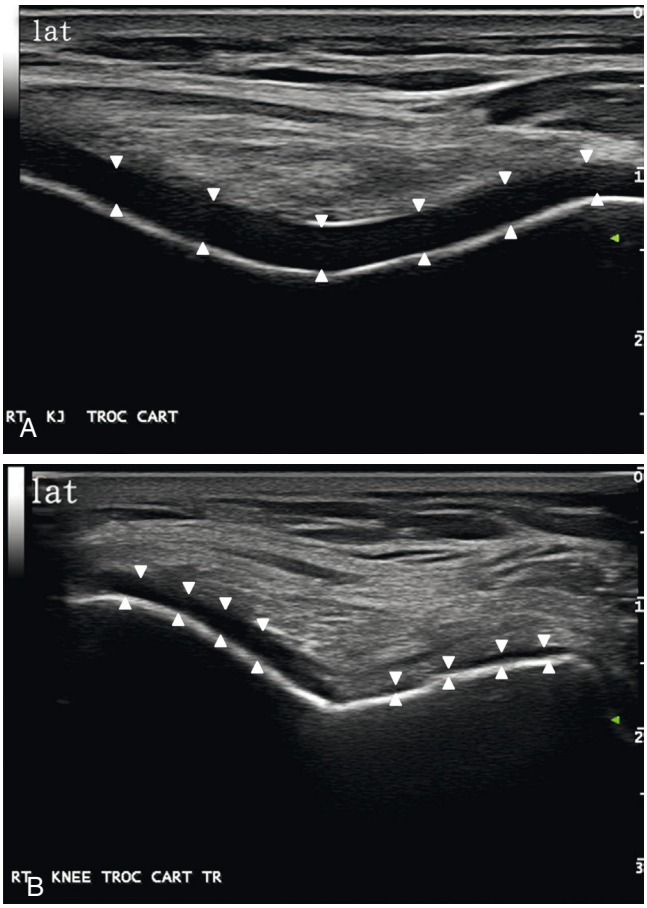
Magnetic resonance imaging (MRI) provides a comprehensive visualization of both osseous and nonosseous articular and periarticular structures relevant to the OA disease process. It offers detailed images of articular cartilage, menisci, ligaments, synovitis, and bone marrow lesions (Fig. 32.3A), which are not clearly visualized on radiographs and are limited on ultrasound.[37]

Prognosis

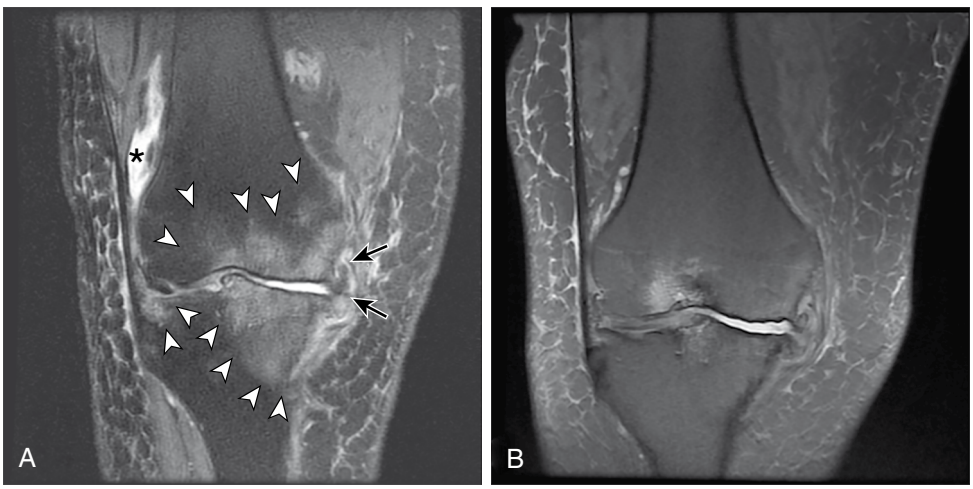
Knee OA progresses slowly. However, if left untreated, it can lead to varying degrees of disability, often manifesting as difficulties in sit-to-stand, walking, climbing stairs, and performing household tasks. The most significant factors associated with a worse prognosis are the severity of joint damage or degeneration visible on x-rays. Other important factors include obesity, older age, presence of comorbid conditions, low physical activity, and poor knee alignment.

Hip Osteoarthritis

Hip OA is the most common joint disorder of the hip and the third most common type of OA, following knee and hand OA. It primarily affects the cartilage and surrounding tissues, with the main symptom being pain. This pain is often felt in the groin, spreading to the thigh, buttocks, or knee, and typically worsens



• **Fig. 32.2** (A) Normal femoral trochlear cartilage in a young male *(arrowheads)*. (B) Severe diffuse focal thinning of the femoral trochlear cartilage at the medial side in a 67-year-old female with osteoarthritis of the knee *(arrowheads)*. Spaces between the arrowheads define the thickness of the trochlear cartilage.



• **Fig. 32.3** (A) A 66-year-old female has been experiencing pain in her right knee, which later extended to both knees over 8 years. She has difficulties squatting and shows limited knee flexion and extension. A coronal intermediate-weighted fat-suppressed magnetic resonance imaging (MRI) of the right knee reveals features of osteoarthritis, including bone marrow edema at the medial femur and medial tibia *(arrowhead),* joint effusion *(asterisk),* and meniscal extrusion *(black arrows)*. (B) Platelet-rich plasma was injected intraosseously into the right femur and tibia, and into the area of medial meniscus extrusion. Three months after treatment, her right knee pain has decreased by 90%. The follow-up MRI showed a significant decrease in joint effusion, along with reductions in bone marrow edema and medial meniscus extrusion signals.

with activity and improves with rest. Other symptoms include stiffness, limited movement, and a crackling sound. Chronic pain from hip OA can also lead to additional pain in other areas, affecting walking and potentially causing knee and lower back pain.

Radiographic signs of hip OA include osteophyte formation, joint space narrowing, and sclerosis of the subchondral bone plate. In more advanced stages, subchondral cyst formation and remodeling of the articular surfaces or deformity may occur. However, hip joint symptoms do not always clearly align with radiographic findings, leading to potential overdiagnosis, as radiographic hip OA is common in the general population and often asymptomatic.

In patient history, three clinical findings associated with the presence of hip OA are a family history of OA, a personal history of knee OA, and pain during activities such as climbing stairs or walking downhill. Physical examination findings are generally more useful than historical features for identifying the presence of OA. The examination often reveals a capsular pattern. Most commonly, internal rotation is the most painful and limited, followed by limitations in flexion, abduction, and extension.

The main risk factor for hip OA is age, as joints wear down over time. This wear affects the cartilage, making it less able to cushion the joints, which can lead to OA. Other risk factors include obesity, previous hip injury, genetics, overuse and stress, sex, and hip development issues.[14]

Hand Osteoarthritis

Hand OA is a common joint disease that typically progresses slowly. The most frequently affected joint in hand OA is the base of the thumb, specifically the trapeziometacarpal (basilar) joint (Fig. 32.4), which is vulnerable because of its unique structure and high load-bearing function. This is followed by the distal interphalangeal (DIP) joint and the proximal interphalangeal (PIP) joint.

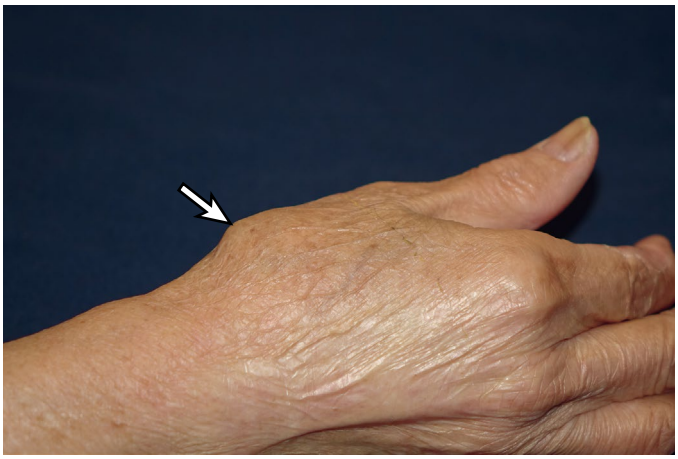
The main symptom of hand OA is pain in the affected joint. Other symptoms include stiffness, weakened hand muscles, and limited hand movement. A key sign of hand OA is squaring at the base of the thumb and the appearance of bumps on the finger joints, known as Heberden nodes at the DIP joints and Bouchard nodes at the PIP joints (Fig. 32.5). The presence of these nodes is a hallmark of hand OA.

The two main risk factors for hand OA are age and sex. Daily wear and tear on the joints, combined with a reduced ability to repair joint tissue, can lead to OA as people age. Females, particularly after menopause, are more likely to develop hand OA possibly caused by hormonal changes that affect the cartilage and joint structures. Other important risk factors include genetics, past injuries, repetitive use of the hands, and obesity. Fat tissue produces inflammatory substances that can cause joint inflammation and damage the cartilage, even in non–weight-bearing joints. There is a strong correlation between the presence of nodules and radiographic evidence of OA; therefore routine imaging is not typically recommended for diagnosing hand OA.

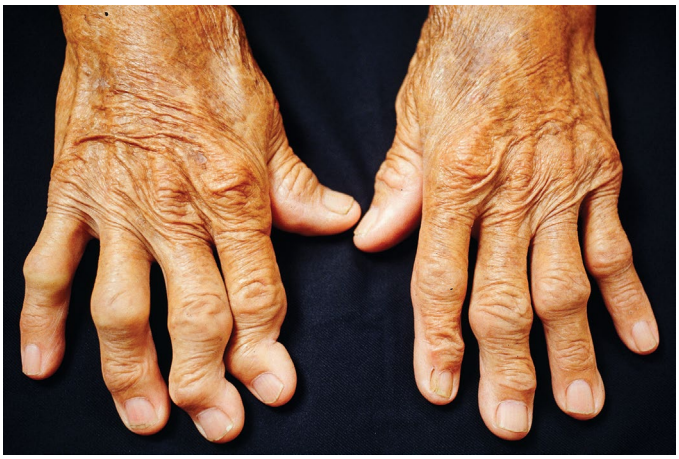
Management of Osteoarthritis

OA-related pain and functional impairment are the hallmarks of OA. The sources of OA-related pain are the bone, synovium, joint capsule, and periarticular structures, including the muscles, tendons, ligaments, and bursae. In addition to local factors, the pain may be influenced by psychosocial and environmental factors, and peripheral and central sensitization of nociceptive pathways may perpetuate the pain and play a role in the chronicity of the disease. Management of OA aims to reduce or eliminate pain, minimize functional impairment and disability, and improve quality of life. Despite numerous efforts, interventions to modify the disease course are still not approved by most evidence-based guidelines. Before treatment, a holistic assessment of people with OA is an essential management strategy. In addition to detailed physical examination of the affected joints, the following crucial items should be addressed: severity and location of joint involvement; causes, frequency, and severity of pain; impact of symptoms on gait and ADLs; impact of participation; psychological effects of OA (e.g., loss of self-esteem, feeling old, sleep disturbance, and depression); assessment of the risk for falls; presence of comorbidities; presence of social support; expectations of treatment; previous treatments; and modifiable risk factors (e.g., obesity, malalignment of joint, and exercise habit). Treatments should be individualized to patients' needs, values, and goals; medical personnel should assist patients in decision-making, with prioritization of patients' safety. Successful treatment depends on patients' adherence, behavior modifications, and optimal implementation of recommendations from medical personnel.

OA management includes nonpharmacological intervention, pharmacological treatment, and surgical options. Nonpharmacological intervention comprises patient education, self-management,



• **Fig. 32.4** Osteoarthritis of the first carpometacarpal joint with subluxation *(arrow)*.



• **Fig. 32.5** Osteoarthritis of the hands involving the proximal and distal interphalangeal joints.

exercise, weight loss management, bracing or splinting, and acupuncture.[6,28] For pharmacological management, acetaminophen, topical or oral nonsteroidal antiinflammatory drugs (NSAIDs), intraarticular corticosteroids injection, and duloxetine are commonly used.

Pharmacological Management

For patients with one or a few joints of involvement, especially hand and/or knee OA, topical NSAIDs are recommended because of similar efficacy compared with oral NSAIDs and better safety profile.[6] Acetaminophen was previously recognized as a safe medication; however, regular use of this medication may cause gastrointestinal blood loss, and its efficacy is not as high as that of other medications. Thus conservative dosing and treatment duration are recommended.

Because of the many side effects of NSAIDs (particularly gastrointestinal, cardiovascular, and renal disorders), treatment with NSAIDs should initially be started on an as-needed basis or be preceded by a trial of topical agents. In addition, to reduce the gastrointestinal side effects, the use of cyclooxygenase-2 (COX-2) inhibitors or addition of misoprostol or proton pump inhibitors to oral NSAIDs may be suggested. The most common side effect of COX-2 inhibitors is the risk of thrombosis, especially coronary artery thrombosis.

Intraarticular corticosteroid injection (Videos 32.5 to 32.9) can reduce inflammation and angiogenesis within the synovium. In randomized clinical trials, intraarticular corticosteroids have been proven effective in the treatment of knee and hip OA; however, their effectiveness is not long lasting. Because of the potential deleterious effects on hyaline cartilage and worsening on OA condition, intraarticular corticosteroid injection is not routinely used; it is used only in inflammatory conditions, especially when there is moderate to severe effusion. The use of oral or intramuscular corticosteroid injections for the management of OA is unproven.

Opioids reduce pain by binding to opioid receptors in the central and peripheral nervous systems. The pain-reducing effect of opioids is modest, and considering their many side effects (e.g., constipation, sedation, psychological effects, and dependence), the use of opioids in OA management is limited. For OA patients with pain sensitization or chronic widespread pain, duloxetine, which is a selective serotonin and norepinephrine reuptake inhibitor antidepressant, may be effective.[7]

Although oral use was postulated to incorporate glucosamine and chondroitin into the articular cartilage, and animal studies have suggested that they may have antiinflammatory effects, in the past few years many nonindustry-funded studies have demonstrated negative results. Thus glucosamine and chondroitin are no longer recommended for OA treatment.

Hyaluronic acid (HA) intraarticular injection (Videos 32.10 to 32.13) is believed to increase the viscosity and elasticity of the OA joint, and previous studies have shown that HA exerts antiinflammatory and antinociceptive effects. HA has been approved by the regulatory agency for use in the treatment of knee OA; however, meta-analyses have shown that HA injection provides either no efficacy or only minimal efficacy. HA injection may also increase the risk of adverse effects (e.g., crystal arthropathy, pseudoseptic joints, and flaring of pain). Therefore recent OA-related academic organizations have either not recommended HA injections for OA management or reported uncertain efficacy.

