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Severe Multidirectional Instability of the Gleno-Humeral Joint

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MULTIDIRECTIONAL instability (MDI) of the gleno-humeral (GH) joint is defined as symptomatic instability in two or more directions. This condition is primarily diagnosed on the basis of clinical findings, which requires a thorough patient history and physical examination.[1-3] MDI typically presents one of three distinct patterns: (a) antero-inferior dislocation with posterior subluxation,(b) postero-inferior dislocation with anterior subluxation, and (c) antero-postero-inferior dislocation.[1-4] Patients with MDI often present GH laxity, symptomatic multi-planar translation, and impingement syndrome.[3,5] The chief complaint is often generalized shoulder pain that is exacerbated by overhead activities and specific arm positions. The GH joint is generally asymptomatic at rest in the anatomical position.[1-3] Clinical findings may include (a) a positive sulcus sign (at least 2mm), (b) positive anterior and posterior apprehension tests, (c) a positive Neer or Hawkins-Kennedy test, and (d) joint glide hypermobility.[3,6,7]

MDI lacks a consistent definition in the literature, which complicates interpretation of epidemiologic studies.[1-4,8,9] Owens et al.[9] reported that females had a greater incidence of MDI events than males had. This difference is likely due to differences in joint laxity between men and women.[10]

Initial management of MDI consists of activity modification and rehabilitation that is focused on strengthening the rotator cuff (RTC) and scapular stabilizers and improvement of GH joint proprioception.[1,3,7,11,12] As many as 89% of patients with MDI may respond favorably to rehabilitation and thereby avoid a surgical intervention.[1,13]

This report reviews the clinical presentation of a patient who had prolonged neurological and functional impairments associated with undiagnosed MDI. The cause of dysfunction was confirmed to be GH laxity, rather than psychosomatic or neurological factors. The patient was diagnosed with severe MDI in all three planes. An open inferior capsular shift was performed to address capsular redundancy.[1,7,11] This surgical procedure has been reported to have an 86% success rate at 38 months postsurgery.[1] The patient was able to return to asymptomatic activities of daily living (ADLs).

Background

A 22 year-old female college athlete, who had left hand dominance, presented symptoms in the right upper extremity. She reported an extensive history of overhead sports activities that included softball, basketball, swimming, and diving. She was found to have congenital GH laxity without any other abnormal clinical examination findings.

The patient experienced progressively greater symptoms of decreased function, pain, spontaneous subluxation, crepitus, and parasthesia that was distributed throughout the brachial plexus dermatomes (C5-T1).[14-16] The terms "numbing, burning, aching, and shooting" were used to describe the nature of pain experienced when sleeping or performing overhead activities. She reported pain during active shoulder range of motion (AROM) and persistent parasthesia that extended to the palmar surface of her right hand. Over a period of two and one-half years, the patient's symptoms of pain, weakness, and disability became progressively more disabling.

Clinical Management

The patient experienced an insidious onset of decreased function (an inability to perform overhead activities or upper extremity weightlifting exercises) and increasing neurological impairment. Her history included a fall on the right upper extremity, with the GH joint in a position of flexion, abduction, and external rotation. Seven months after the traumatic incident, the patient sought care at an emergency room for parasthesia that radiated from the cervical spine to the distal digits of her right upper extremity. She was diagnosed as having an ulnar neuropathy.

Subsequently, the patient was evaluated by an orthopedic surgeon, who diagnosed a rhomboid strain. Scapular winging, crepitus, pain, and audible "popping" within the GH joint were noted. She was referred for six months of conservative rehabilitation, which was primarily focused on GH stabilization, postural correction, and RTC strengthening. The patient did not experience any relief from her neurologic symptoms, instability, or pain.

Her rehabilitation program was modified to incorporate mobilizations of the cervical spine, thoracic spine, and first rib, which provided minimal relief of neurological symptoms and a slight improvement in functional abilities. The patient was then referred to a neurologist, who included thoracic outlet syndrome and multiple sclerosis in the differential diagnosis. The existence of a neurologic disorder was ruled out by physical examination findings, nerve conduction velocity testing, electromyography, blood tests for autoimmune disorders, and an MRI of the brain.

Functional impairments continued to progress, which ultimately led to the inability to perform ADLs and increasing discouragement. Increased radicular symptoms throughout the distribution of the brachial plexus, audible popping and clicking within the GH joint, and pain restricted the use of the right upper extremity. MRI of the GH joint demonstrated mild RTC inflammation and no evidence of a labrum lesion.

