

Acute Bilateral Leg Edema Provoked by Spinal Stenosis with Complex Regional Pain Syndrome Mechanism: A Case Report

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ABSTRACT

Bilateral leg edema can result from various diseases, including Complex Regional Pain Syndrome provoked by spinal stenosis. This case report presents the resolution of acute bilateral leg edema and CRPS in a patient with spinal stenosis. A 51-year-old female presented with acute bilateral leg edema and chronic lower back pain. Imaging revealed minimal anterolisthesis of the L4-L5 vertebra and signs of degenerative disease and CRPS type II. Following vertebral decompression, posterior instrumentation and fusion, the patient experienced a significant reduction in edema and resumed daily activities within two months. Inflammation and the immune cascade post-spinal stenosis can trigger the proliferation of connective tissue cells and the release of inflammatory cytokines. This process activates nociceptive afferents that produce neuropeptides, resulting in edema and other CRPS symptoms. Multimodal analgesia, physiotherapy education and surgery, including posterior decompression with instrumentation and fusion, are effective treatments for spinal stenosis with acute bilateral leg edema due to CRPS.

Keywords: leg, edema, complex regional pain syndromes, spinal stenosis, spondylolisthesis

Introduction

Bilateral leg edema is a common symptom in geriatrics and is often associated with cardiovascular, liver, and renal diseases, drug use, lymphedema, and pregnancy. Leg edema is more prevalent in older adults, affecting approximately 10–30% of those over 65. This is often due to age-related changes in vascular function and an increased likelihood of underlying conditions such as chronic venous insufficiency, heart disease, and kidney dysfunction.

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The prevalence of complex regional pain syndromes (CRPS) is predicted to range from five to 26 instances per 100,000 person-years, with a conservative prevalence rate of 20.57 per 100,000 persons during 11 years (1,2). CRPS can also cause bilateral edema, requiring comprehensive history taking, physical examination, blood tests, and imaging diagnostics to rule out other causes. The Budapest Criteria for CRPS include swollen limbs as a key symptom (3).

Degenerative spondylolisthesis is prevalent among the elderly, particularly in the lumbosacral area, leading to spinal stenosis. Degenerative spondylolisthesis primarily affects adults over 50, with prevalence rates rising significantly in older populations. Studies indicate that it can affect up to 25–30% of people over 65, largely due to age-related degeneration in spinal discs and joints. Symptoms include low back pain and radiating pain into the thighs, with potential neurogenic involvement. This case report discusses CRPS provoked by spinal stenosis (4).

Case Report

A 51-year-old female presented with grade 3 bilateral limb edema, initially reported after visiting a traditional practitioner. The traditional practitioner reportedly only gave a massage and applied oil to the

edematous limb every week for three weeks, but the complaint did not resolve. The patient has not taken any medication or other treatment aside from what was given by the traditional practitioner.

The patient had a history of chronic lower back pain for about 3 months and tingling and burning sensations in both feet, a history of 3 months of bed rest, with minimal relief from physiotherapy, as shown in Figure 1. The patient had only consumed regular analgesics in the last 3 months and had no previous illnesses. Upon presentation, neurologic examination on the lower extremity showed reduced motoric strength to Oxford Motor Scale of 3 on L1-S1, no hypoesthesia/anaesthesia, normal physiological reflex and no pathological reflex was found. Imaging revealed minimal anterior shifting of the L4-L5 vertebra, yellow ligament hypertrophy, and severe right foraminal stenosis, with an SF-36 score pre-operative 24.9%. Thorough preoperative evaluation, such as ECG, serum electrolyte, and coagulation function, showed no abnormalities. The patient was diagnosed with CRPS type II and canal stenosis due to spondylolisthesis of lumbar vertebrae 4-5. The patient has also tried conservative treatment of multimodal analgesia and 1 month of physiotherapy, but there has been no improvement. The patient was then decided to undergo posterior decompression laminotomy using pedicle screws and fusion.