Prolotherapy, also called proliferation therapy, is an injection-based therapy for chronic musculoskeletal pain. Although the application of prolotherapy can be traced back to the 1950s, it has become increasingly popular internationally over recent years. Prolotherapy usually involves the injection of hypertonic dextrose (15% for soft tissues, 25% for joints), morrhuate sodium, phenol-glycerin-glucose, platelet-rich plasma (PRP), or stem cells into the tendon and ligament insertions and intra-articular structures. It has been hypothesized to cause local irritation and subsequent inflammation, followed by tissue healing, although the mechanism of action remains unknown. Prolotherapy (including PRP injection) has shown short- and long-term improvement in pain, function, and even radiological features, but its application in OA has still not been recommended by most OA-related academic organizations. Ultrasound-guided prolotherapy has been shown to improve the accuracy of medicine delivery and clinical outcomes of treatment.[22] Recently, intraosseous injection of PRP or bone marrow concentrate showed promising results in improving OA or avascular necrosis of the hip or knee (Fig. 32.6, see also Fig. 32.3; Video 32.14).[24,33] For patients with moderate to severe OA, or poor response to conventional treatment, transarterial microembolism (TAME, genicular artery embolization for the knee) can also be tried (Fig. 32.7).[41] The mechanism of pain relief for TAME may be caused by reducing numbers of inflammatory cells and unmyelinated sensory nerve fibers. It can also be used in patients with hand OA with intractable pain.[29]

Surgical Procedures

These include total joint replacement, arthroscopic meniscus surgery, osteotomy, and lavage and debridement. Among them, only total joint replacement has been proven effective for OA. Recently, unicompartmental knee replacement has been shown to be effective for end-stage unicompartment knee OA.

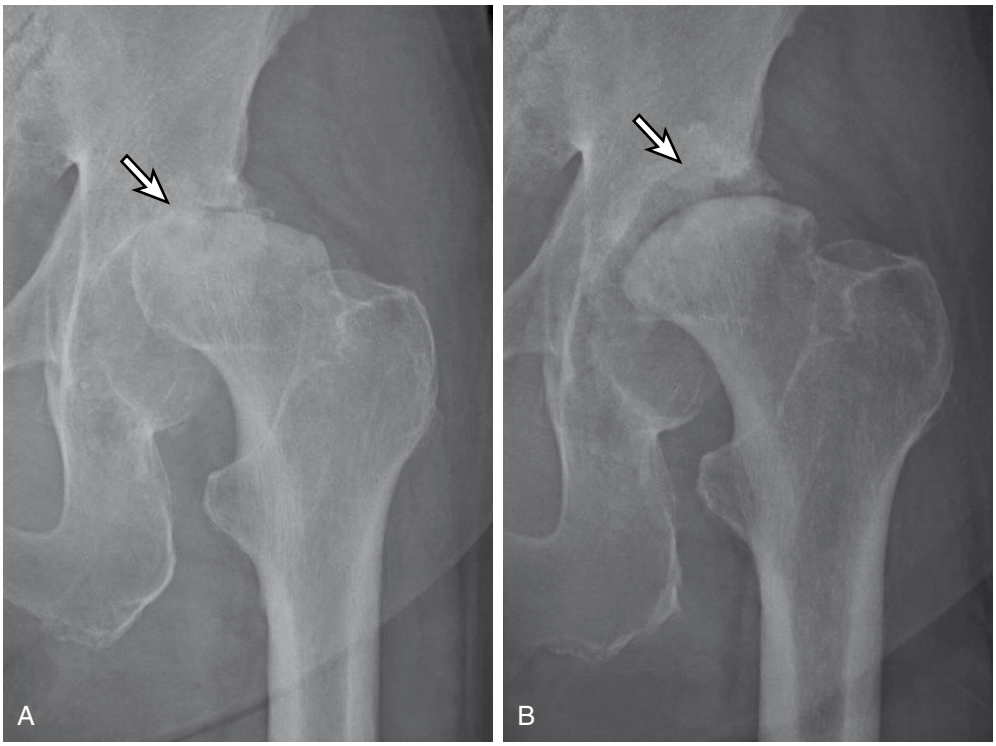
In summary, patients with mild or intermittent pain are likely to require only nonpharmacological treatment and possibly topical NSAIDs, oral analgesics, and intraarticular injection; for patients with frequent pain and more functional limitations, oral NSAIDs, duloxetine, and bracing may also be required; and for patients with prolonged moderate to severe pain and more ADL limitations, stronger pain medications (e.g., opioids) or even surgery may be indicated.

Rheumatoid Arthritis

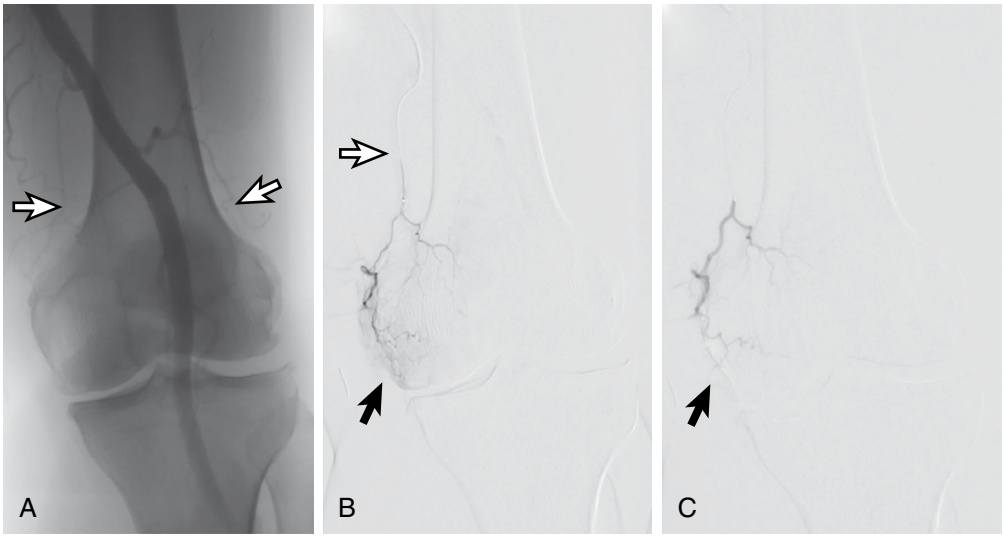
RA is a chronic, systemic inflammatory disease of unknown etiology that primarily involves the joints. It also often involves soft tissues, such as tendon sheaths and bursae. In addition, it may present with extraarticular manifestations. Inflammation and destruction of the joint and soft tissue may lead to joint deformity and loss of physical function. This can happen if the disease is left untreated or when it becomes unresponsive to treatment. The prevalence of RA varies from 0.3% to 1.5% of the population, with a female-to-male ratio of around 3 to 1.

Clinical Characteristics

Typically, the disease's onset is insidious, with pain, stiffness, and swelling of the joints being the predominant symptoms. Morning stiffness, or stiffness after prolonged inactivity, often lasts more than 1 hour in the active inflammatory stage. Up to one-third of patients with RA experience an acute onset of polyarthritis associated with systemic symptoms, including fatigue, myalgia, depression, low-grade fever, and weight loss. The most common joints involved in the early stage of the disease are the metacarpophalangeal (MCP) and PIP joints of the fingers, the interphalangeal (IP)



• **Fig. 32.6** (A) A 76-year-old female presented with osteoarthritis of the hip and femoral head avascular necrosis, exhibiting no joint space with subluxation *(arrow)*. (B) She underwent a series of 13 platelet-rich plasma injections over a span of 6 months. The injections were administered intraosseously in the acetabulum and femoral head, as well as pericapsular injections under reduction. Ultimately, the joint underwent remodeling, resulting in improved status *(arrow)*. (Courtesy Dr. Farn-Wen Tseng.)

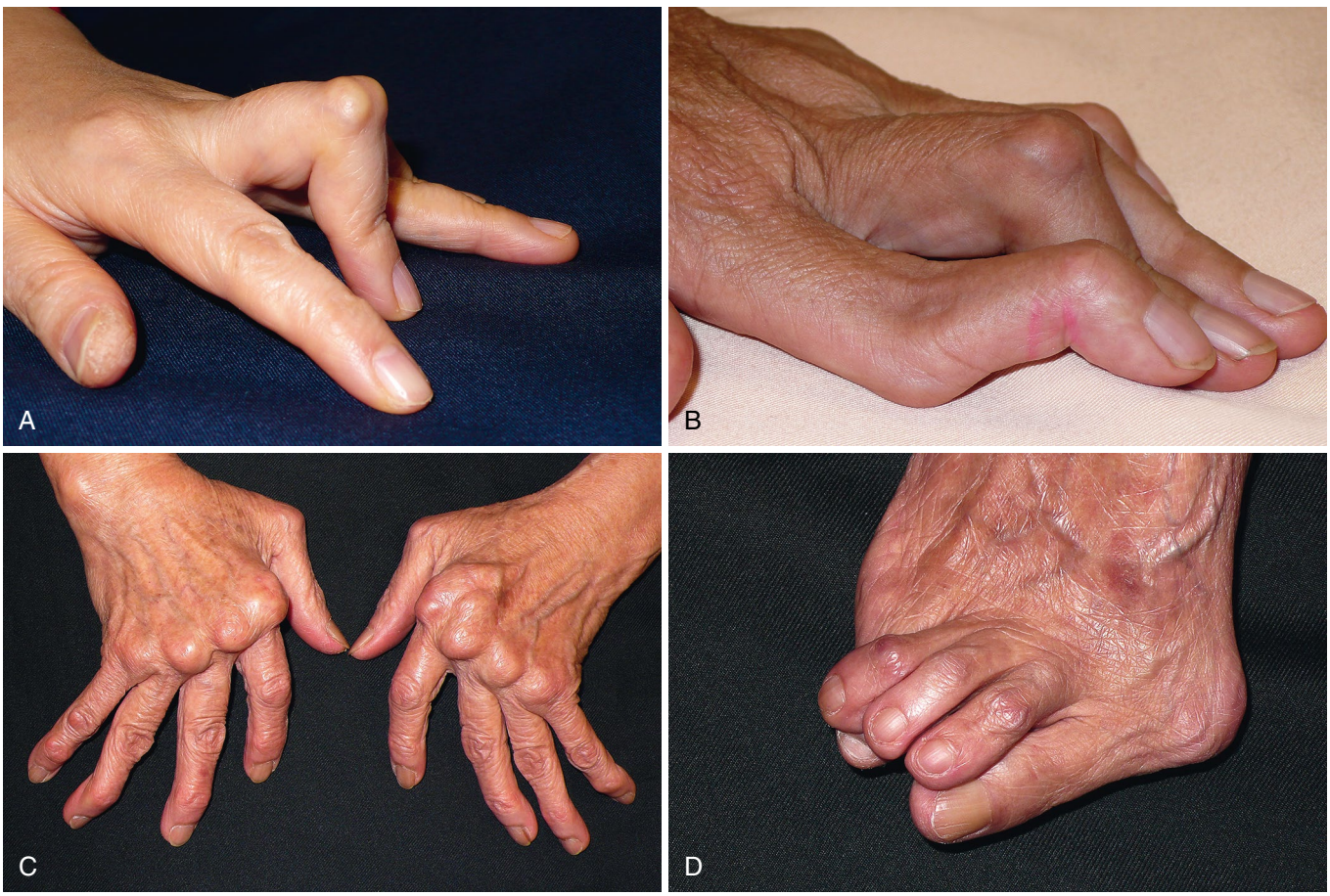


• **Fig. 32.7** Knee joint selective angiography demonstrating successful genicular artery embolization (GAE) with imipenem/cilastatin for abnormal angiogenesis in a 57-year-old male patient with right knee osteoarthritis, Kellgren-Lawrence grade III, and refractory joint pain. The numerical rating scale was improved from 8 to 1.5 after GAE. (A) Angiography via the right distal superficial femoral artery shows the genicular arteries *(white arrows)*. (B) Digital subtraction angiography (DSA) of the descending genicular artery (DGA) via the microcatheter *(white arrow)* reveals abnormal blushlike angiogenesis *(black arrow)*. (C) DSA of the DGA after GAE shows the elimination of the blushlike staining after embolization *(black arrow)*. (Courtesy Dr. Bow Wang.)

joints of the thumb, the wrists, and the metatarsophalangeal (MTP) joints of the toes. Other joints—such as the shoulders, elbows, hips, knees, and ankles—are also frequently affected. Over the whole course of the disease, the facet and atlantoaxial joints of the cervical spine and the acromioclavicular, sternoclavicular, temporomandibular, and cricoarytenoid joints may also be involved. The DIP joints are rarely involved in RA, perhaps resulting from less synovium than the MCPs and PIPs. In addition to involvement of the joints, tenosynovitis is also common in patients with RA and may cause trigger finger (Video 32.15), de Quervain disease (Video 32.16), carpal tunnel syndrome, tendon rupture, and even compression of the cervical cord caused by narrowing of the space available for the upper cervical cord. Baker cyst is also common in RA patients, and aspiration can be performed. Enlargement of the stalk under ultrasound guidance may help obliterate the check valve mechanism, thus preventing the recurrence of the cyst (Video 32.17).

In the late stages of RA, joint deformities commonly occur. Buttonhole (or boutonnière) deformity is flexion of the PIP joints with extension of the DIP joints (Fig. 32.8A). Because the central extensor tendon is destroyed by tenosynovitis, the PIP joints pop up dorsally, resulting in lateral and ventral displacement of the lateral bands of the extensor tendon. In this condition, the lateral bands of the extensor tendon act as flexors of the PIP joints; with tendon shortening, hyperextension of the DIP joints develops. In contrast, the swan-neck deformity is the opposite of the buttonhole deformity, with hyperextension of the PIP joints and flexion of the MCP and DIP joints (see Fig. 32.8B). Shortening of the intrinsic muscle exerts tension on the dorsal tendon sheath, leading to hyperextension of the PIP joints. The lateral bands of the extensor tendon sublux dorsally as the PIP joints herniate in the ventral direction. In addition, shortening of the deep flexor tendons causes flexion of the DIP joints. Other deformities include ulnar deviation (see Fig. 32.8C) of the MCP joints, palmar subluxation of the wrists, arthritis mutilans, hammertoe deformity, claw-toe deformity, flat feet, hallux valgus (see Fig. 32.8D), metatarsal joint subluxation, and Z deformity of the thumb (hyperextension of the IP joint, flexion, and subluxation of the MCP joints).

Owing to the extraarticular foci of the immune response, patients with RA may have different types of extraarticular manifestations during the course of the disease. Common extraarticular features include fatigue, mild normocytic normochromic anemia, rheumatoid nodule (subcutaneous nodule, which occurs in 15%–20% of patients with RA), scleritis, episcleritis, myositis, vasculitis, neuropathy, pericarditis, interstitial pneumonitis and fibrosis, nodular lung disease, myocarditis, cardiac conduction defect, Felty syndrome (RA with neutropenia and splenomegaly), Sjögren syndrome, and amyloidosis. Vasculitis is a serious condition; it can present in five different clinical ways: distal arteritis, cutaneous ulceration, palpable purpura, arteritis of the viscera, and peripheral neuropathy (mononeuritis multiplex or distal



• **Fig. 32.8** Typical deformities of the hands and foot in patients with rheumatoid arthritis. (A) Buttonhole (third finger) deformity. (B) Swan-neck (second finger) deformity. (C) Ulnar deviation. (D) Hallux valgus and toes overriding.

sensory neuropathy). Extraarticular features may be associated with poor prognosis, particularly vasculitis and rheumatoid lung disease. The presence of RF and ACPA or anti-CCP is also common in patients with RA.