The patient was ultimately evaluated by an orthopedic surgeon with shoulder subspecialty expertise, who diagnosed MDI. The GH capsular laxity allowed anterior, inferior, and posterior subluxation without anesthesia. The neurological symptoms were exacerbated by inferior traction, which produced a positive inferior sulcus sign. The physician believed that the neurologic symptoms were caused by the head of the humerus resting on the anterior margin of the glenoid rim, which was responsible for brachial plexus impingement. He recommended an open inferior capsular shift as the best option to address the GH laxity and restore function.

Presurgical Management

The patient elected to delay the surgical intervention for personal reasons. During this period, the patient was instructed to refrain from performance of activities that exacerbated symptoms and to begin a scapular strengthening program. Joint mobilizations were performed in an effort to relocate the position of the head of the humerus, which produced a transient decrease in pain and parasthesia. Her status continued to deteriorate, with development of palmar diaphoresis, continuous numbness and pain, crepitus in the GH joint, and recurring spontaneous subluxations. Two cortisone injections in the posterior GH capsule were administered six weeks apart, which produced a temporary decrease in symptoms and an increase in functional capabilities. Following the injections, the patient was able to move her upper extremity throughout the normal ROM, but audible and palpable clicking and popping were still present. Subsequently, the surgical intervention was performed.

Objective Measures of Status

All ROM measurements were acquired by the same clinician for both an asymptomatic range and the complete symptomatic range. AROM was recorded prior to passive ROM (PROM) with the patient in a supine position and the scapula stabilized to ensure isolation of GH joint motion. End-range PROM was only achieved in forward flexion; the patient could not tolerate extreme GH joint displacement in other planes. The change in values for asymptomatic and symptomatic ROM was substantial (Tables 1 and 2; Figures 1 and 2).

Table 1. ROM Measurements (degrees)

Motion	PROM		AROM	
Pre-Surgery	**Asymptomatic**	**Full ROM**	**Asymptomatic**	**Full ROM**
Forward Flexion (supine)	120	196	90	185
Extension (prone)	42	71	41	58
External Rotation (supine @ 90/90)	73	110	19	79
Internal Rotation (supine @ 90/90)	46	71	35	54
6 Months Post-Surgery				
Forward Flexion (supine)	174	182	162	174
Extension (prone)	51	59	34	43
External Rotation (supine @ 90/90)	79	86	72	77
Internal Rotation (supine @ 90/90)	65	70	56	***
9 Months Post-Surgery				
Forward Flexion (supine)	187*	***	177*	***
Extension (prone)	62	***	54	***
External Rotation (supine @ 90/90)	71	89**	70	79
Internal Rotation (supine @ 90/90)	62	***	54	***

*Stretch anterior, mild parasthesia, discomfort returning

**Stretch at end range, weak returning, general feeling of stability

***No symptoms present

Table 2. Pre-Post Change in Asymptomatic ROM

Motion	PROM (%)	AROM (%)
Forward flexion (supine)	55.8	96.7
Extension (prone)	47.6	31.7
External rotation (supine @ 90/90)	*	268.4
Internal rotation (supine @ 90/90)	34.8	54.3

< 30% change

Treatment Outcome

The patient reported decreased pain and parasthesia immediately following the surgical procedure. As she progressed through the postsurgical rehabilitation program, she reported increased stability and function.

The patient's positive postsurgical outcome confirmed that MDI was the primary cause of dysfunction. The patient's cervical symptoms might have been attributable to upper trapezius spasms and kyphotic posture. Excessive movement of the humeral head was probably responsible for RTC inflammation. After the surgery, the patient developed bicipital tendonitis and RTC impingement, which may have been due to inadequate strength of the scapular stabilizers. As the rehabilitation program progressed, the patient's symptoms resolved and her functional abilities continued to improve.

At a 9-month postsurgery follow-up examination, the patient reported a complete resolution of neurological symptoms and demonstrated normal asymptomatic ROM. The surgery and rehabilitation led to a 96.7% increase in asymptomatic forward flexion AROM and a 268.4% increase in asymptomatic external rotation AROM.

