Ultrasound Features of Hand Involvement in Psoriatic Arthritis and Hand Osteoarthritis: A Comprehensive Review of Differential Imaging Findings and Clinical Applications

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Abstract

Background:

Hand involvement is a major source of pain, functional impairment, and disability in both psoriatic arthritis (PsA) and hand osteoarthritis (HOA). Despite overlapping clinical manifestations, these disorders differ substantially in their underlying pathophysiology, disease progression, prognosis, and therapeutic strategies, making early and accurate differentiation essential for optimal patient management. Conventional radiography remains the standard imaging modality for structural assessment; however, it has limited sensitivity for detecting early inflammatory changes. Musculoskeletal ultrasound (MSUS) has emerged as a valuable imaging technique because of its ability to provide real-time, high-resolution visualization of both inflammatory and structural abnormalities involving joints, tendons, entheses, and periarticular soft tissues.

This review aims to provide a comprehensive overview of the ultrasound features of hand involvement in PsA and HOA, emphasizing the imaging findings that facilitate differential diagnosis and guide clinical decision-making. The review discusses characteristic grayscale and Power Doppler ultrasound abnormalities in both diseases, including synovitis, tenosynovitis, enthesitis, peritendon inflammation, dactylitis, bone erosions, osteophytes, cartilage abnormalities, and new bone formation. The relationship between ultrasound findings and the underlying pathological mechanisms is also explored to enhance understanding of disease-specific imaging patterns. Furthermore, the clinical applications of ultrasound in early diagnosis, disease activity assessment, monitoring therapeutic response, and image-guided interventions are highlighted. Current evidence indicates that ultrasound is highly sensitive for detecting inflammatory lesions in PsA, including subclinical synovitis, flexor tenosynovitis, peritendon extensor inflammation, enthesitis, and active vascular signals on Power Doppler imaging. In contrast, hand OA is characterized predominantly by structural abnormalities such as osteophyte formation, cartilage degeneration, cortical irregularities, and variable degrees of synovitis, particularly in erosive disease. Recognition of these distinctive ultrasound patterns substantially improves diagnostic confidence and assists in distinguishing inflammatory arthropathy from degenerative joint disease, particularly in patients with equivocal clinical findings.

In conclusion, musculoskeletal ultrasound has become an indispensable imaging modality in the evaluation of hand involvement in PsA and HOA. Its ability to simultaneously assess inflammatory activity and structural damage offers significant advantages for differential diagnosis, disease monitoring, and personalized management. Continued advances in ultrasound technology and standardized scanning protocols are expected to further strengthen its role in routine rheumatology practice and improve patient outcomes.

Keywords: Musculoskeletal ultrasound; Psoriatic arthritis; Hand osteoarthritis

Introduction

Hand involvement is a frequent and clinically important manifestation in both psoriatic arthritis (PsA) and hand osteoarthritis (HOA), yet the same symptom complex of pain, stiffness, swelling, and impaired dexterity may arise from very different pathological processes. In PsA, hand symptoms usually reflect immune-mediated inflammation involving synovium, tendon sheaths, entheses, periarticular soft tissues, and bone. In HOA, symptoms are driven mainly by cartilage degeneration, osteophyte formation, subchondral remodeling, and variable secondary synovitis. Because both diseases commonly affect the distal interphalangeal and proximal interphalangeal joints, clinical differentiation may be difficult, particularly in early disease, erosive HOA, or older patients with psoriasis. [1]

Musculoskeletal ultrasound (MSUS) has become especially valuable in this setting because it evaluates the hand as a functional anatomical unit rather than as isolated joints. Unlike radiography, which mainly demonstrates late structural consequences, ultrasound can detect active synovitis, tenosynovitis, enthesitis, peritendon inflammation, soft tissue edema, cortical erosions, osteophytes, cartilage damage, and Power Doppler vascularity during the same examination. This ability to visualize both inflammatory and structural lesions makes ultrasound particularly suited for differentiating PsA from HOA, where the diagnostic question is often whether hand symptoms are predominantly inflammatory, degenerative, or mixed. [2]

The ultrasound phenotype of hand PsA is characterized by multi-compartment inflammation. Synovitis may occur together with flexor tenosynovitis, extensor peritendon inflammation, pulley-related inflammation, dactylitis, entheseal abnormalities, nail unit changes, cortical erosions, and new bone formation. These findings reflect the pathophysiological concept that PsA is not restricted to the synovial membrane but involves the synovio-entheseal and extracapsular soft tissue compartments. Therefore, a limited ultrasound protocol focused only on intra-articular synovitis may underestimate the true inflammatory burden in PsA and miss lesions with high diagnostic value. [3]

