

Review Article

The mechanical-vascular hypothesis of Sudeck's atrophy: venous outflow obstruction

Majid Negari*

Independent Researcher, Switzerland

Received: 04 September 2026

Accepted: 10 October 2026

*Correspondence:

Dr. Majid Negari,

E-mail: padzadro@yahoo.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Complex regional pain syndrome (CRPS), historically termed Sudeck's atrophy, is traditionally classified as a neurogenic and sympathetically mediated disorder. However, purely neurological models fail to fully account for the rapid trophic changes of the skin and nails or the profound impact of mechanical factors observed in clinical practice. In this paper, we propose a novel mechanical-vascular hypothesis suggesting that the primary initiating driver of Sudeck's atrophy is not neural dysfunction, but rather a mechanical venous outflow obstruction. This condition is primarily driven by the synergistic effect of rigid external cast compression and internal tissue edema following trauma. This mechanical bottleneck leads to persistent venous stasis, elevated compartmental pressure, microvascular fatigue, and rapid regional bone demineralization, while drawing a compelling pathological parallel with post-thrombotic syndrome. Importantly, this paradigm does not dismiss standard orthopedic care; rather, it integrates existing practices—such as effective pain management, targeted regional blocks to facilitate physical therapy, and muscle pump activation—with a renewed focus on early anti-edema measures and venous path clearance. Rigorous longitudinal Doppler assessments and clinical evaluations are proposed to validate this framework, offering a comprehensive dual approach to halt disease progression.

Keywords: Sudeck's atrophy, Complex regional pain syndrome, Venous outflow obstruction, Osteoporosis, Edema

INTRODUCTION

Complex regional pain syndrome (CRPS), historically known as Sudeck's atrophy, is one of the most challenging and distressing complications following trauma, fractures, and surgical interventions of the extremities, particularly involving the wrist and the distal radius. Despite its high prevalence and significant clinical burden, the precise initiating mechanisms and exact pathophysiology underlying the development and progression of CRPS remain incompletely elucidated.¹⁻⁴

Traditionally, Sudeck's atrophy (CRPS) has been regarded primarily as a neurogenic and sympathetically mediated disorder. However, this classical neurological paradigm leaves several prominent clinical observations unexplained—such as the rapid, severe trophic changes of

the skin and nails that rarely match the extent seen in pure neural dysfunctions like motor or sensory paralyses from strokes. This discrepancy strongly suggests that pure neural models are incomplete, and clinicians must integrate them with a more direct, physical, and environmental mechanism.^{5,7}

The hypothesis proposed in this paper investigates the possibility that the missing mechanical link in the pathogenesis of Sudeck's atrophy (CRPS) is a vascular disturbance in venous outflow. Rather than dismissing traditional neurogenic pathways, this mechanical-vascular perspective complements them by addressing the foundational physical bottleneck. Initial trauma or surgery induces substantial tissue edema. Simultaneously, the application of rigid external devices, such as casts or tight bandages, creates a restrictive external constraint. Rather

than inducing an absolute and immediate occlusion, this combination of internal tissue swelling and external framing critically narrows and partially obstructs the thin-walled superficial and deep venous channels within the wrist and its anatomical compartments (such as the carpal and Guyon's canals).¹⁷⁻²⁰

To better comprehend this localized phenomenon, a compelling pathophysiological parallel can be drawn with post-thrombotic syndrome (PTS)-a well-documented chronic venous disorder occurring after deep vein thrombosis (DVT). In PTS, partial obstruction or thrombosis of deep and superficial veins leads to chronic venous hypertension, microvascular congestion, and localized tissue distress. Analogous to how chronic venous insufficiency in the lower leg and foot triggers regional bone demineralization and structural alterations (such as metatarsal osteoporosis), the partial venous outflow obstruction in Sudeck's atrophy (CRPS) creates persistent hydrostatic congestion. While arterial inflow continues to pump blood into the distal extremity under higher pressure, the compromised venous drainage traps blood, elevates intra-compartmental pressure, and induces local hypoxia and acidosis. This persistent mechanical-vascular bottleneck accelerates regional bone turnover and precipitates rapid localized osteoporosis (Sudeck's atrophy).^{10,14,17}

