UHN Schroeder Arthritis Institute

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## Schroeder Arthritis Institute

The **Schroeder Arthritis Institute** is the largest multidisciplinary arthritis centre in Canada, integrating medical, surgical and basic science aspects of **Hand, Orthopedics, Osteoporosis and Rheumatology**. All staff in the Institute share the common goal of making a global impact on patient care through discovery, learning and innovative therapeutics.

We hope that these clinical case discussions will provide readers with new insights into the diagnosis and management of complex clinical problems, as well as the benefits of the multi-disciplinary approach that is intrinsic to the care of patients at the Schroeder Arthritis Institute.

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## In This Issue

In **Case 1**, Dr. Leigha Rowbottom and Dr. Lori Albert present the workup and surprising diagnosis in a young man with finger deformities and pain.

In **Case 2**, Dr. Huda Alfaris and Dr. Sindhu Johnson highlight the challenges of managing scleroderma-related digital ischemia and illustrates the effectiveness of advanced therapies in cases resistant to conventional treatment

In **Case 3**, Dr. Abdullah Albihani, Dr. Virginia Carrizo Abarza, Dr. Denis Poddubnyy and Dr. Vinod Chandran present a patient with the challenging problem of chronic back pain, and discuss the key clinical features and imaging findings that can ensure the right diagnosis is made.

# CASE 1 - Cold Consequences

## Case presented by Dr. Leigha Rowbottom and Dr. Lori Albert

**Case Report:** A 28-year-old male presented to the Hand Program clinic with pain, stiffness, and deformities affecting the interphalangeal joints of both hands. There was reduced mobility in the affected digits with functional limitations, particularly with fine motor and grip related activities.

He was previously healthy. Two years earlier he had sustained severe bilateral hand and foot frostbite injuries after prolonged cold exposure, requiring admission to a tertiary care center burn unit. During that admission he was treated conservatively with supportive care and wound management. He was lost to follow up due to subsequent incarceration.

On examination, there were cutaneous changes, including hypopigmentation, atrophy, and thickened skin. There were minor flexion contractures in most digits. There was ulnar deviation at multiple proximal and distal interphalangeal joints. Despite the appreciable deformity, his range of motion was relatively well preserved. X-rays of the hands revealed destructive and erosive changes (Figure 1).

Given his relatively preserved range of motion, despite the deformities, surgical intervention was not deemed to be appropriate. There was concern that his overall function might be reduced by arthrodesis, with digital shortening and reduced range of motion. Replacement arthroplasties were thought to be ill-advised given his young age and heavy use of the hands in manual labour.

In view of the clinical findings and x-ray changes, he was referred to the Rheumatology clinic for consideration of a possible underlying inflammatory rheumatic disease.

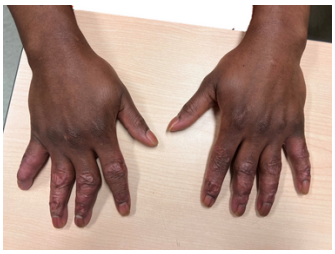

Figure 1. Image of bilateral hands with appreciable deformities affecting the proximal interphalangeal joints, most appreciable on the right.

There was no history of inflammatory joint pain or symptoms prior to the episode of frostbite. There were no extra-articular features of a rheumatic disease or other underlying condition. Importantly, there had been no progression in his symptoms after the development of deforming changes, with no new joints involved and no proximal joint involvement. There were no other medical illnesses and no family history of autoimmune disease. His examination revealed only the changes described above.

Repeat X-rays showed no progression over 4 years when compared to X-rays from another institution, despite the absence of any therapy. Further discussion with the radiologists indicated that the large erosions with central predominance were more in keeping with frostbite arthropathy than immune, inflammatory-mediated changes.

