รายงานผู้ป่วย . Case Report

โรค Neuralgic Amyotrophy แบบไม่มีอาการปวดในผู้หญิงหลังคลอด: กรณีศึกษาและการทบทวนวรรณกรรม

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Painless Neuralgic Amyotrophy in Postpartum Woman: A Case Study and Literature Review

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บทคัดย่อ

หลักการและวัตถุประสงค์: โรค Neuralgic Amyotrophy เป็นความผิดปกติของระบบประสาทส่วนปลายที่พบได้น้อย มักมีลักษณะเด่นคือมีอาการปวดแขนเฉียบพลัน ตามด้วยอาการอ่อนแรงและกล้ามเนื้อฝ่อลีบ การพบผู้ป่วยที่ไม่มีอาการปวดนำมาก่อนเป็นกรณีที่พบได้ไม่บ่อย และอาจทำให้การวินิจฉัยล่าช้า กรณีศึกษานี้จึงมีวัตถุประสงค์เพื่อแสดงถึงอาการของโรค Neuralgic Amyotrophy ที่ไม่มีอาการปวดร่วมด้วยในผู้หญิงหลังคลอดและเน้นถึงความสำคัญของการประเมินทางคลินิก ภาพถ่ายรังสี และการตรวจไฟฟ้าวินิจฉัย

วิธีการศึกษา: กรณีศึกษาในผู้ป่วยเพศหญิงอายุ 34 ปี ที่มีอาการอ่อนแรงของแขนซ้ายโดยไม่มีอาการปวด โดยรวบรวมข้อมูลการตรวจทางคลินิก ผลการตรวจด้วยเครื่องตรวจคลื่นแม่เหล็กไฟฟ้า และการตรวจไฟฟ้าวินิจฉัยจากเวชระเบียน

ผลการศึกษา: ผู้ป่วยมีอาการอ่อนแรงที่แขนซ้ายโดยไม่มีอาการปวด เกิดขึ้นเก้าวันหลังผ่าตัดคลอดบุตรด้วยการระงับความรู้สึกเฉพาะส่วนโดยต้องมีการฉีดยาชาเข้าช่องน้ำไขสันหลัง ผลการตรวจด้วยเครื่องตรวจคลื่นแม่เหล็กไฟฟ้าพบว่าไม่มีความผิดปกติ แต่ผลการตรวจไฟฟ้าวินิจฉัยพบว่ามีพยาธิสภาพที่ร่างแหประสาทแขนซ้ายส่วนบน ซึ่งสอดคล้องกับการวินิจฉัยโรค Neuralgic Amyotrophy ผู้ป่วยรายนี้ได้รับการรักษาฟื้นฟูด้วยการทำกายภาพบำบัดและการทำกิจกรรมบำบัด

สรุป: โรค Neuralgic Amyotrophy ที่ไม่มีอาการปวดอาจทำให้การวินิจฉัยนั้นเป็นไปได้อย่างล่าช้า การประเมินทางคลินิกอย่างครอบคลุมร่วมกับการตรวจไฟฟ้าวินิจฉัยและภาพถ่ายรังสีมีความสำคัญอย่างยิ่งสำหรับการวินิจฉัยที่ถูกต้องและรวดเร็ว การบำบัดรักษาฟื้นฟูอย่างทันท่วงทีเป็นสิ่งที่สำคัญที่มีผลต่อการฟื้นฟูสภาพของผู้ป่วย

คำสำคัญ: Neuralgic amyotrophy, ไม่มีอาการปวด, การแสดงออกของโรคในรูปแบบที่ไม่พบได้ทั่วไป, ผู้หญิงหลังคลอด

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Abstract

Background and Objectives: Neuralgic amyotrophy (NA) is a rare peripheral nerve disorder marked by the acute onset of upper limb pain, followed by weakness and atrophy. A painless presentation is uncommon and may delay diagnosis. This case aims to highlight an atypical presentation of NA in a postpartum woman and underscore the importance of clinical evaluation, imaging, and electrodiagnostic studies.

Methods: A case study was conducted on a 34-year-old postpartum woman with left upper limb weakness, without preceding pain. Clinical examinations, magnetic resonance imaging (MRI), and electrodiagnostic studies were reviewed from medical records.

Results: The patient developed a progressive, painless weakness of the left upper extremity nine days after cesarean section. MRI revealed no abnormalities; however, electrodiagnostic studies confirmed left upper trunk brachial plexopathy, consistent with NA. The patient was treated with physical and occupational therapy.

