Possible Epidemiological Factors Associated with Rupture of the Posterior Tibial Tendon

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ABSTRACT

Rupture of the posterior tibial tendon has been postulated to occur, in part, as a result of degenerative changes to the tendon. This possibility was examined by a review of 67 patients (average age 57 years) diagnosed with rupture of the posterior tibial tendon. Forty-five of the 67 patients (60%) had one or more of the following positive medical histories: (1) hypertension, (2) obesity, (3) diabetes mellitus, (4) previous surgery or trauma about the medial aspect of the foot, or (5) steroid exposure. Thirty-five patients (52%) had either hypertension, diabetes mellitus, or obesity. A statistical correlation was demonstrated between rupture of the posterior tibial tendon and obesity ($P = .005$) and to a lesser extent hypertension ($P = .025$). These disorders have been uniformly associated with an acceleration of the degenerative processes associated with aging, commonly via an acceleration of microvascular and macrovascular diseases. An additional vascular risk is implicated by the known zone of hypovascularity of the posterior tibial tendon and risk of rupture secondary to systemic or local injections of corticosteroids. The prevalence of posterior tibial tendon rupture parallels the degenerative processes of aging, hypertension, diabetes mellitus, and obesity. Additionally, the effects of corticosteroids and local surgical procedures may further be associated with local vascular impairment and eventual rupture.

INTRODUCTION

Rupture of the posterior tibial tendon is one cause of painful acquired flatfoot deformity in adults. The progressive collapse of the medial longitudinal arch leads to the development of several secondary deformities. These include a valgus alignment of the calcaneus, plantarflexion of the talus, abduction of the forefoot, and a fixed forefoot varus-supination deformity.

There is considerable interest within the orthopaedic literature concerning the entity of rupture of the posterior tibial tendon.[1,22,35,36,38] There are several studies describing not only the anatomy and function, but also the management, of rupture of the posterior tibial tendon.[21,30,31,35,36,38,39,44,48] Little is known about those factors which may be associated with or predispose patients to rupture of the posterior tibial tendon. One study indicates a higher frequency of rupture in women over the age of 40.[33] Other series suggest a possible association between rupture of the posterior tibial tendon and rheumatoid arthritis.[1,35] Two relatively large studies in 1983 and 1985 failed to link major trauma about the foot and ankle with the occurrence of rupture.[33,44] In this study, 67 patients diagnosed with rupture of the posterior tibial tendon underwent a review of their medical records with the purpose of identifying possible associated epidemiological factors.

MATERIALS AND METHODS

From January 1978 to December 1986, 79 patients with acquired flatfoot deformity secondary to rupture of the posterior tibial tendon were treated by the one author (R.A.M.). Twelve patients were omitted from the study due to incomplete medical records and the inability to contact them. There were 51 women and 16 men with an average age of 57 years (range 19–87 years). Sixty-five of the 67 patients were Caucasian. One was Hispanic and one was Asian. A detailed medical history was obtained for all patients (see Table 1). All records were assessed for the presence of (1) hypertension, (2) obesity, (3) diabetes mellitus, (4) previous surgery or trauma, and (5) exposure to corticosteroids.

Patients were classified as being hypertensive if they were under the active care of an internist or family practitioner with a specific diagnosis of hypertension. In each instance, the medical records or a patient

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interview confirmed the diagnosis of hypertension and the patient's ongoing involvement in a specific treatment plan of diet, weight, or medication control.

The criteria developed by Craddock[10] was adopted for categorization of obesity for the purposes of this study. In this system, obesity was defined as an excess of adipose tissue reflected in a patient's weight of more than 20% above the normal or predicted weight for a specified age and height. Patients classified as obese were 20% above their predicted body weight and could be clinically described as being moderately obese.[3]

Diabetes mellitus represents a "disturbance of glucose homeostasis."[3] The resultant elevation of serum glucose can be determined by blood glucose level or glucose tolerance test. All patients identified as having diabetes were under the active care of a family practitioner or internist for diabetes mellitus. In each instance, patients were being treated by diet, oral hypoglycemia agents, or insulin.