To manage his symptoms, a variety of analgesic strategies were trialed, including neuropathic pain agents and non-steroidal anti-inflammatory drugs (NSAIDs). He derived the most clinical benefit from as-needed NSAIDs and intra-articular corticosteroid injections of the right index finger PIP. He requested reassessment in the Hand Program regarding surgical management of the dominant index finger specifically. This is a heavy load bearing joint, and as such, he did not meet the criteria for joint arthroplasty and was offered arthrodesis. He deferred surgical intervention and has elected to continue with medical management.

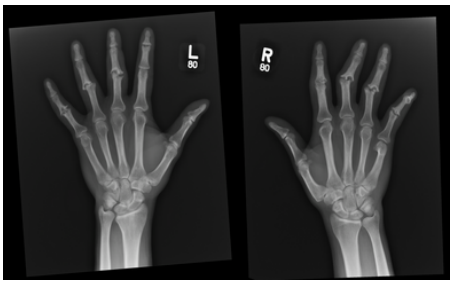


Figure 2. X-ray images of the bilateral hands demonstrating erosive changes of the PIPs bilaterally.

**Discussion:** Frostbite arthropathy is a rare complication of severe thermal injury. There are a small number of cases described in the literature, as outlined in Table 1. Prolonged exposure to subzero temperatures results in tissue freezing and subsequent ischemia. It is generally regarded as an injury to cutaneous structures leading to hypopigmentation, scarring, and thickened skin. However, injury may extend into subcutaneous structures such as muscles, tendons, ligaments and bones. There are several proposed mechanisms of joint injury. Direct cold exposure leads to ice crystal formation within cells, causing mechanical disruption of tissue architecture and necrosis. In addition, vascular ischemia caused by cold-induced vasoconstriction leads to tissue hypoxia and inflammation, which ultimately leads to damage of cartilage and bone.

This rare form of arthropathy typically affects the small joints of the extremities due to their vulnerability to cold exposure. The onset is often delayed after initial frostbite injury. In reported cases, the onset of arthritis symptoms ranged from a few years, to upwards of two decades. Patients present with joint pain, swelling, reduced range of motion, and subsequent deformities. These symptoms are nonspecific and may resemble other forms of arthritis, such as immune-mediated arthritis or osteoarthritis. Thus, the diagnosis of frostbite arthritis may not be clear initially and likely remains a diagnosis of exclusion.

Radiographic imaging may show features such as joint space narrowing, cartilage damage, and erosions. These radiographic features are non-specific, changes may be isolated to a single affected digit, may appear asymmetrical, and may or may not entirely follow the expected joint distribution suggestive of an inflammatory arthritis. Thus, correlation with a history of frostbite is essential for diagnosis.

# CASE 1 - Cold Consequences

**Case presented by Dr. Leigha Rowbottom and Dr. Lori Albert**

This case highlights the delayed onset of joint symptoms, 2 years after severe cold exposure. It was prudent to exclude alternative causes of arthritis, particularly in this young man as it would impact therapeutic strategies. His radiographic changes were in keeping with frostbite arthropathy, but the thorough assessment and consultation with the radiologist were key to confirming this diagnosis.

Given the limited number of cases reported in the literature, there are few clinical strategies for management of patient symptoms, which can be quite severe. A variety of interventions have been used including physical therapy, oral NSAIDs, alternative analgesic strategies, and surgical interventions, with variable success. Consultation with a specialist hand surgeon is important once the diagnosis is made, especially for patients with functional deficits. However, in our patient, surgical intervention was unlikely to provide benefit. The overall management appears to be largely supportive with focus on pain control and strategies to maintain patient function.

**Conclusion:** In conclusion, we present a rare case of frostbite-induced arthropathy, wherein the patient had delayed presentation of painful bilateral hand deformities after previous severe cold induced thermal injury. Importantly, these deformities were non progressive, both clinically and radiographically, which allowed differentiation from immune-mediated arthritis. This case illustrates the importance of multi-specialty collaboration in achieving an accurate diagnosis. However, persistent challenges remain in improving the patient's condition despite appropriate treatment efforts.