Conclusion: Atypical, painless NA presents diagnostic challenges. Accurate diagnosis relies on comprehensive clinical evaluation, electrodiagnostic studies, and imaging. Early recognition and timely rehabilitation are crucial for achieving optimal recovery.

Keywords: meuralgic amyotrophy, painless, atypical presentation, postpartum

Introduction

Neuralgic amyotrophy (NA), also referred to as brachial neuritis or Parsonage-Turner syndrome, is a rare peripheral nerve disorder believed to result from an inflammatory process. The etiology may be idiopathic or hereditary and is often preceded by biomechanical or immunologic triggers. The typical clinical presentation involves the acute onset of severe shoulder pain, followed by muscle weakness and atrophy in the affected, usually unilateral, upper extremity.[1,2] Atypical presentations such as isolated mononeuropathies, painless motor deficits, purely sensory symptoms, or distal involvement have also been reported but are less common.[2,3] This case study presents an atypical, painless manifestation of neuralgic amyotrophy, characterized by progressive left shoulder weakness in a postpartum woman.

Case study

A 34-year-old right-handed Thai woman with a history of bilateral carpal tunnel syndrome during pregnancy, manifested by numbness in both hands, and no history of pregestational or gestational diabetes mellitus, presented with new-onset painless weakness of the left upper limb. The symptoms developed nine days after she delivered a full-term infant via cesarean section under spinal anesthesia. The perioperative course was uneventful, and no complications were noted during her hospital stay. Both mother and infant were discharged after three days of hospitalization.

Upon further evaluation, the patient reported a sudden onset of weakness in the left shoulder and arm upon waking from sleep. The weakness remained static, with neither progression nor improvement, and led to difficulty performing activities such as washing and combing her hair. There were no associated symptoms of pain, numbness, or sensory deficits. There was no reported trauma or mispositioning of the left upper extremity during the procedure. Her family history was negative for neuromuscular disorders.

On physical examination, the patient demonstrated a full range of motion in the neck and shoulder, with no pain on palpation or during resisted testing. Neurological examination revealed hyporeflexia of the left biceps and brachioradialis reflexes. According to the Medical Research Council (MRC) scale, muscle strength in the left infraspinatus was graded 1/5, the deltoid 2/5, and the biceps, brachioradialis, subscapularis, and pronator teres 4/5. No other muscle weakness or sensory deficits were identified. There was no evidence of muscle atrophy or scapular winging. Provocative tests for cervical radiculopathy, musculoskeletal shoulder pathology, and peripheral nerve entrapment were negative, except for a positive Tinel's sign at the wrist and a modified Phalen's test, consistent with her pre-existing diagnosis of carpal tunnel syndrome. A compensatory mechanism involving activation of the left upper trapezius muscle during shoulder abduction was observed (Figure 1A–B).

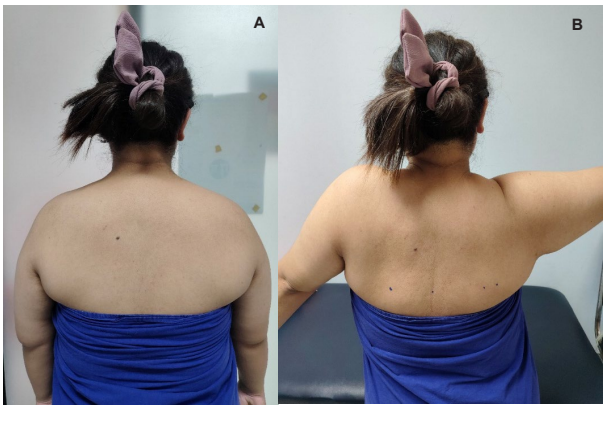

Figure 1 Posterior view of the patient.
(A) Symmetrical scapulae without visible muscle atrophy.
(B) During shoulder abduction, elevation of the left shoulder is observed due to compensatory activation of the left upper trapezius muscle.

Magnetic resonance imaging (MRI) of the brain revealed no mass lesions and no evidence of acute or subacute infarction. MRI of the cervical spine demonstrated mild intervertebral disc degeneration without significant spinal cord or nerve root compression

(Figure 2A). MRI of the left brachial plexus showed no mass lesions or abnormal enhancement (Figure 2B).

An electrodiagnostic study (EDX) was performed 11 days after the onset of symptoms. Nerve conduction studies (NCS) demonstrated delayed sensory and motor latencies in the bilateral median nerves, consistent with carpal tunnel syndrome (Table 1-2). All other nerve conduction parameters were within normal limits. Needle electromyography (EMG) revealed abnormal spontaneous activity and increased insertional activity in the left infraspinatus, deltoid, and biceps muscles (Table 3). These muscles also exhibited polyphasic and complex motor unit action potentials with reduced recruitment. The left brachioradialis muscle demonstrated reduced recruitment without abnormal spontaneous activity or abnormal motor unit morphology. EMG findings in other examined muscles were normal.

