

ISOLATED AND PAINLESS (?) ATROPHY OF THE INFRASPINATUS MUSCLE

LEFT HANDED VERSUS RIGHT HANDED VOLLEYBALL PLAYERS

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SUMMARY - The suprascapular nerve originates from the upper trunk of the brachial plexus or less frequently from the root of C5. It runs a short way and crosses the suprascapular notch. It innervates the supraspinatus muscle and the acromioclavicular and glenohumeral joints. Then, it crosses the lateral edge of the spine of the scapula passing through the spinoglenoid notch, and innervates the infraspinatus muscle. These are potential sites of injury to the suprascapular nerve. Three cases of suprascapular nerve entrapment causing an isolated infraspinatus muscle atrophy in volleyball players were studied. It is suggested the hypothesis that the nature of the smash, in which the athlete uses the arm violently, more than does in volleyball service or in the art of reception, is the key to the pathogenesis of the lesion in volleyball players.

KEY WORDS: volleyball, infraspinatus muscle, suprascapular nerve, entrapment neuropathy, muscular atrophy.

Atrofia isolada e não dolorosa (?) do músculo infraespinhoso: jogadores de voleibol destros versus canhotos

RESUMO - O nervo supraescapular se origina do tronco superior do plexo braquial ou, menos frequentemente, da raiz de C5. Percorre curto caminho e cruza a incisura supraescapular. Daf, o nervo supraescapular dá ramos para o músculo supraespinhoso e para as articulações acromioclavicular e glenoumeral. Então, cruza a borda lateral da espinha da escápula, passando através da chanfradura espinoglenóide e inervando o músculo infraespinhoso. Esses são locais potenciais para lesão do nervo supraescapular. Foram estudados três casos de compressão do nervo supraescapular causando atrofia isolada do músculo infraespinhoso em jogadores de voleibol. É sugerida a hipótese de que a natureza do ataque, na qual o atleta usa o braço violentamente, mais do que no saque ou na recepção, seja a chave da patogênese da lesão em jogadores de voleibol.

PALAVRAS-CHAVE: voleibol, músculo infraespinhal, nervo supraescapular, neuropatia compressiva, atrofia muscular.

Injuries to the suprascapular nerve have been considered rather rare, even in athletes³. In 1963 Kopell and Thompson (quoted by Liveson et al.⁷) first described entrapment of the suprascapular nerve at the suprascapular notch. In 1981, according to Liveson et al.⁷, Ganzhorn et al. described the first case of suprascapular nerve entrapment at the spinoglenoid notch⁷. This sort of lesion has been observed in baseball pitchers², racket players and in volleyball players^{3,9}.

We present three cases of isolated atrophy of the infraspinatus muscle of the dominant shoulder, one of them occurring in a left handed volleyball player. The pathogenesis of this nerve entrapment syndrome is discussed.

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CASE REPORTS

Case AAMS, a 17 year-old single white male, right handed volleyball player had been alerted by teammates to a right scapular atrophy about two months before being examined. He had never noticed any change in his performance besides of restricted inability to lift weight overhead when the right arm was abducted. He could not define the onset of symptoms. He began to play volleyball at the age of 11 and very soon he was in the main team. He is a very powerful "top balls" smasher. The patient had no history of major illness, bone fractures or neuromuscular dysfunction. Examination revealed a well-nourished teenage in a good physical condition. Cranial nerves were normal, reflexes were active and symmetrical, there were no sensory changes and cervical symptoms were absent. It was observed a marked atrophy and weakness of the right infraspinatus muscle, without any involuntary movements such as fasciculations and tremors. The other rotator cuff muscles were normal on examination. Radioimaging exams were normal. The patient was treated conservatively and continued to play volleyball. After six months, the patients began to complain of pain in the dominant shoulder. At this time, the examination revealed a diminution in the muscle bulk and a persistent muscle weakness. The electrophysiologic examination was performed and did not confirm the nerve compression syndrome.

Case TDGC, an 18 year-old single white male, left handed volleyball player was seen with a complaint of asymmetry of the posterior shoulder musculature. He has never noticed any change in his performance. He could not define the onset of symptoms. As a medicine student, he read about this nerve compression syndrome in volleyball players and observed himself, finding an asymmetry of his left scapular musculature. He began to play volleyball at the age of 9 and he is a powerful top balls smasher. Examination revealed a well-nourished male in a good physical condition. Cranial nerves were normal, reflexes were active and symmetrical. No sensory changes or cervical symptoms were noticed. It was observed a marked atrophy, painful and isolated to the left infraspinatus muscle. No weakness was observed on careful manual examination. Radioimaging exams were normal. The electrophysiologic examination confirmed the existence of a suprascapular nerve compression syndrome. The patient was treated conservatively and continued to play volleyball. It was observed an increase of the atrophy after six months.

