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General

Emerging Pain Management Strategies in Müller-Weiss Syndrome: A Narrative Review

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Purpose of the Review: Müller-Weiss syndrome (MWS) is a rare, complex disorder characterized by osteonecrosis and progressive collapse of the tarsal navicular, leading to chronic midfoot pain, altered biomechanics, and significant functional impairment. Recent Findings: Related to insidious onset, MWS is frequently misdiagnosed early on which can lead to prolonged recovery in patients. Pain generation in MWS arises from many different sources, including but not limited to ischemic necrosis, perinavicular joint degeneration, and potential central sensitization. Diagnostic evaluation using weightbearing x-rays are required and are used in conjunction with computed tomography and magnetic resonance imaging to assess structural deformities and rule out other diseases. Summary: Initial management relies on conservative strategies such as orthotic support or NSAIDs, but in the more advanced stages of the condition, pulsed radiofrequency ablation has been performed, as well as various surgical interventions. In the present investigation, pathophysiology, clinical presentation, pain characteristics, diagnostic challenges, and current management strategies are described related to MWS.

1. INTRODUCTION

Müller–Weiss syndrome (MWS) is an uncommon but serious cause of chronic midfoot pain in adults, associated with progressive collapse of the tarsal navicular.[1,2] Although previously described as a variant of adult navicular osteonecrosis, the pathogenesis of MWS is likely multifactorial and involves biomechanical, remodeling, and vascular mechanisms[1,3,4] Clinically, the condition is challenging to recognize, as most patients present with vague symptoms of slowly progressive dorsal foot pain that is poorly localized.[2,5] To complicate matters, radiographic images obtained from these patients usually appear normal during the early stages of the disease.[5,6] Consequently, patients are frequently misdiagnosed and receive inadequate treatment, leading to a prolonged recovery period before appropriate management is initiated.[7,8]

1.1. PAIN CHARACTERISTICS IN PATIENTS WITH MÜLLER–WEISS SYNDROME

Pain is the cardinal feature of MWS and the primary reason patients seek medical attention.[1,2] It is typically activity-related and improves with rest, especially when early in disease.[4,9] Patients often report that symptoms start after prolonged standing or walking and subside when sitting.[8,9] As the condition progresses, pain becomes more constant and less predictable, which limits patients' ability to perform routine activities of daily living.[2,7] In most cases, it is localized to the dorsal aspect of the midfoot, although it may also be present over the medial aspect of the foot.[3,5] Pain is often exacerbated by weight-bearing, and patients may adopt a limp or shorten their stride to reduce the load on the affected foot.[4,7]

The pattern of pain can be quite informative, as most patients with MWS report that the most common site is localized to the dorsal aspect of the midfoot.[3,5] However, the distribution of pain is often not specific, and the condition can be mistaken for numerous other disorders such as midfoot osteoarthritis, stress fracture, and posterior tibial tendon dysfunction.[5,7,10] In addition, plain radiographs may appear normal in the early stages of the disease.[5,6] As a result, patients are often misdiagnosed and receive inadequate treatment.[7,8]

As the disease progresses, patients begin to compensate for the loss of the normal function of the navicular.[1,4] This often leads to the development of additional symptoms. For example, they may shift some of the load from the affected limb to the contralateral side, which can cause lateral foot pain.[4,7] Some patients also develop pain in the hindfoot or knee as a result of biomechanical abnormalities.[1,7] In advanced cases, severe deformity can occur, including paradoxical flatfoot or hindfoot varus.[1,7,11] In such patients, pain is no longer predictable and can be present at all times, independent of activity levels.[2,4] Overall, the pattern of pain can provide valuable information about the

stage of the disease and the nature of biomechanical abnormalities.[1,7]

Chronic pain significantly impacts the health-related quality of life of patients with MWS.[2,12] Severe cases can be especially debilitating, limiting ability to work and engage in physical activities.[2,12] The condition can also have a psychological impact. Patients often report being frustrated or depressed by their limited functional capacity.[12] Furthermore, there is often a disconnect between the degree of radiographic changes and the level of pain experienced by the patient, which suggests that MWS may have a significant neuropathic component that is not captured by conventional radiographic studies.[5,8] Such pain often requires more aggressive management and should be considered when developing a treatment plan.[4,13]

1.2. CLINICAL PRESENTATION AND PROGRESSION OF MÜLLER–WEISS SYNDROME

MWS primarily affects the medial column of the foot, with the tarsal navicular being the key structure that is involved.[1,3] As such, the biomechanical changes that occur in this condition often lead to progressive collapse of the medial longitudinal arch.[1,11] This results in a number of secondary effects, including altered load distribution across the midfoot, compensatory gait abnormalities, and increased stress on the joints of the hindfoot and ankle.[1,4,7] In essence, MWS rarely presents as an isolated midfoot condition, but rather as a complex biomechanical disease that affects the entire lower limb.[2,4]

