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Beyond the "Stinger": Neuralgic Amyotrophy Presenting after Traumatic Tackle in a Collegiate Football Player

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Abstract

Suprascapular neuropathy is an uncommon etiology of shoulder weakness and dysfunction in contact-sport athletes. This case report describes an acute traumatic suprascapular mononeuropathy in a collegiate American football player following tackle-related trauma. On the basis of the patient's history, clinical examination, and imaging studies, the diagnosis was attributed to neuralgic amyotrophy of the suprascapular nerve that was precipitated by a traumatic initial tackle event. Due to its initial symptoms and uncommon occurrence this case presented with a delay in diagnosis, a common challenge in neuralgic amyotrophy. Early recognition through appropriate imaging and electrodiagnostic testing enables accurate prognostication, treatment, and prevents premature return to play before adequate reinnervation occurs.

Keywords

Neuralgic Amyotrophy, Parsonage-Turner Syndrome, Mononeuropathy, Trauma

1. Introduction

There is a high burden of sports-related injuries among young athletes. This is a result of a complex interplay of individual risk factors, including body composition, age, and gender, in combination with sport-specific demands. Contact sports such as football show markedly higher injury incidence [1]. This is readily evident considering the mechanisms of injury inherent to football, including direct trauma, falls, and tackling collisions [2]. Fractures are the most frequently reported injuries among adolescent football players; followed by sprains, contu-

sions, head injuries, and dislocations [3]. A 20-year review of 56,809 football-related fractures demonstrated that young athletes sustained the greatest injury burden accounting for 41.2% of the study population, with upper extremity fractures accounting for 73.7% of cases [4]. While these data highlight the prevalence of bony injury, associated neurologic injuries may be underrecognized.

Peripheral nerve injuries are reported far less frequently in American football and are often underrepresented in large injury surveillance studies and reported cases [5] [6]. When neurologic injuries do occur, they most commonly involve transient brachial plexus neuropraxia, colloquially referred to as "stingers" or "burners" which result from traction or compression of the cervical nerve roots or upper trunk during tackling or collision events [7] [8]. These injuries are typically characterized by acute, unilateral burning pain radiating down the upper extremity with rapid resolution of symptoms and minimal long-term sequelae. Although stingers are relatively common in contact sports, particularly among football players, neuralgic amyotrophy (NA) is rare [9]. This rarity may cause the pathology to be overlooked due to symptom overlap, especially in the absence of hfracture, dislocation, or persistent pain [10]. Recognition and appropriate investigation of these less common neurologic injuries is critical, as delayed diagnosis may result in prolonged weakness, muscle denervation, and functional impairment [11]. The following case presentation describes the occurrence of this uncommon injury in the context of competitive football.

2. Case Presentation

A 21-year-old male with no significant past medical history presented to a Physical Medicine and Rehabilitation clinic with a five-week history of progressive left upper extremity weakness. Approximately five weeks prior to presentation, the patient sustained a tackling collision during a football game, during which he experienced a transient shooting pain radiating down his left arm. He did not note any lasting pain, neurological deficits, or functional limitation immediately following the event and returned to play after a brief rest on the sidelines.

Several days later, while performing an overhead press during strength training, the patient experienced pain in the left shoulder; he was nonetheless able to complete the training session and continued to participate in football practice without restriction. Over the subsequent days, however, he developed a significantly limited range of motion of the left shoulder upon waking. This limitation was functionally apparent when he attempted to play golf and was unable to do so. He also noted that self-massage of the left arm provoked a shooting pain radiating distally, similar to his initial event.

At the time of clinic presentation the patient denied pain at rest. Significant atrophy of the infraspinatus and supraspinatus were noted on exam. He reported weakness with left shoulder abduction, and external rotation, which was confirmed with manual muscle testing revealing a grade of 3/5. Sensation was intact throughout the bilateral upper extremities. Bicep tendon reflex, tricep tendon re-

flex, and brachioradialis tendon reflex were 2/4 and symmetrical bilaterally. Scapular mechanics were grossly unaffected and were symmetrical bilaterally. Empty Can test was positive for weakness but no pain. He was already participating in physical therapy approximately three weeks prior to this visit after visiting an orthopedist. He reported significant improvement in range of motion.