Case SEMN, a 19 year-old right handed volleyball player noticed an asymmetry of the right shoulder. He denied any change in his performance and was still able to play volleyball. He was also weight lifting. The patient denied any history of major illness. On physical examination no neurological deficits were observed his consciousness and mentation were normal, cranial nerves were normal, reflexes were active and symmetrical. Examination also revealed an isolated and painless atrophy of his right infraspinatus muscle. This atrophy was not expressive. The other right and left rotator cuff muscles were normal. No weakness was observed. Radioimaging exams were normal. The electrophysiologic studies did not confirm the nerve compression syndrome. The patient was treated conservatively and continued to play volleyball as a "time balls" smasher with no muscle bulk alterations after six months.

ANATOMY

The anatomy of the suprascapular nerve and mechanisms for its injury have been examined by many authors^{1-4,6,9}.

The infraspinatus muscle is situated in the infraspinatus fossa of the scapula. It is innervated by the suprascapular nerve which takes origin at in the upper trunk of the brachial plexus. Rarely, the suprascapular nerve originates solely from the distal portion of the C5 root, and in 50% of subjects it also receives fibers from the C4 root. It extends laterally across the scalenus medius muscle and under the trapezius and omohyoid muscles as it courses with the suprascapular artery (thyrocervicalis trunk branch) to the suprascapular notch. The suprascapular nerve is separated from the suprascapular artery in the suprascapular notch by the superior transverse scapular ligament. The artery passes across the ligament through the notch and the nerve courses the notch under the ligament providing anatomical predisposition to entrapment. The suprascapular nerve continues deep to the suprascapular muscle, which it innervated with two branches and also gives off sensory filaments to the acromioclavicular and glenohumeral joints.

The nerve leaves the suprascapular fossa and crosses the lateral edge of the spine of the scapula together with the suprascapular artery. In 50% of subjects, an osteofibrous orifice (spinoglenoid notch) is found enclosed by the spinoglenoid ligament, an aponeurotic band that separates the suprascapular and the infraspinatus muscles. Then, the nerve provides two branches to the belly of the infraspinatus muscle and sensory filaments to the glenohumeral joint and scapula (Fig 1).

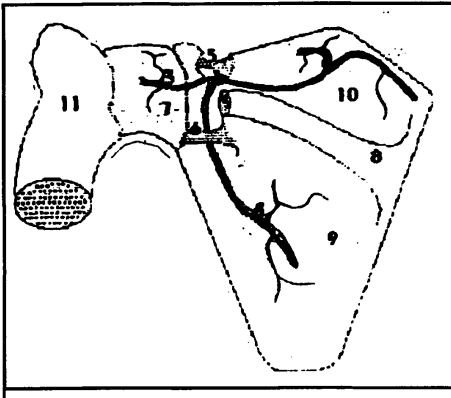


Fig 1. Representation of the anatomy of the suprascapular nerve and its branches.

1) Suprascapular nerve; 2) Branch to supraspinatus muscle; 3) Branch to the joint capsule; 4) Branch to infraspinatus muscle; 5) Supraspinatus notch; 6) Spinoglenoid notch; 7) Joint capsule; 8) Scapular spine; 9) Infraspinatus fossa; 10) Infraspinatus fossa; 11) Supraspinatus fossa; 12) Humerus.

degree, an other had a 2 atrophy degree and the last presented an 1 atrophy degree. This one uses to smash "time balls" (balls lifted next to the upper edge of the net and quickly smashed). The others use to smash "top balls" (lifted much higher from the upper edge of the net and that demand a longer period of time to be smashed). They all had isolated atrophy of the infraspinatus muscle in the dominant shoulder. No atrophy of the supraspinatus muscle was noticed. It places the lesion at the spinoglenoid notch, after the nerve gives off motor branches to the supraspinatus muscle. In two of our cases (Cases 1 and 2) there was a painful atrophy of the infraspinatus muscle irradiating to the shoulder joint and worsening after the games. As quoted by Goss⁴, the suprascapular nerve after passing through the spinoglenoid notch gives off sensory filaments to the scapula and glenohumeral joint. These are the reasons why the atrophy is isolated to the infraspinatus muscle and painful.

Hadley et al.⁶ quoted other possible causes of suprascapular nerve entrapment: lipomas, ganglion cysts, penetrating injuries, arthritic inflammation, systemic lupus erythematosus, surgical dissection and rotator cuff injuries, rupture of the long head of the biceps, degenerative spine disease and C5 radiculopathy. They were all excluded in our cases. Our patients had suprascapular nerve entrapment due to trauma to the shoulder with the injury of the nerve probably caused by eccentric movements of the shoulder while playing volleyball.