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Table 1: Selected Cases from the Literature

| Author (Year) | Patient Demographics | Case Outcomes |
|---|---|---|
| Simonds et al. (1969) | N=1<br>44F | Thermal injury to left hand D2-5 with initial conservative management. Developed visible bony deformity 6 months after injury and subsequently symptoms of isolated osteoarthritis of DIPs of affected digits 27 years later. |
| McCarty et al. (1979) | N=2 children (Case 1 was 14y, Case 2 5y at initial injury) | Case 1 developed swollen DIP and PIP 18 months after initial thermal injury. XR revealed premature epiphyseal closure.<br>Case 2 developed symptoms 20 years after initial injury. XR demonstrated dwarfism of affected digit and chondral loss. Subsequently underwent surgical fusion of PIP 4-5 with good clinical response. |
| McKendry. (1981) | N=1<br>29M | Frostbite injury to bilateral hands at 16yo. Developed pain and swelling of PIP 7 years after initial injury. |
| McCarty et al. (1981) | N=3 young adult patients | Severe frostbite injury in childhood affecting the hands resulting in dwarfing of the middle and distal phalanges, irregular articular surfaces, joint malalignment, evidence of degenerative arthritis in the interphalangeal joints |
| Schwenke. (1984) | N=1<br>38M | Developed isolated osteoarticular changes 10 years after frostbite injury. |
| Smith & Turner. (1998) | N=1<br>36M | Developed unilateral erosive nodal osteoarthritis 14 years after frostbite injury. |
| Bletry et al. (2005) | N=1<br>46F | Bilateral symmetric hand arthritis affecting Ips with history of severe frostbite 20 years earlier. XR demonstrated erosive arthritis with large cystic defects involving PIPs. Non-responsive to NSAIDs. Clinically improved with clodronic acid. |
| Wainapel et al. (2016) | N=1<br>49M | Bilateral erosive arthritis affecting the PIP and DIPs with history of thermal injury 25 years earlier. |

# CASE 2- Management of Digital Ulcers in Systemic Sclerosis

**Case presented by Huda Alfaris MBBS and Dr. Sindhu R Johnson**

**Case Report:** A 24-year-old woman with newly diagnosed limited cutaneous systemic sclerosis presented with severe ischemia and ulceration of the right fourth digit, which did not respond to nifedipine and tadalafil. Initial treatment with aspirin, nitroglycerin patches, and empirical antibiotics for a suspected soft tissue infection proved inadequate, as the ulcers continued to enlarge and produce purulent discharge. Imaging excluded osteomyelitis. She was admitted to the Toronto Western Hospital for intravenous alprostadil infusions for three days, followed by outpatient hyperbaric oxygen therapy, which led to significant improvement in her symptoms. Her management included adequate analgesia, and continuing antibiotics. This case underscores the challenges in managing scleroderma-related digital ischemia and illustrates the effectiveness of advanced therapies in cases resistant to conventional treatment. Figure 1 illustrates the ulcer in two phases: (a) during treatment and (b) following treatment.

**Discussion:** Digital ulcers (DUs) in systemic sclerosis (SSc) represent a significant challenge, contributing to functional impairment, and increased morbidity. The aims of managing digital ulcers are to prevent tissue loss, treat infections, alleviate pain, and reduce ischemia [1] . Effective management requires a comprehensive approach involving early intervention, pharmacological therapy, and non-pharmacological measures.

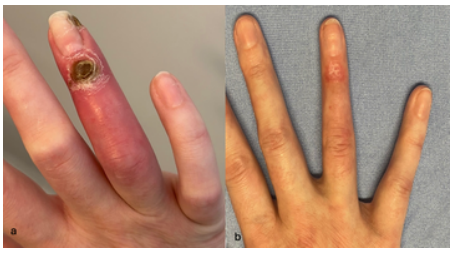

Figure 1 (a) There are two well-defined dry ulcers: one at the tip of the fourth digit and another on the dorsal side of the finger, proximal to the fingernail. Surrounding the ulcer, there is desquamation, erythema extending beyond the proximal interphalangeal joint. Both ulcers show signs of gangrene, with the necrotic core lifting on the lateral side. (b) The fourth digit demonstrates resolution of the digital ulcers and gangrene. There is residual scaring with a healed flat, pink area of skin with smooth, well-defined edges. The surrounding skin appears healthy, with mild erythema.