By contrast, the ultrasound phenotype of HOA is dominated by structural joint damage. Osteophytes, cartilage thinning, cortical irregularity, joint space narrowing, and subchondral remodeling are the principal findings, while synovitis and Power Doppler activity are usually secondary and more prominent in painful or erosive disease. This distinction is clinically important because inflammatory findings in HOA may mimic PsA, but their anatomical distribution and association with dominant degenerative lesions usually differ from the diffuse tendon, entheseal, and periarticular inflammatory pattern seen in psoriatic disease. [4]

The research gap addressed by this review is the insufficient integration of disease-specific ultrasound findings into a practical differential diagnostic framework for hand PsA and HOA. Many published discussions describe PsA imaging or HOA imaging separately, but clinicians frequently face patients in whom these disorders overlap clinically and radiographically. A focused synthesis of gray-scale and Power Doppler features, lesion distribution, structural patterns, and imaging pitfalls is therefore needed to improve diagnostic confidence and guide management in rheumatology and rehabilitation practice. [5]

Accordingly, this review aims to summarize the ultrasound features of hand involvement in PsA and HOA with emphasis on differential imaging findings and clinical applications. Particular attention is given to synovitis, tenosynovitis, peritendon inflammation, enthesitis, dactylitis, nail unit abnormalities, erosions, osteophytes, cartilage damage, and Power Doppler activity. The ultimate purpose is to provide a clinically useful ultrasound-based approach that helps distinguish inflammatory psoriatic hand disease from degenerative osteoarthritic involvement and supports earlier, more individualized patient management. [2]

Overview of Hand Involvement in Psoriatic Arthritis and Hand Osteoarthritis

Hand involvement is a defining feature of both psoriatic arthritis (PsA) and hand osteoarthritis (HOA), although the underlying disease mechanisms, patterns of tissue injury, and long-term outcomes differ considerably. In PsA, inflammation affects multiple musculoskeletal structures, including the synovium, tendon sheaths, entheses, periarticular soft tissues, and bone, resulting in a heterogeneous clinical presentation. Conversely, HOA is primarily characterized by progressive degeneration of articular cartilage accompanied by subchondral bone remodeling, osteophyte formation, and varying degrees of secondary synovial inflammation. Despite these fundamental differences, both disorders frequently present with pain, swelling, stiffness, and impaired hand function, making accurate differentiation a common clinical challenge, particularly during the early stages of disease. [6]

The hands are among the most frequently affected anatomical regions in PsA, with involvement ranging from isolated distal interphalangeal (DIP) joints to extensive polyarticular disease affecting the metacarpophalangeal (MCP), proximal interphalangeal (PIP), and wrist joints. Characteristic manifestations include synovitis, flexor tenosynovitis, dactylitis, enthesitis, peri-extensor tendon inflammation, nail disease, and progressive bone remodeling. Importantly, several of these abnormalities may coexist within the same digit, reflecting the concept of the "functional enthesis" and explaining the diffuse swelling commonly observed in affected fingers. This multisystem involvement distinguishes PsA from many other inflammatory arthritides and contributes to its considerable clinical heterogeneity. [7]

In contrast, HOA predominantly affects the DIP and PIP joints, as well as the first carpometacarpal (CMC) joint, producing progressive structural deterioration that eventually limits dexterity and grip strength. Patients typically develop pain that worsens with activity, short-lasting morning stiffness, joint enlargement, reduced range of motion, and progressive deformity. While cartilage degeneration remains the hallmark pathological process, contemporary evidence recognizes that synovitis, capsular thickening, and inflammatory episodes contribute significantly to pain generation and structural progression, particularly in erosive HOA. These findings have shifted the perception of HOA from a purely degenerative disease toward a complex disorder involving mechanical, inflammatory, metabolic, and biological pathways. [8]

Although clinical examination remains the cornerstone of diagnosis, considerable overlap exists between PsA and HOA. Both conditions may involve the DIP joints, produce finger swelling, and demonstrate structural abnormalities on conventional radiographs. Furthermore, inflammatory HOA may mimic PsA because patients often exhibit joint tenderness, synovial hypertrophy, and intermittent inflammatory flares. Conversely, early PsA may initially present with only a few swollen joints and minimal cutaneous manifestations, increasing the likelihood of diagnostic uncertainty. Consequently, reliance on clinical findings alone may delay appropriate diagnosis and treatment, emphasizing the importance of advanced imaging techniques capable of identifying disease-specific abnormalities before irreversible joint damage develops. [2]