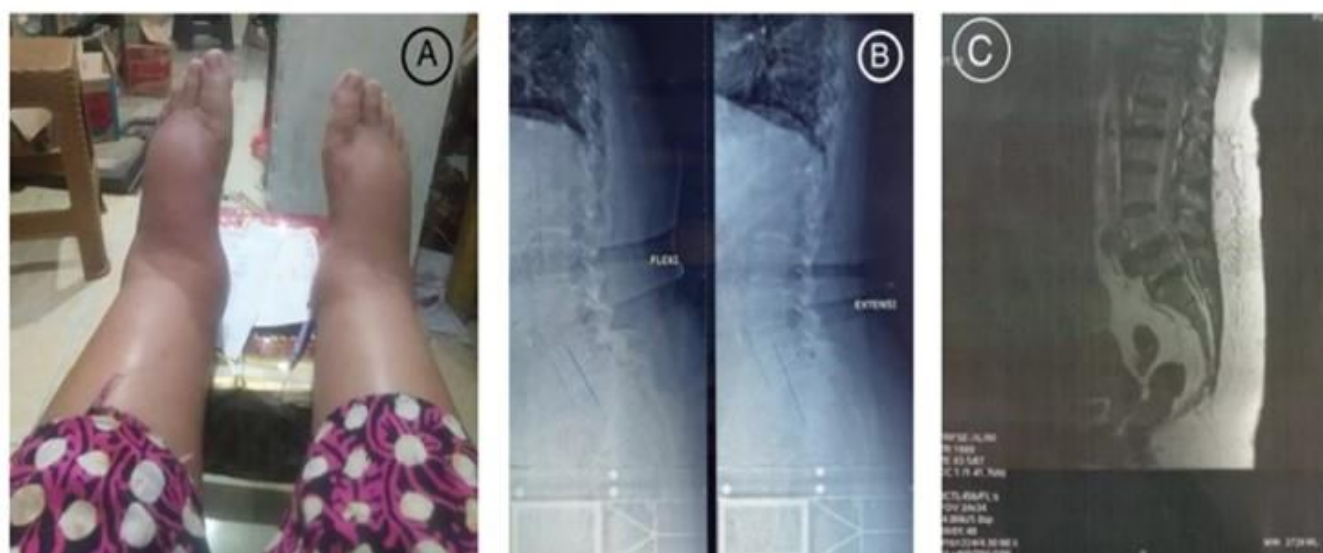


Figure 1. (A) Bilateral edema on lower leg, (B) Lateral lumbosacral X-ray shows spondylolisthesis VL4-5, (C) Lumbosacral MRI shows anterolisthesis VL 4 to VL 5 grade I with right foraminal stenosis VL4-5 and yellow ligament hypertrophy.

Postoperative results showed resolved edema within several days, as shown in Figure 2a, and also X-ray post-operative in Figure 2b. Two months later, the patient resumed normal daily activities.



Fig. 2. (A) Edema on the bilateral limb was resolved (B) postoperative X-ray of the lumbosacral postoperative.

We have done follow-up results within 2 years, there is no pain, or swelling and tingling shown in Figure 3a, and an increase of post-operative SF-36 73%, and union of the vertebra is shown in Figure 3b. The most significant improvement of SF-36 was found in terms of pain (22.5 to 90), physical functioning (30 to 85), and role limitation due to physical health (25 to 100). This is mostly because the patient was not able to walk preoperatively due to the pain. The ability to do activities of daily living independently benefited the patient greatly.

