Ultrasonographic Evaluation of Pes Anserinus Tendino-Bursitis in Patients with Type 2 Diabetes Mellitus

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ABSTRACT. ***Objective.*** To assess musculoskeletal ultrasonographic (US) findings in patients with type 2 diabetes mellitus (DM) with and without pes anserinus (PA) tendinitis or bursitis syndrome; and to determine possible etiologic factors such as systemic diabetic microvascular disease complications in these patients.

Methods. The knee joints were examined with an ultrasound real-time scanner using a 10 MHz electronic linear transducer in 48 patients with type 2 DM and 25 controls. The presence of systemic diabetic microvascular disease complications was evaluated.

Results. On examination 23 (23.9%) knees of the 14 (29.1%) patients with type 2 DM were found to have PA tendinitis or bursitis syndrome. US revealed that only 4 (8.3%) of the diabetic patients with PA tendinitis or bursitis syndrome had PA tendonitis findings. There were no significant differences in the thickness of PA tendons between the diabetic patients with bilateral knee PA tendinitis or bursitis syndrome (9 patients) and controls, or between the asymptomatic and symptomatic knees in patients with unilateral PA tendinitis or bursitis syndrome (5 patients). The prevalence of morphologic changes of the medial meniscus, effusion and synovitis in the suprapatellar recess, popliteal cyst, and radiographic osteoarthritis (OA) in the diabetic patients with PA tendinitis or bursitis syndrome was found to be increased.

Conclusion. The prevalence of PA tendinitis or bursitis syndrome is not uncommon on examination in patients with type 2 DM. However, patients with clinically diagnosed PA tendinitis or bursitis syndrome less frequently have morphologic US changes of the PA tendons. Our results also suggest that structural changes such as meniscus lesions that occur in consequence of OA might have a role in the etiology of medial knee pain in diabetic patients. (J Rheumatol 2003;30:352–4)

Key Indexing Terms:
ANSERINE BURSITIS
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Although type 2 diabetes mellitus (DM) has been shown to be associated with pes anserinus (PA) tendinitis or bursitis syndrome[1], this is the first report to identify the morphological lesions of the anserine insertion and bursa by ultrasound (US) in patients with type 2 DM.

MATERIALS AND METHODS

We prospectively studied 48 patients with type 2 DM diagnosed at the Endocrinology Clinic (34 women, 14 men, mean age 55.1 (9.3) yrs, range 36–77 yrs). Five patients were either on a diet or not taking antidiabetic therapy including insulin or oral antidiabetic drugs. A control group was composed of 25 patients applying to the Physical Medicine and Rehabilitation Clinic who had no DM and no complaints around the knees, and no pathologic findings on examination.

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The effect of control of diabetes was assessed from the concentration of glycated hemoglobin and fasting blood glucose in the diabetic patients[2].

PA tendinitis or bursitis syndrome was clinically diagnosed according to a questionnaire regarding knee pain and PA tendinitis or bursitis examination as described[1]. The questionnaire included the following questions: (1) Have you had knee pain in the past 6 months? (2) Have you had knee pain in the last 2 weeks? (3) Does your knee hurt more when ascending, or descending, stairs? (4) Does your knee hurt more at night or when performing weight-bearing activity? (5) Do you have trouble getting out of a chair? PA tendinitis or bursitis syndrome was diagnosed only if the patient complained of pain attributable to the knee and tenderness was elicited on examination at the PA insertion. Patients with recent knee trauma or surgery, inflammatory rheumatic disease, shoulder tendinitis, trigger finger, Dupuytren's contracture, or hip disease were excluded from the study. In addition, fibromyalgia was excluded by tender point examination.

The knee joints were examined with an ultrasound real-time scanner (Hewlett Packard Image Point, Palo Alto, CA, USA). US was carried out using a 10 MHz electronic linear transducer. PA tendons and presence of PA bursitis was evaluated by US as described[3]. PA tendinitis was described as presence of both thickening and loss of normal fibrillar echotexture. The thickness of subcutaneous medial knee fat and collateral medial ligament was also measured and the presence of morphologic changes was recorded. Both homogeneity and position of the anterior medial meniscus horn were examined. The synovial thickening in the suprapatellar recess was documented as well as the presence of any effusion. The presence or absence of a popliteal cyst and patellar tendon abnormalities were assessed.

Standard weight-bearing anteroposterior radiographs at each knee were

graded for the presence of OA, according to the criteria described by Kellgren and Lawrence[4]. Radiographic OA of the knee was defined as the presence of at least grade 2 radiographic changes.