The patient was initially evaluated by an orthopedic surgeon and subsequently referred to Physical Medicine and Rehabilitation for further workup. Cervical and left shoulder MRI were obtained (**Figure 1** & **Figure 2**). Cervical spine MRI demonstrated tubular structure with increased T2 signal extending from the left neural foramen at C4-C5 to the left subclavian region. Left shoulder MRI revealed a thickened, edematous suprascapular nerve with associated edema and atrophy of the supraspinatus and infraspinatus muscles, with other muscles unaffected. Electrodiagnostic evaluation of the bilateral upper extremities was performed (**Figure 3** & **Figure 4**), yielding an abnormal study consistent with focal left suprascapular mononeuropathy with evidence of active denervation of the supraspinatus and infraspinatus muscles. The patient participated in 3 months of physical therapy with good return of function for everyday activities. The patient was subsequently lost to follow-up and it is therefore unconfirmed what sports activity level he returned to. He did not undergo surgery, or require other interventions.

The patient was diagnosed with Neuralgic Amyotrophy (NA) given these findings. Given the patient had an asymptomatic interval of days, then weakness began with weeks of deterioration (**Figure 5**). It is unlikely that a nerve traction injury occurred as the deficits would have been seen constant from time of injury. This diagnosis was determined as the deltoid was spared therefore it was not likely to be a traction injury as the more proximal muscles would be involved. The changes were isolated to the infraspinatus and supraspinatus leaving only the suprascapular nerve to be affected.

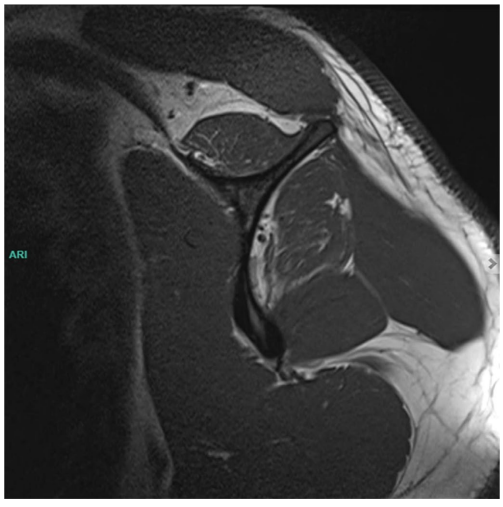

Figure 1. Sagittal view of MRI of left supraspinatus and infraspinatus showing atrophy with evidence of degeneration.

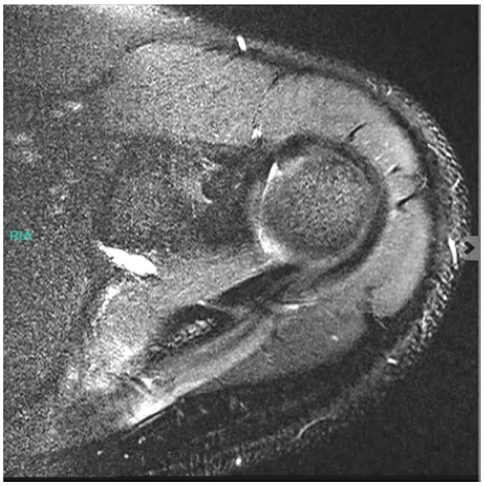

Figure 2. Transverse view of MRI showing left suprascapular nerve enhancement as it crosses the supraspinatus.