The initial symptom in most patients is vague pain on the dorsal aspect of the midfoot that is poorly localized.[5,8] Although it may be sharp and stabbing, it is often described as a dull ache that intensifies with activity.[3,9] In addition, many patients report that the pain is worse on the medial side of the foot.[3,5] These symptoms are often accompanied by swelling of the midfoot and difficulty bearing weight on the affected foot.[7,8] As the condition progresses, patients begin to compensate for loss of function of the navicular, which leads to additional symptoms, including lateral foot pain, as mentioned above.[4,7]

On physical examination, patients with MWS may have swelling and tenderness over the midfoot and the medial aspect of the foot.[7,8] In addition, most patients have a visible deformity of the midfoot, particularly in later stages of the disease.[1,7,11] Gait analysis can also provide valuable information, as many patients exhibit a limp or have a shortened stride on the affected side.[4] Overall, the physical examination is often consistent with a diagnosis of midfoot arthritis, and additional tests are often needed to rule out other conditions.[5,7,10]

Radiographic evaluation typically shows a number of characteristic changes, which can help in the diagnosis of MWS.[5,6] Specifically, there is often a decrease in the height of the medial longitudinal arch and collapse of the tarsal navicular.[5,6,11] The medial cuneiform is often affected too, leading to a decrease in the height of the midfoot.[1,7] The severity of these changes is usually correlated with the stage of the disease, with later stages being associated with more severe deformity.[1,7,9]

1.3. CONSIDERATIONS FOR DEVELOPING A PAIN-CENTERED TREATMENT STRATEGY

The management of MWS is challenging, as most patients present with chronic pain and functional limitations.[2,13] In addition, the disease is often slow to progress, which makes it difficult to determine the optimal time to intervene.[4, 13] The standard of care has historically centered on using orthoses to redistribute pressure and alleviate discomfort in the targeted area.[4,9] Such measures, however, are often insufficient to manage the symptoms of MWS, especially when cases are advanced.[4,13] In addition, they do not address the underlying biomechanical abnormalities, which can lead to the development of other conditions like degenerative joint disease of the hindfoot and ankle.[1,7,14]

It is also important to recognize that most patients living with MWS experience a significant decrease in their overall well-being and daily quality of life.[2,12] Chronic pain and limited mobility can make it difficult for them to work and engage in daily activities.[2,12] Additionally, the disease can have a psychological impact, as many patients report being frustrated and depressed by their condition.[12] Such effects should be taken into consideration when developing a treatment plan, as they may require additional interventions, including counseling or medication.[9,12]

Overall, the current understanding of the natural history of MWS indicates that a pain-centered approach to management is more likely to yield positive outcomes.[4,13] Specifically, treatment should be individualized and take into consideration the stage of the disease, as well as the specific factors that contribute to the patient's pain and functional limitations.[1,7,13] In addition, a multidisciplinary approach that involves the use of orthoses, medication, and surgical intervention when necessary is likely to be more effective than a single modality in most cases.[4,9,13]

2. METHODOLOGY

The present investigation was conducted using the PubMed database to identify relevant studies that examined and focused on the clinical presentation, pathophysiology, biomechanics, and management of Müller-Weiss syndrome. Articles were identified using relevant key words and Boolean combinations. These included "Müller-Weiss syndrome", "Müller-Weiss disease", " navicular osteonecrosis", "pathophysiology", and "central sensitization". There were no strict date inclusions and relevant papers were selected based on their focus on the disease. Additional studies were also selected by manual review of the reference sections of selected articles. There was also priority given to peer-reviewed papers, systematic reviews, and high-level evidence.

3. PATHOPHYSIOLOGY AND PAIN GENERATION

MWS is not just a structural problem with the navicular bone, but has a key degenerative process stemming from pathophysiologic stages that drive the pain associated with the condition.[5] The pathophysiology behind MWS is that there is progressive osteonecrosis from ischemic changes

that eventually leads to structural collapse from fragmentation and sclerosis of the bone.[3] These ischemic changes occur because the central third of the navicular bone is a vascular watershed zone between the dorsalis pedis and the posterior tibial artery, meaning it is naturally susceptible to ischemia when under mechanical load.[15] This ischemia leads to osteocyte death, which leaves the bone unable to handle the chronic compressive forces it normally withstands. This failed response produces degenerative changes concentrated on the lateral half of the navicular bone, generating the classic comma-shaped deformity of the navicular on imaging.[5]

