Orthopedic Surgical Forums

Clinical and Laboratory Parameters in Adult Diabetics With and Without Calcific Shoulder Periarthritis

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Received July 26, 1990, and in revised form November 14, 1990

Summary. The clinical and laboratory parameters of calcific shoulder periarthritis (CSP) were examined in 900 patients with type II diabetes mellitus as well as in 350 age- and sex-matched control subjects. A threefold increased prevalence of CSP in diabetics compared with the control group was associated with the presence of longstanding and poorly controlled diabetes, hypercholesterolemia, and hypertriglyceridemia suggesting pronounced diabetic angiopathy, as well as with minor trauma and hypomagnesemia. Aging and serum calcium concentrations were not related to the presence of CSP. Thirty-two percent of diabetics with CSP were symptomatic; 15% of them presented with severe pain and restriction of shoulder movement. These findings confirm a close pathogenetic interrelation between CSP and diabetes mellitus.

Key words: Calcific shoulder periarthritis – Painful shoulder syndrome – Diabetes mellitus – Hypomagnesemia.

A plethora of rheumatologic syndromes may present in association with diabetes mellitus [1, 2]. Except for neuroarthropathy and osteolysis, which are definitely associated, osteopenia, gout, pseudogout, carpal tunnel syndrome, osteoarthritis, Dupuytren’s contracture, chiroarthropathy, and diffuse idiopathic scleral hyperostosis are all considered as probable or even possible associations [1, 2].

The relation between diabetes mellitus and periarthritis of the shoulder has been discussed in a few studies [3-5]. Bridgman [3] and Lequesne et al. [4] suggested a four- to sixfold increased prevalence of calcifications in diabetes mellitus patients, and Kaklamanis et al. [5] recorded calcifications of the shoulders in a small group of diabetics. As we have reported previously [6], calcific shoulder periarthritis (CSP) was observed in 262 of 824 (31.8%) adult diabetics compared with 33 of 320 (10.3%) controls [6].

The present report is part of our ongoing study which was designed prospectively to assess the clinical, radiographic, and laboratory features of CSP in a large group of adult onset diabetes mellitus patients. Age- and sex-matched control subjects were studied in parallel. In this report, an attempt was made to reveal some clinical and biochemical interrelations between calcific periarthritis and diabetes mellitus.

Materials and Methods

Subjects

This prospective study involved 900 patients with type II diabetes mellitus (371 males and 529 females, aged 36–93, mean age 66.2 years) and 350 age- and sex-matched nondiabetic controls (141 males and 209 females, aged 34–87, mean age 65.7 years). All subjects were inpatients in the Department of Clinical Therapeutics, University of Athens, between 1979 and 1989. Diabetes mellitus diagnosis was made by the clinical history and the abnormal fasting blood sugar values, and the control group comprised subjects with normal fasting blood sugar values and normal glucose tolerance test. Subjects with a family history of diabetes were excluded from the control group. In addition, subjects who had evidence of renal impairment, cerebrovascular disease, peripheral neuropathy, postinfarction shoulder hand syndrome, and those with a history of injuries or fractures in the shoulder were excluded from both groups.

Protocol

Duration of diabetes, details of antidiabetic drugs used (oral drugs or insulin), and duration of the insulin administration were carefully recorded. All subjects had a standard anteroposterior X-ray film of both shoulders. The films were read independently by two observers who did not know the subject’s identity and were evaluated for calcifications indicating CSP. In the group of diabetics with CSP, the longitudinal axis of shoulder calcifications was measured in millimeters. All subjects who showed calcifications had a clinical examination separately by 2 of the authors evaluating the range of shoulder’s movement, as well as a neurologic examination for exclusion of neurologic disease. A detailed clinical medical history of their symptoms and signs (pain in the shoulders either during the study or previously) was obtained from all subjects.

The following were determined in all subjects of the study: serum uric acid, calcium, phosphorus, total cholesterol, and triglycerides, using standard methods. All subjects were on a fat-free diet for 48 hours prior to cholesterol and triglyceride determination. In addition, serum magnesium was randomly determined (normal 18–21 mg/liter) in 73 diabetics.
Table 1: Calcific shoulder periarthritis location and prevalence in diabetics and controls

<table>
<thead>
<tr>
<th>Location</th>
<th>Diabetics (n = 900) (%)</th>
<th>Controls (n = 350) (%)</th>
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<tbody>
<tr>
<td>Right shoulder</td>
<td>127 (14.1)²</td>
<td>14 (4.0)</td>
</tr>
<tr>
<td>Left shoulder</td>
<td>75 (8.3)²</td>
<td>11 (3.1)</td>
</tr>
<tr>
<td>Bilateral</td>
<td>88 (9.8)²</td>
<td>10 (2.8)</td>
</tr>
<tr>
<td>Total</td>
<td>290 (32.2)²</td>
<td>35 (10.0)</td>
</tr>
</tbody>
</table>

* Number of patients and percentage of group total

² P < 0.05 compared with controls

Statistics

Differences between proportions were examined using X² test and differences between mean values were determined by Student’s t test. Data are expressed as mean ± standard error (SE). Statistical significance was inferred for P values of < 0.05.