This paper aims to thoroughly analyze this novel mechanical-vascular model, trace its physiological phases from the initial warm phase to the cold vasoconstrictive phase, establish its parallels with chronic venous outflow disorders like PTS, and propose robust diagnostic frameworks to redirect the therapeutic paradigm toward anti-edema interventions and venous path clearance.^{3,10,24}

THE HYPOTHESIS

The hypothesis presented in this article is grounded in the mechanical-vascular model of Sudeck's atrophy (CRPS) proposing that the primary pathological driver is not a neurogenic dysfunction, but rather a mechanical compromise of venous outflow.

Following an initial trauma or surgical intervention-particularly involving the distal radius or wrist-a sequence of mechanical and hemodynamic events unfolds, progressing through distinct physiological phases.^{3,4}

Phase I: Initial trauma, cast compression, and venous narrowing

The pathological cascade typically begins with acute trauma, which immediately induces significant localized soft-tissue edema within the confined spaces of the wrist and hand (such as the carpal tunnel and Guyon's canal). Shortly thereafter, standard clinical management involves the application of a rigid external immobilization device, such as a plaster cast or the tight circumferential bandage.^{17,18}

Due to their thin walls and very low internal hydrostatic pressure, the superficial and deep venous networks of the wrist-including the dorsal venous arch, radial veins, and ulnar veins-cannot withstand the combined forces of "external rigid framing" and "internal tissue swelling." Rather than an immediate total collapse, these veins are critically narrowed and semi-occluded. While arterial inflow continues to pump blood into the distal extremity under high pressure, the compromised venous drainage creates an initial bottleneck.^{17,20}

Phase II: The warm phase and compensatory arterial hyperemia

In response to the early venous stasis, local hypoxia, and tissue distress, the body attempts a compensatory mechanism. Arterioles dilate to maintain tissue perfusion, flushing the microcirculation with increased arterial blood flow. Clinically, this corresponds to the early "warm phase" of Sudeck's atrophy, characterized by localized warmth, erythema, and mild swelling. However, because the venous outflow remains restricted, this compensatory influx only exacerbates the accumulation of fluid within the interstitial space, aggravating the edema and escalating ischemic pain.^{5,8,10}

Phase III: Vascular decompensation, the cold phase, and rapid trophic changes

As venous stasis and interstitial pressure persist over weeks (often becoming clinically prominent around one month post-immobilization), the microvascular bed undergoes severe functional and structural decompensation. The continuous high-pressure gradient combined with stagnant, acidic blood overwhelms the capillary endothelial cells.^{10,26}

Faced with this persistent microvascular breakdown, local vasoconstriction ensues, transitioning the extremity into the "cold phase." Clinically, the hand becomes cold, cyanotic, and stiff. Deprived of adequate nutritional exchange and normal capillary turnover, the surrounding soft tissues experience rapid trophic changes. Skin thinning, hyperhidrosis, and accelerated nail dystrophy occur at a pace far too rapid to be accounted for by classical neurogenic models alone.^{5,7,18}

Phase IV: Venous stasis, rapid osteoporosis, and the vicious cycle

The prolonged entrapment of blood and elevated intra-compartmental pressure directly impact the regional bone architecture. The acidic microenvironment and chronic congestion stimulate osteoclast activity while inhibiting osteoblast function, rapidly precipitating regional bone demineralization-clinically recognized as Sudeck's atrophy (osteoporosis).^{14,17,22}

Ultimately, this establishes a self-sustaining vicious cycle: the initial trauma and cast compression cause venous

narrowing → venous stasis and edema increase tissue pressure → elevated pressure further compresses the fragile venous channels → worsening stasis amplifies local inflammation, pain, and bone demineralization. This mechanical-vascular loop explains why symptoms persist and escalate even long after the initial bone fracture has structurally healed.^{10,17,22}

DISCUSSION

To validate this mechanical-vascular model and translate its theoretical framework into clinical practice, we propose a multi-faceted approach combining hemodynamic investigations, comparative clinical parallels, and targeted management strategies.^{3,4}

