International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

# Hyperbaric Osteonecrosis in Divers and Compressed-Air Workers: A Narrative Review

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**Abstract**

Dysbaric or hyperbaric osteonecrosis (HBON) is a chronic skeletal complication resulting from repeated exposure to elevated ambient pressure followed by decompression, most commonly affecting divers and compressed-air workers. Despite long-standing recognition, the condition remains underdiagnosed due to its frequently asymptomatic early stages, delayed radiographic findings, and absence of standardized screening and management protocols. This narrative review synthesizes current evidence on the epidemiology, pathophysiology, clinical manifestations, diagnostic approaches, management strategies, prognosis, and emerging advancements in HBON. A comprehensive literature search of PubMed, Embase, and Scopus databases was conducted for studies published up to July 2025, supplemented by manual reference screening of relevant reviews and authoritative texts in diving medicine. The pathogenesis of HBON involves gas and lipid micro embolism, endothelial injury, and impaired intraosseous microcirculation, leading to ischemic bone damage, particularly in weight-bearing joints. Magnetic resonance imaging remains the gold standard for early detection, enabling identification of subclinical lesions and informed risk stratification. Management is stage-dependent, ranging from conservative monitoring and exposure modification in early disease to surgical interventions, including core decompression and joint arthroplasty in advanced stages. Prognosis is strongly influenced by lesion location and timing of diagnosis, with early detection associated with improved outcomes. Emerging advances in imaging techniques, regenerative therapies, and individualized decompression monitoring hold promise but require robust clinical validation. Greater awareness, early diagnosis, and multidisciplinary research efforts are essential to reduce long-term morbidity and occupational disability associated with hyperbaric osteonecrosis.

## Introduction

Osteonecrosis, commonly referred to as avascular necrosis, is characterized by bone tissue death resulting from impaired perfusion, ultimately leading to mechanical failure of the affected bone and progressive joint degeneration. While the condition arises from multiple traumatic and non-traumatic causes, dysbaric—also termed hyperbaric—osteonecrosis constitutes a unique occupational variant. This form is primarily linked to repeated exposure to increased ambient pressure followed by decompression, as encountered by professional divers and compressed-air (caisson) workers.

The underlying pathophysiology is believed to involve the formation of inert gas bubbles during decompression, which can disrupt intraosseous blood flow, elevate marrow pressure, and provoke ischemic injury within bone tissue. Over time, these microvascular insults may culminate in irreversible osteocyte loss and structural compromise. Despite being recognized for several decades, dysbaric osteonecrosis continues to be underdetected, largely because early disease stages are frequently asymptomatic and conventional radiography often fails to reveal abnormalities until advanced damage has occurred.

Advances in magnetic resonance imaging have enabled the identification of subclinical lesions in individuals with occupational hyperbaric exposure, suggesting that the true burden of disease may be underestimated. However, uncertainty persists regarding optimal screening strategies, timing of intervention, and evidence-based management pathways. These unresolved issues highlight the need for focused synthesis of existing knowledge to inform clinical practice and guide future research in hyperbaric osteonecrosis.

**Methods**

This study was designed as a narrative review to consolidate and critically summarize current evidence relating to hyperbaric osteonecrosis in divers and compressed-air workers. A structured literature search was conducted using the PubMed, Embase, and Scopus databases, covering publications available up to July 2025. Search terms included a combination of controlled vocabulary and free-text keywords such as hyperbaric osteonecrosis, dysbaric osteonecrosis, avascular necrosis, decompression illness, divers, compressed-air workers, and hyperbaric exposure. Boolean operators were applied to broaden and refine retrieval of relevant studies.

To ensure comprehensive coverage, bibliographies of eligible articles, prior narrative reviews, and standard references in diving and hyperbaric medicine were manually examined. This strategy facilitated inclusion of foundational literature alongside more recent studies, allowing exploration of both historical perspectives and contemporary understanding of disease mechanisms, clinical features, diagnostic modalities, management options, and long-term outcomes.