Diabetic retinopathy was assessed by direct ophthalmoscopy through dilated pupils. Peripheral neuropathy was assessed by electromyography. Microalbuminuria was defined as urinary albumin excretion > 30 mg/min in 2 consecutive samples or in 2 of 3 consecutive samples.

Statistical analysis was performed using the chi-square test, Mann-Whitney U test, and Kruskal-Wallis test for comparisons between the groups. $P < 0.05$ was established as a level of significance.

RESULTS

On examination 23 (23.9%) knees of the 14 (29.1%) patients with type 2 DM were found to have PA tendinitis or bursitis syndrome. No patient showed signs of fluid distention of the anserine bursae. Four (8.3%) diabetic patients who showed US tendinitis of the PA compared to the opposite knees had the PA tendinitis or bursitis syndrome in both knees on examination (Figures 1, 2). Enlargement of the PA tendons was found in one control subject compared to the opposite knee. All type 2 diabetic patients who showed signs of PA tendinitis on US examination had the PA tendinitis or bursitis syndrome in both knees. But the observed consistency between the clinical and US examinations of the PA tendons was found to be inconsistent (observed consistency 86.3%, kappa 0.243).

We found significant differences between the diabetic patients with and without PA tendinitis or bursitis syndrome and controls with respect to mean thickness of subcutaneous fat, presence of morphologic changes of the medial meniscus, effusion and synovitis in the suprapatellar recess, popliteal cyst, and radiographic OA of the knee joint. The values were greater in the diabetic patients with PA tendinitis or bursitis syndrome.

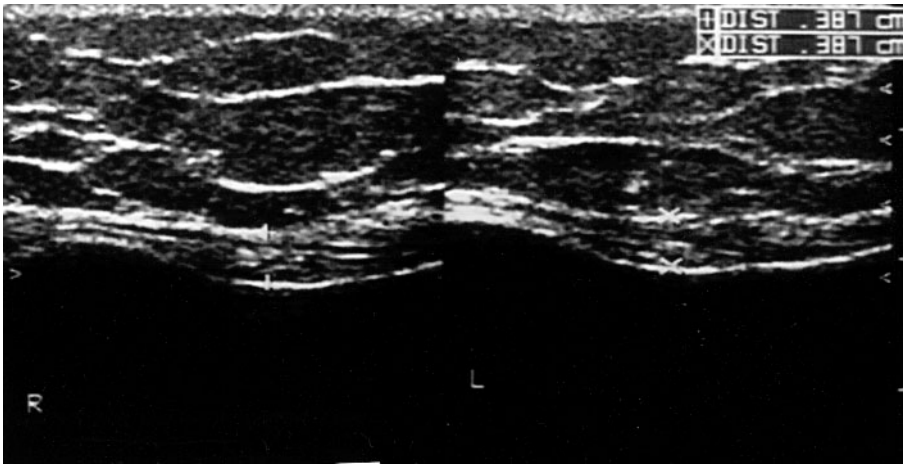

Figure 1. Longitudinal sonographic imaging of the PA tendons in a patient with type 2 DM. Thickness and fibrillar echotexture of the tendons were similar in both knees.

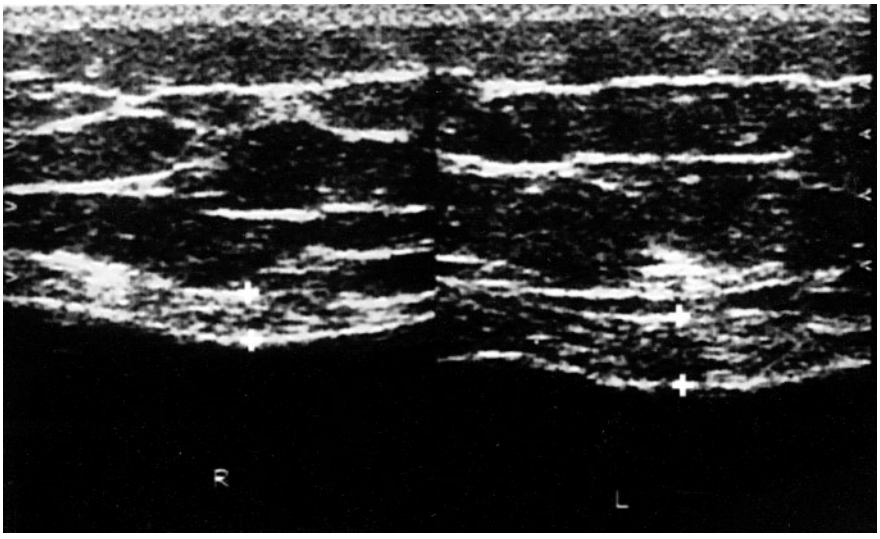

Figure 2. Longitudinal scan of a diabetic patient with PA tendinitis, showing tendon thickening and loss of the normal fibrillar echotexture on the left side compared with the right side.