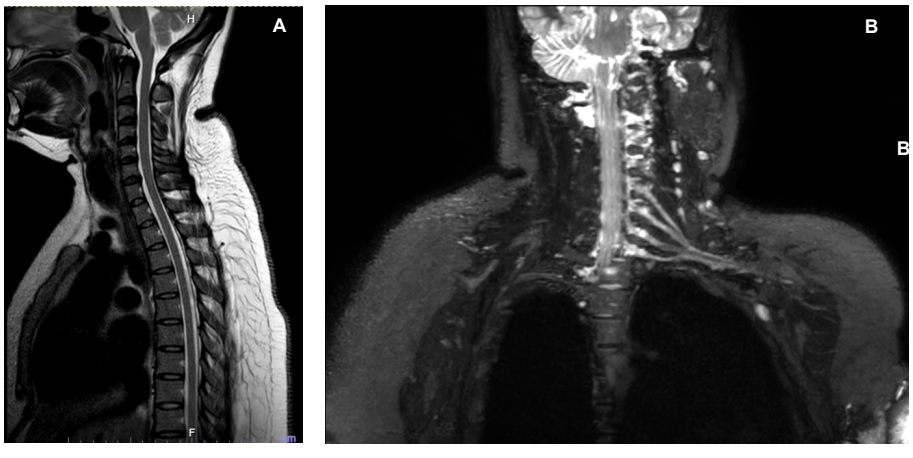

Figure 2 Magnetic resonance imaging (MRI) findings.
(A) T2-weighted sagittal MRI of the cervical spine showing mild intervertebral disc degeneration.
(B) Short tau inversion recovery (STIR) sequence MRI revealed no mass lesion or abnormal signal intensity within the left brachial plexus.

The patient was diagnosed with left-sided atypical neuralgic amyotrophy, primarily involving the upper trunk of the brachial plexus, based on clinical assessment, radiological findings, and supporting electrodiagnostic results. Management was conducted collaboratively by a neurologist and a physiatrist. Neither corticosteroids nor immunosuppressive therapies were administered, and analgesic medications were not prescribed. A comprehensive rehabilitation program was initiated, incorporating both physical and occupational therapy. The therapeutic approach emphasized scapular muscle re-education to reduce compensatory movement patterns that could potentially lead to secondary pain. Additionally, training in activities of daily living (ADLs) was provided to facilitate the patient's functional independence and promote optimal recovery. At the three-month follow-up, the patient reported gradual improvement in muscle strength. Manual muscle testing revealed an MRC grade of 4/5 in both the infraspinatus and deltoid muscles. Scapular motion had returned to normal. However, the patient continued to experience fatigue and reduced endurance during activities involving the affected limb.

A follow-up electrodiagnostic study was conducted to assess reinnervation. Sensory NCS results were within normal limits. Needle EMG showed no abnormal spontaneous activity and normal insertional activity in the left infraspinatus, deltoid, biceps, and brachioradialis muscles (Table 4). These muscles exhibited polyphasic and complex motor unit action potentials with reduced recruitment. Additionally, the left infraspinatus demonstrated giant triphasic motor unit potentials with mildly complex

morphology. These findings are suggestive of ongoing reinnervation. Examination of the contralateral limb was performed to exclude systemic involvement, and the results were normal.

Table 1 Sensory nerve conduction study results.

Nerve	Stimulation Site	Recording Site	Latency (ms)	Amplitude (μV)	Distance (cm)	Velocity (m/s)
Left Median	Wrist	Digit III	6.15	12.2	14	22.76
Right Median	Wrist	Digit III	7.29	13.4	14	19.20
Left Ulnar	Wrist	Digit V	2.40	41.8	12	50.00
Right Ulnar	Wrist	Digit V	2.34	51.9	11	47.01
Left Superficial Radial	Forearm	1st Web space	1.56	64.6	10	64.10
Right Superficial Radial	Forearm	1st Web space	1.67	48.9	10	59.88
Left Medial antebrachial cutaneous	Elbow	Forearm	1.72	13.9	8	46.51
Left Lateral antebrachial cutaneous	Elbow	Forearm	1.67	41.8	7.5	44.91
Right Lateral antebrachial cutaneous	Elbow	Forearm	1.88	34.8	10	53.19

Table 2 Motor nerve conduction study results.