**1. Early Intervention and Infection Control:** Early identification and treatment of digital ulcers are crucial. Initial management focuses on wound care and infection control. Empirical antibiotic therapy should be considered if infection is suspected, guided by wound cultures and local infection protocols. Surgical debridement may be required to remove necrotic tissue and promote healing.

**2. Pharmacological Treatments:**
Vasodilatory therapy is central to managing DUs, as it addresses the underlying ischemia that exacerbates ulceration. Calcium channel blockers, particularly nifedipine, are often the first choice. Nifedipine has demonstrated efficacy in reducing the frequency of digital ulcers and improving healing [2]. For patients who cannot tolerate nifedipine or when it proves ineffective, alternative vasodilators such as alpha-agonists, topical nitrates, phosphodiesterase type 5 inhibitors (PDE-5i) may be used. These PDE-5i such as sildenafil or tadalafil can be effective in enhancing blood flow to the affected areas. In contrast, antiplatelet therapy has proven ineffective for ulcer management. In a placebo-controlled randomized controlled trial of dipyridamole and aspirin, no change in digital ulcer burden was noted [3].

In refractory cases, the more potent vasodilator, prostaglandin analogues are considered. Intravenous iloprost has shown substantial benefits in promoting the healing of DUs and is often used when other treatments are insufficient [2]. Additionally, bosentan, an endothelin receptor antagonist, along with iloprost, effectively decreases the incidence of new DUs [4].

**3. Non-Pharmacological Measures:**
In addition, non-pharmacological strategies play a crucial role in the management of DUs. Smoking cessation is essential as nicotine use exacerbates vasoconstriction. Keeping the hands warm and avoiding cold exposure can also help prevent ulceration. Patients should be advised to avoid triggers such as caffeine, and cough and cold preparations that contain amphetamines or ephedra, which can worsen symptoms [1] .

**4. Advanced Therapies**
For patients with persistent or severe ulcers despite conventional treatment, hyperbaric oxygen therapy (HBOT) can be considered. Although evidence is limited, data suggest HBOT has been shown to improve ulcer healing [5] . Furthermore, digital sympathectomy has also been shown to be effective in promoting healing and preventing the development of digital ulcers [6]. Novel therapies include botox injections and microvascular hand surgery for digital ischemia [6,7].

**Summary:** Effective management of digital ulcers in SSc involves a combination of approaches, tailored to each patient's specific condition and response to treatment. Continued research and development of new therapies are essential for improving outcomes in these challenging cases.

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# CASE 3 - Does she have psoriatic arthritis?

**Case presented by Dr. Abdullah Albihani, Dr. Virginia Carrizo Abarza, Dr. Denis Poddubnyy, and Dr. Vinod Chandran**

**Case Report:** A 41-year-old woman was assessed in the Low Back RAC (Rapid Access Clinic) after referral by her family doctor due to persistent lumbar pain.

She was an office worker and non-smoker. She had been diagnosed with psoriasis and eczema at age 40. She reported that both her parents have back pain attributed to osteoarthritis. She had no family history of psoriasis, axial spondyloarthritis, inflammatory arthritis or inflammatory bowel disease.

She first presented with lower back pain when she was 35 years old. Her back pain was initially attributed to activities such as vigorous lawn mowing, although she did not recall any specific trauma. She had attempted conservative therapies, including physiotherapy, chiropractic care, and acupuncture, with limited symptom relief. Over time, her pain migrated to the thoracic region, becoming constant and severe. Pain episodes would peak at 8/10 on the numeric pain scale, with occasional relief with drugs including non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and muscle relaxants. Spine MRI, a year prior to assessment was reported as demonstrating minimal degenerative disc disease.