Classification Criteria

Until 2010, the classification criteria for RA had been based on the 1987 American Rheumatism Association (ARA) revised criteria, which included four clinical criteria (morning stiffness, arthritis of three or more joint areas, arthritis of hand joints, and symmetrical arthritis), positive RF, the presence of rheumatoid nodules, and radiographic changes (Box 32.1). The four clinical criteria must have been present for 6 weeks.[5] These criteria may be useful for clinical study and can rule out some varieties of transient polyarthritis (e.g., acute viral polyarthritis). However, a major drawback of these criteria is their ineffectiveness in identifying some patients with early disease who subsequently have typical RA because rheumatoid nodules and radiographic erosive changes are usually not present in the early stage of disease. In addition, ACPA testing (which has a similar sensitivity for RF but is much more specific for RA) was not previously available. In contrast, the ARA criteria did not require any exclusion; thus a patient could initially fulfill the diagnostic criteria of RA but evolve into other diagnoses later, particularly Sjögren syndrome, scleroderma, psoriatic arthritis, crystalline arthritis, and systemic lupus erythematosus (SLE). To facilitate earlier diagnosis of RA and thus early effective treatment, the Joint Working Group of the American College of Rheumatology (ACR) and the European Alliance of Associations for Rheumatology (EULAR) developed new criteria for RA in 2010 (Table 32.2).[3] The 2010 criteria comprise four domains: (1) type and number of affected joints, (2) RF and ACPA, (3) acute-phase reactants (CRP and ESR), and (4) the duration of symptoms. For the evaluation of a patient with suspected RA, the highest category within each domain is taken, and the four respective numbers are added. The maximal possible score is 10, where a score of 6 or more indicates the presence of RA. The new criteria place a greater emphasis on serology and imaging studies (ultrasound and MRI), which can also be used to evaluate synovitis.

• BOX 32.1 1987 Revised American Rheumatism Association Criteria for the Classification of Rheumatoid Arthritis

Morning stiffness: Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement.

Arthritis of three or more joint areas: Soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician in at least three joint areas simultaneously. The 14 possible areas are right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints.

Arthritis of hand joints: At least one area swollen (as defined previously) in a wrist, MCP, or PIP joint.

Symmetrical arthritis: Simultaneous involvement of the same joint areas (as defined previously for criterion 2) on both sides of the body (bilateral involvement of PIP, MCP, or MTP joints is acceptable without absolute symmetry).

Rheumatoid nodules: Subcutaneous nodules over bony prominences, on exterior surfaces, or in juxtaarticular regions observed by a physician.

Serum rheumatoid factor: Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in <5% of normal controls.

Radiographic changes: Radiographic changes typical of rheumatoid arthritis on posteroanterior hand and wrist radiographs, which must include erosion or unequivocal bony decalcification localized in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify).

MCP, Metacarpophalangeal; *MTP*, metatarsophalangeal; *PIP*, proximal interphalangeal.

Management of Rheumatoid Arthritis

The comprehensive management of RA requires a combination of nonpharmacological strategies, medical treatment, and surgical interventions. Nonpharmacological measures include patient education,

TABLE 32.2 2010 American College of Rheumatology/European League Against Rheumatism Classification Criteria for Rheumatoid Arthritis Score-Based Algorithm for Classification in an Eligible Patient (Cutpointfor RA: ≥6/10)

Joint Involvement[a]	**(0–5)**
1 medium to large[b] joint	0
2–10 medium to large joints	1
1–3 small[c] joints (with or without involvement of large joints)	2
4–10 small joints (with or without involvement of large joints)	3
>10 joints (at least one small joint)	5
Serology	**(0–3)**
Negative RF **AND** negative ACPA (or anti-CCP)	0
Low[d]-positive RF **OR** low-positive ACPA (or anti-CCP)	2
High[e]-positive RF **OR** high-positive ACPA (or anti-CCP)	3
Acute Phase Reactants	**1**
Normal CRP **AND** normal ESR	0
Abnormal CRP **OR** abnormal ESR	1
Duration of Symptoms	**(0–1)**
<6 weeks	0
≥6 weeks	1

ACPA, Anticitrullinated protein/peptide antibody; *anti-CCP*, anticyclic citrullinated peptide antibody; *CRP*, C-reactive protein; *ESR*, erythrocyte sedimentation rate; *RF*, rheumatoid factor.
[a]*Joint involvement* refers to any swollen or tender joint on examination or evidence of synovitis on magnetic resonance imaging or ultrasonography. Distal interphalangeal joints, first carpometacarpal joint, and first metatarsophalangeal joint are excluded from assessment.
[b]*Medium to large joints* refers to shoulders, elbows, hips, knees, and ankles.
[c]*Small joints* refers to the metacarpophalangeal joints, proximal interphalangeal joints, metatarsophalangeal joints 2–5, thumb interphalangeal joints, and wrists.
[d]*Low titer* means >1 and <3 times upper limit of normal.
[e]*High titer* means ≥3 times upper limit of normal.
Modified with permission from Aletaha D, Neogi T, Silman AJ, et al. 2010 Rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Ann Rheum Dis.* 2010;69:1580-1588.

relative rest with appropriate exercise, physical modalities, occupational therapy, proper orthoses, appropriate shoes, and durable medical equipment. Most of these measures are covered in the domain of rehabilitation management, which is detailed later in this chapter.

Although RA remains incurable, modern therapeutic drugs provide significant control over disease progression and symptoms. The treatment must be prompt, as existing joint damage is irreversible. According to 2021 ACR guidelines, a treat-to-target strategy is emphasized, aiming for low disease activity or remission. If the treatment target is not achieved within 6 months, therapy should be adjusted.[18]

There are three classes of drugs commonly used in treating patients with RA: NSAIDs, glucocorticoids, and disease-modifying antirheumatic drugs (DMARDs).

Nonsteroidal Antiinflammatory Drugs

NSAIDs effectively reduce RA symptoms, improving pain and stiffness as well as joint function. However, they are frequently associated with side effects that affect the renal, hepatic, gastrointestinal, and cardiovascular systems.

Glucocorticoids

Glucocorticoids can quickly reduce inflammation and pain. They may be recommended for short-term use to manage flares or as bridge therapy until DMARDs take effect. However, their disease-modifying effects are minimal. Additionally, prolonged use, especially at doses above 5 mg daily, poses significant risks and should be avoided. Intraarticular or intrasheath corticosteroid injection under palpation or ultrasound guidance is recommended when one or two joints or tendon sheaths are particularly involved and causing symptoms (Videos 32.18 to 32.21).

Disease-Modifying Antirheumatic Drugs

DMARDs can alter the course of the disease, promoting remission by suppressing autoimmune activity and reducing or preventing joint damage. Unlike NSAIDs and glucocorticoids, which do not affect disease progression, it is recommended that patients with RA start treatment with DMARDs early to modify the disease course and encourage remission. DMARDs are subdivided into three categories: (1) conventional synthetic DMARDs (csDMARDs), (2) biological DMARDs (bDMARDs), and (3) targeted synthetic DMARDs (tsDMARDs) (Table 32.3).

csDMARDs. Treatment should be initiated immediately upon diagnosis. Oral methotrexate (MTX) is the preferred first choice because of its safe profile among csDMARDs used for RA. Additionally, MTX offers the most significant efficacy, better dosage flexibility, and lower cost compared with other csDMARDs. It also has a relatively quick therapeutic onset, typically taking effect within 6 to 8 weeks.[18] If MTX monotherapy fails to achieve the treatment target within 3 to 6 months, other csDMARDs, such as hydroxychloroquine, sulfasalazine, or leflunomide, should be considered as part of the initial treatment strategy.[12] Should the combination of MTX and other csDMARDs not yield the desired outcome, it is then appropriate to commence treatment with either bDMARDs or tsDMARDs.[18,34]

bDMARDs. Biological therapies are typically considered when patients show an inadequate response to csDMARDs. These therapies target specific pathways involved in the inflammation process. The currently available drugs for RA target various pathways, as detailed in Table 32.3.

TABLE 32.3 DMARDs Currently Available for Rheumatoid Arthritis

Type		Target	Drugs
Synthetic	Conventional synthetic (csDMARD)		Methotrexate, leflunomide, sulfasalazine, hydroxychloroquine
	Targeted synthetic (tsDMARD)	JAK	Tofacitinib, baricitinib, upadacitinib
Biological	Biological (bDMARD)	TNF	Etanercept, adalimumab, infliximab, golimumab, certolizumab pegol
		B cell	Rituximab, ofatumumab
		T cell	Abatacept, belatacept
		IL-6	Tocilizumab, sarilumab
		IL-1	Anakinra, canakinumab, rilonacept

DMARD, Disease-modifying antirheumatic drug; *IL*, interleukin; *JAK*, Janus kinase; *TNF*, tumor necrosis factor.

tsDMARDs. This newer class of DMARDs, including Janus kinase (JAK) inhibitors (JAKi), blocks the JAK pathways involved in the immune response. The currently available drugs for RA are listed in Table 32.3. They are particularly useful for patients who prefer oral medications or for those who are unsuitable for or have not responded well to bDMARDs.

In patients who have maintained remission for at least 6 months, a stepwise tapering of RA treatment should be considered to minimize both side effects and costs. The current treatment goal is to maintain low disease activity using the minimal effective dose of medication and the fewest possible number of drugs.

Tapering of bDMARD or tsDMARD should be considered first. This can involve reducing the dose or increasing the interval between doses. Complete discontinuation of bDMARD or tsDMARD is generally not advisable because it may lead to a recurrence of the disease in most patients.

Although DMARDs significantly improve symptoms and prevent disease progression in patients with RA, they also come with considerable side effects. MTX, leflunomide, and sulfasalazine have similar adverse effect profiles, including gastrointestinal distress, rash, bone marrow suppression, hepatotoxicity, and an increased incidence of infections. Both MTX and leflunomide can cause alopecia. Additionally, leflunomide may lead to hypertension, peripheral neuropathy, and weight loss, while sulfasalazine is particularly associated with a high risk of gastrointestinal distress.

Hydroxychloroquine has the main advantage that it does not produce side effects related to bone marrow suppression, kidney, or liver function. As a result, hydroxychloroquine has the safest profile among all csDMARDs. The ACR guidelines conditionally recommend hydroxychloroquine over other csDMARDs for patients with low disease activity. However, it is important to note that high

cumulative doses and long-term use of hydroxychloroquine can lead to its most significant side effect, ophthalmic toxicity.[34]

The most concerning adverse effect of both bDMARDs and tsDMARDs is an increased risk of common and serious infections. There is also a risk of reactivating tuberculosis, herpes zoster, and hepatitis B/C. Before starting bDMARD or tsDMARD therapy, all patients should undergo screening for tuberculosis and hepatitis. Ideally, vaccinations, especially live vaccines, should be administered at least 4 weeks before starting bDMARD or tsDMARD therapy. Killed or recombinant vaccines can be given either before or during treatment with csDMARDs, bDMARDs, or tsDMARDs. However, live vaccines should be avoided while patients are being treated with bDMARDs or tsDMARDs.[12,18]

DMARDs, especially bDMARDs, should be avoided in patients with active infection, preexisting bone marrow hypoplasia, leukopenia, or immunodeficiency syndromes. MTX and leflunomide should be avoided in patients with severe liver disease. Tumor necrosis factor inhibitors (TNFi) are not recommended for individuals with a history of congestive heart failure.[18]

During pregnancy and lactation, MTX and leflunomide are contraindicated because of their teratogenic effects. In contrast, hydroxychloroquine is considered safe for use during pregnancy and is strongly recommended for pregnant females. Among biological DMARDs, certolizumab is the only one that can be safely used during pregnancy because it does not transfer across the placenta. Additionally, golimumab is a viable therapeutic option during lactation because of its high molecular weight.

Ankylosing Spondylitis

Seronegative spondyloarthropathy is a type of chronic inflammatory arthritis involving the axial structures; it is manifested by chronic back pain and progressive stiffness of the spine.[38] It can also involve the shoulders, hips, and other peripheral joints. Its manifestations include AS, reactive arthritis, arthropathy of inflammatory bowel disease, psoriatic arthritis, undifferentiated spondyloarthropathy, and juvenile-onset AS. In addition to inflammation of the spine (including the sacroiliac joint), this disease is characterized by the absence of RF, the tendency for familial aggregation, an association with the HLA-B27, inflammation around the entheses (the site of tendon insertion into bone), uveitis, urethritis, and psoriatic skin lesion. AS is the prototypic form and is frequently encountered by physiatrists.

The prevalence of AS has been estimated to be 0.1% to 1.4% of the population (0.2%–0.5% in the United States), and the male-to-female ratio is approximately 2:1 to 3:1 according to modified New York criteria. The prevalence of AS generally mirrors the frequency of HLA-B27 in the population; it is approximately 5% to 6% in people who test positive for HLA-B27. The peak age of onset is between 20 and 30 years.

Clinical Characteristics

The most common presentation is chronic inflammatory back pain. The pain usually starts at the buttock or lower back level, with possible radiation to the posterior thigh. It is a dull pain, insidious in onset and chronic (lasting >3 months). The pain is worse in the later part of the night and the early morning, with morning stiffness lasting more than 30 minutes and often hours. It can be relieved with exercise or activity and worsened with rest (Table 32.4). It is usually improved with the use of NSAIDs. At first, buttock or lower back pain may be unilateral and intermittent; however, within a few months, the pain becomes bilateral and persistent, and the lower back becomes stiff. Gradually, pain and stiffness may ascend from the lower back to the middle back and then to the upper back and neck region. The mobility of the spine can be measured using the modified Schober test, the occiput-to-wall distance, and/or chest expansion (Video 32.22).

TABLE 32.4 Differentiation of Inflammatory vs. Mechanical Low Back Pain

	Inflammatory Pain	Mechanical Pain
Age of onset	<40 years	Middle to old age
Type of onset	Insidious	Acute or insidious
Symptom duration	>3 months	Mostly <3 months
Night pain	Common	Rare
Effect of exercise	Improved	Usually worse (acute pain)
Sacroiliitis	Positive	Negative
Low back mobility	Limited in all motions	Limited in one or some motions
Neurological deficits	Unusual	Possible
CRP or ESR	Increase in active stage	Normal

CRP, C-reactive protein; *ESR*, erythrocyte sedimentation rate.

Patients with AS may experience arthritis outside of the spine, and peripheral arthritis occurs in approximately 35% to 50% of patients with AS over the course of the disease.[38] The most commonly affected joints, in order of frequency, are the shoulders, hips, and knees. Involvement of the ankles, sternoclavicular joints, and temporomandibular joints is also reported. Hip involvement is present in 25% to 35% of patients with AS and is associated with a high degree of physical disability and a poor prognosis. Enthesitis occurs in approximately 40% to 70% of patients with AS at some time during the course of the disease. The most common location of enthesitis in patients with AS is the calcaneal attachments of the Achilles tendon. Other locations include the calcaneal attachments of the plantar fascia, shoulders, costochondral junctions, sternoclavicular and manubriosternal joints, and superior iliac crest. Dactylitis (or "sausage digits") is characterized by diffuse swelling of the toes or fingers and is present in 6% to 8% of patients with AS.