These pathophysiologic changes set off a progressive collapse of the medial column architecture. As the lateral navicular collapses, the remaining medial fragment subluxates dorsomedially,[16] pushing the talus into lateral deviation and produce a hindfoot varus deformity of the affected foot.[16] In addition, this same collapse of the navicular bone undermines the medial longitudinal arch, generating the flatfoot with varus deviation seen in MWS.[2] Furthermore, in severe cases, sufficient navicular destruction may result in the talus directly articulating with the lateral cuneiforms, resulting in complete collapse of the medial column.[17]

These structural changes result in altered load transmission through the midfoot joints. The structural changes to the navicular shift most of the compressive forces from the first metatarsal and tarsometatarsal joints directly onto the second metatarsal joint.[18] This overloads the second metatarsal joint leading to hypertrophic bone changes and articular joint degeneration.[18] As mentioned previously, there will be progressive arch failure which will force the tibia into external rotation that can predispose to poor knee mechanics and eventually osteoarthritis.[2]

The pain behind MWS stems from both biomechanical and neurobiological pain mechanisms. In addition to the metatarsal load shift, there is abnormal stress distribution across the talonavicular and surrounding joints.[1] While the talonavicular joint normally distributes force equally across the navicular when under stress, the navicular deformity in MWS disrupts this stress distribution, creating pathologic stress at the articular surface.[5] This asymmetric stress causes subchondral bone stress, in addition to marrow edema and cortical erosions at the talonavicular joint which serves as a direct source of pain.[19] The deformed navicular surface therefore accelerates cartilage wear and leads to a cycle of mechanical pain when the foot is loaded.

As the cartilage continues to degrade under load, the talonavicular joint releases inflammatory cytokines that further accelerate joint destruction, making the arthritis a self-sustaining degenerative process.[20] Specifically, IL-1β and TNF-α are the main cytokines in this process.[21] They both upregulate matrix metalloproteinases that degrade the cartilage matrix while also inhibiting collagen synthesis from chondrocytes that are needed for repair.[21] Not just the talonavicular joint is affected, as progressive disease can spread to the naviculocuneiform and other perinavicular joints, resulting in a broad zone of pain across the foot.[20] This osteoarthritic pain is different from the initial ischemic pain from osteonecrosis. The progressive arthritic pain is load-dependent and can worsen with increased activity and typically improves with rest, distinguishing it from the initial ischemic pain.[22]

In addition to the static weight-bearing pain, there is a dynamic pain cycle that is exacerbated by a normal walking gait. During the midstance of the gait cycle, the talar head is firmly seated into the navicular and forms an arch that supports the full body weight on the foot.[23] A deformed navicular cannot accommodate this load without an abnormal stress concentration.[24] This instability carries into the push off phase, where normally, the midtarsal joint firmly locks so that the foot is rigid for the propulsion needed.[23] The structurally altered navicular inhibits this locking mechanism which forces the surrounding joints to compensate and carry a disproportionate load.[24] Together, these two phases result in inadequate load transfer that causes repetitive microtrauma with every step that further exacerbates the pain.[24]

In addition to the biomechanical pain associated with MWS, there is also a neurobiological pain mechanism that further exacerbates this condition. While the molecular pain mechanisms specific to MWS have limited studies, they are likely analogous to similar degenerative bone and joint conditions. Over time, subchondral bone is damaged by the abnormal stress caused by the deformed navicular bone, and new sensory nerve fibers may grow into the bone and directly increase pain signaling.[25] This process may be driven by nerve growth factor (NGF) that is released during the extensive bone remodeling that takes place in MWS , heightening the sensitivity of peripheral nociceptors to pain.[25] Furthermore, the inflammatory mediators released by the surrounding bone don't just damage the tissue but also lower the activation threshold of the nociceptors so that the slightest joint movement can be intensely painful.[26] The combination of new nerve growth triggered by NGF and increased peripheral nociceptor sensitization likely explains why MWS can cause pain that is out of proportion to what's expected from the degree of navicular damage.

In addition to the heightened pain from peripheral nerves, it is possible there is also central sensitization in chronic cases of MWS. Prolonged peripheral nociceptor activation from the degenerative navicular and surrounding joints can drive central nervous system changes in the dorsal horn of the spinal cord.[27] The neurons in this area can become hyperexcitable from an upregulation of NMDA receptors which will lead to a lower signal threshold needed to trigger pain.[28] This central sensitization may be the reason why the pain with MWS can be triggered by what would normally be non-painful input and results in pain out of proportion to the degree of tissue destruction.