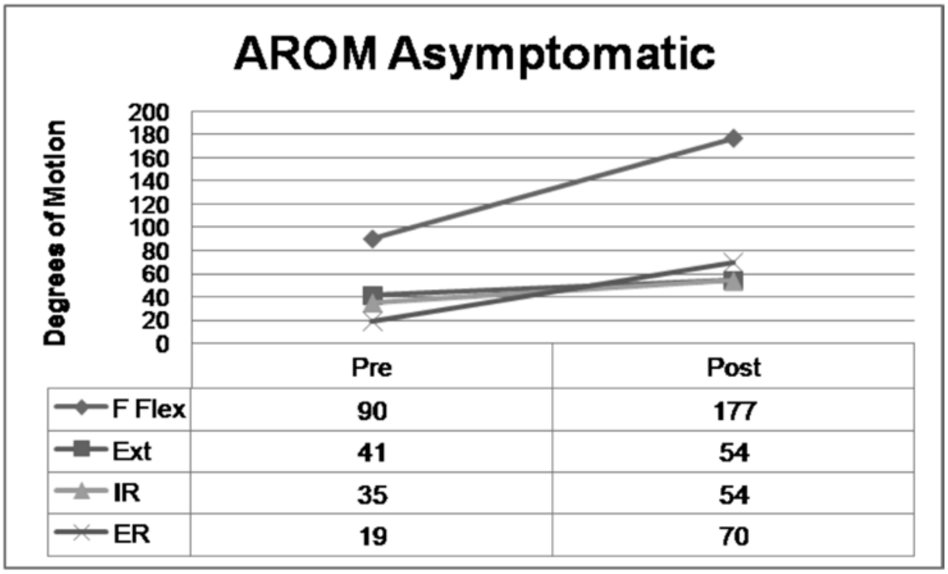

Figure 1 AROM asymptomatic.

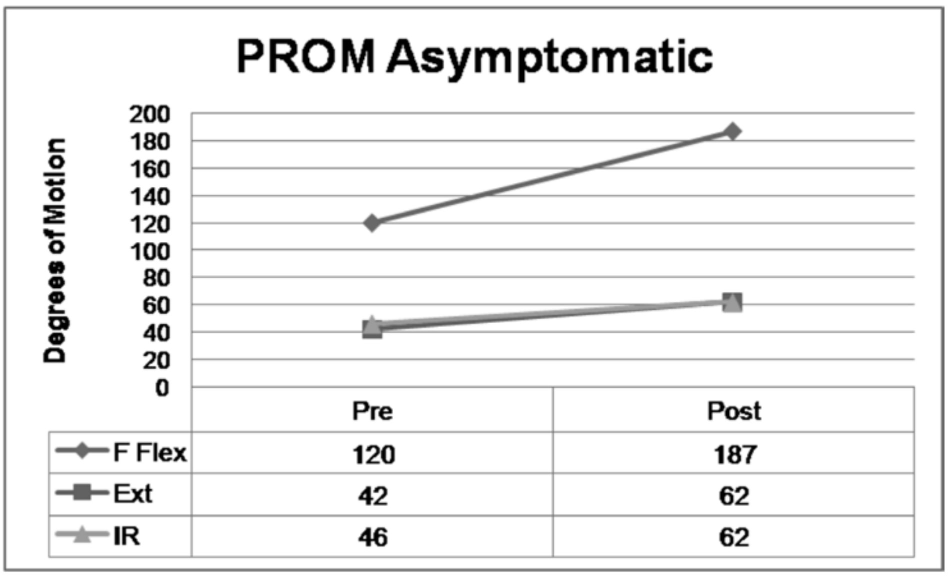

Figure 2 PROM asymptomatic.

Uniqueness of the Case

The patient's clinical care was initially based on symptoms, rather than identification of the cause of dysfunction.[2,4] The lack of an accurate diagnosis led to a prolonged course of ineffective treatment. The severe loss of function and neurologic symptoms represent an unusual presentation for MDI. The anterior, posterior, and inferior subluxations were easily reduced, which was evidence of severe laxity of the GH joint. The patient experienced an immediate decrease in symptoms following the surgical intervention and she was able to perform normal ADLs within 4 months. She was allowed to resume performance of overhead activities at 6 months postsurgery.

Summary

This case emphasizes the importance of careful consideration of the patient's description of symptoms and concerns, and the performance of a thorough physical examination. The postsurgical resolution of symptoms and restoration of function confirmed that the cause of dysfunction was GH instability.

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