Pathophysiology and Natural history of the Stiff Shoulder

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I. The stages of adhesive capsulitis of the shoulder

Stage 1: 0-3 months

Pain with active and passive of motion (Limitation of FF, ABD, IR, ER)

Exam under anesthesia: Normal or minimal loss of ROM

Arthroscopy: Diffuse GH synovitis, most pronounced in the anterior superior capsule

Pathology: Hypertrophic, hypervascular synovitis, rare inflammatory cell infiltrates, normal underlying capsule

Stage 2: **"freezing stage"** 3-9 months

Chronic pain with active and passive ROM (Significant limitation of FF, ABD, IR, ER)

Exam under anesthesia: ROM essentially identical to awake ROM

Arthroscopy: Diffuse, pedunculated synovitis (christmas tree synovitis), tight capsule with rubbery or dense feel on insertion of arthroscope

Pathology: Hypertrophic, hypervascular synovitis with perivascular and subsynovial scar, significant fibroplasia and scar formation in the underlying capsule

Stage 3: **"frozen stage"** 9-15 months

Minimal pain except at end ROM (Significant limitation of ROM with rigid "end feel")

Exam under anesthesia: ROM identical to awake ROM

Arthroscopy: No hypervascularity seen, remnants of fibrotic synovium can be seen. The capsule feels extremely dense and thick on insertion of the arthroscope and there is a diminished capsular volume.

Pathology: "Burned out" synovitis without significant hypertrophy or hypervascularity. Underlying capsule demonstrates dense scar formation

Stage 4: **"thawing phase"** 15-24 months

Minimal pain, Progressive improvement in range of motion

EUA/arthroscopy/pathology: not generally available

II. Pathologic Mechanism

Autoimmune factor [1,4,5,6,12]

- High incidence of HLA B27[5], not confirmed this association.[13,20,25]
- Lower serum IgA levels & increased immune complex, CRP levels [1,4,6]
- In general, sufficient evidence to support immunologic therapy has been lacking.

A relation to myofascial pain syndrome

- Travell :The subscapularis trigger points → influence on the sympathetic vasomotor activity → hypoxia of the periarticular tissues → a local proliferation of fibrous tissue about the shoulder capsule.[29]
- Palpable bands of muscle fibers → a local twitch response [27,31]

A biochemical factor

- Lundberg: Increase in glycosaminoglycans and a decrease in glycoprotein content.
  → consistent with the process of fibrosis,
  → may represent the effect of frozen shoulder, rather than its cause.[17,18]
- Rodeo: The specific cytokines in the initiation and evolution of the fibrotic process in adhesive capsulitis of the shoulder.

Neurologic dysfunction

- Suprascapular compression neuropathy : but electromyography (EMG) and nerve conduction studies have not supported this theory.[14]
- Autonomic dysfunction (a form a reflex sympathetic dystrophy)[24]

Various endocrine disorders

- **Hyperlipidemia**: increased serum triglyceride and cholesterol levels **(Bunker)** [7].
- Diabetes mellitus[3,15], Thyroid disorders[2,10], hypoadrenalism[30],corticotropin deficiency[8]

Trivial trauma

- An important factor, particularly when it is followed by a prolonged period of immobilization(major & minor trauma). [22]
- "Constitutional" predisposition: bilateral frozen shoulders.[5,18,21,23,32]

Psychologic factors

- **Coventry**:periarthritic personality.[9,10,16,24]
- No evidence for a characteristic personality disorder.[21,32]

Fibromatosis

- Response to cytokines, lymphocyte, or monocyte products. Platelet-derived growth factor is a potent mytogenic polypeptide for mesenchymal cells.
- **Bunker and Anthony** report that the pathologic process is active fibroblastic proliferation accompanied by some transformation to a smooth-muscle phenotype (myofibroblasts). -- very similar to those seen in Dupuytren's disease of the hand, with no inflammation and no synovial involvement. The contracture acts as a checkrein against external rotation, causing a loss of both active and passive movement.[7]

Secondary frozen shoulder includes intrinsic, extrinsic, or systemic disorders.