As a result of central sensitization, the pain associated with MWS may transition from primarily peripheral nociceptive pain associated with the structural damage, to a mixed nociplastic pain phenotype. This occurs when sustained nociceptive pain input over time starts to shift towards a mixed nociplastic-nociceptive pain phenotype where pain becomes out of proportion and exceeds the typical nociceptive pain that structural findings alone usually

cause.[29] This type of pain is widespread beyond the navicular and its surrounding joints, with hypersensitivity to normally non-painful stimuli and usually persists chronically.[30] Ultimately the combination of peripheral nociceptive pain, potential central sensitization, and the eventual mixed nociplastic pain phenotype make MWS unique. It is not just a structural problem causing the pain, but with prolonged exposure, can become a self-sustaining process of chronic pain independent of the original navicular pathology.

4. CLINICAL PRESENTATION AND DIAGNOSTIC EVALUATION

4.1. SYMPTOM PROFILE

MWS is insidious in nature. It often presents in 40 to 50 year old patients with no traumatic history.[31] Even though it has been determined that trauma does not cause MWS , it has been found to expedite the degenerative process in this syndrome.[6] The typical patient demographic for MWS is often a female who is in their fourth to sixth decade of life and has a high body mass index.[32] MWS often begins with a vague pain in the midfoot area. Patients will often report lateral ankle instability, peroneal tendonitis, and limited subtalar mobility.[33] Most painful symptoms occur during weight-bearing activity.[6] One of the main drivers of pain is the alteration of pressure distribution: reduced pressure on the toes with an increase in plantar pressure on the midfoot produces pain. Patients often present with chronic pain on the dorsum of both feet, usually in an asymmetric manner.[1] Bilateral distribution is more common than unilateral, which also supports the notion that MWS is a primary, spontaneous disease of osteonecrosis.[34] Pain in the knee joint can also be a presentation due to the altered mechanics of the whole lower limb, a secondary manifestation being changes in the foot.[33]

4.2. PHYSICAL EXAM FINDINGS

There is a wide range of physical exam findings from severe midfoot pain with restricted mobility to severe foot deformities with no pain.[31] When palpating the foot over the dorsomedial aspect of the navicular, swelling and tenderness is seen in majority of patients.[35] Subtalar varus causes a lateral column overload, so there could also be tenderness over the sinus tarsi, calcaneocuboid and peroneal tendons.[32] The hallmark of this MWS is paradoxical pes planus: midfoot medial arch is collapsed causing a flatfoot deformity paired with a rearfoot subtalar varus.[11] Although, depending a severity, the medial arch of the foot could be preserved, flat, or even high in conjunction with a rearfoot varus.[1] When a patient is being physically examined, their gait should be observed without footwear, so the foot and ankle can be visualized. Persistent collapse of the medial arch and rearfoot varus throughout the entire transition of toe-off up until the whole foot is planted is a key finding. Additionally, an anatomical inspection of the foot should be done to check for abnormalities: pes cavovarus, brachymetatarsia of the first metatarsal, and metatarsus adductus.[32] Even though patients develop a paradoxical pes plano-varus, rearfoot alignment is rarely a significant issue.[7] Rearfoot varus may not be apparent on inspection and might only be detected through palpating the rearfoot during heel rise.[1,31] The prominence of the navicular on the medial aspect of the foot often causes a false identification of rearfoot valgus.[22] As the navicular bone collapses and pes planus varus deformity develops in the disease process, the tibia externally rotates, changing lower limb biomechanics and increasing the likelihood of the development of knee arthritis.[5]

4.3. IMAGING AND STAGING

To make a clinical diagnosis of Müller Weiss Syndrome, diagnostic imaging is required. A weightbearing x-ray of both feet is first line when ruling out MWS.[11] The radiological findings may mimic osteonecrosis.[33] The normal bone morphology of the navicular bone is characterized by a pyriform shape.[36] In contrast, the navicular displays a comma-shaped configuration, decreased size, and increased radiodensity.[6] These characteristics can be visualized from an anteroposterior view. The changes in the navicular shape are due to the collapse and fragmentation of its lateral portion. Perinavicular arthritis is a common finding displaying dorsolateral fragmentation of the navicular, osteophytes, and sclerosis.[33]