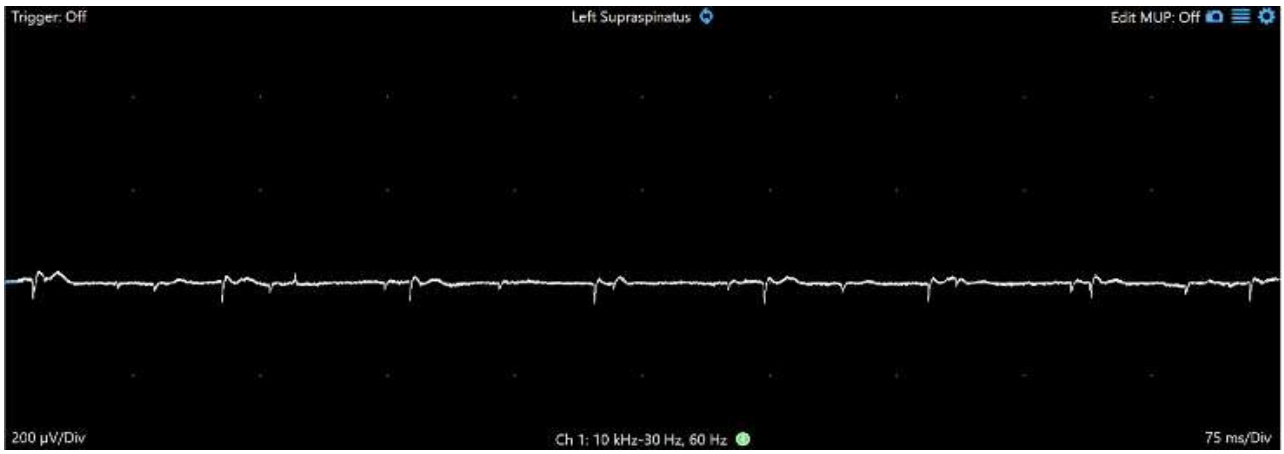

Figure 3. Needle EMG tracing of Left Supraspinatus showing fibrillations, confirming active denervation.

EMG+

Side	Muscle	Nerve	Root	Ins.Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int.Pat	Comment
Left	Deltoid	Axillary	C5-C6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Infraspin	Suprascapular	C5-C6	Nml	**2+**	Nml	Nml	Nml	0	Nml	Nml	
Left	Supraspin	Suprascapular	C5-C6	Nml	**2+**	Nml	Nml	Nml	0	Nml	Nml	
Left	Pronator teres	Median	C6-C7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Cervical PSP upper	Rami	C1-C3	Nml	Nml	Nml						
Left	Brachiorad	Radial	C5-C6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	APB	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-C8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Biceps	Musculocut	C5-C6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	

Figure 4. EMG table showing needle testing of all LUE muscles.

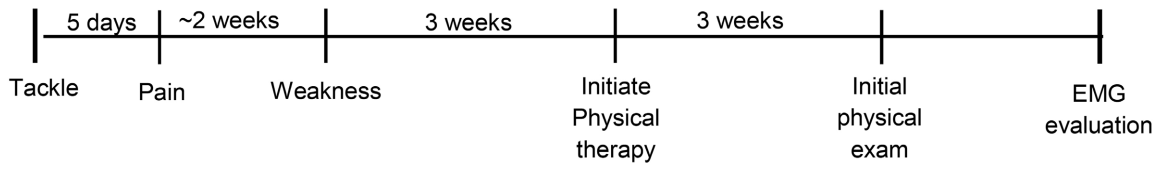

Figure 5. Timeline course of events.

3. Discussion

Parsonage-Turner syndrome, also known as neuralgic amyotrophy (NA), is an idiopathic peripheral neuropathy most commonly affecting nerves of the shoulder girdle and upper extremity. It occurs in around 1.6 - 3 per 100,000 individuals per year, but is likely underdiagnosed [12] [13]. It results in muscle weakness of downstream muscles of the affected nerve but is commonly preceded by severe pain. The pain is often described as unrelenting sharp, severe pain that can last hours to weeks [14]. The etiology is unknown but is thought to be autoimmune in nature [15]. The diagnosis is clinical, based on history and physical exam. Determining other similarly presenting conditions that can mimic the presentation is an essential part of the medical investigation. Objective outcome measures applicable to NA include manual muscle testing for shoulder flexion, abduction, and external rotation; goniometric measurement of shoulder range of motion; validated patient-reported instruments such as the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, and the American Shoulder and Elbow Surgeons (ASES) standardized shoulder assessment. Both DASH and ASES have been used extensively to track outcomes in suprascapular neuropathy [16]. The diagnosis can be corroborated and supported with EMG and MRI findings [13] [14].

The most commonly affected nerves include the long thoracic, suprascapular, axillary, anterior interosseous, and phrenic nerves [13]. The suprascapular nerve provides motor innervation to supraspinatus and infraspinatus muscles, which are responsible for shoulder abduction and external rotation respectively. The anatomical course of the nerve beneath the superior transverse scapular ligament (TSL) at the suprascapular notch creates vulnerability to traction injury during forceful shoulder movements [17]. Suprascapular neuropathy has been well-documented in overhead sports (*i.e.* volleyball and baseball) from repetitive microtrauma or compression from paralabral cysts [10] [18]-[20]. Upon a literature review, no published cases were identified describing suprascapular neuropathy resulting from a football tackle mechanism.