Given the exploratory nature of this review, no rigid inclusion or exclusion criteria were imposed beyond relevance to hyperbaric osteonecrosis. A wide range of study types—including observational studies, case series, clinical recommendations, and expert consensus statements—were incorporated to provide a broad and integrative overview. Research involving non-diving populations was selectively included when it offered mechanistic or conceptual insights applicable to dysbaric osteonecrosis.

Owing to marked variability in study designs, populations, and reported outcomes, statistical pooling and meta-analysis were not undertaken. Formal assessment of methodological bias was likewise not performed. Instead, findings were synthesized qualitatively, with emphasis on recurring themes, areas of agreement, and notable inconsistencies in the literature. This narrative approach enabled contextual interpretation of evidence while identifying knowledge gaps requiring further investigation.

**Ethical Use of Artificial Intelligence**

Artificial intelligence tools, specifically OpenAI ChatGPT (GPT-5), were utilized solely to support manuscript preparation by assisting with language refinement, structural organization, and clarity of expression. All AI-assisted outputs were critically reviewed and substantially edited by the authors to ensure scientific accuracy, originality, and adherence to academic standards. AI was not used for data analysis, interpretation, or generation of scientific conclusions. No patient-specific or confidential data were involved. Full responsibility for the content, references, and interpretations presented in this manuscript rests with the authors.

**Epidemiology**

Dysbaric osteonecrosis (DON) is widely recognized as an occupational disease associated with repeated or prolonged exposure to increased ambient pressure. It has been documented in a range of populations, including caisson workers, military and commercial divers, indigenous or artisanal divers, recreational divers, and individuals engaged in technical diving. Historically, the condition has been reported more frequently among caisson workers than among military or commercial divers, likely reflecting differences in exposure patterns, safety oversight, and decompression practices.

A particularly high burden of disease has been observed among indigenous and artisanal diving communities. These divers often operate with minimal formal training, limited understanding of decompression physiology, and inadequate safety equipment. The use of improvised surface-supplied "hookah" systems without standardized decompression protocols significantly increases risk. For example, studies of artisanal fishing divers in Mexico have demonstrated a notably high prevalence of dysbaric osteonecrosis, underscoring the vulnerability of this group.

With the growing popularity of technical diving, concerns have been raised regarding a potential future rise in DON incidence within this population. Technical dives are characterized by greater depths, extended bottom times, and increased inert gas loading, often involving complex gas mixtures. When combined with insufficient decompression knowledge or unsafe ascent practices, these factors may predispose divers to chronic skeletal complications, including DON.

Across both divers and caisson workers, several risk factors consistently correlate with disease development. These include cumulative exposure to pressure, increased depth and duration of exposure, frequent dives or shifts, inadequate or delayed decompression, unsafe diving practices, advancing age, and poor awareness of preventive measures. Lifestyle and medical factors such as chronic alcohol consumption and long-term corticosteroid use are also believed to increase susceptibility to osteonecrosis and may act as additional contributors in dysbaric settings.

Cardiac right-to-left shunts, particularly a patent foramen ovale (PFO), have been strongly associated with decompression illness. Individuals with a PFO have a significantly higher risk—reported to be up to fivefold—of developing decompression-related complications, as venous gas emboli may bypass pulmonary filtration and enter the systemic circulation. Larger PFOs appear to confer a greater risk of severe decompression illness. While the U.S. Navy estimates a relatively low prevalence of DON among military divers, studies from Japan have reported markedly higher rates, in some cases approaching 50% among commercial divers, reflecting differences in exposure intensity and operational practices.

The disease predominantly affects men, consistent with the male predominance in occupations involving compressed-gas exposure. Multifocal disease typically presents between the third and fifth decades of life. Geographic variation in prevalence has been reported, with higher rates documented in regions such as Japan, Korea, Turkey, and Hawaii. Although modern dive tables, computers, and safety protocols have substantially reduced the incidence of acute decompression sickness, the risk has not been eliminated. In recreational diving, decompression sickness is estimated to occur in approximately 3 per 10,000 dives, whereas commercial diving carries a higher incidence, ranging from 1.5 to 10 cases per 10,000 dives. The likelihood of decompression illness increases with dive depth and duration, and males are reported to be approximately 2.5 times more likely than females to be affected.