Additionally, it is common to see a hypertrophic long second metatarsal. The rest of the metatarsals look atrophic because of the increased load across the second metatarsal.[32] There are several signs visible on a lateral view x-ray as well: the sinus tarsi “see-through” sign due to supination and varus of the rearfoot, a laterally displaced and widened talar head, and a double talar dome shadow with posterior shifting of the fibula due to external rotation of the rearfoot and ankle.[1] The first tarsometatarsal joint is positioned perpendicular to the longitudinal axis of the foot instead of its normal medial position. The second tarsometatarsal joint is externally rotated instead of its normal perpendicular orientation to the longitudinal axis of the foot.[32] “Cuboid sign” is also seen: medial shift of the cuboid in relation to the calcaneus.[22]

Weightbearing x-rays are usually sufficient, but CT scans are useful for assessing bone stock and preoperative planning.[37] The CT is useful in identifying degenerative changes in the subtalar joint and determining if an arthroscopic triple arthrodesis would be the next best plan of action in treatment.[7] Standard CT doesn’t provide appreciable information about the functionality of the rearfoot in relation to the midfoot, so if a CT is done, it must be weightbearing to provide useful information regarding bony alignment under functioning conditions.[22]

Magnetic resonance imaging (MRI) can also be used, but MRI is most useful for preoperative planning and the exclusion of other disease processes.[31] MRI assists in ruling out other causes of midfoot pain: stress fractures, osteonecrosis, and osteomyelitis.[32,38] As the condition worsens, an MRI is usually done to monitor the progression, which can reveal a bone marrow edema compatible with osteonecro-

sis.[6] An MRI is most helpful when assessing soft tissue structures and detecting perinavicular arthritic changes early. Bone marrow edema, loss of bone marrow intensity, and effusion in adjacent joints are the main conditions able to be appreciated in an MRI.[22] In regards to understanding the progression of this syndrome, MRIs and CTs provide a better representation of progressive changes.[35]

5. PAIN PHENOTYPES AND MECHANISTIC STRATIFICATION IN MWS

5.1. PAIN PHENOTYPES

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with or resembling that associated with actual or potential tissue damage".[39] Current pain classification frameworks divide pain into nociceptive, neuropathic, and nociplastic phenotypes based on localization, sensation, symptoms, and surrounding tissue damage.[40] Some patients have been found to show a mixed phenotype consisting of multiple types of pain together.[41] Nociceptive pain comes from damage or threat of damage to non-neural tissue. Neuropathic pain is from somatosensory nervous system damage and often involves feelings of pins, needles, numbness, and muscle weakness. Nociplastic pain consists of altered nociception with no evidence of tissue damage activating nociceptors and often involves fatigue, sleep problems, and cognitive issues.[30,39-41] Phenotyping pain in musculoskeletal disorders can provide direction for pain management, improve patient understanding, and help guide patient lifestyle and exercise recommendations.[41]

Describing MWS in a pain phenotype framework can be valuable for both patients and clinicians. Current literature does not specifically categorize pain in MWS as one certain phenotype. However, using descriptions of pain and symptoms from MWS studies in conjunction with expert consensus features for pain phenotype classification, it appears that most MWS literature documents pain that is predominantly consistent with the nociceptive phenotype.[1,12,22,40,42,43] In MWS studies, patients often present with localized tenderness, chronic midfoot pain that is worsened by weight-bearing, and functional limitation from structural deformity and degenerative changes.[1,12,42,43] These features, along with symptomatic improvement after offloading strategies and arthrodesis, support a nociceptive phenotype for MWS pain.[22,43] Neuropathic pain symptoms such as burning, numbness, and paresthesia have not been reported in the available MWS literature. However, one recent MWS case report identified imaging findings suggestive of chronic Baxter neuropathy secondary to altered foot mechanics with no neuropathic pain reported.[44] A radiologic study reported that symptom severity may not always correlate with radiographic stage, suggesting that factors beyond structural pathology may contribute to pain, such as potentially nociplastic mechanisms in select patients.[35] However, no other evidence suggestive of nociplastic pain, such as widespread pain or central sensitization, was reported.[35] Therefore, based on the current evidence, pain from MWS appears to be mostly nociceptive, with neuropathic and nociplastic pain possible, but not widely documented.