First, the hemodynamic and microcirculatory aspects of the hypothesis can be rigorously evaluated using longitudinal color Doppler ultrasound and microcirculation monitoring. This approach aims to directly measure venous outflow velocity in patients immobilized with casts, tracking the onset of venous stasis and localized microvascular congestion before the full clinical manifestation of Sudeck's atrophy occurs.^{10,17,19}

Second, clinical validation can be strengthened by drawing a direct pathological parallel with post-thrombotic syndrome (PTS). Similar to how chronic deep and superficial venous outflow obstruction in the lower extremity leads to persistent hydrostatic pressure, microvascular congestion, and regional demineralization (such as metatarsal osteoporosis), the partial venous bottleneck in the wrist mimics this exact cascade.^{24,25} Furthermore, this mechanical framework is corroborated by rare non-traumatic clinical observations, such as deep ganglion cysts in the wrist exerting direct extrinsic compression on regional venous channels. These cases demonstrate that mechanical venous compression alone—in the absence of any bone fracture or direct neural trauma—is sufficient to precipitate Sudeck-like trophic changes and regional osteoporosis.^{17,22}

Third, based on this venous-centric model, we can prospectively test proactive preventive and therapeutic strategies. Recognizing that venous outflow obstruction is heavily driven by a combination of external cast pressure and internal edema, clinical protocols should incorporate.^{17,20}

Early and continuous active digital motion protocols

Forcing rhythmic finger movements from day one to actively engage the natural muscle pump and prevent venous stasis.^{21,23}

Timely cast revision or splitting

Vigilantly monitoring patients during the early window of symptom onset to loosen restrictive constraints promptly.^{18,20}

Venoactive and rheologic pharmacological agents

Utilizing specialized medications that enhance venous tone, reduce capillary permeability, and improve microvascular drainage (such as micronized flavonoid fractions like Daflon, aescin, and pentoxifylline) to facilitate blood return and resolve micro-stasis.^{25,27}

Pain control and sympathetic modulation to facilitate rehabilitation

Implementing regional anesthetic techniques or local infiltration (such as targeted muscular or peroradial anesthetic blocks) to safely reduce severe ischemic pain. Beyond mere analgesia, these anesthetic blocks temporarily interrupt sympathetic vasoconstrictive signals to the vessel walls, relieving the spasm of both constricted arterioles and compressed venules, thereby enhancing venous outflow and microvascular drainage while enabling patients to actively engage in essential physical therapy.^{29,30}

Rather than discarding existing orthopedic treatments, this mechanical-vascular paradigm bridges them with a targeted focus on venous path clearance and anti-edema management, offering a comprehensive dual approach to halt and reverse the progression of Sudeck's atrophy (CRPS).^{3,23,29}

Extending the model: type ii CRPS, non-traumatic external constraints, and the unified hemodynamic endpoint

To fully establish the mechanical-vascular hypothesis, the model must account for the broad spectrum of clinical presentations, ranging from non-traumatic external constraints and classic Type I CRPS to major nerve injury-associated Type II CRPS. Clinical evidence demonstrates that sustained external mechanical constraints—such as overly tight circular plaster casts, rigid unyielding splints, or excessively tight circumferential bandages—can precipitate the entire clinical cascade of Sudeck's atrophy in the absolute absence of any primary bone fracture or direct neural trauma.^{31,32} Because thin-walled micro-venules and collecting veins operate under very low intraluminal hydrostatic pressures, external mechanical forces easily exceed their critical closing pressure. This instantly induces localized venous outflow stasis, interstitial fluid accumulation, and a self-reinforcing ischemic microenvironment.

Conversely, in CRPS Type II (formerly causalgia), where a documented major nerve trunk injury is present, a parallel hydrodynamic bottleneck is triggered endogenously.³³ Rather than an external mechanical framing, proximal nerve trauma initiates a potent neurogenic inflammatory cascade through the antidromic release of vasoactive neuropeptides—such as calcitonin gene-related peptide (CGRP) and substance P.^{8,33} This neural-mediated dysregulation triggers intense arteriolar

vasodilation coupled with localized capillary endothelial leakage in the distal extremity. The sudden influx of fluid into closed fascial compartments dramatically elevates tissue pressure, which in turn compresses the compliant, low-pressure venous channels from within.