There are many hypotheses that try to explain the pathogenesis of this sort of nerve compression syndrome observed in volleyball players^{3,9}. Picot, quoted by Tengan et al.⁹ hypothesized that the nerve could be injured during movements of the shoulder as the nerve slides back and fourth through the foramen. Tengan et al.⁹ considered that this nerve lesion might be caused by the act of reception of the ball named "manchete". During this act of reception, the athletic would cause an eccentric contraction of the infraspinatus muscle that would increase the distance between the points of origin and termination of the nerve and might cause lesion of the nerve in its terminal branches. Due to this act of reception, there would be an unilateral lesion always located in the right shoulder as a consequence of directing the ball to the lifter that is frequently located to the right of the receptor, and it would cause higher tension to the right shoulder.

KINESIOLOGY

Two fixed points and two critical points have been identified along the course of the suprascapular nerve^{1,9}. It is fixed in its origin from the upper trunk of the brachial plexus or, less frequently, from the root of C5, and its termination in the infraspinatus muscle. Between these two fixed points there are two critical points that represent potential sites of injury to the nerve, the suprascapular notch and the lateral blunt edge of the spine of the scapula. Tengan et al.⁹ consider that there are three critical points of injury to the suprascapular nerve: the suprascapular notch, the lateral edge of the spine of the scapula, and the spinoglenoid notch.

COMMENTS

Patients reported were three athletic males between 17 and 19 years old. They were all volleyball players, playing as smashers. Two of them were the best smashers of their team, according to the coach. In a scale from 0 to 5 to the muscle bulk, one player had a 3 atrophy

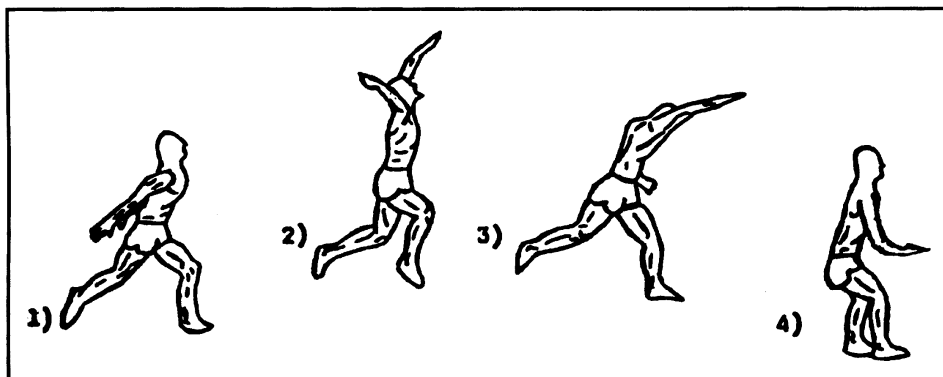


Fig 2. Four stages of the volleyball smash: 1) wind-up; 2) cocking; 3) acceleration; 4) follow-through.

There was a left handed volleyball player we examined that had an isolated and painful atrophy of the left infraspinatus muscle, and he was the athlete who had the biggest atrophy among our cases (he is one of the authors) (Fig 3). This case, for sure, excludes the “manchete” hypothesis.

The pathogenesis should, then, be attributed to two asymmetrical and powerful movements that are typical to the game: the service and the smash. Biomechanically, the smash and the service are similar to the act of pitching in baseball⁹. They are divided into four stages: wind-up or preparation (some authors consider the wind-up as belonging to the cocking phase⁸), cocking, acceleration, and follow-through⁵ (Fig 2).

