

Atypical Delayed Brachial Plexopathy with Dysautonomia: A Case of Minor Trauma with Major Sequelae

INTRO

- Brachial plexopathies are commonly associated with high-impact trauma with acute neurologic deficits.
- In rare instances, minor injuries can trigger a slow, insidious cascade of neurological dysfunction.
- We present a unique case of chronic brachial plexopathy with evolving dysautonomia several years after a seemingly negligible shoulder injury.

METHODS

- Consent for the report was obtained from the patient.

CASE REPORT

- 36-year-old female PMH of eosinophilic esophagitis and depression presented with chronic right shoulder and clavicular pain for 3 years after attempt to force open a shut door with her body. No fracture or dislocation were noted upon initial injury, and she had an unrevealing exam with improvements in pain over the first couple months.
- At 6-9 months, she noted shoulder drooping, fatigue, radiating numbness to the spine, hand, and fingers. Scapular winging, intermittent “shocking”, right axillary and scapular hyperhidrosis, early satiety, and diarrhea followed after 1 year. She denied allodynia, swelling, skin color or temperature changes.
- MRI showed mild disc bulges at C5-C7 without spinal stenosis or neural foraminal narrowing and mild supraspinatus tendinosis.
- EMG revealed grade 3 fibrillation potentials in the trapezius without activation, concerning for right spinal accessory mononeuropathy due to disruption in nerve continuity vs. poor activation due to pain.
- MR neurogram demonstrated right trapezius and SCM muscle atrophy but no signal abnormality along the brachial plexus or spinal accessory nerve.
- She was referred to the Peripheral Nerve Injury Clinic. A stellate ganglion block was scheduled to manage pain and autonomic symptoms.

A rare case of **progressive dysautonomia** emerging years after minor shoulder trauma, revealing an underlying nerve injury.



Figure 1 Demonstration of the patient's physical exam findings at rest and when doing a wall push.

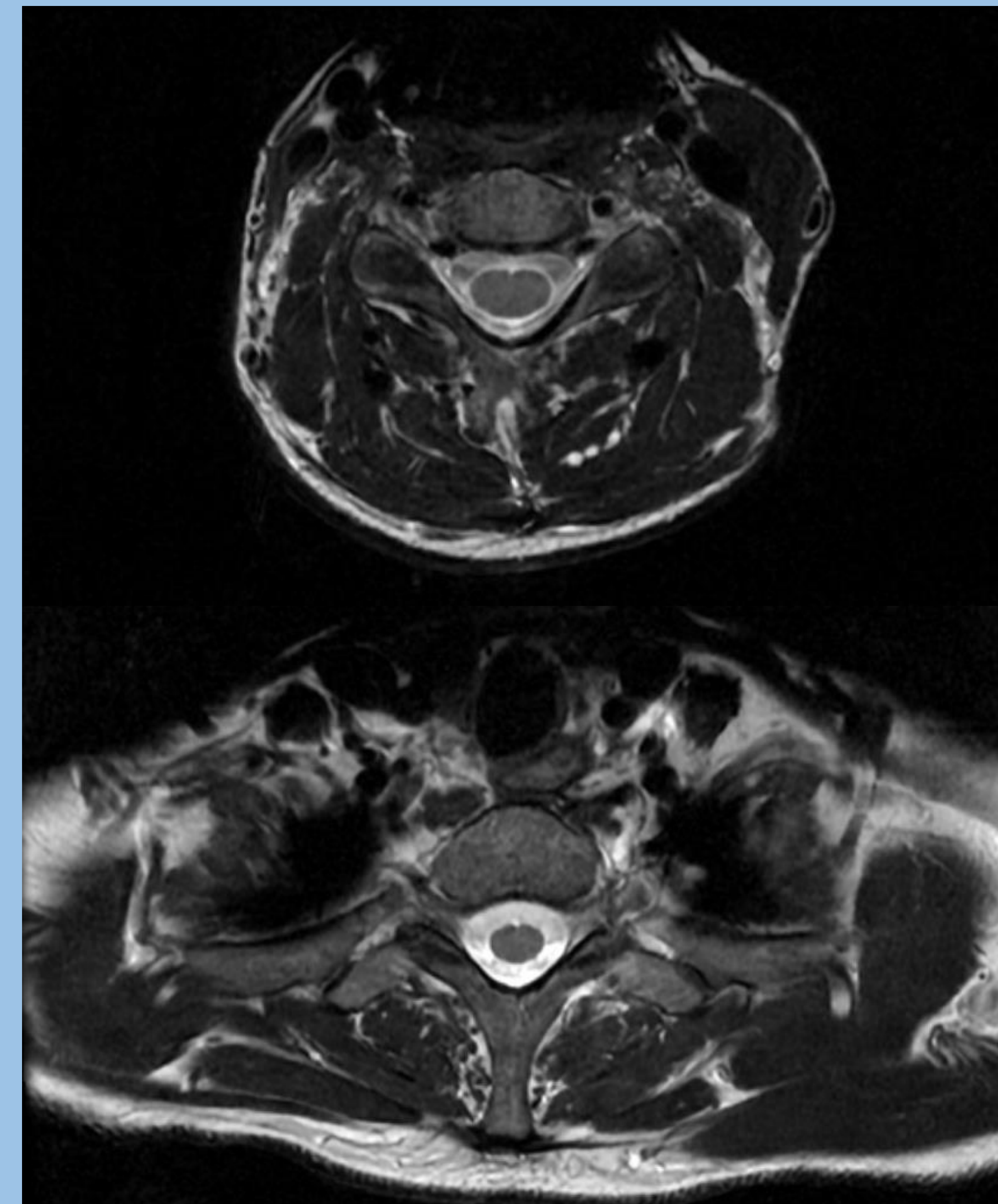


Figure 2 MRI C-spine at C5 and T1 which reveals both sternocleidomastoid and trapezius muscle atrophy.

DISCUSSION

- Deafferentation from nerve injury, as well as damage to C-fibers, can lead to dysregulation of afferent and efferent pain signals, resulting in persistent neuropathic pain.
- The gradual onset of peripheral neuropathy and long-term sequelae following axonotmesis complicated early recognition.
- The development of autonomic features including hyperhidrosis, early satiety, and diarrhea suggests a sympathetically mediated pain source.
- In rare cases, neuropathies can progress to central sensitization or complex regional pain syndrome (CRPS).
- However, the absence of allodynia, swelling, and skin changes, which are hallmarks of Budapest criteria for CRPS, reduce suspicion for extensive sympathetic injury.
- Identifying the primary pain generator is imperative for developing effective treatment strategies. Conservative treatment with physical therapy, anti-inflammatories, and nerve pain medications aims to reduce inflammation and promote healing.
- Stellate ganglion blockade can be both diagnostic and therapeutic by modulating sympathetically mediated pain and alleviate autonomic dysfunction.
- Ultimately, this case demonstrates how even minor injuries can precipitate complex pathologies requiring comprehensive, individualized management.



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