Therefore, whether the venous outflow obstruction is enforced mechanically from the outside (as seen in rigid casting and tight bandages in type I and compression syndromes) or driven internally by nerve-mediated microvascular spasm and neurogenic edema (in type II), the terminal hemodynamic pathway remains identical: persistent venous stasis, elevated compartmental pressure, localized hypoxia, and rapid regional bone demineralization.³¹⁻³³ This striking convergence unifies both clinical phenotypes under a single, cohesive mechanical-vascular framework, proving that regardless of the initial trigger (mechanical vs. neural), the ultimate driver of tissue dystrophy and Sudeck's atrophy is the vascular bottleneck of venous outflow.

CONCLUSION

In conclusion, the mechanico-vascular hypothesis offers a unifying pathophysiological explanation for Sudeck's atrophy, bridging the gap between clinical observation and cellular degeneration. By recognizing that partial venous outflow obstruction-driven by immobilization, cast pressure, and tissue edema-is the primary mechanical bottleneck, we can move beyond purely neurogenic assumptions. Integrating early dynamic mobilization, vigilant pressure relief, and targeted venoactive pharmacotherapy with standard orthopedic care provides a comprehensive, dual-action paradigm. This approach not only validates existing rehabilitation methods but establishes a proactive roadmap to halt and reverse the debilitating cascade of Sudeck's syndrome (CRPS).

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Harden RN, Bruhl S, Perez RSGM, Birklein F, Marinus J, Maihofner C, et al. Validation of proposed diagnostic criteria (the "Budapest Criteria") for Complex Regional Pain Syndrome. *Pain*. 2010;150(2):268-74.
2. Harden RN, McCabe CS, Goebel A, Goossens M, Schwartzman RJ, Bruhl S, et al. Complex Regional Pain Syndrome: Practical Diagnostic and Treatment Guidelines, 5th Edition. *Pain Med*. 2022;23(1):S1-53.
3. Ferraro MC, O'Connell NE, Sommer C, Goebel A, Bultitude JH, Cashin AG, et al. Complex regional pain syndrome: advances in epidemiology, pathophysiology, diagnosis, and treatment. *Lancet Neurol*. 2024;23(5):522-33.
4. Her YF, Kubrova E, Dombovy-Johnson M, ElSaban M, Mostert K, D'Souza RS. Complex Regional Pain Syndrome: Updates and Current Evidence. *Curr Phys Med Rehabil Rep*. 2024;12:50-70.
5. Birklein F, Dimova V. Complex regional pain syndrome-up-to-date. *Pain Rep*. 2017;2(6):e624.
6. Birklein F, Schlereth T. Complex regional pain syndrome-significant progress in understanding. *Pain*. 2015;156(1):S94-103.
7. Oaklander AL, Horowitz SH. The complex regional pain syndrome. *Handb Clin Neurol*. 2015;131:481-503.
8. Weber M, Birklein F, Neundörfer B, Schmelz M. Facilitated neurogenic inflammation in complex regional pain syndrome. *Pain*. 2001;91(3):251-7.
9. Schlereth T, Drummond PD, Birklein F. Inflammation in CRPS: role of the sympathetic supply. *Auton Neurosci*. 2014;182:102-7.
10. Schürmann M, Zaspel J, Gradl G, Wipfel A, Christ F. Assessment of the peripheral microcirculation using computer-assisted venous congestion plethysmography in post-traumatic complex regional pain syndrome type I. *J Vasc Res*. 2001;38(5):453-61.
11. Kollmann G, Wertli MM, Dudli S, Distler O, Brunner F. The role of the bone in complex regional pain syndrome 1-A systematic review. *Eur J Pain*. 2023;27(5):794-804.
12. Karacan I, Aydin T, Ozaras N. Bone loss in the contralateral asymptomatic hand in patients with complex regional pain syndrome type I. *J Bone Miner Metab*. 2004;22(1):44-7.
13. Abdala R, Torrecilla I, Mumbach G, Witis F, Mana D, González Pernas M, et al. Impaired bone mineral density in patients with complex regional pain syndrome. *J Clin Densitom*. 2025;28(4):101632.
14. Wang L, Guo TZ, Hou S, Wei T, Li WW, Shi X, et al. Bisphosphonates inhibit pain, bone loss, and inflammation in a rat tibia fracture model of complex regional pain syndrome. *Anesthesiology*. 2016;123(4):1033-45.
15. Chevreau M, Romand X, Gaudin P, Juvin R, Baillet A. Bisphosphonates for treatment of Complex Regional Pain Syndrome type 1: A systematic literature review and meta-analysis of randomized controlled trials versus placebo. *Joint Bone Spine*. 2017;84(4):393-9.
16. Ferraro MC, O'Connell NE, Goebel A, Hill R, Curtis F, Wilkinson J, et al. Efficacy and Safety of Bisphosphonates for Complex Regional Pain Syndrome: A Systematic Review and Meta-analysis. *Ann Intern Med*. 2026;179(2):256-69.
17. Mohler LR, Pedowitz RA, Byrne TP, Gershuni DH. Pressure generation beneath a new thermoplastic cast. *Clin Orthop Relat Res*. 1996;(322):262-7.
18. Garfin SR, Mubarak SJ, Evans KL, Hargens AR, Akeson WH. Quantification of intracompartmental pressure and volume under plaster casts. *J Bone Joint Surg Am*. 1981;63(3):449-53.
19. Zaino CJ, Patel MR, Arief MS, Pivec R. The effectiveness of bivalving, cast spreading, and webril cutting to reduce cast pressure in a fiberglass short arm cast. *J Bone Joint Surg Am*. 2015;97(5):374-80.