From a functional perspective, hand involvement substantially compromises activities of daily living in both disorders. Progressive impairment of grip strength, pinch function, manual dexterity, and fine motor coordination interferes with self-care, occupational performance, and social participation. In PsA, persistent inflammation may lead to irreversible joint destruction, ankylosis, tendon dysfunction, and severe deformities such as arthritis mutilans. In HOA, progressive osteophyte formation, joint instability, and structural remodeling similarly contribute to chronic disability, although the mechanisms differ from those observed in inflammatory arthritis. Therefore, early recognition of hand involvement has important implications for preserving long-term function and improving patient quality of life. [9]

Given the overlapping clinical presentations and divergent therapeutic approaches, accurate characterization of hand pathology is essential. Musculoskeletal ultrasound has emerged as an ideal imaging modality because it simultaneously evaluates synovial inflammation, tendon pathology, enthesitis, cartilage integrity, cortical bone changes, osteophytes, erosions, and vascular activity using Power Doppler imaging. This comprehensive assessment enables clinicians to distinguish inflammatory

lesions typical of PsA from the predominantly degenerative abnormalities observed in HOA, thereby improving diagnostic confidence and facilitating individualized management strategies. [10]

Pathophysiological Basis of Ultrasound Findings

Understanding the pathophysiological mechanisms underlying PsA and HOA is essential for interpreting their ultrasound appearances. Musculoskeletal ultrasound depicts the structural and inflammatory consequences of disease rather than the molecular processes themselves; therefore, the observed sonographic abnormalities directly reflect the underlying pathological events occurring within the synovium, tendons, entheses, cartilage, cortical bone, and periarticular soft tissues. The distinct biological mechanisms driving PsA and HOA explain their characteristic imaging patterns and provide the basis for differentiating these disorders during ultrasound examination. [11]

Psoriatic arthritis is a chronic immune-mediated inflammatory disease characterized by complex interactions between genetic susceptibility, environmental triggers, and dysregulated innate and adaptive immune responses. Activation of inflammatory pathways involving tumor necrosis factor (TNF), interleukin (IL)-17, IL-23, and multiple immune cell populations promotes persistent inflammation at the synovium, tendon sheaths, entheses, and adjacent bone. Unlike rheumatoid arthritis, where synovitis predominates, PsA demonstrates inflammation involving multiple anatomical compartments simultaneously, explaining the coexistence of synovitis, tenosynovitis, enthesitis, peritendon inflammation, and new bone formation that can be readily visualized using ultrasound. [12]

One of the distinguishing pathological features of PsA is enthesitis, which is increasingly recognized as a primary site of inflammation rather than merely a secondary manifestation. Mechanical stress at tendon and ligament insertions, together with immune activation in genetically susceptible individuals, initiates inflammatory cascades that subsequently extend to adjacent synovium, tendon sheaths, bone marrow, and periosteum. This "synovio-entheseal complex" concept explains why ultrasound frequently detects thickened entheses, erosions, enthesophytes, adjacent bursitis, and increased Power Doppler vascularity even in patients with limited clinical symptoms. Recognition of these findings is particularly valuable because enthesitis is relatively uncommon in primary hand OA and therefore contributes substantially to differential diagnosis. [13]

Another hallmark of PsA is pathological bone remodeling characterized by the coexistence of bone erosion and excessive new bone formation. Persistent inflammation stimulates osteoclast-mediated bone resorption while simultaneously promoting osteoblast activation and periosteal bone proliferation. Consequently, patients may develop cortical erosions adjacent to areas of periosteal new bone formation, ankylosis, and characteristic deformities involving the distal interphalangeal joints. Ultrasound is capable of detecting these cortical abnormalities with high sensitivity, particularly in small peripheral joints where early structural damage may not yet be visible on conventional radiography. [14]

In contrast, HOA develops through a multifactorial process involving aging, repetitive mechanical loading, genetic susceptibility, metabolic disturbances, and low-grade inflammation. The initial pathological event is disruption of cartilage extracellular matrix homeostasis, resulting in progressive degradation of collagen and proteoglycans. As cartilage integrity declines, abnormal mechanical forces are transferred to the subchondral bone, leading to sclerosis, cyst formation, osteophyte development, and remodeling of the joint margins. Although inflammation is generally less prominent than in PsA, synovial activation contributes significantly to pain generation and disease progression, particularly in erosive HOA. [15]

Current evidence indicates that inflammation in HOA is mediated by activation of synovial macrophages, fibroblast-like synoviocytes, and chondrocytes, which release inflammatory cytokines, chemokines, and matrix-degrading enzymes, including matrix metalloproteinases. These mediators accelerate cartilage degradation while promoting synovial hypertrophy and episodic increases in vascularity that may be detected by Power Doppler ultrasound. Consequently, ultrasound findings of synovitis are not exclusive to inflammatory arthritis and should always be interpreted alongside the distribution of structural abnormalities and accompanying tendon or entheseal involvement to avoid diagnostic misclassification. [16]