Due to her relatively younger age, presence of psoriasis and minimal degenerative change in the spine, out of keeping with her symptoms, she was referred to the Psoriatic Arthritis (PsA) clinic for further evaluation.

**Clinical Evaluation:**
Our musculoskeletal examination demonstrated tenderness in the thoracic region, normal range of spinal mobility, and plantar fasciitis. Neurological examination did not demonstrate neurological deficits. Examination of the skin revealed pustular psoriasis affecting the feet. There was no nail involvement.

**Investigations**
**Laboratory Tests:**
Routine blood work, including complete blood count, liver function tests, renal function, and inflammatory markers, were within normal limits. HLA-B27 was negative.

**Imaging:**
Plain radiographs of the pelvis (Figure 1) revealed minimal sclerosis of the right sacroiliac joint. There was no evidence of sacroiliitis. The lumbar spine X-Ray (Figure 2) revealed some degenerative changes with an isolated finding of new bone formation at T11-12 (of questionable significance in isolation).

However, a more recent MRI of the spine (Figure 3) revealed bone marrow edema at the anterior margins of T6/T7, anterior aspect of T4, and around the superior endplate of T9 accompanied by structural changes – fat lesions and ankylosis at T5/T6. These changes were highly suggestive of inflammatory spondylitis. MRI of the sacroiliac joints (Figure 4) showed no evidence of sacroiliitis.

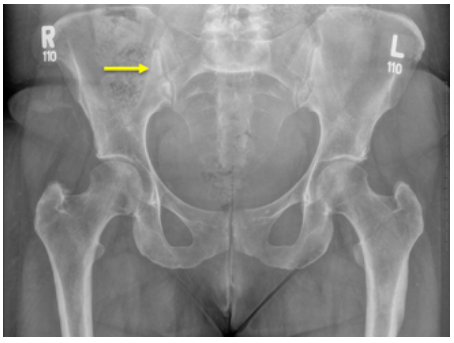


Figure 1. Pelvic X-Ray (September 2023): Minimal sclerosis on the right sacroiliac joint demonstrated (yellow arrow). There is no evidence of definite radiographic sacroiliitis.

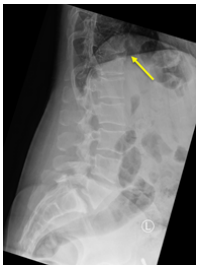

Figure 2

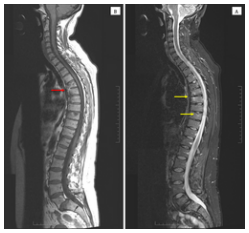

Figure 3

Figure 2. Lumbar Spine X-Ray (September 2023): There is disc space narrowing at the L5-S1 level. There is disc space narrowing at the T10-11 and T11-12 levels. New bone formation between T11-12 (yellow arrow).

Figure 3. MRI spine with sagittal T1 (A)/STIR (B) sequences (February 2024): There is bone marrow edema at the anterior margins of T6/T7 and around the superior endplate of T9 (yellow arrows) accompanied by structural changes – fat lesions and ankylosis T5/T6 (red arrow). Intervertebral disc height loss and desiccation from T5/6 down to T9/10.

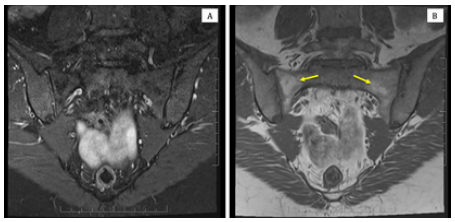


Figure 4. MRI sacroiliac oblique coronal STIR (A)/T1 (B) sequences (February 2024): There is no evidence of active sacroiliitis, no bone marrow edema identified. There are regions of fatty metaplasia in the sacral area (yellow arrows) that is likely physiological.