Patients with AS may have extraarticular comorbidities, including anterior uveitis, inflammatory bowel disease, psoriatic skin lesions, aortic regurgitation, cardiac conduction disturbance, restrictive lung disease, apical pulmonary fibrosis, immunoglobulin nephropathy, or renal amyloidosis. Among these conditions, anterior uveitis (or iritis) is the most common, occurring in 25% to 40% of patients with AS. Anterior uveitis presents as acute eye pain, redness, photophobia, increased lacrimation, and blurring of vision. Treatment of acute uveitis should begin as early as possible to avoid complications such as glaucoma or vision loss.

Recent data suggest that the prevalence of osteoporosis and vertebral fractures in patients with AS is 25% and 10%, respectively. Approximately 65% of fractures are associated with a neurological deficit. Spinal cord injury is 11 times more common in

patients with AS than in the general population and affects the cervical spine more often than the thoracic and lumbar spine. Spontaneous subluxation of the atlantoaxial joint and cauda equina syndrome may also occur in patients with AS.

Laboratory findings are generally nonspecific for AS. An elevated ESR or CRP is present in approximately 50% to 70% of patients who have AS with active disease. Normochromic normocytic anemia is occasionally seen, and HLA-B27 is present in 90% to 95% of patients of European ancestry with AS.

Imaging

Plain radiography and MRI are the principal imaging techniques used to evaluate patients with AS, especially for sacroiliac joints. In general, radiographic changes take a few years to develop. Radiographic findings of sacroiliitis (pelvis, anteroposterior view) include narrowing of the joint space, sclerosis, erosion, and bony ankylosis. These have been divided into five grades (Box 32.2, Fig. 32.9). Other findings on plain radiograph of the pelvis include erosions and osteitis at the ischial tuberosity, femoral trochanter, iliac crests, and symphysis pubis. Radiographic changes of the spine may include squaring of the vertebral bodies, syndesmophytes (Fig. 32.10), ankylosis of the facet joints, and calcification of the anterior longitudinal ligament.

If a plain radiograph of the pelvis does not fulfill the criteria for sacroiliitis but the suspicion of AS remains high, the next step is MRI. In the case of active sacroiliitis, MRI can show bone marrow edema in the bones adjacent to the affected joints, as shown in short tau inversion recovery images (Fig. 32.11) or T2-weighted images with fat suppression (not seen in T1-weighted images). MRI has been shown to be more sensitive than conventional radiography, bone scintigraphy, and computed tomography scans in the detection of sacroiliitis.

Diagnostic Criteria

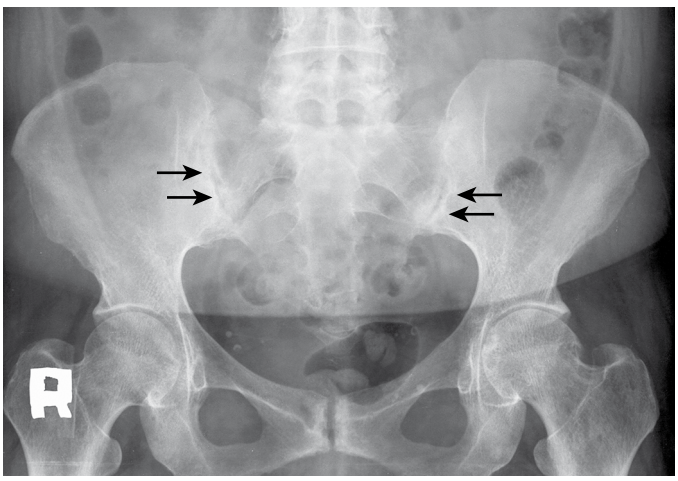
Diagnosis (or classification) of classic AS is based on the 1984 modified New York criteria, which requires the symptoms of lower back pain, limited lumbar spine motion and chest expansion, and plain radiographs showing sacroiliitis (Box 32.3).[20] A stage at which the clinical symptoms and signs of patients fulfill the clinical criteria of AS but do not suggest sacroiliitis on radiographs and may reveal sacroiliitis on MRI is called nonradiographic axial spondylarthritis (nr-axSpA). The proportion of patients with nr-axSpA who experience disease progression is unknown; however, factors related to disease progression include male sex, HLA-B27, disease duration, the extent of inflammation on MRI, and persistent systemic inflammation.[38] Current treatment strategies for patients with nr-axSpA with high disease activity (symptoms and signs) are the same as those for patients with established AS.[42]

Management of Ankylosing Spondylitis

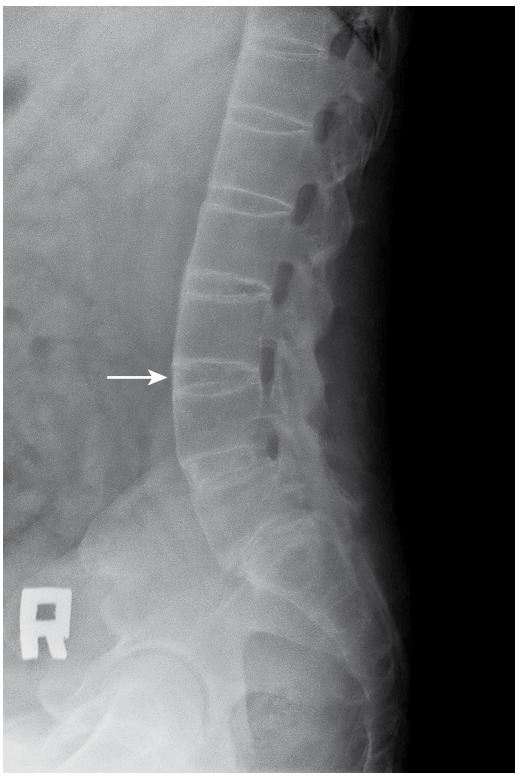
The management goals for patients with AS include (1) relieving symptoms, (2) maintaining functional abilities, (3) preventing progressive structural damage to the spine and joints, and (4) minimizing comorbidities that affect areas beyond the spine and joints. Effective management requires a combination of nonpharmacological and pharmacological treatments. This includes participating

• BOX 32.2 Radiographic Grading of Sacroiliitis

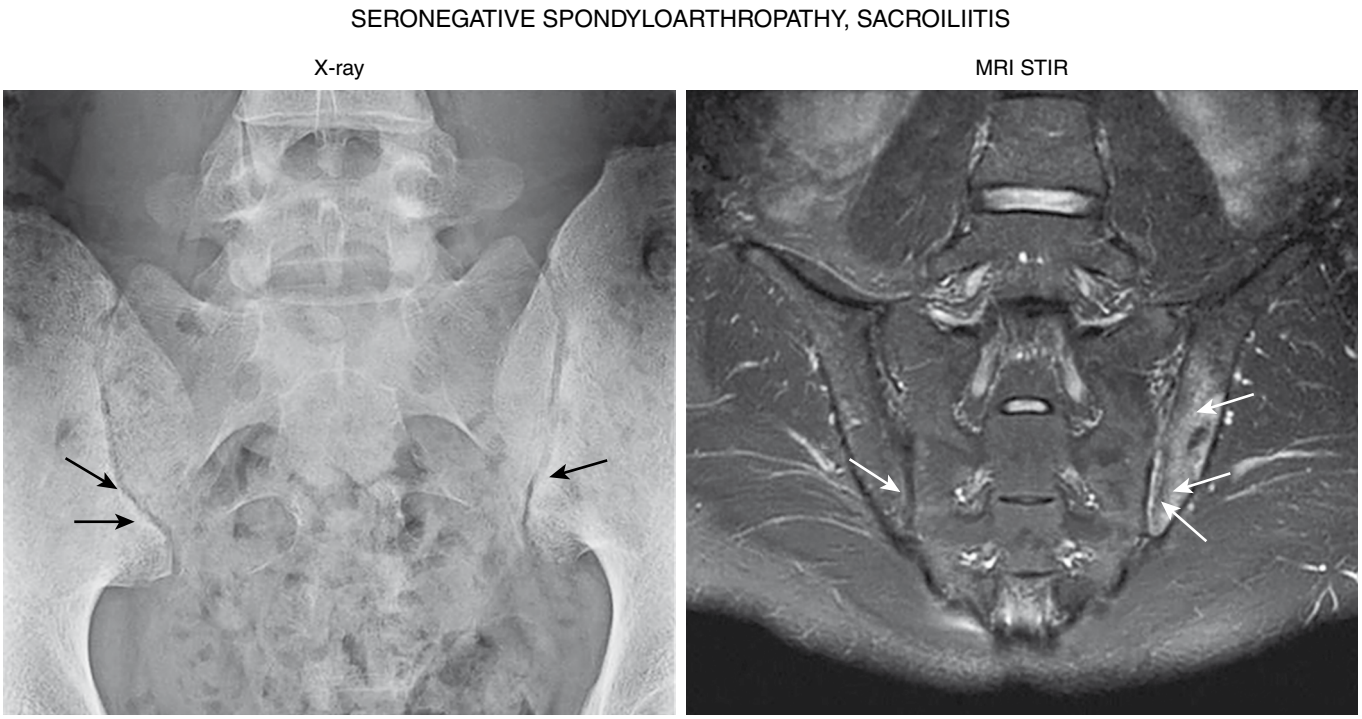
Grade 0: Normal
Grade 1: Suspicious changes
Grade 2: Minimal abnormality—small, localized areas with erosions or sclerosis without alteration in the joint width
Grade 3: Unequivocal abnormality—moderate or advanced sacroiliitis with erosions, sclerosis, widening, narrowing, or partial ankylosis
Grade 4: Total ankyloses



• **Fig. 32.9** Anteroposterior view of the pelvis in a patient with ankylosing spondylitis shows grade 3 sacroiliitis bilaterally *(arrows).*



• **Fig. 32.10** Lateral view of the thoracolumbar spine in a patient with ankylosing spondylitis shows syndesmophytes *(arrow)* and a bamboo spine.



• **Fig. 32.11** Comparison of anteroposterior view of the pelvis *(left)* and magnetic resonance imaging (*MRI*; short tau inversion recovery *[STIR]*, *right*) of the sacroiliac joint in a patient with early ankylosing spondylitis. MRI shows left sacroiliitis *(white arrows),* which is not shown on the plain film *(black arrow).* (Courtesy Dr. Hung-Ta Wu.)

• BOX 32.3 1984 Modified New York Criteria for Ankylosing Spondylitis

Clinical Variables
- Inflammatory back pain >3 months
- Limitation of motion of the lumbar spine in both the sagittal and frontal planes
- Limitation of chest expansion relative to normal values

Radiological Variables (Plain Radiographs)
- Sacroiliitis grade ≥2 bilaterally
- Sacroiliitis grade 3–4 unilaterally

Definite Diagnosis
- At least one clinical variable plus at least one radiological variable

in a rehabilitation program (see later), undergoing medication therapies, and, if necessary, surgery. It is also essential for patients to receive education about their disease, engage in regular exercise, and cease smoking to improve their outcomes.

The treatment goal should be to achieve clinical remission or to ensure that musculoskeletal symptoms and extraarticular manifestations are inactive. A treatment target is defined as the lack of clinical and laboratory evidence indicating significant disease activity. This should be determined based on clinical symptoms, signs, and acute phase reactants.[35]

Nonsteroidal Antiinflammatory Drugs

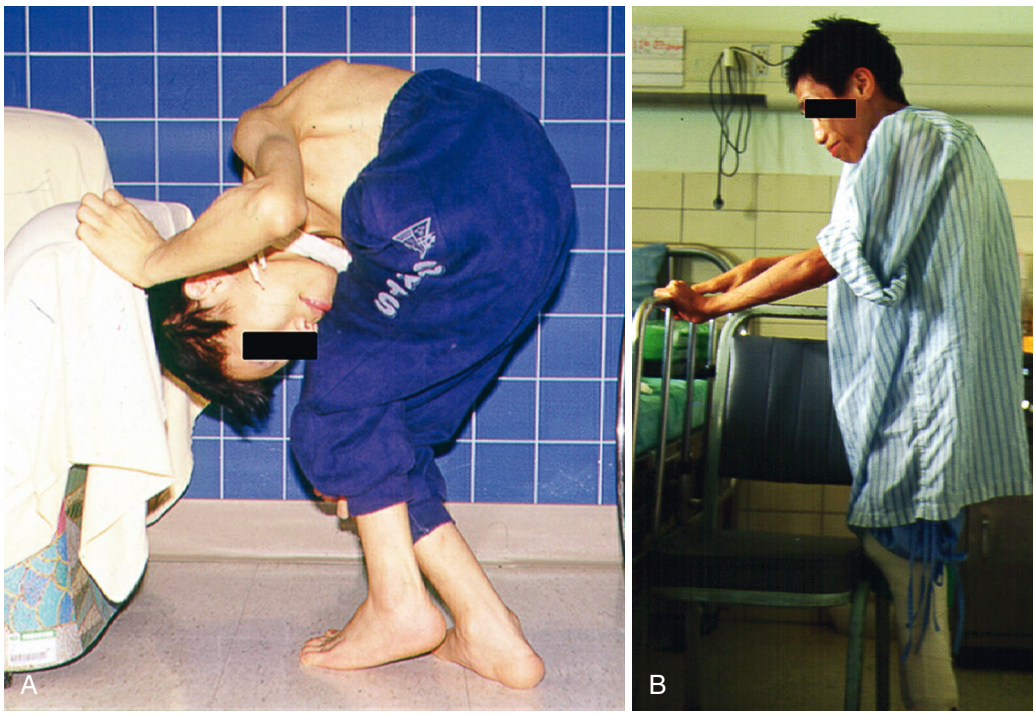
Patients experiencing pain and stiffness should consider NSAIDs as their primary medication. The dosage should be increased to the maximum tolerated level, after carefully weighing the potential risks against the benefits. For patients who benefit from NSAIDs, continued use is recommended. The dosage should be adjusted based on the severity of their symptoms. Any NSAID, including those that inhibit COX-2, can be effective for treatment. However, other pain relievers, such as paracetamol and opioid-like drugs, usually do not help and are often not well tolerated.[42]

Glucocorticoids

Local corticosteroid injections may be recommended for persistent peripheral arthritis, sacroiliitis, or enthesitis, except in the Achilles, patellar, or quadriceps tendons because of the risk of tendon rupture. Injections at other locations, such as the greater trochanter, pelvic rim, and attachment point of the plantar fascia, are considered relatively safer. Short-term use of systemic corticosteroids may be helpful in patients with purely axial disease for symptomatic control; however, their long-term use is discouraged.