The differing pathological mechanisms of PsA and HOA ultimately produce distinct sonographic signatures. PsA predominantly demonstrates active inflammatory lesions affecting multiple musculoskeletal tissues simultaneously, whereas HOA is characterized mainly by structural degeneration with secondary inflammatory changes. Appreciating these biological differences enables clinicians to correctly interpret grayscale and Power Doppler abnormalities, distinguish inflammatory arthropathy from degenerative disease, and optimize subsequent diagnostic and therapeutic decisions. [2]

Ultrasound Features of Hand Psoriatic Arthritis

Ultrasound assessment of hand PsA should begin with the recognition that the disease is not confined to the intra-articular synovium. Rather, PsA frequently involves the joint, tendon sheath, enthesis, peri-tendinous soft tissue, nail unit, and adjacent cortical bone as interconnected anatomical targets. This explains why a patient with apparently localized finger pain may demonstrate multiple ultrasound abnormalities within the same digit. In the hand, this multi-compartment pattern is particularly useful diagnostically, because it supports an inflammatory psoriatic process rather than a purely degenerative disorder. [17]

Synovitis is one of the most frequently detected ultrasound abnormalities in hand PsA. On grayscale imaging, it appears as synovial hypertrophy, joint effusion, or capsular distension, most commonly involving the MCP, PIP, and DIP joints. Power Doppler signal within hypertrophied synovium indicates active inflammatory vascularization and may be present even when clinical swelling is mild or uncertain. This ability to detect subclinical synovitis is clinically important, especially in patients with psoriasis who report hand pain but do not yet fulfill clear clinical criteria for arthritis. [18]

Tenosynovitis is another major ultrasound feature of hand PsA and is particularly relevant when patients present with finger swelling, pain during movement, or impaired grip. Flexor tenosynovitis appears as hypoechoic or anechoic tendon sheath distension, tendon sheath thickening, and sometimes Power Doppler signal surrounding the tendon. In PsA, tenosynovitis may occur together with synovitis and soft tissue edema, contributing to the diffuse swelling of the digit. This finding is especially important because tendon involvement may be more extensive than suggested by clinical examination alone. [19]

Peritendon extensor inflammation is a distinctive ultrasound lesion described in PsA, particularly around the extensor tendon at the dorsal aspect of the MCP and interphalangeal joints. Unlike classic tenosynovitis, this abnormality involves inflammatory changes around tendons that lack a true synovial sheath, appearing as hypoechoic thickening and Power Doppler signal in the peritendinous soft tissue. Its recognition is diagnostically valuable because it reflects the extrasynovial inflammatory pattern of PsA and helps differentiate PsA from rheumatoid arthritis and degenerative hand disease. [20]

Dactylitis represents one of the most characteristic clinical and ultrasound manifestations of PsA. Sonographically, dactylitis is not a single lesion but a combination of flexor tenosynovitis, soft tissue edema, synovitis, pulley inflammation, and sometimes enthesitis or bone changes. Acute dactylitis commonly demonstrates prominent hypoechoic soft tissue swelling and Power Doppler activity, whereas chronic dactylitis may show more fibrotic thickening with less vascularity. Ultrasound is therefore useful not only for confirming dactylitis but also for identifying its dominant inflammatory component and monitoring response to therapy. [21]

Enthesitis in the hand may be subtle but remains central to the psoriatic disease concept. Ultrasound features include entheseal thickening, hypoechogenicity, cortical irregularity, erosions, enthesophytes, calcifications, and Power Doppler signal close to the bony insertion. In PsA, entheseal abnormalities may occur at extensor tendon insertions, collateral ligament insertions, and periarticular functional entheses around small joints. Because clinical palpation of small hand entheses is difficult and often nonspecific, ultrasound provides a more precise method for detecting entheseal involvement. [22]

Structural damage in hand PsA reflects the coexistence of destructive and proliferative bone responses. Ultrasound can detect cortical bone erosions as intra-articular discontinuities visible in two

perpendicular planes, while new bone formation may appear as irregular bony prominences, periosteal reaction, or entheseal bony proliferation. This mixed pattern of erosion and new bone formation is important because it differs from the predominantly osteophyte-driven remodeling of hand OA. When present alongside Doppler-positive synovitis, tenosynovitis, or enthesitis, these structural findings strongly support PsA. [23]