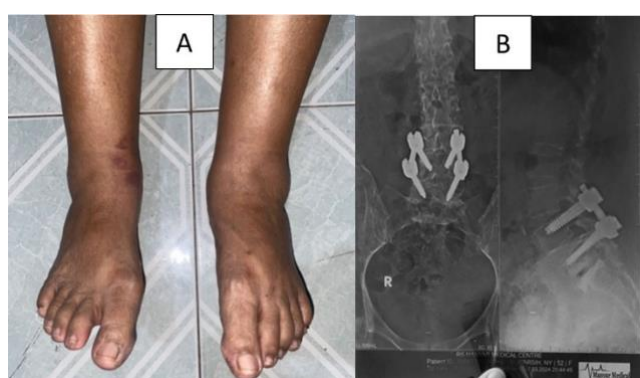


Fig. 3. (A) Clinical picture 2 years postoperative, (B) X-ray of the lumbosacral 2 years postoperative.

Discussion

Leg edema is a prevalent symptom that can be caused by various diseases and conditions, including issues with the venous and lymphatic systems,

excessive fluid volume, heightened capillary permeability, and reduced oncotic pressure (5).

According to a comprehensive survey conducted between 2000 and 2016, peripheral edema was observed in approximately 19% to 20% of persons in the United States. Peripheral edema is correlated with advanced age, low socioeconomic status, non-Caucasian ethnicity, female gender, obesity, diabetes, hypertension, pain, sedentary lifestyle, and limited mobility (6).

Degenerative spondylolisthesis (DS) refers to the slippage of one vertebra over another, first described by Newman and Stone in 1955. The etiology differs from disc degeneration, arthritis of the facet joints, and ligament and muscle dysfunction. Studies indicate that DS predominantly occurs at the L4-L5 level, with a ratio of 6:1 among women compared to men, and a prevalence of 25% in women versus 19.1% in men. Degenerative changes in the spine, particularly in the facet joints and intervertebral discs, contribute to vertebral instability and forward movement without any defect in the pars interarticularis (7).

While a study in 2017 linked pitting edema to lumbar disc herniation, the direct connection between spinal stenosis and bilateral leg edema remains unclear. In this case, bilateral leg edema developed following a prolonged period of lower back pain accompanied by tingling and cold sensations. Through analysis and exclusion of other potential causes of leg oedema, a diagnosis of CRPS was considered (8).

Furthermore, the Budapest Criteria may be used to confirm the diagnosis of CRPS clinically. In our case, the patient had a continuing pain which is disproportionate to the initiating event, sensory problem (tingling in both feet), vasomotor problem (differences in skin coloration between both limbs), bilateral limb edema, and motoric problem (decreased range of movement). In addition, other diagnoses cannot better explain the problem since the patient does not have significant comorbidities or other accompanying symptoms. From the lumbosacral MRI we found that there were degenerative disease of the spine at levels VL 1-2, VL2-3, VL 4-5, VL5-S1 accompanied by right-left yellow ligament

hypertrophy, right and left facet joint arthrosis which causes moderate compression anterior thecal sac, severe right foramina stenosis, and moderate left foramina stenosis with anterolisthesis VL 4 to VL 5 grade I.

Complex Regional Pain Syndrome is a persistent pain disorder that is defined by symptoms including heightened sensitivity to pain, discomfort caused by non-painful stimuli, swelling, changes in skin color, changes in tissue health, and abnormal muscle contractions, often affecting the limbs. CRPS is categorized into two subtypes: Type 1, which does not involve any nerve injury, and Type 2, which is linked to underlying nerve damage. The pathophysiology of Complex Regional Pain Syndrome Type II (CRPS II), particularly in its second stage, is complex and multifactorial, involving a combination of neurogenic inflammation, central and peripheral sensitisation, and immune dysregulation. Neurogenic inflammation plays a pivotal role in the progression of CRPS II. Following nerve injury, primary afferent sensory neurons release neuropeptides such as Substance P (SP) and Calcitonin Gene-Related Peptide (CGRP). These neuropeptides contribute to vasodilation, increased vascular permeability, and recruitment of immune cells. In CRPS II, the expression of neuropeptides is significantly upregulated not only at the injury site but also in adjacent tissues, indicating their role in propagating inflammation and hypersensitivity. Elevated levels of pro-inflammatory cytokines like TNF- α , IL-1, IL-6, and IL-8 are found in the serum and affected tissues, contributing to ongoing inflammation and pain (9,10).

