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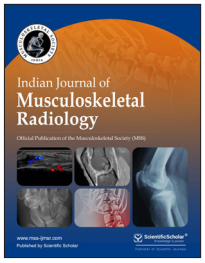

*Case Report*

# Isolated infraspinatus atrophy in elite volleyball player- Understanding the biomechanics, imaging and outcome

Nafisa Shakir Batta[1], Mukul Mittal[2], Vikas Batra[1], Vineet Jain Jain[2]

Departments of [1]Radiology, Mahajan Imajing, [2]Orthopedics, Safdarjung Hospital, Sports Injury Centre, New Delhi, India.

***Corresponding author:**
Nafisa Shakir Batta,
Department of Radiology,
Mahajan Imajing, Safdarjung Hospital, Sports Injury Centre,
New Delhi, India.

nafisa.shakir@gmail.com

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## ABSTRACT

Over 800 million people worldwide play volleyball, either recreational or competitive. Even though all throwing athletes suffer from upper extremity injuries caused by overuse, volleyball additionally involves certain prototypical throwing biomechanics, not seen in other overhead sports. Complex biomechanics and the balanced interplay between the glenohumeral and scapulothoracic muscles ensure velocity and torque displacement in spike and jump-serve maneuvers (formerly known as smash) which are exclusive to volleyball. In this case report, we have described in detail the entity of isolated infraspinatus atrophy secondary to suprascapular nerve neuropathy.

**Keywords:** Infraspinatus atrophy, Suprascapular neuropathy, Volleyball injuries, Spiking injury

## INTRODUCTION

The throwing biomechanics seen in volleyball differs from other overhead sports such as cricket, baseball, basketball, and cricket. Suprascapular neuropathy (SSN) is a rare cause of shoulder dysfunction seen in volleyball players causing isolated infraspinatus atrophy (IIA).[1] Because of overlapping symptoms with other more common shoulder pathologies such as rotator cuff tendinopathy and labral tears, the incidence of reporting remains low.[2] A high degree of suspicion is therefore essential for early diagnosis, treatment, and prevention of this injury.

## CASE REPORT

A 24-year-old right-hand dominant, national-level male volleyball player presented with complaints of right shoulder pain of 6 months duration, gradual and progressive, which was diffuse and predominantly posterior, worsened with play especially during contact with the ball in abducted and externally rotated position.

On inspection, there was hollowing indicating wasting of infraspinatus muscle [Figure 1]. Range of motion (ROM) testing, done using digital goniometry suggested increased external rotation (ER) with ROM (114 degrees) and slightly decreased internal rotation (78 degrees), consistent with changes observed in elite throwing athletes. Manual muscle testing was graded on a 5-point scale. Weakness of infraspinatus muscle was noted on the right side (4/5). Rest of the rotator cuff

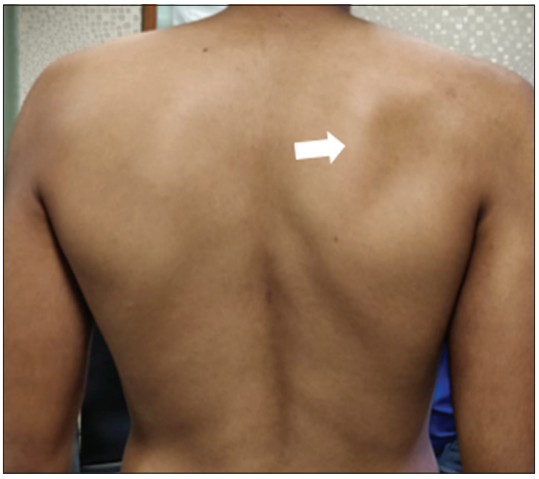

**Figure 1:** Posterior shoulder girdle view with visible atrophy in the infraspinatus fossa of the right shoulder (white arrow).

muscles revealed pain-free, full strength. There was slight pain on passively abducting the shoulder in an externally rotated position. Mild discomfort was noted on cross-arm adduction. Tests for external impingement described by Hawking and Kennedy, Neer, and Welsh were negative. There was no evidence of scapular malposition or dyskinesia. O'Brien test for a superior labral tear and Spurling's test to assess cervical radiculopathy were negative.

The patient underwent radiographic examination of the shoulder and spine which revealed no abnormality. Magnetic resonance imaging shoulder was performed on a 1.5 Tesla scanner and revealed diffuse denervation edema within the infraspinatus muscle belly, which showed loss of volume (more than 50%). No space-occupying lesion was seen in the region of the spinoglenoid notch [Figure 2a-c]. The rest of the rotator cuff was normal concluding IIA. No labral or bony pathology was observed.

EMG/NCV demonstrated isolated denervation in the IS consisting of fibrillation potentials, positive sharp waves, and single unit recruitment of normal motor unit potentials (MUP). MUPs did not suggest any myopathy compound muscle action potentials.

The patient was put on a program of posterior capsular stretching with focus on strengthening external rotators in both adducted and abducted shoulder positions. Axioscapular, scapulohumeral, and axiohumeral muscles were strengthened in a progressive manner. The training regimen was modified and the player was restricted from spiking and blocking during play. At 6 months follow-up, patient's symptoms had improved, and he was involved in unrestricted play at national level.

