Published in final edited form as:

J Clin Rheumatol. 2010 September ; 16(6): 300–301. doi:10.1097/RHU.0b013e3181ef6ee1.

Diffuse Palpable Tendon Friction Rubs in a Patient with Seronegative Erosive Polyarthritis

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Key Indexing Terms

Tendon Friction Rub; Polyarthritis; Systemic Sclerosis

INTRODUCTION

Palpable tendon friction rubs were first reported by Westphal in 1876, who described “coarse cracking and crepitus” in a 23-year-old woman with diffuse cutaneous systemic sclerosis (SSc). [1] The pathology of tendon friction rubs in SSc was subsequently attributed to non-inflammatory fibrinous deposits on the surface of tendon sheaths and the overlying fascia.[2] Rodnan and Medsger used sound tracings to identify tendons as the source of the “leathery crepitus”. [3] In addition to SSc, tendon rubs have been described in eosinophilic fasciitis and traumatic tendinopathies.[4] However, the occurrence of diffuse tendon rubs is thought to be limited to SSc. The presence of friction rubs in SSc is associated with diffuse cutaneous disease, increased disease severity and functional disability [5], and poor survival.[4] We now describe a patient with diffuse tendon friction rubs coexistent with symmetric polyarticular erosive arthritis. Tendon friction rubs, although highly characteristic of SSc, are not unique to this disease.

CASE REPORT

A 62 year-old man with a history of coronary artery disease and stroke, presented with painful symmetrical swelling of his wrists, shoulders, knees, ankles, metacarpophalangeal (MCP), and proximal interphalangeal (PIP) joints. He also noted fatigue, morning stiffness lasting 60 minutes, a 30 pound weight loss, and Raynaud phenomenon not associated with ischemic complications. He denied skin tightness, dyspepsia, weakness, or shortness of breath. Physical examination revealed striking audible and palpable tendon friction rubs at his finger flexors and extensors, wrists, elbows, knees, and ankles. Prominent synovitis was evident over his MCPs, PIPs, wrists, and knees bilaterally. He had no scleroderma, sclerodactyly, nailfold capillary changes, telangiectasias, finger tapering, cuticular alterations, calcinosis, ischemic ulcers, digital pitting, or pterygium inversum unguis.

Laboratory evaluation revealed anemia and mildly elevated acute phase reactants (Table 1). Rheumatoid factor, anti-nuclear, anti-centromere, anti-topoisomerase, and anti-cyclic citrullinated peptide autoantibodies were negative (Table 2). A high resolution computed

tomography scan of the chest showed clear lungs, and pulmonary function testing revealed normal lung mechanics and volumes. Magnetic resonance imaging of the hands, wrists, and knees demonstrated erosive arthropathy with extensive synovitis and tenosynovitis (Figure 1). Treatment with methotrexate and tapering doses of corticosteroids resulted in an incomplete clinical response. Addition of adalimumab resulted in significant symptomatic improvement, and near-complete resolution of the tendon friction rubs.

DISCUSSION

Tendon friction rubs are prominent and thought to be diagnostic of SSc.[4] In a careful evaluation of 36 patients with SSc, those with tendon rubs showed thickened and non-inflamed tendon sheaths with varying degrees of surface fibrin deposition. Specimens from patients without tendon rubs ranged from normal synovial tissue to dense inflammatory infiltrates to synovial fibrosis.[3]

Our patient presented with symmetric polyarticular erosive arthritis and diffuse tendon friction rubs. While erosive arthritis can occur rarely in SSc [6], our patient had only Raynaud phenomenon and displayed no other features of SSc. He does fulfill 5 of the 7 ACR criteria for rheumatoid arthritis (RA).(7) We believe that his clinical presentation is most consistent with the diagnosis of seronegative RA. Treatment with methotrexate and adalimumab led to improvement of the polyarthritis and tendon rubs. Based on the present observations, we propose that while the existence of diffuse tendon friction rubs should alert clinicians to the diagnosis of SSc, erosive arthritis ought to be considered in the differential diagnosis.

Acknowledgments

Grant Support: NIH Grant: AR-42309

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Figure 1.

Figure 1a. Erosive arthritis of the hand. Sagittal T1 weighted MRI with fat suppression of the left hand. The MRI demonstrates metacarpal head erosions (solid arrows) and volar subluxations of the metacarpophalangeal (MCP) joints. There is extensive MCP synovitis with prominent flexor (dashed arrows) and extensor tenosynovitis (not depicted).

Figure 1b. Erosive arthritis of the knee. Coronal T2 weighted MRI of the left knee. The MRI demonstrates moderately thickened and inflamed synovium (dashed arrow) with a 9 mm erosion in the medial aspect of the lateral tibial plateau (solid arrow).

Table 1

General Laboratory Evaluation

Laboratory Test	Laboratory Value	Normal Range
Leukocytes	8.7	(3.5–10.5) k/ μ L
Hemoglobin	12.2	(13.0–17.5) k/ μ L
Eosinophils	0.2	(0.0–0.6) k/ μ L
Platelets	296	(140–390) k/ μ L
Serum Creatinine	0.8	(0–1.7) mg/dL
TSH	0.89	(0.4–5.0) mIU/mL
AST	24	(0–40) U/L
ALT	18	(0–48) U/L
Total Bilirubin	0.7	(0–1.3) mg/dL
Alkaline Phosphatase	77	(30–115) U/L
Albumin	3.6	(3.4–5.0) g/dL
ESR	21	(3–10) mm/hr
CRP	4.4	(<0.8) mg/dL
CPK	26	(0–200) U/L
BNP	72	(0.2–100) pg/mL
Urinalysis	Negative for RBC, WBC, protein, casts, & blood	
C3	173	(82–235) mg/dL
C4	45	(16–70) mg/dL

Table 2**Serum Antibody Profile**

Laboratory Test	Laboratory Value	Normal Range
ANA	<7.5	(<7.5) IU/mL
Rheumatoid Factor	<20	(<20) IU/mL
Anti-CCP IgG	16	(<20) U
Anti-dsDNA	<5	(<5.0) IU/mL
U1 RNP/SNRNP IgG	<5	(<5.0) U
Smith IgG	<5	(<5.0) U
SS-A IgG	<5	(<5.0) U
SS-B IgG	<5	(<5.0) U
SCL-70 IgG	<5	(<5.0) U
Anti-Centromere	<7.5	(<7.5) IU/mL
Beta2 Glycoprotein IgG	<20	(<20) U/mL
Beta2 Glycoprotein IgM	<10	(<10) U/mL
Beta2 Glycoprotein IgA	<10	(<10) U/mL
Anti-Thyropoxidase	34	(<60) U/L
Anti-Ribosomal P Protein	<5	(<5.0) U
Hepatitis C Antibody	NR	Nonreactive
Hepatitis B Surface Antibody	NR	Nonreactive
Hepatitis B Core Antibody	NR	Nonreactive
Hepatitis B Surface Antigen	Negative	Negative
Lyme Disease Antibody	Negative	Negative
Quantiferon	Negative	Negative
Parvovirus B19 IgG	5.65	(>1.11) IU
Parvovirus B19 IgM	0.09	(<0.89) IU
Anti-Streptolysin O	165	(<200) IU/mL