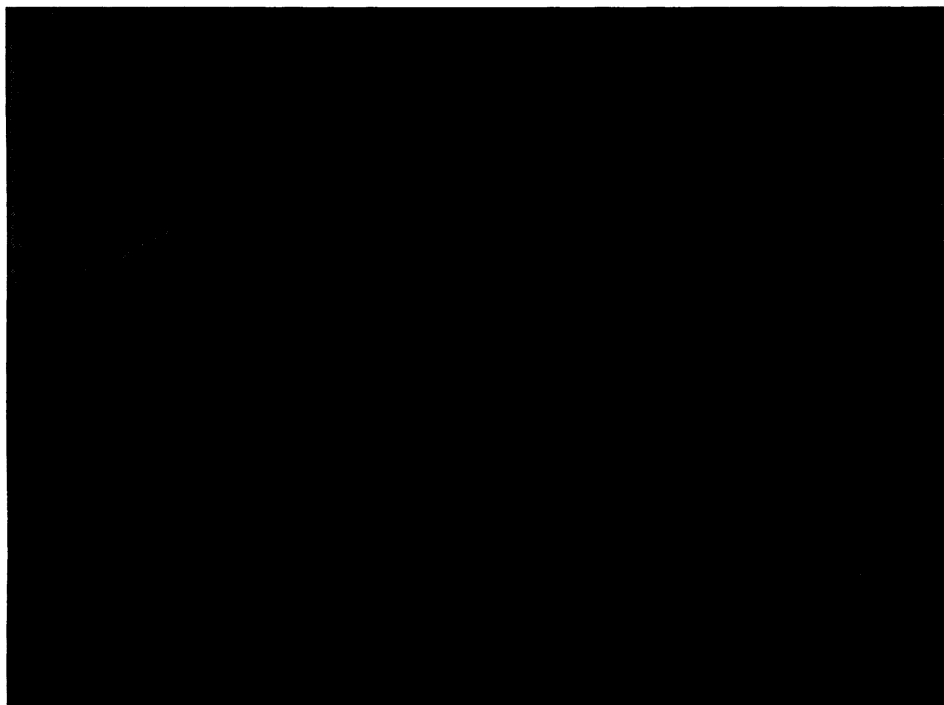


Fig 3. Case 2. Isolated infraspinatus muscle atrophy of the left shoulder. Posterior view.

- (1) Wind-up or preparation phase: preliminary activity; a period dominated by upper extremity flexion with both arms hyperextended. It ends when a jumping action is fulfilled. The wind-up places the body to a position so that all segments of the body may contribute to the jumping action.
- (2) Cocking phase: a period of dominant shoulder abduction, external rotation and hyperextension, and dominant elbow flexion. The nondominant arm acquires a flexion position, like aiming the ball. It begins when the athlete jumps and ends when maximum rotation of the shoulder is attained.
- (3) Acceleration phase: starts with the dominant shoulder abduction, external rotation and hyperextension. The dominant shoulder will be, now, flexed, adducted and internally rotated, and the dominant elbow will be extended. This phase ends, in the service, when the athlete hits the ball. In the smash, the acceleration phase ends a few seconds after hitting the ball.
- (4) Follow-through phase: it is a deceleration phase. In the service, after ball-hit, a braking action is provided by the posterior muscles of the shoulder. In the smash, a few seconds after ball-hit, the arm is cross-body adducted and the posterior muscles of the shoulder must provide a braking action.

Because of an eccentric position acquired by the dominant arm, characterized by maximum external rotation, 20° of hyperextension and 90° of abduction, during the cocking phase of the smash, as in the service, mostly caused by external rotation, the terminal branches of the nerve to the belly of the infraspinatus muscle are shifted medially by the contraction of the muscle. Probably, it causes tension in the nerve itself at the lateral edge of the spine of the scapula³.

According to Ferretti et al.³, the final phase of the service offers maximum stress in the trunk of the nerve. They consider that the movement of the scapula - fulfilling abduction itself, allowing maximum up-ward projection of the arm, associated to the eccentric contraction of the infraspinatus muscle that is required to put a brake on the movement of the scapula - increases the distance between the upper trunk of the brachial plexus and the infraspinatus muscle, its origin and termination, respectively. It will, then, stretch the nerve across the lateral edge of the spine of the scapula.

This kind of injury has been found in baseball athletes, a sport in which athletes throw or serve more often and more violently than do volleyball players². This led us to the belief that the nature of the smash, where the athlete uses the arm violently, more than in the volleyball service, is the key to the pathogenesis of the lesion.

During the final phase of the smash, the dominant arm is put into a cross-body adduction. It is associated, as in the service, with the braking action that is provided by posterior shoulder musculature, mostly and roughly, the infraspinatus muscle. This action increases the distance between the points of origin and termination of the nerve and may cause lesion of the nerve in its terminal branches. Also, in our cases, the atrophy was predominantly present in more powerful smashers and in "top balls" smashing volleyball players. This last, probably, because of the necessity to smash balls that are lifted higher than in "time balls" smashers, which also increases the distance between the upper trunk of the brachial plexus and the infraspinatus muscle.

It is hard to tell which mechanism is responsible for this kind of sports nerve injury but we assume that the smash with their powerful movements, is one of the keys to the pathogenesis of the lesion.

The electrophysiological examination did not confirm the atrophy in two of our cases. Although, in one case, the left handed volleyball player, the conduction velocity of the suprascapular nerve to the infraspinatus muscle was altered, the latency being higher than to the supraspinatus muscle, which was normal. It confirmed the compression syndrome of the suprascapular nerve at the spinoglenoid notch.

Our patients continued to play normally without any change in their performances and they were not submitted to exploratory surgery.

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