5.2. MECHANISTIC STRATIFICATION

Several overlapping mechanisms appear to contribute to nociceptive pain in MWS. The predominant driver of pain may evolve as the disease progresses. In earlier stages, pain is likely primarily related to mechanical stress from abnormal load transmission and navicular compression.[1,22,45] As structural deformity progresses, degenerative changes involving the talonavicular and naviculocuneiform joints likely become greater contributors to nociceptive pain.[1,43,45] Osseous abnormalities, soft tissue damage, and compensatory biomechanical changes likely become stronger contributors to pain as MWS progresses further.[1,35,44,46] Although neuropathic and nociplastic mechanisms have not been widely documented in MWS patients, it is possible that select patients may experience these pain mechanisms in chronic disease.[35,44]

One of the strongest nociceptive pain generators in MWS is mechanical dysfunction. Alterations in the normal load transfer across the midfoot cause excessive pressure on the navicular bone resulting in progressive deformation, lateral shift of the talar head, and hindfoot varus.[1,45] One proposed biomechanical mechanism for MWS is the "nutcracker effect," where a shortened first metatarsal bone causes compressive forces to be transferred to the second, third, and fourth metatarsals. This squeezes the lateral region of the navicular and causes the talar head to override the calcaneus, which may result in subtalar varus and damage to the lateral structures of the ankle and foot.[46] Collectively, the mechanical changes associated with MWS contribute to flattening of the medial longitudinal arch, midfoot collapse, perinavicular degeneration, talonavicular and naviculocuneiform arthritis, and/or paradoxical pes planus varus.[38,45] Plantar pressure studies further support mechanical causes of nociceptive pain by demonstrating abnormal pressure distribution and altered loading patterns in patients with MWS.[42] The biomechanical abnormalities in MWS likely generate nociceptive pain through ongoing mechanical stimulation of peripheral nociceptors during weight-bearing activities.[1,42,47]

Degenerative changes are another likely cause of nociceptive pain in MWS. Progressive collapse and deformation of the navicular alter joint congruity and promote degeneration of adjacent articulations. This promotes perinavicular osteoarthritis and chronic pain.[1,22,45] Degeneration most commonly occurs at the talonavicular and naviculocuneiform joints, where secondary arthritis contributes to progressive deformity, loss of function, and weight-bearing pain.[35,38,43,45] Imaging studies show osteoarthritic changes, cartilage degeneration, subchondral cyst formation, and joint effusions in cases of advanced disease, which support degenerative changes as a likely cause of pain in these patients.[35] As disease severity progresses, perinavicular osteoarthrosis and collapse of the medial arch become increasingly apparent, further exacerbating pain and functional impairment.[35,46] These degenerative changes likely

cause nociceptive pain through ongoing nociceptive input associated with chronic tissue damage and joint inflammation.[35,47]

Osseous pathology of the navicular bone also likely contributes to pain in MWS. Progressive compression, collapse, and fragmentation of the navicular may contribute to pain through abnormal loading and bone damage.[1,45] Several authors have proposed osteonecrosis or ischemic injury as contributing factors in MWS progression. However, histological confirmation remains inconsistent.[33,45,48] Imaging studies show bone marrow edema, cystic changes, and other reactive osseous abnormalities within the navicular of patients with MWS, which support bone pathology as a contributor to their pain.[22,35,44] MRI findings of increased T2 signal intensity, subchondral changes, and fragmentation in patients with advanced disease also support progressive structural deterioration and osseous failure as potential pain generators.[35,44] The osseous abnormalities in MWS likely cause nociceptive pain through persistent nociceptive input from ongoing structural deterioration and tissue injury within the navicular.[35,44,47]

Secondary pathology beyond the navicular bone may also contribute to pain in MWS patients. Imaging studies have shown soft tissue edema, joint effusions, synovitis, and adventitial bursitis in patients with advanced MWS. This suggests that inflammation and periarticular irritation may worsen pain.[35,44] Adjacent structures may also be involved, with peroneal tendon and calcaneocuboid pain commonly reported in patients with MWS.[46] Knee pain and degenerative knee changes have also been documented in patients with MWS, suggesting that the effects of the disease can extend beyond the foot, possibly due to lower-limb compensation and altered biomechanics.[1,33,46] Secondary pathologies from chronic MWS may contribute to nociceptive pain through inflammation, periarticular irritation, and mechanical stress that activate peripheral nociceptors in structures beyond the navicular itself.[35,44,46,47]

6. CONSERVATIVE AND PHARMACOLOGIC MANAGEMENT

Mechanical offloading is a significant factor in management of MWS designed to reduce stress on the damaged navicular bone while also alleviating pain.[31,45] The first step of conservative treatment is to use orthotic supports and gait aids to reduce the stress on the talonavicular joint and the naviculocuneiform joint.[45,49] However, for more advanced conditions, it is also important to form a combined therapy of orthotic support with a rocker sole shoe.[45] Orthotic support has been shown to reduce the need for surgery by 80% regardless of how advanced the MWS is.[45,50] In addition to orthotic supports, patients are encouraged to modify activities that exacerbate symptoms, such as prolonged standing, high impact exercise, or repetitive weight bearing tasks.[9] The use of orthotic supports in addition to activity modifications and mechanical offloading may reduce symptomatic flare-ups and improve overall management of MWS. Lastly, appropriate footwear has been shown to reduce joint motion and improve mechanical stress across the damaged navicular bone.[9,31,45] Shoes with rigid soles or rocker bottom designs will promote an efficient walking pattern that will reduce pain and improve symptoms of MWS without the need for surgical intervention.[31,45]