**Pathophysiology**

Dysbaric osteonecrosis is considered a chronic skeletal manifestation of decompression illness, arising from microvascular compromise following exposure to rapid or inadequate decompression. The central pathogenic mechanism involves the formation of gas and lipid microemboli that obstruct the blood supply to bone, particularly in the juxta-articular regions of long bones.

During exposure to increased ambient pressure at depth, inert gases—primarily nitrogen—dissolve in body tissues in proportion to the surrounding pressure. When ascent occurs gradually, dissolved gases are safely eliminated via the lungs. However, during rapid ascent or insufficient decompression, ambient pressure falls abruptly, preventing adequate gas elimination. This results in the formation of nitrogen bubbles within tissues and the circulation, which may cause both mechanical obstruction of blood vessels and endothelial injury.

These intravascular gas bubbles can impair intraosseous microcirculation through direct occlusion, activation of coagulation pathways, and inflammatory endothelial damage. The resulting ischemia compromises osteocyte viability and, over time, leads to bone necrosis. In addition to gas embolism, lipid-related mechanisms are thought to play a significant role in the development of DON.

Experimental animal studies and postmortem human investigations have demonstrated that rapid decompression can cause marked expansion of marrow adipocytes. With further decompression stress, these enlarged fat cells may rupture, releasing lipid droplets and cellular debris into the marrow and vascular spaces. Fat emboli can accumulate within medullary sinusoids and small epiphyseal and diaphyseal vessels, producing severe intravascular obstruction. The combined effects of gas and fat embolization exacerbate ischemia, particularly in areas with limited collateral blood supply.

The femoral head is the most frequently affected site, largely due to its relatively tenuous vascularity, which renders it especially vulnerable to ischemic injury. Other commonly involved locations include the proximal humerus, tibia, shoulder girdle, and hip region. Repeated exposure to known risk factors—such as rapid ascent, increased dive depth and duration, high-frequency exposure to pressure, and inadequate decompression—further amplifies the likelihood of developing dysbaric osteonecrosis.

**Clinical Presentation**

Dysbaric osteonecrosis demonstrates a wide clinical spectrum, ranging from completely asymptomatic lesions identified incidentally to advanced, symptomatic disease associated with significant joint dysfunction. In its earliest stages, the condition is frequently silent and is most often detected during routine imaging performed as part of occupational surveillance programs in military, commercial, or offshore diving populations. At this stage, patients typically lack pain or functional impairment despite clear radiological evidence of ischemic bone injury.

As the disease progresses, clinical symptoms gradually emerge. The most common presenting complaint is deep, poorly localized joint pain that initially occurs with activity and weight bearing and later progresses to persistent pain at rest. Associated symptoms include joint stiffness, reduced range of motion, and functional limitation. Weight-bearing joints—particularly the femoral head—are most frequently involved, followed by the shoulder, knee, and proximal humerus. Pain may be exacerbated by prolonged standing, walking, or lifting, and symptoms often worsen over time as structural integrity deteriorates.

Patients commonly present in early to mid-adulthood, typically between 30 and 50 years of age, reflecting the cumulative effect of repeated hyperbaric exposure. A detailed occupational history is essential and often reveals prolonged employment as a professional diver, caisson worker, or involvement in technical or indigenous diving practices with inadequate decompression protocols. Multifocal disease is a recognized feature of dysbaric osteonecrosis, particularly in individuals with long-term or high-intensity

exposure, and may result in simultaneous or sequential involvement of multiple joints.
Clinically, the disease follows a stepwise progression from an initial preclinical phase to advanced structural failure. Early disease is characterized by preserved joint architecture and minimal symptoms, while intermediate stages show increasing pain and functional impairment. In advanced disease, subchondral fracture and collapse of the articular surface lead to secondary osteoarthritis, marked loss of joint function, and significant disability. Once collapse occurs, symptoms typically intensify rapidly, and joint-preserving treatment options become limited.
When compared with osteonecrosis caused by corticosteroid use, chronic alcohol consumption, or trauma, dysbaric osteonecrosis shares similar clinical manifestations but differs in several key aspects. Steroid- and alcohol-related osteonecrosis often presents bilaterally and may progress more rapidly, whereas traumatic osteonecrosis is usually localized to the site of injury and temporally related to a specific event. Dysbaric osteonecrosis is distinguished by its strong occupational association, frequent multifocal involvement, and prolonged asymptomatic phase. Recognition of these clinical patterns, combined with careful exposure history, is crucial for early diagnosis, appropriate risk stratification, and timely intervention.