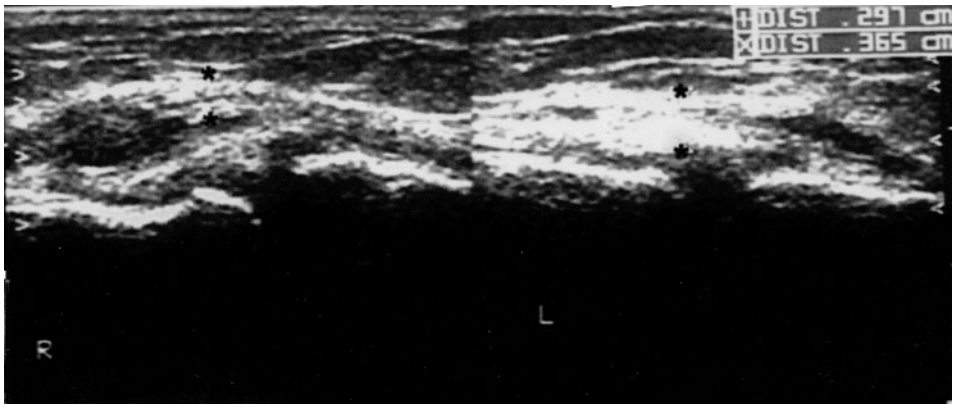

Figure 3. Longitudinal medial scans of a diabetic patient with bilateral PA tendinitis or bursitis syndrome on examination. Meniscal cyst on the right side and thickening of the medial collateral ligament on the left side compared with right side.

The ultrasonographic measurements of the PA insertion, subcutaneous medial knee fat, and medial collateral ligament were similar in the symptomatic knees compared to the opposite painless knees in the diabetic patients with unilateral PA tendinitis or bursitis syndrome (5 patients).

We found significant differences between the diabetic patients with bilateral PA tendinitis or bursitis syndrome (9 patients) and controls in respect to morphologic changes of the medial meniscus, effusion and synovitis in the suprapatellar recess, popliteal cyst, and radiographic OA in the knee joint (Figure 3). We did not detect a significant difference between the diabetic patients with bilateral knee PA tendinitis or bursitis syndrome and controls in respect to the thickness of PA tendons, subcutaneous medial knee fat, and medial collateral ligament.

Radiographic OA was found in 15 (65.2%) of 23 knees in diabetic patients with PA tendinitis or bursitis syndrome compared with 28 (38.3%) of 73 knees in the patients without PA tendinitis or bursitis syndrome, and the difference between the groups was statistically significant. Joint effusion, synovitis, popliteal cyst, and medial meniscus lesions were detected more frequently in the knees of diabetic patients with radiographic OA. Metabolic control showed no differences between the diabetic patients with and those without PA tendinitis or bursitis syndrome. No significant differences were observed between the groups for microvascular complications such as retinopathy, microalbuminuria (nephropathy), or neuropathy.

DISCUSSION

This study corroborated previous observations that prevalence of PA tendinitis or bursitis syndrome is not uncommon on examination in patients with type 2 DM; however, patients with clinically diagnosed PA tendinitis or bursitis syndrome less frequently have morphologic US changes of the PA tendons (29.1 vs 8.3%)[1,3]. Our findings also suggest that structural changes such as meniscus lesions that occur in consequence of OA might have a role in the etiology of medial knee pain in patients with type 2 DM.

US evaluation is superior to clinical examination for diagnosis of PA tendinitis or bursitis syndrome since it depicts morphologic changes in PA tendons and other structures around the knee. Hence, musculoskletal US examination should be routinely used as a diagnostic tool to improve the assessment of joints and soft tissues.

REFERENCES

1. Cohen SE, Mahul O, Meir R, Rubinow A. Anserine bursitis and non-insulin dependent diabetes mellitus. J Rheumatol 1997;24:2162-5.
2. The Diabetes Control and Complications Trial Research Group. The relationship of glycemic exposure (HbAiC) to the risk of development and progression of retinopathy in the Diabetes Control and Complications Trial. Diabetes 1995;44:968-83.
3. Uson J, Aguado P, Bernad M, et al. Pes anserinus tendino-bursitis: what are we talking about? Scand J Rheumatol 2000;29:184-6.
4. Croft P. Osteoarthritis. Br J Rheumatol 1990;29:391-5.