- *Intrinsic* shoulder abnormalities: rotator cuff tendinitis, rotator cuff tears, tendinitis of

the long head of the biceps tendon, calcific tendinitis, and acromioclavicular arthritis.

- *Extrinsic* disorders: ischemic heart disease and myocardial infarction, pulmonary disorders including tuberculosis, chronic bronchitis, emphysema, and tumor, cervical disc disease and radiculopathy, cerebral vascular hemorrhage, previous coronary artery bypass graft surgery, previous breast surgery, lesions of the middle humerus,123 and central nervous system disorders, such as Parkinson's disease.
- *Systemic* disorders: diabetes mellitus, hypothyroidism, hyperthyroidism, and hypoadrenalism.

III. Natural History

- Epidemiologically, the exact prevalence and incidence of frozen shoulder are not known: minimum of 2%.[18]
- The fourth and sixth decades of life, and it is more common in women than men.
- The nondominant extremity appears to be more commonly involved.[18]Bilateral involvement occurs in 6% to 50% of cases, although only 14% of these bilateral cases manifest simultaneously.[1,6,18,24] When a history of bilateral involvement is identified, the possibility of a constitutional predisposition should be explored.[18,24]
- There is significant controversy over the natural history of frozen shoulder relative to both objective and subjective outcomes. Several investigators using a variety of treatment methods have reported that a high percentage of affected patients achieve full range of motion.[11] In addition, they have found complete or near complete symptomatic relief.[11,12] More recent investigations have questioned the early optimistic reports, finding measurable restriction at follow-up in 39% to 76% of patients[23] and persistent symptoms in up to 45%.[1,22]
- The time course of adhesive capsulitis has been described as classically lasting 18 to 24 months.[1] Recent studies have challenged this commonly held belief.

  Reeves: 1) the mean duration of symptoms was 30 months.
  2) some restriction in shoulder motion in more than 50% of patients in a 5- to 10-year follow-up, but functional impairment was identified in only 7%.[23]

  Clark: 42% of patients had persisting limitations of motion after 6 years of F/U.

  Binder: 90% of patients did not regain the minimum range of motion.[1]

  Schaffer: Almost half remained symptomatic many years after the onset of symptoms, and up to 56% had residual restriction in one or more planes.[27]

  Regardless of objective restriction or the presence of symptoms, few patients are reportedly functionally restricted to any significant degree.[1,23] Symptomatic patients frequently have no measurable restricted range of motion in any plane. Conversely,

those patients with the most significant motion restriction were often pain-free. Whether this is due to adaptation to such restriction, or restriction in motion is unimportant for daily living activities is an unresolved issue. In Schaffer's report, the author states that the preeminent importance of forward flexion and elevation in daily activities superseded the findings of restriction predominantly in the abducted and externally rotated positions, which resulted in little functional impairment.[27]

In general, the natural history of frozen shoulder is uncertain, and additional randomized, prospective studies are needed. Difficulty exists in performing these studies owing to the ethical dilemma of assigning patients to an untreated group.