Disease-Modifying Antirheumatic Drugs

Patients with purely axial disease should not use csDMARDs because of their minimal effectiveness. For patients experiencing peripheral arthritis, sulfasalazine is the only recommended csDMARD. Additionally, for patients who continue to have high disease activity despite NSAID treatment, bDMARDs should be considered. According to the 2022 Assessment of Spondylarthritis International Society–EULAR recommendations, for patients with persistently high disease activity, consideration should be given to using bDMARDs, such as TNFi, interleukin-17 inhibitors (IL-17i), or JAKi. All these drug classes are highly effective for reducing inflammation and relieving symptoms, halting bone destruction, slowing radiographic progression, and potentially modifying the disease. However, they cannot prevent the ossification of already damaged



• **Fig. 32.12** Severe deformity of the spine in a 29-year-old patient with ankylosing spondylitis. (A) Before surgery. (B) After surgery. Substantial improvement after spinal osteotomy (90-degree correction) and total hip replacement (60-degree correction). (Courtesy Dr. Ing-Ho Chen.)

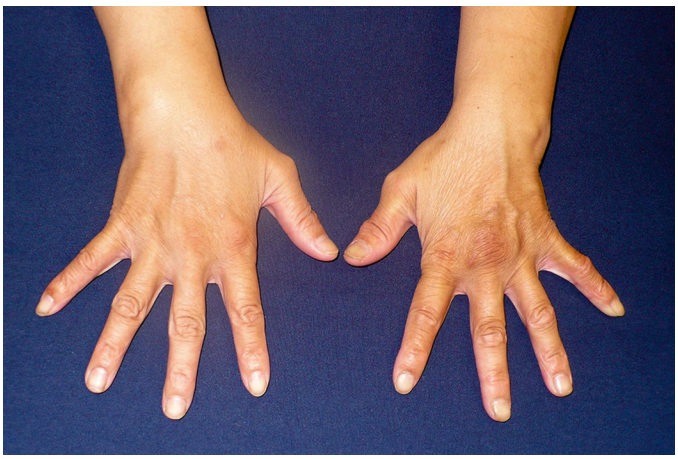
bone. The current clinical practice typically involves starting treatment with either a TNFi or an IL-17i. If a patient has a history of recurrent uveitis or active inflammatory bowel disease, a TNFi should be the preferred treatment. For patients with significant psoriasis, an IL-17i may be the better choice.[35]

If a patient does not respond well to a bDMARD or a JAKi, the physician should reevaluate the patient's condition. It is wise to reconsider whether the initial diagnosis was accurate. If the diagnosis is confirmed and the initial treatments are definitely ineffective, it is recommended to consider switching to another bDMARD, such as a TNFi or IL-17i, or a JAKi. Additionally, if a patient has been in sustained remission for at least 6 months, gradually reducing the dosage of a bDMARD can be considered.[35]

If a patient experiences refractory hip pain or disability, along with radiographic evidence of structural damage, total hip arthroplasty should be considered, irrespective of the patient's age. For those with severe and disabling spinal deformities, spinal corrective osteotomy (Fig. 32.12) may be an option. Additionally, if there is a significant change in the course of a patient's disease, potential noninflammatory causes, such as a spinal fracture, should be explored. Appropriate evaluations, including imaging tests, should be conducted. Patients with AS who have spinal fusion or advanced spinal osteoporosis should avoid spinal manipulation that involves high-velocity thrusts.

Psoriatic Arthritis

Psoriatic arthritis is a member of the seronegative spondyloarthropathy family and is defined as an inflammatory arthritis associated with psoriasis. It is usually negative for RF and affects both sexes equally. The prevalence of psoriatic arthritis is approximately 1 to 2 per 1000, and the incidence is approximately



• **Fig. 32.13** Swelling of right wrist and left third metacarpophalangeal joint in a patient with psoriatic arthropathy.

6 per 100,000 per year. It is estimated that 4% to 30% of patients with psoriasis have psoriatic arthritis.

Patients with psoriatic arthritis present with symptoms and signs of joint, entheseal, and spinal inflammation. They usually complain of pain and stiffness in the affected joints. Morning stiffness often lasts for longer than 30 minutes; it is accentuated by prolonged immobility and is relieved by physical activity. Moll and Wright have described five clinical patterns of psoriatic arthritis: (1) asymmetrical oligoarthritis (Fig. 32.13), (2) polyarthritis, (3) predominant DIP joint involvement, (4) destructive arthritis (arthritis mutilans), and (5) predominant spondylarthritis. Polyarthritis is the most common (63%), followed by oligoarthritis

• BOX 32.4 Classification for Psoriatic Arthritis Criteria

1. Inflammatory musculoskeletal disease (peripheral arthritis, spondylitis, or enthesitis) *and*
2. At least 3 points from the following:
   - Current psoriasis (2 points), a personal history of psoriasis (1 point), or a family history of psoriasis (1 point)
   - Typical nail lesions (1 point): onycholysis, pitting
   - Dactylitis (1 point): present or past, documented by a rheumatologist
   - Negative rheumatoid factor (1 point)
   - Juxtaarticular bone formation (1 point): on hand or foot radiographs

From Taylor W, Gladman D, Helliwell P, et al. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum.* 2006;54:2665-2673.

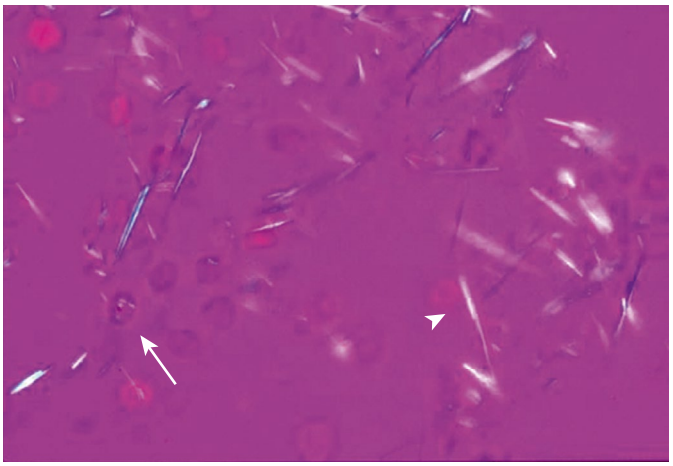
(13%), and predominant DIP involvement (<5%). Although spinal involvement is found in 40% to 70% of patients with psoriatic arthritis, spinal involvement alone occurs in only 2% to 4% of patients with psoriatic arthritis. Some patients present with more than one pattern, and the classification is not fixed. The pattern of disease may fluctuate. Other musculoskeletal features include dactylitis (sausage digit, resulting from inflammation of the soft tissue and joints), enthesitis, tenosynovitis, nail lesions (pits and onycholysis), and pitting edema in the hands or feet.

The most typical radiological finding is the coexistence of erosive changes and new bone formation in the DIP joints. Other changes include fluffy periostitis with new bone formation at the site of enthesitis, lysis of the terminal phalanges, gross destruction of isolated joints, "pencil in cup" appearance, and the presence of both joint lysis and ankylosis. MRI may be more sensitive than conventional radiography in detecting articular, periarticular, and soft tissue inflammation. In recent years, the Classification of Psoriatic Arthritis study developed new criteria for patients with psoriatic arthritis (Box 32.4). Patients with inflammatory musculoskeletal disease can be classified as having psoriatic arthritis if they have a total of at least three of the five categories.

Treatment for psoriatic arthritis includes therapy for both skin and joint disease. The treatment usually starts with NSAIDs. If the arthritis is not well controlled by the NSAIDs, the next step is a nonbiological DMARD, preferably MTX, leflunomide, or sulfasalazine. For patients whose joint condition does not improve after 3 months of treatment, a TNFi may be added, usually etanercept, adalimumab, or infliximab. TNFi has been demonstrated to reduce the radiographic progression of disease; the effect is superior to that of MTX. In case of intolerance or inadequate response to TNFi or in patients with severe skin involvement, IL-17i or even IL-12/23i may be considered.[40] The use of oral corticosteroids in patients with psoriatic arthritis should be avoided. Joint replacement may be suggested when psoriatic arthritis causes damage that limits movement and impairs function.

Gout and Other Crystal-Related Arthropathies

Crystal-related arthropathies are disorders characterized by the deposition of minerals in the joints. These crystals include monosodium urate (MSU), calcium pyrophosphate dihydrate, hydroxyapatite, and others. Intraarticular crystals can lead to both acute and chronic inflammation.



• **Fig. 32.14** Monosodium urate crystals from a joint with acute gouty arthritis, viewed under polarized light microscopy. Note that both intra- *(arrow)* and extracellular *(arrowhead)* crystals are present (×1000, with a first-order red compensator).

Gout, the most common crystal-related arthropathy and the most prevalent inflammatory arthritis worldwide, occurs when serum urate levels become excessively high. This condition leads to the formation of MSU crystals (Fig. 32.14) in the joints and surrounding tissues, triggering gout flares. Gout prevalence ranges from 0.9% to 6.1% in various countries. It is more common in males than in females. In females, gout flares usually occur after menopause. Often, patients with gout are not diagnosed early because the condition can affect areas such as the Achilles tendon and may be mistaken for tendinitis or traumatic arthritis after trauma. Therefore physiatrists should be familiar with gout.

Clinical Characteristics

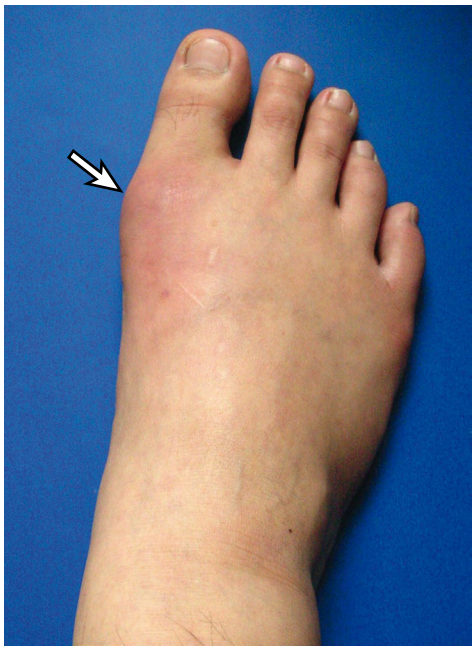
Gout occurs after long-term hyperuricemia, defined as a serum urate level greater than 7 mg%. The likelihood of developing gout correlates well with serum urate levels and the duration of hyperuricemia. However, most people with hyperuricemia remain asymptomatic throughout their lives, with only about 10% eventually developing gout.

A gout flare is characterized by sudden pain, erythema, swelling, and warmth over a joint or bursa. Initially, it often affects a single joint, primarily in the lower limbs, with the first MTP joint being the most frequently affected (Fig. 32.15). Gout flares most commonly occur overnight and in the early morning. The pain can be so severe that even light touch or pressure is intolerable, significantly impairing the patient's ability to walk or use of the affected joint. Symptoms typically peak within 24 hours and resolve within 14 days, with no symptoms between episodes.

Tophaceous gout, characterized by the deposition of urate crystals primarily in subcutaneous tissues and joints, can manifest when gout management is suboptimal or neglected. These urate deposits, or tophi, may lead to bone erosion, articular deformities, and, in advanced cases, impaired joint mobility (Fig. 32.16). The principal determinant of tophaceous formation is hyperuricemia, while the chronicity of gout serves as a secondary but crucial factor.

Comorbidities

Gout is associated with several cardiovascular risk factors, including hypertension, hyperlipidemia, obesity, and diabetes. It is also an independent risk factor for all-cause mortality. Among these causes, cardiovascular disease is the most common, while chronic kidney disease (CKD) is another significant cause.[2] Patients with early onset or the presence of tophi are more likely to exhibit comorbidities, which are markers of gout severity and should prompt earlier treatment.



• **Fig. 32.15** A gout flare is characterized by sudden pain, erythema, swelling, and warmth over the first metatarsophalangeal joint *(arrow)*.

Management

Patients should be informed about comorbidities related to gout and advised to lead a healthy lifestyle, which includes avoiding alcohol and heavy meals, controlling body weight, and exercising regularly. It is essential for the management of gout to integrate screening and treatment of associated comorbidities. Although dietary modifications may only yield small changes in serum urate levels, certain dietary factors can trigger flares.

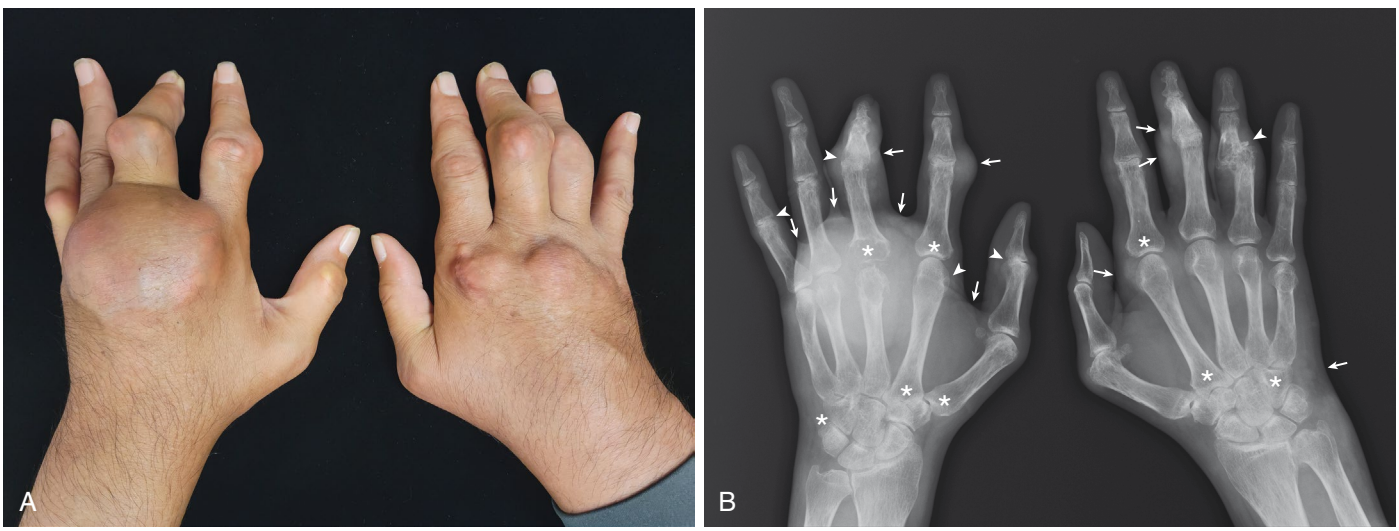
Gout Flare

Acute gout flares should be treated as early as possible with NSAIDs, colchicine, or glucocorticoids (oral or intraarticular) as first-line therapies. NSAIDs should be prescribed at a high dose initially unless contraindicated. Different types of NSAIDs do not exhibit significant differences in effectiveness. Colchicine should be used at lower doses in patients with CKD and the elderly. If a patient does not respond well to or is contraindicated for NSAIDs, glucocorticoids (oral, intraarticular, or intramuscular) can be used as first-line therapy.

During a gout flare, the serum urate level should not be altered, as both increases and decreases may precipitate or prolong the flare. Therefore urate-lowering drugs should not be added if the patient was not taking them before the flare. Conversely, if the patient was already on such medication, it should be continued at the same dose. Prophylactic therapy should be delayed until the complete resolution of all signs of inflammation. Dietary therapies are ineffective in managing symptomatic outcomes.