Results

Table 1 shows the location of CSP and its prevalence in diabetics and controls. A significantly higher number of calcifications was observed in the right shoulder of diabetics compared than in the left shoulder (127/290 versus 75/290, P < 0.05). Moreover, when diabetics were separated into right-handed (n = 791) and left-handed (n = 109) patients, the overall incidence of calcifications in the right shoulder compared with the left was higher in right-handed (24.4% versus 18.1%, P < 0.05), but not in left-handed patients (19.2% versus 19.2%, P: NS).

The mean age of diabetics with CSP was not significantly different from the mean age of those without CSP. However, the disease duration was shorter (P < 0.001), the percentage of patients treated with insulin was lower (P < 0.001), and duration of insulin therapy was shorter (P < 0.001) in this subgroup compared with the CSP subgroup (Table 2).

The size of calcifications (longitudinal axis) in diabetics ranged from 3 to 23 mm; patients were symptomatic when the size exceeded 16 mm. Among the 290 patients with CSP, 93 (32.6%) were symptomatic either before or during the study. Fifteen percent of them presented with acute CSP characterized by sudden and severe pain in the shoulder with total restriction of movement. The remaining 85% had a chronic form of CSP characterized by exacerbations and remissions. These patients complained of a dull ache, not easily localized, worsening with active shoulder movements.

Table 3 shows the mean values for serum uric acid, calcium, phosphorus, cholesterol, and triglycerides in the subgroups of diabetics and controls (with and without CSP). All mean values in these subgroups are within normal limits except for abnormally high cholesterol triglyceride levels in diabetics with CSP. No statistical difference could be demonstrated between any subgroup either for uric acid, calcium, or phosphorus. However, diabetics with CSP had significantly higher cholesterol and triglyceride levels when compared with diabetics without CSP (P < 0.001 and P < 0.001, respectively), and when compared with controls with CSP (P < 0.001 and P < 0.001, respectively) and controls without CSP (P < 0.001 and P < 0.001, respectively).

Determination of serum magnesium levels in a random sample of 73 diabetics revealed abnormally low magnesium values (<18 mg/liter) in 32 patients; 20 of them had CSP. Moreover, the mean value for magnesium in the group of diabetics with CSP (35 of 73) was significantly lower compared with the mean value for magnesium in the remaining 38 diabetics without CSP (17.58 ± 0.40 versus 18.86 ± 0.42 mg/liter, respectively, P < 0.04).

Discussion

Calcific shoulder periarthritis is a term that refers to inflammatory disorders of structures surrounding the shoulder—tendons, tendon sheaths, capsules, and bursae—associated with local calcium deposits [7]. It is also known as calcific tendinitis or tendinobursitis and even though many have assumed that this condition occurs as a consequence of degenerative tendinitis [8, 9], other studies suggest that calcium deposits may occur in the apparent absence of local degeneration [10, 11]. The precise composition of these deposits is not yet fully elucidated and even though the different phases of the disease have been histologically documented, a recent study failed to reveal any significant difference in crystal structure and composition of deposits between the acute and chronic phase of calcific shoulder periarthritis [12]. Most studies of the pathogenesis of this condition have focused on local factors that predispose periarticular tissues to calcification but the precise mechanism remains unknown [9, 13–17].

As we have reported previously, the prevalence of calcific shoulder periarthritis is three times higher in patients with diabetes mellitus than in control subjects [6]. Further evidence for a possible pathogenetic association between these two entities is provided by the present report. Our data show that the presence of CSP, in the absence of neurologic disease, is highly correlated with longer duration of diabetes as well as with longer insulin therapy, though it is not related to aging. In addition, diabetics with periarthritis have higher cholesterol and triglyceride levels compared with either diabetics without periarthritis or with the periarthritis control group. These findings suggest that altered tendon physiologic features, possibly produced by changes in local blood flow, may promote the development of periarthritis in longstanding and poorly controlled diabetes. As it is well known, in such cases diabetic angiopathy is more common and severe; hyperlipidemia precipitates atherosclerotic vascular lesions in general [19]. In addition, high triglyceride levels is an independent risk factor for atherosclerosis in diabetes [20]. On the other hand, experimental studies have shown that decreased blood flow in the shoulders facilitates and precipitates the calcification of the surrounding tendons [7, 18]. Moreover, the microvascular pattern of the supraspinatus tendon in 18 human anatomic specimens has been investigated recently and the presence of a hypovascular zone close to the insertion of this tendon into the humeral head was confirmed [21]. Consequently, it is reasonable to suggest that the high prevalence of shoulder calcifications in diabetics is due to the compromised vascular supply of this region. Repeated minor trauma may have an additive effect [22] as calcifications were observed more frequently in the right shoulder and most of our population is right handed. Moreover, the normal levels of calcium and phosphorus in the diabetics with CSP, which were similar with the levels in the other groups (Table 3), confirm that calcifications in this case do not result from a generalized disturbance in calcium and phosphorus metabolism but follow some local abnormality of the affected connective tissue [23].

Defective formation of glycosaminoglycan in diabetes mellitus is suggested by the work of Schiller and Drofman [24], who showed that insulin is necessary for the production