Central sensitization refers to the heightened sensitivity of the central nervous system (CNS) to nociceptive stimuli. The activation of glial cells (microglia and astrocytes) within the spinal cord is critical for maintaining this sensitization. These cells release pro-inflammatory mediators that exacerbate pain signalling pathways, leading to a vicious cycle of pain amplification (10).

Recent studies have highlighted the role of immune dysregulation in CRPS II. An increase in CD4⁺ and CD8⁺ T lymphocyte populations suggests

an antigen-mediated immune response that may contribute to chronic inflammation. Autoantibodies against adrenergic receptors have been identified, indicating a potential autoimmune component that sensitizes nociceptors and exacerbates pain responses. CRPS II often includes autonomic symptoms such as temperature changes, sweating abnormalities, and changes in skin color (1,10).

The pathophysiology of CRPS involves abnormal tissue response to injury, increased sensitization of the peripheral and central nervous systems, inflammatory changes, and autonomic dysregulation. In this patient, exacerbation of spinal stenosis following non-medical procedures likely triggered the inflammation and immune cascade characteristic of CRPS. Nociceptive C-fibres begin to produce inflammatory neuropeptides, activating mast cells and releasing chemical mediators that cause acute symptoms such as edema, skin redness, warmth, and hair growth (1,11,12).

To enhance patient outcomes, it is essential to promptly diagnose and refer individuals to physical therapy, occupational therapy, and chronic pain management. This patient is CRPS II stage 1, which is the acute phase, a duration within 1-3 months from the onset, and there was edema, increasing pain and burning sensation, also there were bony changes. CRPS II, also known as causalgia, is marked by intense pain that can be disproportionate to the initial injury (9,13).

During the early stages of CRPS treatment, a comprehensive approach is used, which includes the use of corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), and substances that remove harmful free radicals (such as dimethylsulfoxide or N-acetylcysteine). Additional therapies include the use of gabapentin, vasodilatory medicines, physiotherapy, and sympathetic nerve blocks. The effectiveness of narcotic pain medicines in treating CRPS is limited. Vitamin C may have a positive effect in preventing the occurrence of CRPS (12,14). In this patient, the edema is resolved within several days.

More invasive treatment includes spinal cord stimulation, sympathetic blocks and sympathectomy.

These interventional treatments are usually considered if conservative treatment fails, but in our case, surgical treatment becomes essential if the syndrome is secondary to the spinal stenosis. A similar case had been reported previously by Kim et al. (17). In this case, a 22-year-old man had a chief complaint of persistent back and leg pain along with discoloration and swelling in the right leg. The complaint was in accordance with the MRI of central disc herniation of VL4/5. The patient was treated with percutaneous nucleoplasty after 2 months of failed conservative treatment. Other similar cases were reported by Ozturk et al. (16). In this case, a 37-year-old female had a chief complaint of constant burning, tingling, and severe pain in both hands, but with no swelling. The MRI showed a disc protrusion on C5-C6. In this case, the patient was successfully treated conservatively with

analgesics for 2 weeks. VAS was reduced from 10 to 5. From these other cases, the author believes that vasomotor problems as a distinguishing symptom for CRPS compared to regular pain caused by disc herniation, and in such cases, it is imperative to prepare the patient for invasive treatments, as the pain tends to be greater than usual and surgical treatments result in a significant reduction in pain (15-17).

Conclusion

Complex regional pain syndromes need good diagnosis and treatment. Assessment of CRPS should be considered in patients presenting with bilateral leg edema. Such a case can be treated well with multimodal analgesia, physiotherapy education and also surgery, including posterior decompression with instrumentation and fusion.

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