Urate-Lowering Therapy

According to the 2020 ACR gout guideline, urate-lowering therapy (ULT) for prophylaxis is strongly recommended for patients with any of the following: one or more subcutaneous tophi, radiographic damage attributable to gout, or two or more gout flares annually. ULT is also advised for patients experiencing their first flare if they have moderate to severe CKD (stage ≥3), a serum



• **Fig. 32.16** A 46-year-old male has been experiencing gout for 16 years. He has not previously taken urate-lowering medication. Subcutaneous tophi have developed on his hands, feet, and knee joints for the past 5 years. (A) There are several subcutaneous tophi of various sizes on the hand, resulting in joint restriction and finger deformity. (B) The radiography image demonstrates soft tissue tophi *(arrow),* extensive bone erosions *(arrowhead),* and intraosseous bone lesions *(asterisk).*

urate level above 9 mg%, or urolithiasis.[17] For patients on ULT, the serum urate level should be kept below 6 mg% to prevent recurrent flares. For those with tophi, the target serum urate level is below 5 mg% to promote faster dissolution of crystals. However, maintaining serum urate levels below 3 mg% chronically is not recommended. Serum urate levels should be maintained for life.

Allopurinol, a xanthine oxidase inhibitor, is the first-line drug for all patients, including those with moderate to severe CKD (stage ≥3). It is favored because of its efficacy, tolerability, safety, and lower cost. Allopurinol should be started at a low dose (50–100 mg daily) to minimize side effects and potential flares, then increased in 100-mg increments every 2 to 4 weeks until the serum urate target is achieved. Doses often exceed 300 mg/day, up to a maximum of 800 mg/day. In patients with renal impairment, both the starting dose and increments should be lower (50 mg), though the serum urate target remains the same. Side effects include skin rash and gastrointestinal discomfort, although these are generally rare. However, severe cutaneous adverse reactions, as well as liver and renal toxicity, are potentially life threatening but rare. Allopurinol should be avoided in patients carrying the HLA-B*5801 allele because of a significantly increased risk of adverse events. If allopurinol is contraindicated or not tolerated, febuxostat can be used as a second-line alternative.

Uricosuric agents such as sulfinpyrazone, probenecid, and benzbromarone may be used in patients who are resistant to or intolerant of allopurinol. Benzbromarone is preferred and can be administered when the estimated glomerular filtration rate is as low as 20 mL/min. Treatment combining allopurinol and benzbromarone has shown to be more effective than either agent alone.

ULT does not decrease the risk of acute flare in the first 6 months. Therefore colchicine 0.5 mg once or twice daily should be considered as prophylaxis against acute flares and continued for up to 6 months. When a patient cannot tolerate colchicine, a low-dose NSAID can be used as an alternative drug.

Rehabilitation

Regular aerobic exercise can help lower serum urate levels, reduce inflammation, maintain body weight, and improve overall metabolism, thereby decreasing the risk of comorbidities. However, patients with gout should avoid exercising during a flare to prevent worsening pain.

During a gout flare, it is beneficial to elevate and rest the affected joint. Applying an ice pack to the painful area for 20 minutes several times a day may help decrease hyperemia. This can ease pain and inflammation, reducing the severity of the flare. Do not use heat on an already inflamed joint because it can worsen the symptoms.

Systemic Lupus Erythematous

SLE is a chronic autoimmune disease that can affect many organ systems. It predominantly affects females of child-bearing age, with the peak age between 15 and 40 years. The classic triad of fever, joint pain, and rash in a female of child-bearing age should prompt investigation into an SLE diagnosis. The 2019 EULAR/ACR classification criteria for SLE require a positive antinuclear antibody (ANA) test at least once as a mandatory first step. This is followed by additional criteria that are categorized into seven clinical areas, such as musculoskeletal and neuropsychiatric, and three immunological domains that include antiphospholipid antibodies, complement proteins, and SLE-specific antibodies.[4]

Musculoskeletal Manifestation

Joint involvement is the most common feature in patients with SLE, observed in up to 95% of cases. Joint symptoms often serve as the initial symptom of SLE and are present in about 75% of patients at the time of diagnosis. It most typically presents as symmetrical polyarthritis, most commonly involving the MCP, PIP, and knee joints. Any young female patient with arthritis should be evaluated for a possible diagnosis of SLE.

The new classification criteria by EULAR/ACR in 2019 recommended that joint involvement is either (1) synovitis involving two or more joints characterized by swelling or effusion or (2) tenderness in two or more joints and at least 30 minutes of morning stiffness.[4] Unlike in RA, the arthritis of SLE is typically nonerosive. Approximately 5% to 40% of patients with SLE may develop Jaccoud arthropathy, characterized by ulnar deviation of the fingers, swan-neck deformities, and other joint deformities that are usually reducible and attributed to joint laxity.[39]

Neuropsychiatric Manifestation

Neurological and psychiatric features of SLE refer to disorders complicated with SLE (NPSLE), which are severe but potentially treatable. NPSLE may be the first clinical manifestation of SLE, with a 10-fold increase in the mortality rate compared with those without NPSLE; therefore clinicians should be aware.

Patients may experience a variety of neuropsychiatric symptoms that are often nonspecific and not particularly useful for diagnosis. These symptoms can be focal or diffuse, including headache, anxiety, depression, cognitive dysfunction, seizures, psychosis, mononeuritis multiplex, myelitis, peripheral or cranial neuropathy, and acute confusion state. At times, it may be challenging to differentiate whether the neuropsychiatric findings are a result of SLE or other causes. In 2019, the new classification criteria for SLE proposed by the EULAR/ACR defined only delirium, psychosis, and seizures as neuropsychiatric manifestations to improve diagnostic specificity for SLE.[4]

Cerebrovascular disease, neuropathies, delirium, and seizure disorders are the most frequent manifestations of NPSLE. NPSLE commonly occurs early, within the first or second year after SLE diagnosis. Approximately 28% to 40% of NPSLE-related manifestations develop before or at the time of SLE diagnosis. The pathogenesis of NPSLE is multifactorial, with collusion of ischemic and inflammatory mechanisms. Autoantibodies play key roles in mediating both mechanisms. Most cerebrovascular disorders in NPSLE are caused by antiphospholipid antibodies, which are associated with the development of thrombosis.[19]

Transverse myelitis, characterized by paraplegia and sensory impairment, is a rare but severe complication of SLE. This condition results from inflammatory demyelination or necrosis of the spinal cord, triggered by various autoimmune mechanisms. Symptoms typically develop rapidly, often reaching their peak severity within the first 24 hours. The condition may start with general symptoms such as fever, headaches, and vomiting, and progress to paresthesia and weakness in the lower limbs. Transverse myelitis usually manifests within the first 5 years after the onset of SLE; in about half of these cases, it may be the initial sign of the disease. There is frequent involvement at the thoracic level, where smaller-caliber vessels make the region more vulnerable to thrombosis. Specifically, the thoracic segment, particularly from T5 to T8 and most notably T7, is most frequently affected.[11]

Septic Arthritis

Septic arthritis refers to an infection in a joint caused by bacteria, viruses, mycobacteria, or fungi. Bacteria are the most common cause, typically affecting elderly people and very young children. Bacterial arthritis can lead to severe morbidity and mortality, and delayed or inadequate treatment may cause irreversible joint destruction.

Risk Factors

The most significant risk factor is preexisting joint disease. Patients with RA, OA, gout, diabetes mellitus, recent trauma, recent joint surgery, skin infections, those older than 80 years, and those using immunosuppressive medications all face high risks. Among these factors, RA is the most common and is associated with worse outcomes.

Clinical Presentation

The most common symptoms include the acute onset of joint pain, erythema, heat, and restricted joint mobility. Fever is detected in only about 40% to 60% of patients, indicating that an increase in temperature is not a prerequisite for diagnosis. Sweats or rigors are less common. Septic arthritis typically affects a single joint; however, multiple joints are involved in 20% of cases. In adults, the knee is the most frequently affected joint, involved in almost half of all cases, followed by the hip, shoulder, elbow, and ankle. In children, the hip is more frequently affected.[23] Infections of axial joints, such as the sternoclavicular or sacroiliac joints, are more common in patients with a history of intravenous drug use.

Microbiology

The most common causative organism across all age categories and risk groups is *Staphylococcus aureus*, followed by *Streptococcus*. Gram-negative organisms are less common. In sexually active adolescents and young adults, *Neisseria gonorrhoeae* should be considered. Intravenous drug users are susceptible to infections from a mix of bacterial organisms as well as fungal infections and unusual pathogens.

Diagnosis

A positive synovial fluid culture is key to a definitive diagnosis. Synovial fluid analysis typically shows a highly elevated white blood cell (WBC) count of over 50,000/μL and a polymorphonuclear leukocyte count of over 90%. Gram staining provides immediate results; however, its sensitivity is low, while its specificity is nearly 100%. Therefore Gram staining alone is unreliable for early decision-making in patients requiring urgent surgical drainage and washout. Lower WBC counts, a negative Gram stain, or subsequently negative synovial fluid cultures do not rule out the diagnosis but make it less likely. ESR and CRP levels are each more than 90% sensitive for detecting septic arthritis, helpful for ruling out the condition. Procalcitonin shows greater specificity than CRP and is considered the most sensitive systemic marker for distinguishing septic from aseptic arthritis.[43] Although WBC count, ESR, and serum CRP levels are nonspecific for bacterial arthritis, they can be used to monitor response to treatment.

Infection can be introduced into a joint either through hematogenous spread or direct inoculation, such as from penetrating trauma or therapeutic joint injections. Hematogenous spread is the most common route. Penetrating trauma, meanwhile, is the predominant method of introducing septic arthritis in the small joints of the hands and feet. Because pathogenesis may be hematogenous, blood cultures are positive in 25% to 50% of patients with septic arthritis.

The most critical differential diagnosis for bacterial arthritis is gout, which can present with similar symptoms. Gout is diagnosed by polarizing microscopic visualization of MSU crystals (see Fig. 32.14). However, the presence of gout does not exclude the possibility of a concurrent bacterial joint infection because both conditions can coexist.

Management

The cornerstone of treatment is the prompt removal of intraarticular pus and appropriate antibiotic therapy.[36] Needle aspirations should be repeated daily until effusions resolve and cultures return negative results. Arthroscopic drainage can provide rapid recovery and low morbidity. Because septic arthritis can rapidly destroy joint structures, broad-spectrum antibiotics are usually necessary until culture data become available. Vancomycin is often the initial antibiotic choice. For immunocompromised patients or intravenous drug users, vancomycin plus a third-generation cephalosporin should be administered. Treatment typically continues for up to 6 weeks, starting with intravenous antibiotics for the first 2 weeks followed by oral antibiotics.[36]

Rehabilitation

Patients are recommended to wear a splint to maintain the functional position of the affected joint to reduce pain and tissue damage. However, wearing a splint for long periods of time can limit the ROM and weaken the muscles around the affected joint. Therefore patients should not wear the splint continuously and should be passively mobilized, followed by active mobilization once their pain has decreased. Subsequently, it is crucial to implement aggressive rehabilitation to prevent muscle wasting and joint contractures.[36]

Rehabilitative Management of Rheumatic Diseases

People with rheumatic diseases report experiencing pain, fatigue, reduced mobility, impaired hand function, stress, and anxiety. These symptoms contribute to decreased participation in daily activities, including work and leisure. Rehabilitation of the patient with a rheumatic disease can pose a challenge because of the need to address various biopsychosocial aspects. The goals of a comprehensive rehabilitation program, consisting of patient-centered services, are listed in Box 32.5.

Rehabilitation Evaluation of Patients with Rheumatic Diseases

A holistic examination assists professionals in appropriate goal setting and treatment planning. According to the World Health Organization framework for measuring health and disability, the ICF outlines the key components of the rehabilitation evaluation for patients with rheumatic diseases (Box 32.6). For specific evaluation purposes and target populations, clinical practitioners may select from available standardized assessment tools (Table 32.5).

Rehabilitation Intervention of Patients With Rheumatic Diseases

In addition to the medical treatments mentioned earlier, nonpharmacological interventions should also be included. Recognizing the importance of holistic care, the 2022 ACR Guideline for Exercise, Rehabilitation, Diet, and Additional Integrative Interventions for Rheumatoid Arthritis and the 2019 ACR Guideline for the Management of Osteoarthritis of the Hand, Hip, and Knee highlight the significance of these interventions.[16,28] These nonpharmacological approaches include exercise, appropriate physical modalities, orthoses, patient education (i.e., joint protection techniques and activity pacing), functional activities or mobility training, assistive devices, adaptive equipment, environmental modifications, and vocational rehabilitation. All of these should be considered for patients.

These interventions are typically delivered through comprehensive rehabilitation programs, including occupational therapy, physical therapy, and/or hand therapy. An interprofessional team-based approach to rheumatic diseases management is necessary. It should begin at an early stage to help patients identify important management strategies to prevent unnecessary impairment or functional decline. The rehabilitation intervention must be individualized and monitored with periodic reevaluations.

• BOX 32.5 Rehabilitation Goals for Rheumatic Diseases

1. To alleviate pain and reduce inflammation
2. To improve and maintain joint function and mobility
3. To enhance muscle strength and endurance
4. To restore and enhance hand function to improve daily activities
5. To minimize fatigue and improve overall energy levels
6. To facilitate self-management and successful adaptation to the disease
7. To maintain participation in daily life and social roles, including work and leisure activities
8. To reduce stress and anxiety to achieve sense of self-efficacy and well-being

Exercise

Consistent engagement in exercise is strongly recommend over no exercise.[16,28] The purposes of exercise programs for patients with arthritis are to (1) increase and maintain ROM, (2) improve muscle strength and endurance, (3) increase aerobic capacity, (4) increase bone density, (5) improve functional ability, and (6) improve psychological function. In addition, through exercise programs, the joints of patients with arthritis can be made to function better biomechanically. With improvements in aerobic capacity, cardiovascular morbidity and mortality are reduced.[31]

Before exercise therapy is recommended, it is important to evaluate the patient's joint and periarticular conditions as well as cardiopulmonary function and other systemic features. Common exercise interventions for patients with arthritis include mobilization exercise, strengthening exercise, aerobic (or conditioning) exercise, and recreational exercise.[10]

Mobilization exercise can be performed by ROM exercise, stretching exercise, proprioceptive neuromuscular facilitation (PNF) technique, or joint mobilization. In actively inflamed joints, gentle active or active-assistive ROM through the possible range should be performed. Passive stretching should be applied with extreme caution because it may be associated with rupture of the joint capsule with large effusion or may induce an inflammatory response in otherwise controlled joints. While inflammation

• BOX 32.6 Key Components of the Rehabilitation Evaluation of Patients With Rheumatic Diseases

Disease and Comorbidities
Current diagnosis
Current disease activity
Cardiovascular comorbidities
Depression
Other

Body Function and Structure
Pain
Tender/swollen joint count
Nodules and nodes
Range of motion
Deformities
Muscle strength
Fatigue

Activities: Execution of a Task or Action
Reaching
Manipulation
Hand function
Mobility
Ambulation

Participation: Involvement in Life Situations
Daily routines
Self-care
Domestic life
Education
Leisure
Work

Environmental Factors: Physical, Social, and Attitudinal World
Home physical environment
Workstation setup
Assistive devices and mobility aids
Interpersonal environment
Social support
Societal policies and regulations

Personal Factors: Features of Individual and Not Part of Health Condition
Personal identity
Occupation
Rehabilitation goals
Beliefs about arthritis
Coping style
Self-efficacy
Religious and spiritual beliefs

Modified from Toledo SD, Trapani K, Feldbruegge. Rheumatic diseases. In: Braddom RL, ed. *Physical Medicine and Rehabilitation.* 4th ed. WB Saunders; 2010:769-784.