**Diagnosis:**
Although the patient had psoriasis, her clinical presentation of chronic non-specific back pain and initial imaging findings were not diagnostic of PsA. Subsequent MRI findings were highly suggestive of a diagnosis of PsA, specifically with axial involvement. She was also diagnosed with calcaneal enthesitis at the Achilles tendon and plantar fascia insertion sites, further supporting a psoriatic etiology.

**Management:**
The patient was counselled about her diagnosis and the management of PsA. Physiotherapy was strongly recommended. Given that she had not responded to NSAIDs, and had bothersome pustular psoriasis, biologic therapy with interleukin-17A inhibitors (secukinumab or ixekizumab) was recommended.

**Discussion:**
The presence of thoracic inflammation confirmed by imaging and enthesitis in a person with psoriasis supports a diagnosis of axial PsA. Her presentation aligns with the phenotype of axial PsA, which often appears later in life, particularly in women, and is characterized by asymmetric spinal and sacroiliac involvement [1]. This phenotype differs from other forms of axial spondyloarthritis such as ankylosing spondylitis (radiographic axial spondyloarthritis), which are more commonly HLA-B27 positive and symmetrical. Recent studies indicate that axial PsA may present with involvement of any segment of the spine including isolated thoracic inflammation sometimes without sacroiliitis, necessitating advanced imaging like MRI to confirm the diagnosis and distinguish it from other spondyloarthritides.

# CASE 3 - Does she have psoriatic arthritis?

**Case presented by Dr. Abdullah Albihani, Dr. Virginia Carrizo Abarza, Dr. Denis Poddubnyy, and Dr. Vinod Chandran**

To optimally assess axial PsA, MRI should include specific sequences that best highlight inflammatory and structural changes. STIR (Short Tau Inversion Recovery) and T1-weighted sequences are essential, as STIR is highly sensitive to inflammation (showing bone marrow edema) while T1 helps evaluate structural changes. Specifically in the context of axial PsA, sacroiliac as well as whole-spine rather than segmental imaging is recommended, since patients may have spinal involvement without sacroiliitis (as demonstrated in this case). Moreover, the site of pain may not correspond to site of structural or inflammatory changes on MRI. Radiography alone may not capture inflammatory/structural changes needed for diagnosis[1].

Treatment of axial spondyloarthritis including axial PsA includes education and physiotherapy to reduce pain, maintain spinal mobility and posture and improve health-related quality of life and function, and pharmacotherapy with NSAIDs[3] . If the patient continues to have active spinal disease despite NSAIDs, targeted biological or small molecule Janus kinase (JAK) inhibitors are recommended. Tumour necrosis factor (TNF) inhibitors were among the first biologic therapies shown to be effective in treating axial SpA.

More recently, interleukin-17A (IL-17A) inhibitors and Janus kinase (JAK) inhibitors have become available and are equally efficacious for both axial and peripheral manifestations of SpA, including psoriatic arthritis[2]. The choice of therapy depends on whether the patient has other manifestations including significant peripheral arthritis, enthesitis, dactylitis, and extra-musculoskeletal manifestations including inflammatory bowel disease, uveitis and of course, psoriasis. In our patient, we recommended interleukin-17A inhibitors since TNF inhibitors may lead to de novo or exacerbate pustular psoriasis[4] .

**Conclusion:** PsA must be considered in the differential diagnosis when patients present with chronic back pain. The presence of psoriasis, enthesitis and MR imaging findings suggestive of inflammatory spondylitis clinched the diagnosis despite the absence of sacroiliitis on plain radiography and MR imaging.

**Take Home Message**

1- Chronic thoracic pain should prompt evaluation for axial involvement in psoriatic arthritis.

2- Psoriatic arthritis can present with both peripheral (enthesitis, arthritis) and axial symptoms.

3- Magnetic resonance imaging is key to diagnosis of axial involvement in PsA.

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