## Diagnosis

### Imaging Modalities

Imaging constitutes the cornerstone of diagnosis and staging in dysbaric osteonecrosis, particularly because early disease is frequently clinically silent. Conventional radiography is typically the first-line investigation due to its wide availability; however, its diagnostic yield is limited in early stages. Radiographic abnormalities generally appear only after irreversible structural damage has occurred and may include areas of sclerosis, cyst-like lucencies, subchondral radiolucent lines consistent with the crescent sign, and eventual collapse of the articular surface.
Magnetic resonance imaging (MRI) is considered the most sensitive modality for early detection and is therefore regarded as the reference standard. MRI can identify ischemic changes within the bone marrow before alterations become apparent on plain radiographs. Characteristic findings include serpiginous low-signal intensity bands on T1-weighted sequences, corresponding high-signal intensity on T2-weighted or STIR images, and surrounding marrow edema. In addition to early diagnosis, MRI enables accurate assessment of lesion size, anatomical distribution, and extent of involvement, all of which are essential for staging, prognostication, and therapeutic decision-making.
Computed tomography (CT) provides excellent visualization of cortical integrity and trabecular architecture and is particularly useful for detecting subtle subchondral fractures and assessing the degree of mechanical compromise. Although CT is less sensitive than MRI for early marrow changes, it serves as a valuable adjunct in surgical planning, especially when joint-preserving procedures are being contemplated.
Bone scintigraphy was historically used to identify early disease by demonstrating increased radionuclide uptake in areas of altered bone metabolism. However, its relatively poor specificity and limited ability to define lesion boundaries have resulted in a decline in its clinical utility, with MRI largely replacing scintigraphy in contemporary diagnostic algorithms.

### Screening Programs

Given its strong association with occupational exposure to compressed air, dysbaric osteonecrosis has prompted the development of targeted screening strategies in certain military and commercial diving populations. MRI-based screening programs have demonstrated effectiveness in detecting asymptomatic lesions before clinical deterioration or joint collapse occurs. Early detection facilitates stratification of risk, modification of exposure patterns, and timely implementation of preventive or therapeutic measures, potentially mitigating long-term disability and occupational loss. Despite these advantages, the absence of standardized screening intervals, uncertainty regarding cost-effectiveness, and limited availability of resources remain barriers to widespread adoption.

**Laboratory Investigations**

Currently, no laboratory tests or biomarkers are established for the diagnosis or monitoring of dysbaric osteonecrosis. Research efforts have explored indicators of bone turnover, endothelial injury, coagulation abnormalities, and inflammatory pathways; however, none have demonstrated sufficient reliability or clinical applicability for routine use. Consequently, diagnosis continues to rely primarily on a combination of exposure history, clinical assessment, and imaging findings.

**Management**

**Conservative Management**

Treatment approaches for dysbaric osteonecrosis are individualized based on disease stage, anatomical location, symptom severity, and functional limitation. Early-stage or asymptomatic lesions without evidence of structural collapse may be managed non-operatively with close clinical and radiological follow-up. Avoidance or significant reduction of further hyperbaric exposure is a critical component of management to prevent disease progression. Supportive measures, including activity modification and structured physiotherapy, may help preserve joint mobility and function. Temporary limitation of weight-bearing has been advocated to reduce mechanical stress on vulnerable joints, although evidence supporting durable benefit remains largely observational and inconclusive.