REFERENCES

1. Binder A, Bulgen DY, Hazleman BL. Frozen shoulder: a long-term prospective study. Ann Rheum Dis 1984; 43: 361.
2. Bowman CA, Jeffcoate WJ, Pattrick M, et al. Bilateral adhesive capsulitis, oligoarthritis and proximal myopathy as presentation of hypothyroidism. Br J Rheum 1988; 27: 62.
3. Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. Ann Rheum Dis 1972; 31: 69.
4. Bulgen DY, Binder AI, Hazleman BL, et al. Immunological studies in frozen shoulder. J Rheumatol 1982; 9: 893.
5. Bulgen DY, Hazleman BL, Voak DL. HLA-B27 and frozen shoulder. Lancet 1976; 1: 1042.
6. Bulgen DY, Hazleman BL, Ward M, et al. Immunological studies in frozen shoulder. Ann Rheum Dis 1978; 37: 135.
7. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. J Bone Joint Surg [Br] 1995; 77: 677-83.
8. Choy EH, Corhill M, Gibson T, et al. Isolated ACTH deficiency presenting with bilateral frozen shoulder. Br J Rheum 1991; 30: 226.
9. Coventry MB. Problem of painful shoulder. JAMA 1953; 151: 177.
10. Fleming A, Dodman S, Beer TC, et al. Personal in frozen shoulder. Ann Rheum Dis 1975; 35: 456.
12. Grey RG. The natural history of idiopathic frozen shoulder. J Bone Surg 1978; 60: 564.
13. Hazleman BL. The painful stiff shoulder. Rheumatol Phys Med 1972; 11: 413.
14. Kessel L, Bayley I, Young A. The upper limb: the frozen shoulder. Br J Hosp Med 1981; 25: 334.

15. Kopell AP, Thompson WAL. Pain and the frozen shoulder. Surg Gynecol Obstet 1959; 109: 279.
16. Lequesene M, Dang N, Bensasson M, et al. Increased association of diabetes mellitus and capsulitis of the shoulder and shoulder-hand syndrome. Scand J Rheumatol 1977; 6: 53.
17. Lorenz TH, Musser MJ. Life, stress, emotions, and the painful stiff shoulder. Ann Arthritis Med 1952; 37: 1232.
18. Lundberg BJ. Glycosaminoglycans of the normal and frozen shoulder joint capsule. Clin Orthop 1970; 69: 279.
19. Lundberg BJ. The frozen shoulder: clinical and radiographical observations. The effect of manipulation under general anesthesia: structure and glycosaminoglycan content of the joint capsule. Acta Orthop Scand [Suppl] 1969; 119: 1-9.
20. Neer CS II. Frozen shoulder. In: Neer CS, ed. Shoulder reconstruction. Philadelphia: WB Saunders, 1990: 422.
21. Noy S, Dekel S, Orgad S, et al. HLA-B27 and frozen shoulder. Tissue Antigens 1981; 17: 251.
22. Oesterreicher W, Van Dam G. Social and psychological researches into brachialgia and periarthritis. Arthritis Rheum 1964; 7: 670.
23. Quigley TB. Checkrein shoulder, a type of frozen shoulder: diagnosis and treatment by manipulation under and ACTH or cortisone. Clin Orthop 1982; 164; 4.
24. Reeves B. The natural history of the frozen shoulder syndrome. J Rheumatol 1975; 4: 193.
25. Rizk TE, Pinals RD. Frozen shoulder. Semin Arthritis Rheum 1982; 11: 440.
26. Rizk TE, Pinals RS. Histocompatibility type and racial incidence in frozen shoulder. Arch Phys Med Rehabil 1984; 65: 33.
27. Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder: a long-term follow-up. J Bone Joint Surg [Am] 1993; 74: 738.
28. Simmons DB. Myofascial pain syndromes: where are we? where are we going? Arch Phys Med Rehabil 1988; 69: 209-12.
29. Travell JG, Simmons DG. Myofascial pain and dysfunction: trigger point manual. Baltimore: Williams & Wilkins, 1983: 410-24.
30. Wohlgethan JR. Frozen shoulder in hyperthyroidism. Arthritis Rheum 1987; 30: 936.
31. Wolfe F. Fibrositis, fibromyalgia and musculoskeletal disease: the current status of fibrositis syndrome. Arch Phys Med Rehabil 1988; 69: 527-31.
32. Wright V, Haq AM. Periarthritis of the shoulder: I. Aetiological considerations with particular reference to personality factors. Ann Rheum Dis 1976; 35: 213.