Nerve	Sites	Latency (ms)	Amplitude (mV)	Distance (cm)	Velocity (m/s)
Left Median- APB	Wrist	6.92	7.3	8	
	Elbow	10.96	6.8	19	47.0
Right Median-APB	Wrist	6.33	7.8	8	
	Elbow	10.54	6.2	20	47.5
Left Ulnar-ADM	Wrist	1.98	7.0	8	
	RCG	5.48	6.7	21.5	61.4
Right Ulnar-ADM	Wrist	2.02	6.7	6	
	RCG	5.58	6.1	23	64.6

Abbreviations: APB, Abductor pollicis brevis muscle; ADM, Abductor digiti minimi muscle; RCG, Retrocondylar groove

Table 3 Needle electromyography findings at initial evaluation

Muscle	IA	Spontaneous			Motor unit action potential			Recruitment
		Fib	PSW	Fasc	Morphology	Amp (uV)	Duration	Pattern
Left Deltoid	Increased	None	2+*	None	Triphasic, Polyphasic	800-1,200	Increased	Reduced
Left Biceps brachii	Increased	None	2+*	None	Triphasic, Complex	1,000-1,500	Increased	Reduced
Left Brachioradialis	Normal	None	None	None	Triphasic, Mildly complex	1,000-1,500	Normal	Reduced
Left Triceps brachii	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal
Left Pronator teres	Normal	None	None	None	Triphasic	800-1,200	Normal	Normal
Left Flexor pollicis longus	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal
Left Extensor indicis proprius	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal
Left First dorsal interos-seous	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal
Left Rhomboid major	Normal	None	None	None	Triphasic	800-1,200	Normal	Normal
Left Infraspinatus	Increased	None	2+*	None	Triphasic, Complex	1,000-1,500	Increased	Reduced
Left Cervical paraspinals	Normal	None	None	None	Triphasic	800-1,200	Normal	NA

Abbreviations: IA, Insertional activity; Fib, Fibrillation; PSW, Positive sharp wave; Fasc, Fasciculation; Amp, Amplitude; NA, Not available

* Spontaneous size 100-300 μV

Table 4 Needle electromyography findings at follow-up evaluation

Muscle	IA	Spontaneous			Motor unit action potential			Recruitment
		Fib	PSW	Fasc	Morphology	Amp (uV)	Duration	Pattern
Left Deltoid	Normal	None	None	None	Triphasic, Polyphasic	1,000-1,500	Increased	Reduced
Left Biceps brachii	Normal	None	None	None	Complex, Polyphasic	1,000-1,500	Increased	Reduced
Left Brachioradialis	Normal	None	None	None	Triphasic, Complex	1,000-1,500	Increased	Reduced
Left Infraspinatus	Normal	None	None	None	Triphasic, Mildly complex	>5,000	Normal	Reduced
Right First dorsal interosseous	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal
Right Flexor carpi radialis	Normal	None	None	None	Triphasic	1,000-3,000	Normal	Normal
Right Biceps brachii	Normal	None	None	None	Triphasic	1,000-1,500	Normal	Normal

Abbreviations: IA, Insertional activity; Fibrillation; PSW, Positive sharp wave; Fasc, Fasciculation; Amp, Amplitude

Discussion

Neuralgic amyotrophy (NA) is an underrecognized inflammatory disorder of the peripheral nervous system. A recent prospective cohort study reported its incidence as approximately 1 in 1,000 individuals.[4] NA is observed more frequently in males than in females, with an estimated male-to-female ratio of 2:1. The disorder affects a broad age range, with a median age of onset around 40 years in idiopathic cases and approximately 25 years in those with the hereditary.[3] The etiology of NA can be classified as either idiopathic or hereditary. Hereditary neuralgic amyotrophy (HNA), the inherited form, follows an autosomal dominant pattern and is most associated with mutations in the SEPT9 gene located on

chromosome 17q25. Individuals with HNA have an increased risk of recurrent episodes compared to those with idiopathic NA.[5–7]

The pathophysiology of NA remains incompletely understood but is believed to involve a multifactorial interplay of genetic susceptibility, mechanical stress, and environmental or immunologic triggers.[2] Among environmental factors, infections are the most commonly reported triggers, accounting for 43.5% of cases. Mechanical factors such as strenuous upper limb activity represent the second most frequent cause (17.4%), followed by immune-mediated events including surgery (13.9%), the peripartum period (8.7%), and vaccination (4.3%).[2,3] In the present case, the onset of symptoms following cesarean section under spinal anesthesia suggests a possible immune-related trigger.