**Medical Therapy**

Pharmacological strategies aimed at slowing disease progression have been investigated, though none are currently supported by high-quality evidence specific to dysbaric osteonecrosis. Bisphosphonates have been studied for their ability to inhibit osteoclast-mediated bone resorption and potentially reduce the risk of subchondral collapse. While some reports suggest benefit in early-stage femoral head osteonecrosis, findings are inconsistent, and their role in dysbaric disease remains uncertain.

Other medical interventions, including anticoagulants, antiplatelet agents, and vasodilatory drugs, have been proposed to enhance intraosseous perfusion and counteract ischemic mechanisms. Despite a sound theoretical basis, clinical evidence remains insufficient to support routine use, and these therapies are best regarded as experimental.

**Surgical Management**

Surgical treatment is generally reserved for patients with symptomatic disease, progressive lesions, or a high likelihood of joint collapse. Core decompression is the most commonly utilized joint-preserving procedure in early stages and aims to reduce intraosseous pressure, promote neovascularization, and stimulate bone repair. Clinical outcomes are most favorable when intervention occurs prior to the development of subchondral fracture or articular surface collapse.

In selected cases, bone grafting—either non-vascularized or vascularized—may be employed in conjunction with core decompression to provide mechanical support and enhance revascularization of necrotic bone. Corrective osteotomy has also been used to redistribute load away from affected regions; however, its role has diminished with advances in arthroplasty techniques.

For advanced disease marked by articular collapse and secondary degenerative changes, total joint arthroplasty represents the definitive treatment option. While arthroplasty offers reliable pain relief and functional improvement, considerations regarding implant durability and the likelihood of future revision are particularly important in younger, active patients.

**Emerging Therapies**

Recent research has increasingly focused on biologic and regenerative strategies aimed at preserving joint integrity in early disease. The use of autologous bone marrow–derived mesenchymal stem cells in combination with core decompression has shown encouraging results in reducing pain, slowing disease progression, and promoting bone regeneration in early-stage osteonecrosis. Additional investigational approaches include targeted growth factor delivery, scaffold-based tissue engineering, and gene-modulating therapies. Although preliminary outcomes are promising, further large-scale randomized studies with long-term follow-up are necessary before these modalities can be incorporated into routine clinical practice.

**Prevention**

Prevention of dysbaric osteonecrosis primarily focuses on minimizing decompression-related stress and reducing cumulative hyperbaric exposure. The implementation of safer decompression protocols remains the cornerstone of prevention. Modern dive tables and dive computers, which incorporate conservative ascent profiles, staged decompression stops, and real-time monitoring of depth and bottom time, have significantly reduced the incidence of decompression-related complications. Adherence to established decompression limits, avoidance of rapid ascents, and incorporation of adequate surface intervals are essential measures to limit inert gas supersaturation and bubble formation.

Oxygen pre-breathing has emerged as an additional preventive strategy, particularly in professional and military diving operations. Pre-exposure inhalation of oxygen reduces the body’s nitrogen load by promoting inert gas washout, thereby decreasing the risk of intravascular and extravascular bubble formation during decompression. While oxygen pre-breathing has demonstrated benefit in reducing acute decompression illness in controlled settings, its role in preventing long-term skeletal complications such as dysbaric osteonecrosis is less clearly defined and requires further study.

Education and training play a critical role in prevention, especially in high-risk groups such as indigenous, artisanal, and technical divers. Comprehensive instruction on decompression physiology, safe ascent practices, recognition of early symptoms, and the risks of cumulative exposure can significantly reduce unsafe behaviors. Increased awareness is particularly important in settings where informal diving practices, limited equipment, and economic pressures contribute to high-risk exposure patterns.