The nail unit is anatomically and functionally connected to the distal interphalangeal joint and extensor tendon enthesis, explaining the close association between nail psoriasis and DIP involvement in PsA. Ultrasound may show nail plate thickening, loss of trilaminar nail structure, nail bed thickening, and Doppler signal within the nail bed or matrix region. Although nail ultrasound is not yet universally standardized in routine practice, it can provide supportive evidence of psoriatic disease, particularly when DIP pain or swelling raises the differential diagnosis between PsA and HOA. [24]

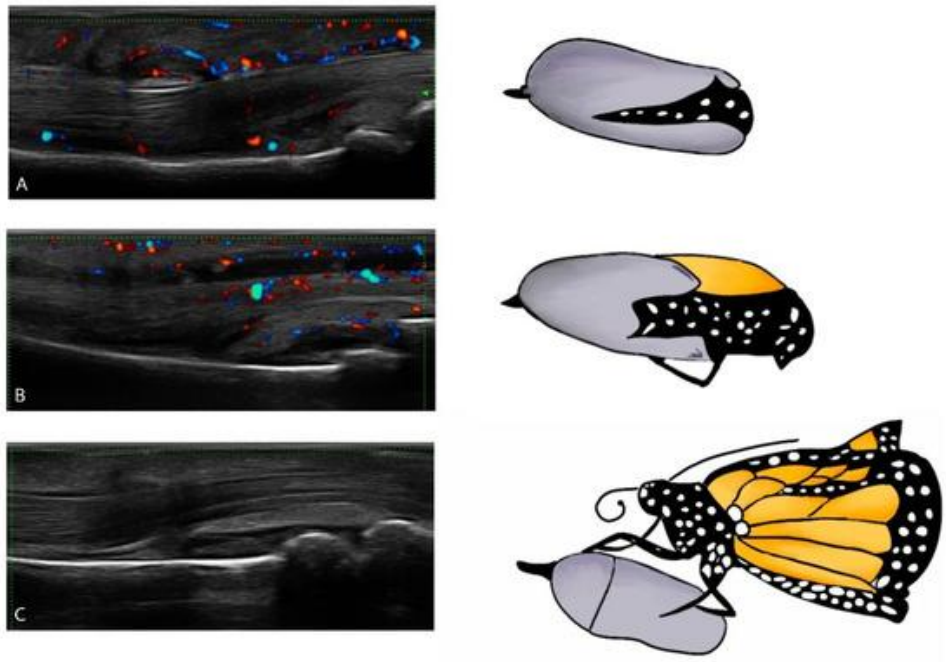


Figure 1. Evolution of dactylitis. Inflammation initially compromises the extracapsular structures and subsequently extends to the joint structures. The US lesions underlying dactylitis change over time, like evolving from a chrysalis to a butterfly. In particular: a) in the early phase flexor tenosynovitis and soft tissue oedema are prevalent. Joint synovitis is often absent; b) in the intermediate phase, both flexor tenosynovitis and synovitis may be present; c) in the late-chronic phase, joint synovitis is prevalent while flexor tenosynovitis is often absent or present but of minimal degree. [24]

Ultrasound Features of Hand Osteoarthritis

Hand osteoarthritis (HOA) is characterized by a combination of structural degeneration and variable inflammatory changes that can be effectively evaluated using musculoskeletal ultrasound. Unlike PsA, in which inflammation is the primary pathological process, ultrasound findings in HOA predominantly reflect cartilage degeneration, osteophyte formation, subchondral bone remodeling, and joint margin changes, with synovitis representing a secondary phenomenon. The ability of ultrasound to simultaneously visualize both inflammatory and structural abnormalities has improved understanding of HOA as a whole-joint disease rather than a disorder limited to articular cartilage. [25]

Osteophytes are the hallmark ultrasound finding in HOA and are identified as cortical protrusions extending beyond the normal bone contour. They are most frequently observed at the distal interphalangeal (DIP), proximal interphalangeal (PIP), and first carpometacarpal (CMC) joints, corresponding to the typical distribution of the disease. Ultrasound is highly sensitive for detecting small osteophytes, particularly during the early stages of disease when conventional radiographs may underestimate their presence. Their size and distribution often correlate with disease severity, joint

deformity, and progressive limitation of hand function. [26]

Cartilage abnormalities represent another important sonographic feature of HOA. Healthy hyaline cartilage appears as a smooth, homogeneous, anechoic layer covering the cortical bone, whereas osteoarthritic cartilage demonstrates progressive thinning, loss of surface regularity, increased echogenicity, and focal defects. As degeneration advances, ultrasound may reveal complete loss of the normal cartilage interface together with cortical irregularity and narrowing of the joint space. These structural changes reflect the progressive deterioration of cartilage extracellular matrix that underlies HOA pathogenesis. [27]