As this was the patient's first episode, a detailed family history was obtained to evaluate for HNA. No similar events, either postoperatively or postpartum, were identified among family members. Therefore, a diagnosis of HNA is considered unlikely. Nonetheless, ongoing monitoring is recommended considering the potential for recurrence following future triggering exposures.

The classic presentation of NA typically begins with the sudden onset of severe pain in the shoulder or upper arm, intensifying within hours. Pain may occur in the shoulder, neck, scapular region, or medial lower arm. Weakness typically follows within hours to days, and paresthesia may be felt along the lateral shoulder and arm. Notably, pain and paresthesia typically do not align with the territory of weakness. The classic phenotype of NA primarily affects the upper and middle brachial plexus, with the suprascapular and long thoracic nerves being the most involved. Atypical presentations, such as painless weakness as observed in this case, account for up to 21.9% of cases. Other uncommon variants include pure sensory symptoms, distal limb involvement, isolated mononeuropathies, and autonomic dysfunction.[1–3, 8–10]

Examination should begin with visual inspection and sensory evaluation. Muscle atrophy should be assessed, particularly in the shoulder girdle, scapular region, and forearm. Manual muscle testing should be conducted to identify specific muscle weakness. Scapular motion during abduction and anteflexion should be carefully evaluated for signs of scapular winging or dyskinesis.[1, 2, 9, 11]

Currently, no standard immunological tests exist to confirm NA. Routine laboratory investigations, including inflammatory markers, are typically unremarkable. Genetic testing for SEPT9 mutations may be warranted when HNA is suspected.[2,9]

Radiological evaluation plays an important role in supporting the diagnosis of NA. MRI of the brachial plexus may demonstrate T2 hyperintensity in affected trunks or denervated muscles.[9] Advanced imaging, including magnetic resonance neurography (MRN) and neuromuscular ultrasound (NMUS), can detect structural abnormalities in up to 90% of cases within the first month of symptom onset.[1, 11, 12] MRN has also been shown to identify pathological changes earlier than conventional MRI.[13]

EDX studies are crucial for confirming the diagnosis of NA, as they typically reveal features consistent with peripheral neuropathy, primarily axonal degeneration. Needle EMG, particularly when conducted 10 to 15 days after symptom onset, often demonstrates a neurogenic pattern and is highly informative. Given NA's predilection for motor nerves, it is not uncommon for sensory NCS to yield normal results. Therefore, a normal sensory NCS does not exclude the diagnosis of NA. Similarly, motor nerve conduction studies may appear normal, with amplitudes remaining within normal limits in some cases.[1, 2, 9] This was exemplified in the present case, in which sensory NCS findings were normal, while needle EMG revealed abnormalities consistent with upper trunk involvement of the brachial plexus.

Key differential diagnoses include cervical radiculopathy and shoulder joint pathologies, as symptom overlap can lead to misdiagnosis. A

definitive diagnosis requires integration of clinical presentation, imaging, and electrodiagnostic results.[1, 2, 9]

Effective pain management is essential for patients experiencing significant pain. Symptomatic treatment typically includes a combination of analgesics, gabapentinoids, tricyclic antidepressant, and non-steroidal anti-inflammatory drugs (NSAIDs), which are often sufficient. However, in more severe cases, opioid analgesics may be necessary.[1,2] Currently, there is insufficient evidence to support the routine use of intravenous immune globulin (IVIG) or corticosteroids.[14,15]

Rehabilitation management involves a multidisciplinary approach with shared goal setting between clinicians and the patient. Physical therapy primarily aims to restore motor control, particularly scapular positioning, coordination, and motion, often incorporating supportive feedback mechanisms. Emphasis is placed on optimizing movement quality rather than strength training. Occupational therapy addresses ADLs by implementing energy conservation techniques, ergonomic principles, activity modification, and the use of assistive devices to promote functional independence.[1, 2, 10]

Surgical neurolysis may be considered in cases of complete paralysis with no signs of recovery after six months.[10] Alternatively, NMUS or MRN can be performed at three months in patients lacking clinical improvement, to identify possible nerve constriction and guide surgical decision-making.[11]

The prognosis for NA is generally favorable approximately 80–90% of patients achieve substantial recovery within 2–3 years. However, a subset may continue to experience chronic pain, fatigue, or functional limitations.[2]

Conclusion

This case highlights a rare, atypical presentation of NA in the postpartum period, characterized by painless onset and upper trunk involvement. Such presentations may be easily overlooked or misdiagnosed, underscoring the importance of thorough clinical evaluation supported by electrodiagnostic studies and advanced imaging modalities. Early recognition and initiation of a multidisciplinary rehabilitation program are key to improving functional outcomes.

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