Occupational health surveillance programs provide an additional layer of prevention by facilitating early detection and risk mitigation. Structured medical evaluations, periodic imaging—particularly MRI in selected high-risk populations—and documentation of cumulative exposure allow identification of asymptomatic disease and enable timely modification of work practices. Such programs have been successfully implemented in certain military and commercial diving sectors, although their broader adoption is limited by cost, resource availability, and lack of standardized guidelines. Strengthening preventive strategies through a combination of safe decompression practices, education, and surveillance is essential to reducing the long-term burden of dysbaric osteonecrosis

**Prognosis and Long-Term Outcomes**

The clinical course of hyperbaric osteonecrosis is variable and largely determined by the site of involvement, lesion size, and stage at which the disease is identified. Lesions detected at an early stage—particularly those discovered incidentally through surveillance programs in military or offshore divers—often demonstrate a relatively benign course. Long-term observational data indicate that many small, asymptomatic lesions, especially those located in non–weight-bearing bones such as the humerus, may remain radiologically stable for many years without progression.

In contrast, involvement of weight-bearing or mechanically stressed joints carries a substantially poorer prognosis. Lesions affecting the femoral head and shoulder are more likely to enlarge and progress to subchondral fracture and articular collapse. Longitudinal cohort studies have reported progression to joint surface failure in a significant proportion of hip lesions over a 10-year period, frequently necessitating surgical intervention.

Once structural collapse has occurred, therapeutic options become limited, and joint replacement is often required to achieve pain relief and restore function. Although arthroplasty can be effective from a musculoskeletal perspective, the occupational implications are profound. Individuals who undergo joint replacement are permanently excluded from further diving or compressed-air work, resulting in loss of professional identity, financial strain, and long-term vocational limitations.

Beyond physical impairment, the disease exerts a substantial psychosocial impact. Affected individuals commonly experience chronic pain, reduced mobility, and restrictions in daily activities. Sudden cessation of professional diving careers has been associated with psychological distress, including anxiety and depressive symptoms. Quality-of-life assessments consistently demonstrate lower physical and mental health scores among divers with advanced hyperbaric osteonecrosis compared with unaffected counterparts.

Overall, prognosis is closely linked to the timing of diagnosis. Lesions identified early through magnetic resonance imaging and managed with exposure modification and preventive strategies generally have a favorable outlook. Conversely, delayed diagnosis increases the likelihood of irreversible joint damage, invasive surgical intervention, and long-term disability.

**Current Advancements**

Recent progress in the understanding, detection, and prevention of dysbaric osteonecrosis spans several domains, including advanced imaging, experimental biologic therapies, and occupational health innovations. Emerging imaging techniques have shown promise in identifying pathological changes at a stage even earlier than conventional MRI. Magnetic resonance spectroscopy (MRS), for example, has been used to evaluate bone marrow composition in divers, revealing alterations in lipid metabolism and early marrow edema in symptomatic individuals before structural abnormalities become apparent on standard MRI or radiographs. These findings suggest that MRS may detect early microvascular stress and metabolic remodeling preceding overt osteonecrosis.

Experimental work has also explored alternative pathways for detecting inert gas burden. In animal models, ultrasound-based identification of lymphatic gas bubbles has demonstrated that interstitial gas may enter both venous and lymphatic systems before ultimately returning to the systemic circulation. Image brightness analysis of lymph nodes has correlated darker signal patterns with more severe decompression sickness, raising the possibility of indirect bubble detection outside the vascular compartment.

Cardiac imaging has further contributed to understanding decompression physiology. Transthoracic echocardiography using standard clinical ultrasound systems can visualize circulating gas emboli within the right heart chambers. Notably, detectable venous gas emboli are frequently observed in divers even in the absence of clinical decompression sickness, highlighting the complexity of bubble dynamics and individual tolerance.

At the molecular level, preclinical studies have investigated protective strategies against decompression-related injury. In rodent models, pretreatment with normobaric oxygen combined with pharmacologic modulation of intracellular signaling pathways has been shown to reduce oxidative stress, inflammation, and apoptosis, thereby lowering the incidence of spinal decompression injury. While these results are encouraging, translation to human application has yet to be established.

Insights have also been drawn from comparative models. Gas bubble disease in fish, which similarly involves intravascular gas embolism, has been associated with upregulation of heat shock proteins such as HSP70, reflecting cellular stress and injury. Although these biomarkers are well characterized in aquatic species, their relevance and utility in human decompression-related disorders remain unclear.