Synovitis is increasingly recognized as an important component of HOA and contributes significantly to pain and disease progression. On grayscale ultrasound, synovitis appears as hypoechoic synovial hypertrophy with or without joint effusion, while Power Doppler imaging may demonstrate increased vascularity indicating active inflammation. Although Doppler activity is generally less intense and less extensive than in PsA, inflammatory changes are relatively common in symptomatic and erosive HOA, emphasizing that ultrasound findings should always be interpreted alongside structural abnormalities rather than vascularity alone. [28]

Erosive hand osteoarthritis represents a more aggressive inflammatory subset characterized by episodic inflammatory flares and accelerated structural destruction. Ultrasound typically demonstrates central cortical erosions, marked synovial hypertrophy, joint effusion, and variable Power Doppler activity superimposed on established osteophytes and cartilage loss. Because these inflammatory findings may resemble PsA, careful assessment of erosion morphology, joint distribution, associated tendon abnormalities, and absence of characteristic entheseal or diffuse extrasynovial inflammation is essential for accurate differential diagnosis. [29]

Subchondral bone remodeling is another characteristic feature of HOA that can be appreciated sonographically through cortical irregularity, marginal remodeling, and progressive osteophyte development. Although ultrasound cannot directly visualize deep subchondral bone architecture, cortical changes provide indirect evidence of the remodeling process associated with chronic mechanical loading and cartilage degeneration. These structural alterations usually evolve gradually and are accompanied by relatively limited periarticular soft tissue inflammation compared with the multifocal inflammatory lesions observed in PsA. [30]

An important strength of ultrasound in HOA is its ability to detect inflammatory activity before substantial radiographic progression becomes evident and to monitor changes over time. Several studies have demonstrated associations between ultrasound-detected synovitis or Power Doppler signal and subsequent structural progression, suggesting that inflammatory activity contributes to disease evolution even in a traditionally degenerative disorder. Consequently, ultrasound provides clinically relevant information beyond conventional radiography by identifying patients with more active disease who may require closer follow-up and individualized therapeutic strategies. [31]

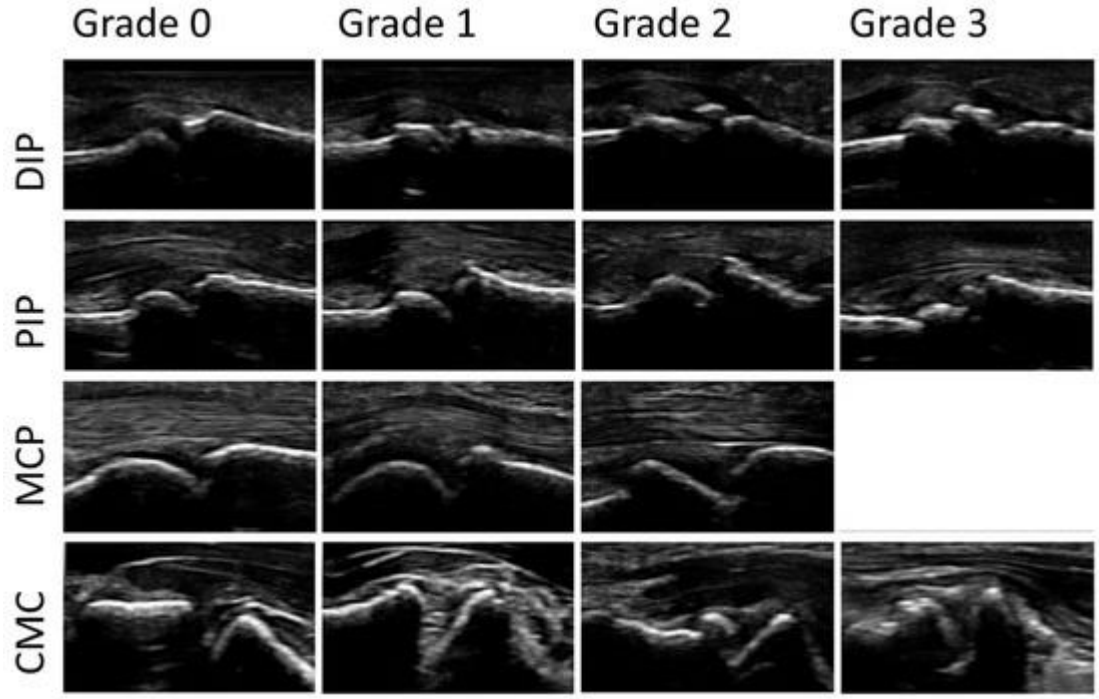


Figure 2. Examples of the different grades of osteophytes (0–3) for each joint group (DIP = distal interphalangeal joint; PIP = proximal interphalangeal joint; MCP = metacarpophalangeal joint; CMC = carpometacarpal joint in palmar view). No grade 3 osteophyte was found at any MCP joint. [31]