TABLE 32.5 Common Impairment and Functional Assessment Tools in Rheumatic Diseases

Domain	Assessment Tools	RA	OA	AS
Disease activity	2010 American College of Rheumatology/ European League Against Rheumatism Classification Criteria for RA	v		
	Ankylosing Spondylitis Disease Activity Score			v
	Bath Ankylosing Spondylitis Disease Activity Index			v
	28-Joint Disease Activity Score	v		
Pain	Visual Analog Scale Numerical Rating Scale	v	v	v
Fatigue	Fatigue Severity Scale	v	v	v
	Multidimensional Assessment of Fatigue	v	v	v
Joint function and mobility	Bath Ankylosing Spondylitis Metrology Index			v
	Chest Expansion Test			v
	Modified Schober Test			v
	Occiput-to-Wall Distance Test			v
	Range of Motion Tests	v	v	v
	Timed Up and Go Test	v	v	v
	6-Minute Walk Test	v	v	v
Muscle strength and endurance	Hand Grip Strength Test	v	v	
	Sit-to-Stand Test	v	v	v
Hand function	Australian/Canadian Osteoarthritis Hand Index	v	v	
	Arthritis Hand Function Test	v	v	
	Disabilities of the Arm, Shoulder, and Hand	v	v	
	Michigan Hand Outcome Questionnaire	v	v	
Activity and participation	Arthritis Impact Measurement Scale II	v	v	
	Craig Handicap Assessment and Reporting Technique	v	v	
	Health Assessment Questionnaire	v	v	v
	Knee Injury and Osteoarthritis Outcome Score		v	
	Sickness Impact Profile	v	v	v
	Western Ontario and McMaster Universities Arthritis Index		v	
Psychological well-being	Beck Depression Inventory	v	v	v
	Hospital Anxiety and Depression Scale	v	v	v
	Rheumatoid Arthritis Quality of Life	v		
Overall health status	Short Form Health Survey 36	v	v	v
	World Health Organization Disability Assessment Schedule	v	v	v

AS, Ankylosing spondylitis; *OA*, osteoarthritis; *RA*, rheumatoid arthritis. *V,* denotes suitability for assessing patients with the specified diagnosis.

subsides, the joint should be moved through the full range, possibly with assistance (active-assistive exercise). PNF techniques, such as the hold-relax and slow reversal-hold-relax technique, may be applied to improve exercise efficiency.

Aquatherapy is also useful for mobilizing joints because buoyancy in water reduces the effect of gravity and thus unloads the joints. Mobilization exercise should commence in the early stages of arthritis, when just a few repetitions through the full ROM are sufficient; if contracture or ankylosis of joints develops, it is difficult to restore the full ROM.[25]

The objective of strengthening exercise is to restore and maintain optimal muscle strength and endurance. There are three types

of muscle contraction: isometric (static) contraction, isotonic (dynamic) contraction, and isokinetic (dynamic) contraction. During isometric contraction, there is no change in the muscle length and joint position. The advantage of isometric contraction is minimal joint stress during muscle contraction; thus it is suited to sufferers of arthritis with mechanically deranged joints. It may also be used in an acute inflammatory joint or immediately after surgery. During isotonic contraction, the load is fixed, but muscle length and joint position are changed. If the muscle length is shortening during isotonic contraction, it is concentric contraction while the muscle length is lengthening for eccentric contraction. Isotonic contraction is suited for training of isotonic tasks and for patients without acute inflamed or biomechanically deranged joints; it should be avoided in patients with active arthritis because isotonic contraction stresses the joint through its ROM. During isokinetic contraction, the velocity of muscle contraction is constant, but the muscle contraction force is changeable (accommodative resistance). Isokinetic contraction usually requires maximal force of contraction, which in most cases is not recommended for patients with arthritis (unless the joints are in very good condition).

The purpose of aerobic exercise is to increase aerobic capacity, which increases work capacity and promotes optimal function; as such, it may reduce cardiovascular morbidity and mortality. Previous studies have shown that aerobic exercises increase aerobic capacity and functional ability in patients with RA, OA, AS, and fibromyalgia. Before the initiation of aerobic training, joint swelling and disease activity should be controlled. For patients with arthritis, the type of exercise should involve only low joint stress, such as walking, cycling, swimming, water aerobics, or low-impact aerobic dance. The exercise intensity, duration, and frequency can be adapted to those of cardiac rehabilitation. For water exercise, the temperature should be 83°F to 88°F (28°C–31°C). Tai-chi exercise can also be used but should be modified according to the patient's condition. Both land- and water-based aerobic exercise programs lasting 6 weeks to 3 months can have a positive effect on aerobic capacity, muscle strength, or functional ability.[25] During acute flares and periods of inflammation, strenuous exercises should be avoided. If joint pain persists for 2 hours after exercise and exceeds the pain severity before exercise, the intensity and/or duration of exercise should be reduced.

Recreational exercise is often a combination of mobilization exercise, strengthening exercise, and aerobic exercise, which are also all fun to do. It is usually a group exercise—that is, patients with a similar disease often exercise together.

Exercise programs may comprise a combination of land- and water-based activities (Fig. 32.17). Through recreational exercise, patients may improve muscle strength, aerobic capacity, and psychosocial well-being. It may provide social contact and thus have an antidepressant effect.

Kinesiology tape (KT) (Fig. 32.18) is a therapeutic elastic tape that can be applied on the skin to reduce pain, increase ROM, reduce swelling, and provide mechanical support. The hypothesized therapeutic mechanism of KT is an improvement in the local circulation in the taped area by increasing the cutaneous and subcutaneous interstitial area and a facilitation of lymphatic drainage. Moreover, the KT provides sensory feedback to patients who have fear of movement and facilitates small muscle contraction by generating a pull on the fascia, which may increase muscle strength. Therefore the KT can be used for treating arthritis to alleviate pain and improve joint function.

• **Fig. 32.17** A group pool exercise for patients with rheumatoid arthritis.

• **Fig. 32.18** Kinesiology tape.

Physical Modalities

Physical modalities such as thermotherapy (heat and cold therapy), electrical therapy, and low-power laser are commonly used in rehabilitation practice to relieve pain and increase the flexibility of joints and soft tissues in patients with rheumatic disorders.

Heat

Heat therapy (including superficial or deep heat) can increase both skin and joint temperature and the viscoelastic properties of collagen. Clinically, these two effects can relieve stiffness of joints and soft tissue, thus enhancing the efficacy of stretching. In addition, both superficial and deep heat can raise the pain threshold, producing analgesia and a sedation effect by acting on A-delta and C fibers and muscle spindles. Conversely, heat therapy can increase joint swelling, elevate leukocyte counts in the joint fluid of patients with arthritis, and may cause ischemic necrosis of the synovium by increasing its metabolic demand.

The most commonly used heat modalities in rheumatic diseases are moist heating pads, paraffin (Video 32.23), electrical heating pads, infrared heat, ultrasound, and shortwave diathermy. Most studies conclude that heat modalities do not alter the disease process. There is also concern about the use of paraffin in systemic sclerosis because microvascular pathology may create heat dissipation impairments. However, paraffin baths are recommended for short-term benefits in arthritic hands.

Therapeutic ultrasound

The thermal effects of ultrasound include an increase in metabolic activity and blood flow, a decrease in subacute and chronic inflammation and muscle spasm, and an increase in the extensibility of collagenous structures. The analgesic effect might be a result of increased microvascular permeability and cell metabolism.[10] Continuous-mode ultrasound generates the thermal effect, which increases nerve conduction and elevates the pain threshold. Pulsed-mode ultrasound stimulates cartilage repair and increases antiinflammatory and analgesic effects. Ultrasound has been applied for treatment of tennis elbow, OA, calcific tendinopathy of the shoulder, and other disabilities; however, scientific evidence for its usefulness is still not well recognized.

Cold

The effects of cold therapy are to reduce skin and joint temperature, reduce joint swelling and cell count in synovial fluid, decrease the metabolic demand of the synovium, and inhibit collagenase activity. It can also raise the pain threshold and inhibit muscle spindle activity. Therefore it can provide pain relief and reduce inflammation in patients with acute arthritis. Common cold modalities include a cold pack, cold air, ice pack, or cold bath. Cold should not be used in patients with cold hypersensitivity, Raynaud phenomenon, cryoglobulinemia, or paroxysmal cold hemoglobinuria.

Electrical therapy

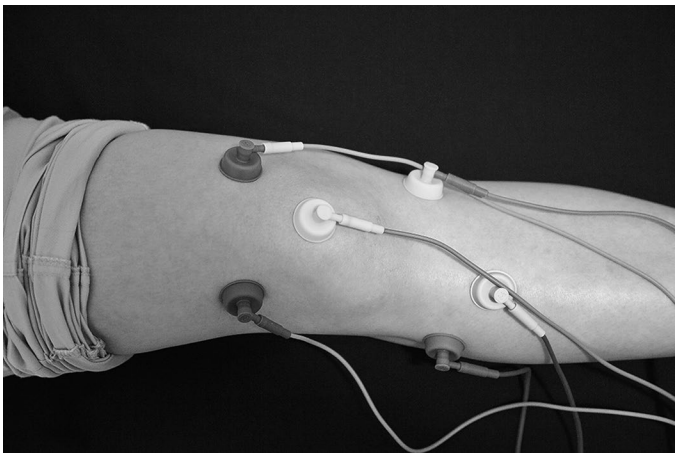
The purposes of electrical therapy are pain control and muscle stimulation. There are different types, including transcutaneous electrical nerve stimulation (TENS) (Video 32.24) and middle-frequency interferential therapy. These modalities are commonly used in the treatment of rheumatic diseases. Electrical therapy has been shown to have a clinically beneficial effect on grip strength for patients who have RA with hand atrophy. Another study showed that TENS may be more effective than the intraarticular injection of HA in patients with OA of the knee (Fig. 32.19). Although evidence of the efficacy of electrical therapy for rheumatic diseases is still lacking, electrical therapy can reduce joint and soft tissue pain and may thus reduce the dosage of pain medications.

Low-level (or low-power) laser therapy

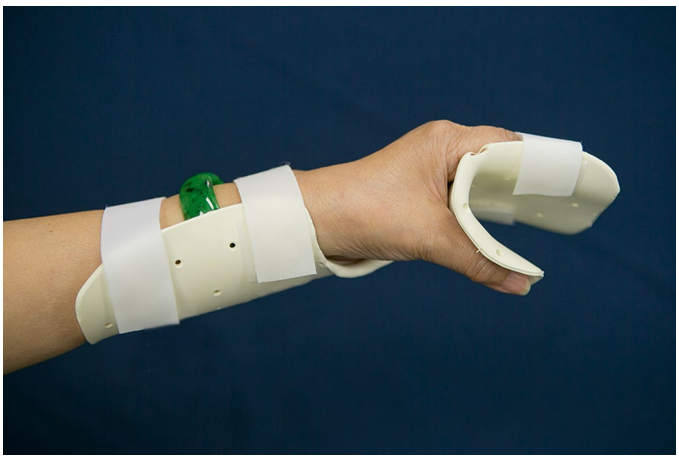
Low-level laser therapy has been used in the treatment of rheumatic diseases, including RA, OA, lower back pain, and carpal tunnel syndrome, for more than 20 years. Six studies of medium quality, testing over 220 patients with RA, showed that low-level laser therapy decreased pain and morning stiffness more than placebo laser therapy. No side effects were reported.

Orthoses

Orthotic positioning, especially hand splints, is usually considered and may have different benefits for patients at different stages, such as supporting and aligning weakened joints, reducing pain or instability, improving joint alignment, and assisting with movements (Videos 32.25 to 32.31).[1,8] For example, it is common to prescribe a resting hand splint to provide support and alleviate pain for patients in the acute stage (Fig. 32.20). During the postinflammatory process, an orthosis (e.g., the thumb-post splint) (Fig. 32.21) is used for external support to enhance stability of unstable or subluxed joints.



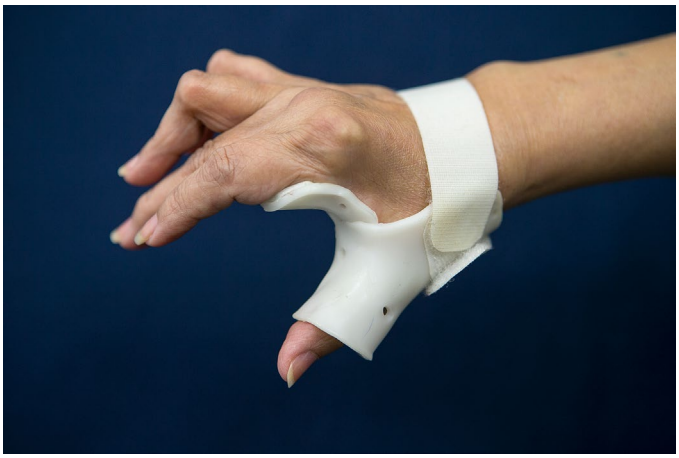
• **Fig. 32.19** Transcutaneous electrical nerve stimulation for arthritis of the knee. (Courtesy Archives of Physical Medicine and Rehabilitation.)



• **Fig. 32.20** Resting splints.

To prevent undesirable displacement or movement, a three-point oval-8 splint (Fig. 32.22) may be recommended for wear during activities, allowing flexion but preventing hyperextension of the finger PIP joints (swan-neck deformity). The MCP joint ulnar deviation orthosis may permit continued use of the IP joints of the hand without aggravating the deformity of the MCP joints. The orthosis can be made of lightweight thermoplastics, or soft materials can be used to counteract the deforming forces.

There are findings suggesting that splints are effective in reducing pain both immediately after provision of the splint and after splinting over a period of time. A systematic review also indicated the grip strength is temporarily increased while a splint is worn. However, there is insufficient evidence to show that wearing a splint for a long time can decrease the severity of hand deformities or preserve hand function.[1,16,28] Patients with foot and/or ankle involvement are recommended to use bracing and orthoses.[16]



• **Fig. 32.21** Thumb post splint for arthritis of the first carpometacarpal joint.

• **Fig. 32.22** Orthotic positioning of a swan-neck deformity. *Inset,* The oval-8 splint prevents proximal interphalangeal hyperextension and allows flexion.