Advances in regenerative medicine represent another promising avenue. Experimental studies using oxygen-releasing biomaterials in combination with mesenchymal stem cell therapy have demonstrated reduced apoptosis, enhanced angiogenesis, and improved bone regeneration in ischemic animal models. Additionally, qualitative audio Doppler self-monitoring has emerged as a practical post-dive tool for detecting venous gas bubbles, offering divers a simple safety check that may support risk awareness and behavioral modification.

**Research Gaps and Future Directions**

Despite decades of recognition, hyperbaric osteonecrosis remains relatively understudied, and substantial gaps in knowledge persist. Much of the existing literature consists of small case series and retrospective analyses, with a paucity of large, prospective, longitudinal studies. As a result, the true prevalence, natural history, and long-term outcomes of the disease—particularly in the context of modern dive computers and individualized decompression profiles—are not fully understood.

Individual susceptibility represents a critical area of uncertainty. Emerging evidence suggests that genetic and molecular factors, including variations in coagulation pathways, endothelial function, and vascular repair mechanisms, may influence risk. However, these associations have not been confirmed in adequately powered prospective studies. Clarifying the biological basis of susceptibility could enable targeted screening and personalized preventive strategies.

Technological advances in imaging offer considerable promise but remain largely investigational. Modalities such as diffusion-weighted imaging, quantitative T2 mapping, and ultra–high-field MRI have demonstrated potential for detecting microstructural bone changes before conventional imaging abnormalities appear. Artificial intelligence–driven image analysis is also being explored for automated screening and lesion characterization. Robust validation, standardization, and cost-effectiveness analyses are required before these tools can be incorporated into routine occupational health programs.

Therapeutic innovation is another area requiring rigorous evaluation. Regenerative approaches—including mesenchymal stem cell therapy, platelet-rich plasma, and bioengineered scaffolds—have shown encouraging early results, but most studies are limited by small sample sizes, lack of controls, and short follow-up periods. Well-designed randomized controlled trials are essential to determine their safety, efficacy, and durability.

Finally, optimization of decompression strategies remains a priority. Current decompression models are largely based on population averages and do not account for individual variability in physiological response. Integration of wearable sensors capable of monitoring real-time indicators of decompression stress may enable the development of personalized decompression algorithms, potentially reducing disease risk in susceptible individuals.

Future progress will depend on coordinated, multidisciplinary collaboration involving diving medicine specialists, orthopedic surgeons, radiologists, geneticists, and biomedical engineers. Establishing large prospective registries, validating advanced imaging and molecular biomarkers, and rigorously testing regenerative and preventive interventions are essential steps toward transforming hyperbaric osteonecrosis from an underrecognized occupational hazard into a predictable, preventable, and effectively managed condition.

**Conclusion**

Dysbaric or hyperbaric osteonecrosis remains an important yet underrecognized occupational complication of repeated exposure to compressed-air environments. Its insidious onset, prolonged asymptomatic phase, and delayed radiographic manifestations contribute to underdiagnosis and missed opportunities for early intervention. Advances in imaging, particularly magnetic resonance imaging, have improved the ability to detect subclinical disease, emphasizing the value of early surveillance in high-risk populations. Disease progression and long-term outcomes are strongly influenced by lesion location, cumulative exposure, and timing of diagnosis, with weight-bearing joints carrying the highest risk of structural collapse and permanent disability.

Management remains largely stage-dependent, ranging from conservative observation and exposure modification in early disease to surgical intervention in advanced cases. Preventive strategies—including adherence to safe decompression protocols, education, oxygen pre-breathing, and structured occupational health surveillance—play a critical role in reducing disease burden. Emerging advances in imaging, biologic therapies, and personalized decompression strategies offer promise but require robust clinical validation.

Addressing the persistent gaps in epidemiological data, individual susceptibility, and therapeutic efficacy will require coordinated, multidisciplinary research efforts. Large prospective studies, standardized screening protocols, and evidence-based preventive strategies are essential to improving early detection, preserving joint function, and minimizing the long-term physical, psychological, and occupational consequences of dysbaric osteonecrosis.

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