Ultrasound-guided Cryoneurolysis for Peripherally Induced Dystonia after Non-neurological Surgery

Arun Kalava, MD[1] • Alvaro Soto Rincon, MD[2] / [1]TampaPainMD, Tampa, FL • [2]Larkin Community Hospital, Department of Neurology, South Miami, FL

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INTRODUCTION

- **Acquired dystonia** is defined by CNS-centric etiologies; the 2025 MDS consensus retains "acquired" without specifying anatomic level or mechanism.[1]
- **Dystonia is routinely mistaken for spasticity** (velocity-dependent hypertonia from stretch-reflex hyperexcitability, UMN lesions); dystonia is sustained involuntary contraction producing abnormal posture.[1]
- **Largest cohort (n=713):** fixed dystonia was the most common peripherally induced movement disorder-related dystonia
- **Peripheral trauma raises dystonia risk** (adjusted HR 3.12; 95% CI 2.30–4.24); the "second-hit" hypothesis: nerve trauma unmasks dystonia in a susceptible host via spinal/central maladaptive plasticity.[3]
- **DNBs localize the peripheral motor generator[5]; cryoneurolysis targets it directly[4]** . First reported use of DNB-directed cryoneurolysis for refractory PID.

MATERIALS & METHODS

- **Three women (ages 37–63)** with treatment-refractory post-surgical PID after non-neurological surgery; Retrospective, consecutive case series (CARE/EQUATOR, HIPAA-compliant, written consent).
- **US-guided diagnostic nerve block (DNB)** with electrical stimulation localized the peripheral motor generator in each patient.[5]
- **Percutaneous cryoneurolysis** (Iovera, Pacira Biosciences) under high-frequency US guidance (15–4 MHz), following 1% lidocaine skin wheal and 18-G introducer placement.
- **Cryoablation:** ≈−88°C (1-s pre-warm, 60-s freeze, 45-s thaw) targeting the same motor branches identified by DNB; produces reversible axonotmesis while preserving endoneurial architecture.[4]
- **Outcomes:** NRS pain, motor/functional examination, photographs, and durability of relief.

RESULTS / CASE DESCRIPTION *Table 1. Master Summary*

Characteristics	Patient 1	Patient 2	Patient 3
Age / Sex	52 y, F	37 y, F	63 y, F
			
Surgical trigger (interval)	Foot reconstruction (16 y)	Revision 1st-rib resection + nerve graft (neurogenic TOS); hematoma/lymphatic leak, 3 reop. (3 y)	Sacral fall + right THA (4 y)
Phenotype	CRPS-I, fixed equinovarus dystonia, allodynia	Chest-wall neuropathic pain, ICN spasm, pectoral hypertonicity, ↓ UE ROM	Groin pain, PF hypertonicity, vulvar dysesthesia, dyspareunia, sitting intolerance; glut. med/min wasting
UMN signs	None	None	None
Failed prior therapy	Baclofen, tizanidine, diazepam, gabapentinoids, BTX, ketamine, SCS, bracing, PT	Transdermal fentanyl, steroids, clonidine, serial nerve blocks, IV ketamine	NSAIDs, gabapentin, PF PT, BTX, pudendal decompression, steroid injections
DNB target → response	Gastrocnemius, soleus, FDL, TP → 80–90% equinovarus correction	T3–T6 ICN, LTN, TDN, MPN/LPN → near-complete relief w/ pectoral blocks; 50–100% w/ ICN blocks	Pudendal + PFCN partial; obturator (ant+post) → >50% improvement, tx redirected
Cryoneurolysis target	Same as DNB targets	Same as DNB targets	Pudendal, PFCN, obturator (ant+post)
NRS (pre → post)	9 → 3	8 → 3	8 → 2
Functional outcome	Sustained plantigrade alignment, improved gait, ↓ brace dependence	Fentanyl discontinued, improved arm ROM & chest-wall expansion, ICN spasm ↓ >80%	Improved gait/posture/sitting tolerance, dyspareunia resolved
Durability / repeat	>9 mo; no repeat	>6 mo; repeat cryo of LTN, TDN, MPN/LPN, T3–T6 ICN at 3 mo	>9 mo; no repeat
Complications	None	Transient neuritis (resolved); no scapular winging/shoulder weakness	Transient hip-flexor spasm (resolved); no gait instability/adduction weakness

ABBREVIATIONS

BTX = botulinum toxin; CRPS = complex regional pain syndrome; DNB = diagnostic nerve block; FDL = flexor digitorum longus; ICN = intercostal nerve; IV = intravenous; LPN = lateral pectoral nerve; LTN = long thoracic nerve; MPN = medial pectoral nerve; NRS = numerical rating scale; NSAIDs = nonsteroidal anti-inflammatory drugs; PF = pelvic floor; PFCN = posterior femoral cutaneous nerve; PT = physical therapy; ROM = range of motion; SCS = spinal cord stimulation; TDN = thoracodorsal nerve; THA = total hip arthroplasty; TOS = thoracic outlet syndrome; TP = tibialis posterior; UE = upper extremity; UMN = upper motor neuron.

FIGURE 1. PATIENT 1

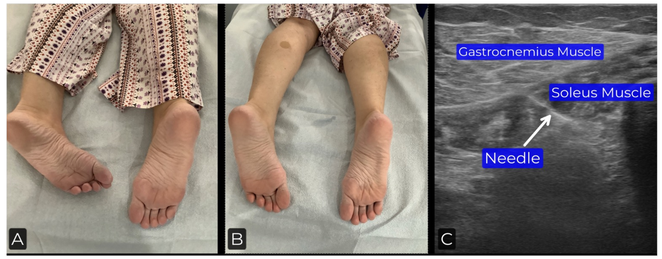


Figure 1. Patient 1: CRPS-associated fixed equinovarus dystonia (prone). **(A)** Pre-procedure fixed left equinovarus deformity (sole facing medially) with cold, dusky-purple discoloration of cold-type CRPS; right foot neutral, normally perfused. **(B)** Plantigrade alignment restored after US-guided diagnostic nerve block with electrical stimulation of tibial motor branches; purple discoloration resolved with return to normal color and warmth (restored perfusion after sympathetic blockade). **(C)** US: cryoprobe needle (arrow) between gastrocnemius and soleus at an intramuscular motor branch; each branch (gastrocnemius, soleus, FDL, tibialis posterior) treated in separate passes (FDL, tibialis posterior not shown).

DISCUSSION & CONCLUSIONS

- **Three PID phenotypes** treated with DNB-guided cryoneurolysis achieved relief lasting >6–9 months.[4,5]
- **P1:** CRPS fixed dystonia from aberrant Golgi tendon organ feedback
- **P2:** sequential insults ~ "second-hit" (untested)[3]; resting pectoral hypertonicity met dystonia[1]
- **P3:** spread beyond index injury; superior gluteal injury disrupted proprioception; DNB reidentified obturator as dominant generator.[5]
- **Peripherally Induced Dystonia (PID)** may be a cumulative spectrum of aberrant sensorimotor compensation after peripheral nerve injury in a susceptible host (genetic, environmental, psychological). The injury drives spinal/central maladaptive plasticity (a "second hit"), integrating nerve dysfunction, altered proprioception, and central sensitization into a specific motor phenotype.

SCAN FOR EXPANDED VERSION & REFERENCES

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