The present case highlights a clinically meaningful peripheral nerve injury whose recognition and classification are essential for prognosis, management, and functional recovery [21] [22]. Peripheral nerve injuries are traditionally categorized using the Seddon classification which include neurapraxia, axonotmesis, and neurotmesis. These classifications describe increasing neuronal damage severity based on the degree of myelin sheath, axonal disruption and connective tissue involvement [23]. Sunderland later expanded this framework into a five-degree system, providing greater anatomic and prognostic specificity by distinguishing injuries with preserved endoneurial, perineurial, and epineurial integrity from those with neurotmesis [24] [25]. These classification systems remain central to contemporary clinical practice as they guide diagnostic evaluation, electrodiagnostic interpretation, and expectations for recovery.

Treatments for NA involve a multidisciplinary approach encompassing pain management, immune therapy, rehabilitation, and in select cases surgical inter-

vention [26]. For the acute phase, pain management and immune modulation are key. Corticosteroids can be used within the first month of onset, or in more extreme cases intravenous immunoglobulin may be considered within the first 2 weeks of onset [27]-[29]. For pain management, neuropathic pain agents in conjunction with nonsteroidal anti-inflammatory medications, and sometimes opiate medications are required. In chronic management comprehensive rehabilitation is key. A complete program should include physical therapy and occupational therapy. These will help the patient relearn motor patterns, help scapular coordination, and teach energy conservation management [26] [29]. Infrequently, prolonged chronic cases patients may require surgical options including neurolysis, nerve transfers, or tendon transfers [26] [29]-[31].

The prognosis of suprascapular mononeuropathy is closely tied to the severity of nerve injury as classified by the Seddon-Sunderland framework, the promptness of diagnosis, and the etiology of the injury. Poor prognostic indicators for conservative management include long duration of symptoms prior to treatment, marked muscle atrophy at presentation, and the presence of a persistent compressive lesion [17]. NA recovery is variable, cases are typically managed conservatively as they are usually identified in the chronic phase. Functional outcome is variable with recovery typically ranging from 8 - 18 months with varying and often incomplete recovery [12] [26] [32] [33]. Typically axonal regeneration proceeds at an estimated rate of around one millimeter per day along their previous course, but can vary from nerve to nerve [34].

Return to unrestricted sport should be guided by symmetrical shoulder strength, pain-free execution of sport-specific skills, and where possible, electrodiagnostic evidence of reinnervation without ongoing active denervation [12] [26] [35]-[37]. Activity modifications during the recovery period are an important adjunct to formal rehabilitation. For football, this most commonly includes limiting blocking and tackling mechanics that load the ipsilateral shoulder into traction or depression until adequate strength is restored and potentially modifying shoulder pad fitting to reduce compressive forces at the suprascapular notch. In athletes who are otherwise asymptomatic, concurrent participation in a structured rehabilitation program while continuing to compete with modified activity is a reasonable approach provided symptoms do not progress and serial monitoring is maintained [38].

4. Conclusion

Parsonage-Turner syndrome, also known as neuralgic amyotrophy (NA) is an idiopathic mononeuropathy usually occurring within shoulder or upper extremity neurovasculature [12]. This case highlights an uncommon presentation of NA in a collegiate American football player following tackle-related traction injury. Transient neurologic symptoms in American football are frequently attributed to benign brachial plexus neuropraxia or “stingers” [7] [8]. The diagnostic course in this patient underscores the importance of correlating clinical history with ad-

vanced imaging and electrodiagnostic testing to accurately localize the lesion, determine injury severity, and guide prognosis [21] [22]. This case serves to increase awareness of suprascapular neuropathy as a potential sports-related injury in collision athletics, despite its rarity in football. Timely recognition is essential for early referral to a structured rehabilitation program and appropriate management. This allows for progressive functional recovery while avoiding premature return to unrestricted play. This approach optimizes recovery, minimizes long-term functional deficits, and facilitates safe return to sport [26].

Author Contributions

Cameron Widhalm, DO—Conceptualization, data curation, investigation, project administration, writing—original draft, writing—review & editing, visualization, supervision;

Faredun Dungore, OMS-IV—Conceptualization, data curation, writing—original draft;

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Brian Khin, DO—Conceptualization, data curation, writing—review & editing;

Petro Zaroukian, MD—Conceptualization, data curation, writing—review & editing;

Justin Phillips, MD—Investigation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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