Non-steroidal anti-inflammatory drugs (NSAID's) have been shown to reduce inflammation and aid in chronic pain in amongst many types of diseases.[51] They decrease the formation of arachidonic acid derivatives by inhibiting cyclooxygenase 1 (COX-1) and cyclooxygenase 2 (COX-2) enzymes.[52] However, like many chronic conditions, NSAIDs fall in the conservative treatment category for MWS and there is minimal research displaying their relative efficacy at treating this condition. Due to the rarity of MWS , there has not been any sufficient data implicating the pharmacologic benefit of NSAIDS, opioids, or adjuvant neuropathic agents.

7. INTERVENTIONAL AND SURGICAL PAIN MANAGEMENT

7.1. IMAGE-GUIDED INTERVENTIONS

Conservative management remains the first-line approach for MWS, particularly during the early stages of disease progression. Common non-surgical treatments include physiotherapy, activity modification, orthotic support, and mechanical offloading to reduce stress on the affected joints.[13,45] Although these interventions do not reverse the underlying disease process, they can relieve symptoms. Image-guided interventions also play an important role in diagnosis and symptom management, with corticosteroid injections and diagnostic anesthetic blocks being the most frequently used procedures.

Corticosteroid injections have long been used in the management of musculoskeletal disorders to reduce joint inflammation and may provide short-term pain relief, particularly when combined with other conservative measures.[53]

Diagnostic anesthetic blocks represent another useful image-guided intervention in the management of MWS by helping identify the specific joint responsible for a patient's symptoms before surgical intervention and guide treatment decisions.[54] Image-guided techniques using fluoroscopy, computed tomography (CT), or MRI provide improved visualization of the joint space, allowing for more precise needle placement.

As a result, image-guided diagnostic blocks improve both diagnostic reliability and therapeutic effectiveness compared with blind injection techniques.[54]The role of image-guided interventions remains limited and is largely dependent on disease stage. As MWS progresses to stages III and IV, conservative management alone often becomes insufficient, and surgical procedures such as talonavicular arthrodesis or triple arthrodesis become increasingly appropriate. In advanced disease, image-guided interventions are primarily used as adjunctive therapies for symptom control rather than definitive treatment.[13]

Pulsed radiofrequency ablation is another interventional pain procedure that has been performed for foot pain related to MWS. This treatment modality involves the use of radiofrequency waves to reduce pain around the navicular bone without affecting motor nerves. In this regard, world champion Rafael Nadal, who suffers from MWS, had successful pulsed radiofrequency ablation back in June of 2022, just prior to winning the French Open.

7.2. SURGICAL OPTIONS

While image-guided interventions and other conservative therapies can relieve symptoms during the early stages of MWS, they are often insufficient to prevent disease progression in advanced cases. Surgery is therefore considered when pain, deformity, or functional impairment persist despite conservative management.[55] The goals of surgery are to restore alignment, stabilize the medial column, relieve pain, and improve overall function.

The choice of surgical procedure depends on the stage of disease, degree of deformity, joint involvement, and the presence of osteoarthritis. Joint-sparing procedures may be considered during the early stages of MWS , when preservation of the native joints remains possible. One minimally invasive technique is percutaneous decompression of the navicular bone, which can be performed before joint fusion becomes necessary.[5] However, the evidence remains limited, as isolated percutaneous decompression was identified in only one publication included in the systematic review.[13] Another joint-preserving procedure is calcaneal osteotomy, which may correct hindfoot malalignment, redistribute mechanical loading, and relieve pain while preserving joint motion.[13]

As MWS progresses, collapse of the navicular bone and secondary degeneration of the surrounding joints often make joint-preserving procedures insufficient. In these cases, arthrodesis becomes the preferred surgical treatment to stabilize symptomatic degenerative joints, restore the longitudinal arch, and relieve pain.[5] The choice of fusion procedure depends on the joints involved, the severity of deformity, and the degree of osteoarthritis present.[13] Arthrodesis is the predominant surgical treatment for advanced MWS, accounting for approximately 80% of the surgical procedures described in the current literature.[13]