## DISCUSSION

In volleyball players complex and balanced interplay between the glenohumeral (deltoid, supraspinatus, infraspinatus, teres

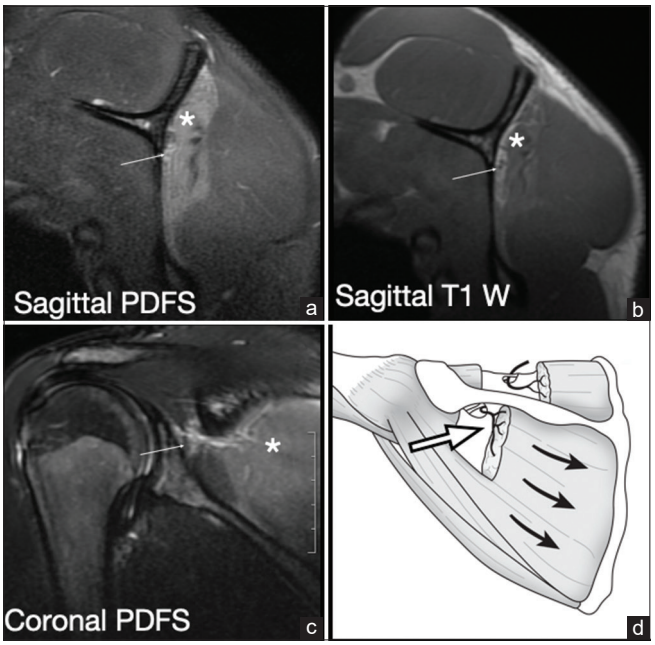


**Figure 2:** (a) Oblique sagittal T1 weighted images at the level of the mid-belly of the infraspinatus, shows moderate volume loss (*) and fatty atrophy. The distal branches of the suprascapular nerve and vessels are well delineated at the undersurface of the infraspinatus, superficial to the scapular cortex (white arrow in Figure 2a-d), (b) Oblique sagittal proton density fat-saturated (PDFS) images at the level of the mid-belly of infraspinatus, shows isolated moderate volume loss (*) and diffuse edema consistent with denervation pattern. Normal muscle bulk and signal intensity of the supraspinatus and teres minor are seen. (c) Oblique coronal PDFS images at the spinoglenoid notch clearly depict the isolated infraspinatus atrophy and diffuse denervation edema through its muscle fibers. (d) Illustration depicting suprascapular nerve traction injury due to active contraction of infraspinatus (Original illustration by Mr. Madhu Mangal Singh).

minor, and subscapularis) and scapulothoracic (trapezius, rhomboids, serratus anterior, pectoralis minor, and levator scapulae) muscles ensure velocity and torque displacement in spike and jump-serve maneuvers (formerly known as smash) which are exclusive to volleyball.[1,3] Even mild functional impairments of muscle activation, in the setting of repetitive trauma, can significantly hamper the scapulohumeral rhythm causing SICK scapula syndrome (scapular malposition, inferior medial border prominence, coracoid pain, and malposition and dyskinesia of scapular movement).[3,4]

There are two potential sites of supraspinatus nerve compression; the suprascapular notch and the spinoglenoid notch.[2] The suprascapular notch compression causes atrophy of both supraspinatus and infraspinatus muscles causing weakness in both abduction and ER.[2,5] Compression at spinoglenoid notch as in the case of volleyball players causes isolated atrophy of infraspinatus and weakness of only ER.[4,6]

Etiology of SSN is still unknown, however, a few theories have been postulated. Anatomical variants of spinoglenoid ligaments, anomalous course of the nerve, ganglion cysts arising from labral tears could be predisposing factors and should be screened using imaging.[7,8]

The "float" serve aims to impart a floating trajectory to the ball making it difficult to receive. During this serve, the server must contact the ball sharply at the center, immediately arrest follow up, and retract the arm. This requires first eccentric and then concentric activation of posterior shoulder muscles especially the infraspinatus and teres minor [Figure 2d]. This repetitive mechanism leads to impingement of terminal branches of supraspinatus nerve at spinoglenoid notch.[8]

ER of the shoulder with the elbow in 90° during a jump, when the player is on his feet, uses the force of the entire body to rotate and sweep the ball downwards from the hitting arm into an explosive dropdown.[9]

During this spiking movement, fixity of the SSN at the spinoglenoid notch, predisposes it to be held against the violently contracting muscle belly of the infraspinatus, these actions are exhaustively repeated during training and competition causing compression neuropathy which eventually causes denervation edema and atrophy within the infraspinatus muscle, worsening the scapulothoracic imbalance.[3,9]

Ferretti *et al*. examined 96 elite volleyball players and concluded that repetitive stress due to stretching of the SSN during cocking of the arm and follow-through at serve as the probable causative factor for IIA.[7]

A study of 16 Belgian male professional volleyball players correlating electromyographic investigation, a clinical shoulder examination, and isokinetic concentric peak torque shoulder internal/ER strength testing, suggesting an association between an increased ROM of the shoulder joint and the presence of isolated paralysis of the infraspinatus muscle.[9,10]

Contemori and Biscarini. highlighted in their study, altered shoulder muscle activity levels, scapulothoracic muscles imbalances, and abnormal scapulothoracic recruitment patterns in the hitting shoulder of professional volleyball players with IIA, secondary to SSN neuropathy.[6]

## CONCLUSION

Shoulder abduction in the scapular plane coupled with a high degree of ER in elevation, during volleyball serve and spike can cause SSN and IIA. The correct diagnosis followed by shoulder strengthening, pre-play stretching, and engaging the shoulder girdle in the optimal scapulohumeral rhythm for correct spiking technique, combined, all can result in a return to play at the pre-injury level.

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**Declaration of patient consent**

Patient's consent not required as patients identity is not disclosed or compromised.

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**Conflicts of interest**

There are no conflicts of interest.

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