Differential Ultrasound Features Between Hand Psoriatic Arthritis and Hand Osteoarthritis

Although hand PsA and HOA may share several clinical manifestations, including pain, swelling, stiffness, and distal interphalangeal (DIP) joint involvement, musculoskeletal ultrasound provides important disease-specific features that facilitate differential diagnosis. Rather than relying on a single abnormality, ultrasound enables comprehensive assessment of joints, tendons, entheses, periarticular soft tissues, cartilage, and cortical bone within the same examination. The pattern, distribution, and combination of sonographic abnormalities are considerably more informative than any isolated lesion and often provide the key to distinguishing inflammatory from degenerative hand disease. [32]

The principal distinction between the two disorders lies in the predominance of inflammation versus structural degeneration. In PsA, ultrasound typically demonstrates active synovitis with prominent Power Doppler vascularity, flexor tenosynovitis, peritendon extensor inflammation, enthesitis, dactylitis, and erosive bone changes associated with new bone formation. In contrast, HOA is characterized mainly by osteophytes, cartilage thinning, cortical remodeling, joint space narrowing, and variable synovitis, while tendon and entheseal abnormalities are generally less extensive and less inflammatory in nature. Recognition of this overall disease pattern substantially improves diagnostic confidence during routine clinical practice. [33]

Enthesitis represents one of the most valuable ultrasound findings for differentiating PsA from HOA. In PsA, inflammatory changes at tendon and ligament insertions frequently demonstrate entheseal thickening, hypoechogenicity, erosions, enthesophytes, and Power Doppler signal adjacent to the cortical insertion, reflecting active inflammation of the enthesis. Conversely, entheseal abnormalities in HOA are usually degenerative, with minimal vascularity and without the diffuse inflammatory soft tissue changes characteristic of psoriatic disease. Therefore, identification of active entheseal inflammation strongly favors PsA, particularly when associated with synovitis or tenosynovitis. [34]

Tendon involvement also differs substantially between the two conditions. Flexor tenosynovitis and peritendon extensor inflammation are common findings in PsA and frequently contribute to dactylitis

and diffuse finger swelling. Ultrasound often demonstrates circumferential tendon sheath thickening, hypoechoic inflammatory tissue, and increased Doppler vascularity surrounding the tendons. In HOA, tendon abnormalities are generally secondary to mechanical degeneration or adjacent osteophytes and rarely exhibit the extensive inflammatory pattern observed in PsA. Consequently, diffuse inflammatory tendon involvement is considerably more suggestive of PsA than of degenerative osteoarthritis. [27]

Bone abnormalities provide another important point of distinction. PsA characteristically exhibits the coexistence of cortical erosions and new bone formation resulting from simultaneous osteoclastic and osteoblastic activity. By comparison, HOA predominantly demonstrates marginal osteophytes, cortical remodeling, cartilage loss, and subchondral structural changes related to chronic mechanical degeneration. Although erosive HOA may produce cortical defects that resemble inflammatory erosions, these lesions usually occur in conjunction with prominent osteophytes and advanced degenerative remodeling rather than widespread inflammatory soft tissue abnormalities. [29]

Power Doppler imaging further enhances differential diagnosis by demonstrating the distribution and intensity of active inflammation. Patients with active PsA frequently exhibit Doppler signals within synovium, tendon sheaths, entheses, peritendinous tissues, and occasionally the nail apparatus, reflecting persistent immune-mediated inflammation. In contrast, Doppler activity in HOA is generally less extensive and primarily confined to inflamed synovium during symptomatic or erosive phases. Accordingly, diffuse multisite Doppler positivity involving several anatomical compartments strongly supports a diagnosis of PsA over HOA. [24]

Despite these characteristic differences, overlap between PsA and HOA remains possible, particularly in elderly patients, individuals with long-standing psoriasis, or those with erosive hand OA. For this reason, ultrasound findings should always be interpreted alongside clinical history, physical examination, laboratory investigations, and established classification criteria. When integrated with clinical assessment, musculoskeletal ultrasound substantially improves diagnostic accuracy, facilitates earlier recognition of inflammatory arthritis, and supports more appropriate therapeutic decision-making in patients presenting with hand symptoms. [2]