Among the available fusion techniques, talonavicular-cuneiform fusion was the most frequently used for patients with Maceira stage III–V disease.[13] It addresses the structural deformity and mechanical instability associated with progressive navicular collapse by restoring the alignment of the medial longitudinal arch, improving foot biomechanics, relieving pain, and re-establishing medial column height and stability.[55,56] Triple arthrodesis is generally reserved for patients with advanced-stage MWS involving multiple degenerative joints and, in some cases, is combined with talonavicular arthrodesis to maximize correction of the deformity. This combined approach has demonstrated significant radiographic correction while improving overall foot alignment and function.[13]

Despite favorable clinical outcomes, surgical intervention should not be viewed as a complete cure for MWS . Rather than eliminating pain entirely, the primary objective of surgery is to restore structural alignment, stabilize the foot, slow disease progression, and improve overall function. While surgery provides structural correction and improved biomechanics, postoperative pain management and rehabilitation remain essential to optimizing recovery and long-term outcomes.

7.3. POST-SURGICAL PAIN MANAGEMENT

Although surgical intervention restores structural stability and improves foot alignment, successful treatment of MWS extends beyond the operation itself. Effective postoperative pain management requires a comprehensive approach that begins before surgery, continues throughout the perioperative period, and extends into long-term rehabilitation. Enhanced Recovery After Surgery (ERAS) protocols, multimodal analgesia, physical therapy, and patient education all contribute to optimizing recovery while minimizing postoperative pain and complications.[57]

ERAS is a multidisciplinary, evidence-based protocol designed to reduce the physiologic stress associated with surgery while improving postoperative recovery.[57] Patients who receive comprehensive counseling regarding their diagnosis, surgical procedure, expected recovery, and postoperative rehabilitation are generally better prepared to participate in their recovery and may experience greater satisfaction with their surgical care.[57] Multimodal analgesia (MMA) is another key component of perioperative pain management. Rather than relying solely on opioids, MMA combines medications with different mechanisms of action, including acetaminophen, NSAIDs, gabapentin, regional nerve blocks, and local anesthetic infiltration.[57] This approach provides superior pain control while significantly reducing postoperative opioid requirements.

Successful surgical correction of MWS does not immediately restore normal foot mechanics. Following procedures such as talonavicular or triple arthrodesis, patients must gradually adapt to altered biomechanics resulting from joint fusion and changes in foot alignment.

Physical therapy plays a central role in postoperative rehabilitation by helping patients regain strength, improve balance, restore gait mechanics, and safely adapt to changes in foot alignment after surgery. Structured rehabilitation and appropriate gait training facilitate a safe return to mobility while promoting functional recovery.[58] Even when surgery is technically successful, structural correction does not eliminate the risk of persistent postoperative pain. Recent evidence from foot and ankle surgery suggests that pain outcomes are influenced by multiple factors, including preoperative pain severity, perioperative pain management strategies, and postoperative rehabilitation.[59] This delayed intervention may partially contribute to persistent postoperative pain despite successful structural correction.

Ultimately, successful management of MWS extends beyond surgical correction of the deformity. Continued pain management, rehabilitation, and patient education remain integral components of long-term care.

CONCLUSION

MWS is a complex and progressive disorder that causes substantial chronic pain and functional impairment through a combination of tarsal navicular collapse, altered biomechanics, joint degeneration, and inflammation. Treatment should be individualized based on the patient's clinical presentation, as MWS can vary between patients. Its insidious presentation and nonspecific symptoms can delay diagnosis, highlighting the importance of appropriate clinical evaluation and weightbearing imaging. Earlier recognition is important as it can allow for timely intervention that preserves function and slows down disease progression before substantial deformity and joint degeneration develop. Therefore, more reliable methods for detecting MWS at an early stage are needed. Further research into MWS can also help better determine whether different pain phenotypes are present. MWS pain currently appears predominantly nociceptive, although neuropathic and nociplastic categorization may be possible if more studies are conducted. Current management of the condition is primarily focused on symptom control and preservation of function, with conservative strategies such as orthotic support, activity modification, and mechanical offloading serving as initial approaches. Image-guided interventions may provide additional symptomatic relief, while surgical correction is generally reserved for patients with persistent pain who have exhausted other treatment options. Imaging with CT or MRI may provide additional information regarding structural deformity and preoperative planning.

Although surgical intervention can improve the functional outcomes, postoperative pain management and rehabilitation remain essential to achieving optimal results. Given the rarity of MWS and the limited evidence available, further research is needed to better characterize its pain phenotypes and enable earlier detection. The current literature of MWS is also mainly case reports and small observational studies, which makes it difficult to establish standardized diagnostic and treatment protocols. Larger prospective studies are needed to better evaluate the effectiveness of available interventions and identify factors associated with improved long-term outcomes.

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