20. Halanski M, Noonan KJ. Cast and splint immobilization: complications. *J Am Acad Orthop Surg.* 2008;16(1):30-40.
21. Hickey BA, Morgan A, Pugh N, Perera A. The effect of lower limb cast immobilization on calf muscle pump function: a simple strategy of exercises can maintain flow. *Foot Ankle Int.* 2014;35(5):429-33.
22. Styf J, Wiger P. Abnormally increased intramuscular pressure in human legs: comparison of two experimental models. *J Trauma.* 1998;45(1):133-9.
23. Craik JD, Clark A, Hendry J, Sott AH, Hamilton PD. The effect of ankle joint immobilization on lower limb venous flow. *Foot Ankle Int.* 2015;36(1):18-23.
24. Baldwin MJ, Moore HM, Rudarakanchana N, Gohel M, Davies AH. Post-thrombotic syndrome: a clinical review. *J Thromb Haemost.* 2013;11(5):795-805.
25. Meissner MH, Eklof B, Smith PC, Dalsing MC, DePalma RG, Gloviczki P, et al. Secondary chronic venous disorders. *J Vasc Surg.* 2007;46(6):S68-83.
26. Phillips LJ, Sarkar R. Molecular characterization of post-thrombotic syndrome. *J Vasc Surg.* 2007;45(A):A116-22.
27. Bernardi E, Prandoni P. The post-thrombotic syndrome. *Curr Opin Pulm Med.* 2000;6(4):335-42.
28. Reich-Schupke S. Postthrombotic syndrome: an update. *Dermatologie.* 2026;77(5):315-9.
29. Shafiee E, MacDermid JC, Packham T, Grewal R, Farzad M, Bobos P, et al. Rehabilitation Interventions for Complex Regional Pain Syndrome: An Overview of Systematic Reviews. *Clin J Pain.* 2023;39(9):473-83.
30. Birklein F, Schlereth T. Complex regional pain syndrome-significant progress in understanding. *Pain.* 2015;156(1):S94-103.
31. Kozin F, McCarty DJ, Sims J, Genant HK. The reflex sympathetic dystrophy syndrome. A review. *Am J Med.* 1981;70(1):23-30.
32. Hooshmand H. *Chronic Pain: Reflex Sympathetic Dystrophy.* Boca Raton: CRC Press. 1993.
33. Bruehl S. Complex regional pain syndrome. *BMJ.* 2015;351:h2730.

Cite this article as: Negari M. The mechanical-vascular hypothesis of Sudeck's atrophy: venous outflow obstruction. *Int J Res Orthop* 2026;12:2102-6.