Current Limitations and Future Perspectives

Despite the growing integration of musculoskeletal ultrasound into routine rheumatology practice, several limitations continue to affect its widespread implementation and interpretation. One of the principal challenges is operator dependency, as image acquisition and interpretation require substantial training and experience. Variations in probe positioning, machine settings, scanning protocols, and examiner expertise may influence the detection of subtle inflammatory lesions, particularly low-grade synovitis, enthesitis, and Power Doppler signals. Consequently, standardized training programs and adherence to validated scanning protocols remain essential for ensuring reproducibility and diagnostic accuracy across different clinical settings. [35]

The overlap of ultrasound abnormalities between inflammatory and degenerative diseases also presents a diagnostic challenge. Synovial hypertrophy and Power Doppler activity are not exclusive to PsA and may be observed in symptomatic or erosive HOA, while osteophytes and degenerative cortical irregularities may coexist with inflammatory arthritis in older patients. Therefore, ultrasound findings should not be considered in isolation but rather integrated with patient history, physical examination, laboratory investigations, and established classification criteria. A comprehensive, pattern-based interpretation remains the most reliable approach for differentiating hand PsA from HOA. [18]

Technological advances are expected to further enhance the diagnostic capabilities of musculoskeletal ultrasound. The development of ultra-high-frequency transducers has significantly improved spatial resolution, allowing more detailed visualization of superficial structures such as nail units, pulley systems, collateral ligaments, and small entheses. In parallel, advances in Doppler sensitivity permit detection of very low-volume vascular flow, improving identification of subclinical inflammatory activity that may previously have been undetectable using conventional equipment. These innovations may contribute to earlier diagnosis and more accurate monitoring of disease activity in both PsA and HOA. [36]

Emerging technologies, including three-dimensional ultrasound, automated image analysis, quantitative ultrasound measurements, and artificial intelligence (AI)-assisted image interpretation, represent promising future directions in musculoskeletal imaging. Machine learning algorithms have the potential to improve lesion recognition, reduce interobserver variability, standardize scoring systems, and facilitate objective assessment of inflammatory and structural changes. Although these technologies remain largely investigational, they may ultimately support precision medicine by providing more reproducible imaging biomarkers for diagnosis, prognosis, and therapeutic monitoring. [37]

Future research should focus on establishing standardized ultrasound protocols specifically for hand arthritis, validating disease-specific scoring systems, and conducting longitudinal studies that correlate ultrasound abnormalities with clinical outcomes, structural progression, and treatment response. Greater integration of ultrasound findings into treat-to-target strategies may further optimize patient management by allowing earlier therapeutic intervention and more accurate assessment of remission and residual inflammatory activity. As evidence continues to accumulate, musculoskeletal ultrasound is expected to assume an increasingly central role in the multidisciplinary care of patients with PsA and HOA. [38,40]

Conclusion

Musculoskeletal ultrasound has become an indispensable imaging modality for evaluating hand involvement in both psoriatic arthritis and hand osteoarthritis by providing comprehensive assessment of inflammatory activity and structural joint damage in a single examination. Compared with conventional imaging, ultrasound offers superior sensitivity for detecting early synovitis, tenosynovitis, enthesitis, dactylitis, cortical erosions, osteophytes, cartilage abnormalities, and Power Doppler vascularity, thereby facilitating earlier diagnosis and more accurate characterization of disease processes.

Although PsA and HOA may share several clinical manifestations and certain sonographic findings, each disorder demonstrates a characteristic pattern of ultrasound abnormalities that reflects its underlying pathophysiology. PsA is distinguished by multisite inflammatory involvement affecting the synovium, tendon sheaths, entheses, periarticular soft tissues, and nail apparatus, frequently accompanied by active Doppler vascularity and a combination of erosive and proliferative bone changes. In contrast, HOA is characterized predominantly by cartilage degeneration, osteophyte formation, cortical remodeling, and secondary synovitis, particularly in erosive disease. Recognition of these disease-specific imaging patterns substantially improves differential diagnosis and supports appropriate therapeutic decision-making.

Beyond its diagnostic value, musculoskeletal ultrasound has become an essential tool for monitoring disease activity, evaluating treatment response, guiding minimally invasive procedures, and identifying subclinical inflammation that may not be apparent on clinical examination. Continued technological innovations, together with standardized scanning protocols and validated scoring systems, are expected to further enhance its diagnostic performance and clinical applicability. Consequently, musculoskeletal ultrasound should be considered an integral component of the comprehensive evaluation and personalized management of patients with hand involvement due to psoriatic arthritis and hand osteoarthritis.

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