Patient Education

People with rheumatic disease learn to manage this lifelong disease to continue performing a range of activities and social roles. Patients must understand that joint deformities can occur without appropriate antiinflammatory or disease-modifying medication. Interventions require teaching the patient and family about the disease, its symptoms, and the potential irreversible damage from chronic synovitis. They also need to learn adaptive skills, including joint protection and activity pacing techniques, the use of assistive devices, and environmental modifications to maintain meaningful occupational functioning (Videos 32.32 to 32.38). In addition, exercise, diet, weight control (if obese), fatigue management, stress management/relaxation, and social support are usually included in the educational program. Enhancing the patient's self-efficacy to adhere to treatment at home is important.[30]

Improve or Maintain Functional Mobility

Joint protection is a self-management technique widely taught to people with rheumatic diseases, especially RA and OA (Box 32.7). Joint protection principles are ideally initiated early in the disease process to reduce loading to vulnerable joints by using proper joint and body mechanics. Strategies involving the use of assistive devices and alternative movement patterns for affected joints can help patients perform daily activities while preserving joint integrity, thus relieving joint pain during activities.[8,9,13]

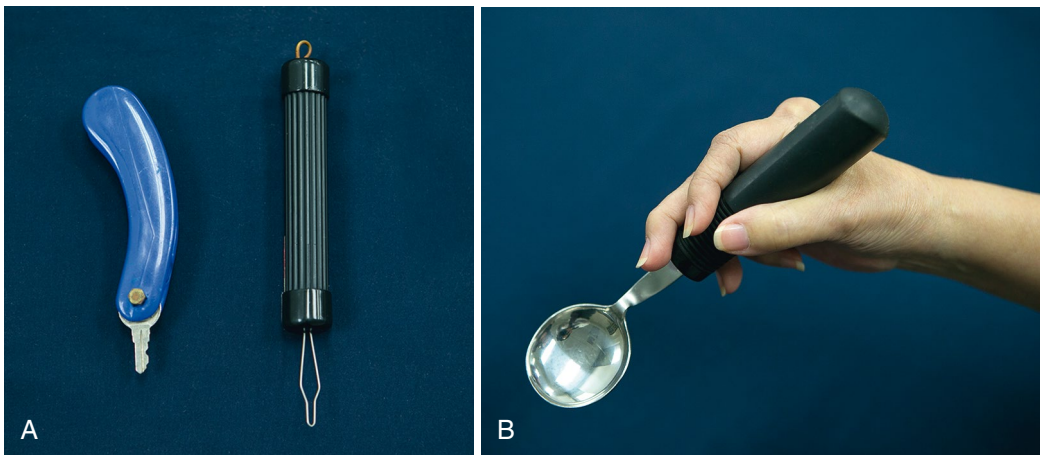
Joint protection education, typically provided by an occupational therapist, is delivered individually or in groups. Previous studies showed that psychoeducational interventions, including patient education, self-management, and cognitive-behavioral approaches, improve pain, self-efficacy and psychological status for up to 12 months.[21] It is important to note that the education should not rely solely on written information and verbal explanations. More effective behavior change techniques, such as skills practice, goal setting, and home programs, are required. However, recent evidence indicates that the effects of joint protection programs on pain and hand function in people with hand arthritis are minimal and not clinically significant.[9] Despite this, the ACR guideline recommends joint protection education in the early stages of RA and OA.[16,28] It is important to note that education should not rely solely on written information and verbal explanations.

Energy conservation principles and fatigue management are usually incorporated into the education. Fatigue has multifactorial contributors, including physiological, behavioral, and environmental factors. To manage the effects of fatigue on everyday activities, therapists teach patients to analyze their daily activities to identify what increases pain and fatigue. Activity pacing, by balancing activity and rest, is often recommended to accomplish tasks efficiently. Additionally, work simplification and the use of assistive or adaptive devices are useful strategies for fatigue management.

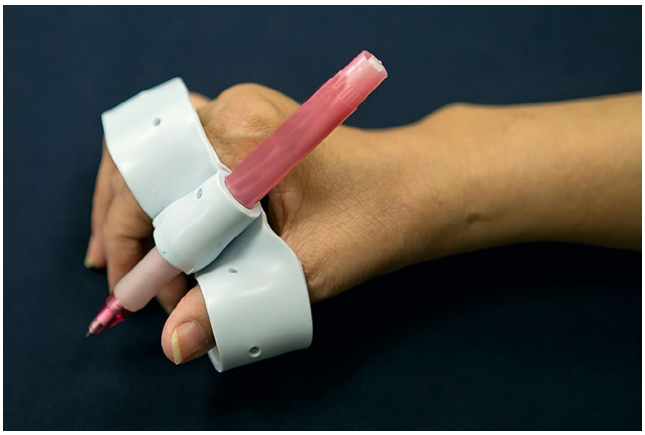
• BOX 32.7 Principles of Joint Protection for Rheumatic Diseases

1. Respect pain as a signal to stop the activity.
2. Maintain muscle strength and joint range of motion.
   - Maintaining daily activities within the limitations of the patient's pain helps prevent disuse atrophy.
   - Strengthening around an unstable joint can increase stability and reduce pain.
3. Use each joint in its most stable anatomical and functional plane.
4. Avoid positions of deformity and forces in their direction (e.g., turning resistive round doorknobs in an ulnar direction should be avoided by use of a lever door opener for patients with rheumatoid arthritis).
5. Use the largest, strongest joints available for the job (e.g., using a belted waist pack rather than holding purse with hook grasp).
6. Ensure correct patterns of movement.
7. Avoid staying in one position for long periods.
8. Avoid starting an activity that cannot be stopped immediately if it proves to be beyond your capability.
9. Balance rest and activity.
10. Reduce the force.
    - Building up handles to avoid tight grasp.
    - Using assistive devices, such as jar openers, to reduce the stress of the hand and wrist joints.
    - Using alternative methods to accomplish the task (e.g., using the handrail to reduce the impact load to involved knee joints while going up and downstairs).

Modified from Cordery J, Rocchi M. Joint protection and fatigue management. In: Melvin J, Jensen G, eds. *Assessment and Management.* vol. 1. American Occupational Therapy Association; 1998:279-322.



• **Fig. 32.23** (A) Assistive devices with built-up handles to avoid tight grasp. (B) Easy-to-hold utensils.



• **Fig. 32.24** Writing aid to reduce dexterity demands and stress on hand joints.

Assistive Devices and Adaptive Equipment

Many devices and equipment can assist patients with rheumatic diseases, particularly RA and OA, by limiting joint stress and promoting functional independence. For patients with limited ROM or pain in the hands, assistive devices with built-up handles and modifications to avoid tight grasps, such as broad keyholders, buttoning and zipping aids, and easy-hold utensils (Fig. 32.23), provide increased leverage and reduced dexterity demands. Devices such as writing aids (Fig. 32.24) can enhance functional performance. Extended-handle devices, such as long-handled combs, reachers (Fig. 32.25), and sock aids, benefit patients with limited shoulder, elbow, hip, or knee ROM. Mobility aids, including walkers, crutches, wheelchairs, and scooters, can accommodate various levels of ambulation difficulty and environmental requirements. An arthritic crutch with hand grip and forearm support is highly recommended to reduce stress on hand or wrist joints during ambulation. Appropriate footwear or insoles can enhance comfort during ambulation. Equipment such as a lift chair, shower chair, commode, and elevated toilet seat can reduce effort on the lower extremities and alleviate stress on the hands. Professional advice is generally required for selecting these products.[8]

Environmental Modification

Adapting the environment to enhance safety involves various measures, such as adaptations for toileting (toilet safety rail), showering (handheld shower, walk-in bath), and installing grab bars, ramps, stair lifts, etc.

Vocational Rehabilitation

Training programs aim to overcome barriers to successful employment. Worksite evaluation and modifications involve assessing and adjusting worksite conditions and duties for safety and well-being. For patients currently employed or seeking employment, vocational rehabilitation, worksite evaluations, and/or modifications are recommended to support employment.

The previously mentioned interventions provided through comprehensive rehabilitation (mainly occupational and physical therapy) programs are recommended for their benefits on pain, physical function, preserving independence, remaining in work, and safety.

Rehabilitation Intervention for Knee or Hip Osteoarthritis

Knee or hip OA may cause pain, swelling, limitation in ROM, walking difficulty, and ADL dependence. If moderate effusion is observed, joint aspiration is indicated. Intraarticular corticosteroid injection may follow if the joint fluid is clear (see Videos 32.5, 32.6, and 32.9). Physical modalities, such as cold therapy, electrical therapy, and local massage to muscles in spasm, may also be applied during the acute stage of arthritis.

• **Fig. 32.25** Extended-handle devices to decrease the demands of range of motion at the shoulder and hip joints. (A) Long-handled comb. (B) Reacher. (C) Sock aid.

A short course of oral NSAIDs may also be administered in patients with mild knee pain; local NSAID patches or gels may also be applied, especially in patients with gastrointestinal problems. Patient education on joint protection and exercise is crucial once acute arthritis is under control. If a patient is obese or overweight, weight reduction programs, including nutrition consultation and aerobic exercise, are strongly suggested. The use of a cane on the less affected side can relieve joint force up to 20%. In patients with unilateral tibiofemoral OA of moderate severity, a valgus knee brace may be suggested for medial compartment knee OA, and a varus knee brace may be suggested for lateral compartment knee OA[15]; however, this condition is less common. Theoretically, a lateral wedge insole may be helpful for medial compartment knee OA; however, recent clinical guidelines no longer recommend the use of the insole. Patellar taping may help to relieve knee pain in patients with patellofemoral OA. In addition, a structured exercise program (Video 32.39) that includes flexibility, strength, aerobic, balance, and proprioceptive training or even tai chi exercise is strongly suggested, and compliance with exercise regimens is essential. In patients with contracture of the affected joint, heat therapy, hydrotherapy (Video 32.40), and diathermy may be applied, followed by joint mobilization and stretching exercises. The rehabilitation process of hip OA is similar to that of knee OA; however, well-controlled studies on hip OA are few. In addition, topical medications are less effective for hip OA, and intraarticular injection into the hip joint may depend more on imaging (e.g., ultrasound) guidance.

Rehabilitation Intervention for Rheumatoid Arthritis

Acute Stage

In the acute stage, patients with RA often have arthritis involving multiple joints; this may be accompanied by fever, weight loss, anemia, and fatigue. In this stage, complete bedrest for a few days may help relieve joints and systemic symptoms. If one or two joints are more severely affected, resting splints are recommended. Application of a cold pack or TENS over the inflammatory joints may help reduce joint swelling and relieve joint pain. If there is severe spasm and pain in the nearby muscles, local hot packs or gentle massage would be beneficial. Bedrest should be as short as possible because prolonged bedrest may lead to deconditioning.

For prevention of joint and soft tissue contracture, actively inflamed joints should be moved gently through the possible range by the patient (active ROM) or with assistance from another person (active-assisted ROM). Three repetitions for each joint once or twice daily are suggested. As joint inflammation subsides, the range may be increased gradually to the full range, possibly with assistance. A PNF technique could be applied early on for selected muscle groups. Passive stretching is usually not performed in an inflammatory joint; if performed, it should be done with extreme caution because it may worsen or prolong the inflammatory process or lead to subluxation or rupture of a joint.

For minimizing muscle atrophy, isometric exercise at submaximal effort is recommended in the inflammatory condition.

At first, a few nonresistive repetitions should be performed with a gradual increase in repetition and resistance. Isotonic exercise is not recommended in the acute inflammatory stage. Patient education (including joint protection and energy conservation techniques) should also commence.

Subacute and Chronic Stages

After the acute stage subsides, joint pain, morning stiffness, and joint swelling diminishes. Isotonic exercise (Video 32.41) can be added to the exercise program gradually, with increases in repetition and resistance. Local cold therapy can be changed to hot therapy if swelling of the joint subsides. Splints, foot orthoses, or assistive devices can be prescribed if indicated.

A forearm support crutch or cane is preferable to a traditional wrist support device. If a patient's condition improves, endurance training, aerobic exercise, and recreational exercise can be added to the exercise program.

Rehabilitation Intervention for Ankylosing Spondylitis

The main symptoms of AS include chronic back pain, spinal deformity, limited chest excursion, and pain and contracture of the involved peripheral joints (especially the hips). The characteristic posture of patients with AS is loss of lumbar lordosis, increased thoracic kyphosis, compensatory extension of the cervical spine, and flexion of hip and knee joints. The center of mass of the trunk in the sagittal plane shifts forward and downward. The main aims of management for patients with AS are pain relief, improvement of posture and mobility, and maintenance or improvement of respiratory function.

Pain relief may be achieved with the use of antiinflammatory drugs and physical modalities, including hot or cold packs, hot baths, hydrotherapy, spa therapy, diathermy, electric therapy, and mobility exercise. Maintenance of good mobility of the spine and peripheral joints not only relates to physical function but also affects relief of pain and stiffness. Before mobility exercise, a hot pack, or hot bath (shower), followed by 5 to 10 minutes of warmup exercise, is suggested. The target areas for stretching are the suboccipital muscles, pectoral muscles, hamstrings, hip flexors, and spinal rotators. The ROM exercise should include extension of all segments of the spine and full ROM of the neck, shoulders, and hips. It is advisable for patients to perform the exercises at a time of day when they feel the least pain or fatigue. PNF techniques provided by therapists are also suggested if available. Strengthening exercises for the major muscle groups, including back extensors, shoulder retractors, hip extensors, and other postural muscles, are also recommended (Videos 32.41 and 32.42). In addition, aerobic training with brisk walking, stationary cycling (with upright handlebars), swimming, or other aquatic therapy can help to increase muscle endurance and reduce cardiovascular morbidity and mortality. Exercise programs combining flexibility, muscle strength, and aerobic exercise are effective on patient's function, mobility, and disease activity.

Inflammation of the costochondral joints or costovertebral joints or entheses may cause chest pain and inhibit deep breathing. Treatment with antiinflammatory drugs and heat therapy may help relieve pain and improve breathing patterns. Daily deep-breathing exercises with an emphasis on full ribcage expansion are encouraged.

Education about correct posture is very important for patients with AS. They should be instructed how to check their postural alignment (e.g., measure body height, touch the occipital protuberance to the wall while standing) and avoid positions that encourage a stooped posture or spinal flexion for prolonged periods. A firm mattress and a small pillow or neck support are desirable for sleep. Lying on the side in a curved position should be avoided. Daily prone lying for 10 to 15 minutes or more is recommended. Because patients with AS often have a fragile and osteoporotic spine, contact sports, cervical traction, or spinal manipulation should be avoided. Minor trauma that results in fracture or dislocation of the spine with spinal cord compression is not uncommon. For enhancing independence in ADLs, prism glasses, long-handled devices, or a cane may be needed.

Summary

In view of the growing number of patients presenting with rheumatic diseases, research on prevention and treatment is evolving. Less invasive surgical options and expanding conservative treatments to combine allopathic and naturopathic medicine is the current trend. Physiatrists will continue to be an integral part of the management. The chronicity and disabling nature of these diseases will continue to challenge physiatrists toward innovation and creativity.

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