The presence of corticosteroid exposure was based upon a history of local injections about the posterior tibial tendon or the oral intake of corticosteroids. Some reasons for the administration of corticosteroid injections were persistent medial foot and ankle pain secondary to posterior tibial tendinitis and nonspecific soft tissue trauma. In most instances, patients received multiple injections in or about the medial aspect of the foot. The injections preceded the diagnosis of rupture of the posterior tibial tendon by a period of months to years. With respect to oral corticosteroids, some patients had been prescribed systemic corticosteroids for various medical illnesses, such as bronchitis, polymyalgia rheumatica, and rheumatoid arthritis. Patients taking estrogen replacement for the treatment of menopausal and postmenopausal symptoms were not tabulated with those patients receiving injection or oral corticosteroids.

Finally, medical records were examined for the presence of prior major trauma or surgical procedures about the medial aspect of the foot and ankle. The criteria for inclusion in this group were: (1) previous fractures about the medial aspect of the foot and ankle, or (2) surgery about the medial aspect of the foot and ankle.

The incidence of hypertension, obesity, diabetes mellitus, trauma, and corticosteroid administration in these 67 patients was compared with the incidence of these disorders in the population at large. The Chi-square test for goodness of fit was utilized in the statistical analysis of the significance of various disorders in patients with rupture of the posterior tibial tendon in comparison with these disorders in the population at large.[15]

The diagnosis of posterior tibial tendon rupture was based upon a thorough history and physical examination. All patients were examined by the senior author (R.A.M.). The chief complaint was that of pain localized to the medial aspect of the foot. Commonly, there was an associated loss of the medial longitudinal arch of the foot. Inspection demonstrated a swelling that corresponded to the distal aspect of the proximal stump of the ruptured tendon. Patients lacked the ability to simultaneously plantarflex and invert the forefoot. There was a profound weakness in the patient's effort to adduct the plantarflexed and abducted forefoot against resistance. Simultaneous palpation of the posterior tibial tendon distal to the medial malleolus demonstrated its attenuation of function. Perhaps the most sensitive test of rupture of the posterior tibial tendon was to have the patient attempt single leg-toe stance on the injured side. Fifty-one of the 67 patients underwent surgical exploration and reconstruction. The remaining 16 patients were treated by nonoperative means.

RESULTS

The data collected from the review of medical records and patient interviews are summarized in Table 1. Information was recorded for the duration of symptoms, history of trauma, and the presence of hypertension, obesity, diabetes mellitus, steroids, previous surgery or injury, and other significant medical illnesses. The majority (76%) of patients were women. The average age of the patients was 57 years (range 19–87 years). The mean duration of symptoms before the diagnosis was 25.7 months. Thirty-eight of the 67 patients (approximately 3:2) had no history of trauma immediately prior to the onset of collapse of the medial longitudinal arch. Of the 29 patients who did have trauma, the event generally consisted of a minor twisting injury of the foot or ankle.

A summary of various diagnoses of patients with ruptures is outlined in Table 2. Thirty-four percent had a diagnosis of hypertension. Twenty-three patients (34%) were obese by the Craddock criteria. Thirteen patients had been administered corticosteroids orally or via injections. Seven patients had prior surgery or major trauma to the medial aspect of the foot or ankle. Patients with lower extremity trauma and surgery not localized to the medial aspect of the foot were not included for consideration within this group. Finally, six patients were diabetic.

Various miscellaneous medical diagnoses are also indicated in Table 1. Case 4 had a history of arrhythmias. Another case (case 20) with a diagnosis of hypothyroidism was treated with Synthroid (Boots Pharmaceuticals, Lincolnshire, IL) for a period of 10 years. Case 50 had rheumatoid arthritis and, additionally, was prescribed oral corticosteroids. Case 60 had a history

TABLE 1
Clinical Data and Results*

Case*	Sex	Age	Symptom duration	Trauma history	HTN	OB	DM	SURG	STER	Other
1	F	67	3	—	—	—	—	—	—	—
2*	F	59	12	—	—	—	—	+	—	—
3	M	60	24	—	—	—	—	—	—	—
4*	F	54	1	+	—	—	—	—	+	+
5*	F	57	30	—	+	—	—	—	+	—
6*	F	39	48	+	—	—	—	+	+	—
7*	F	21	8	—	—	—	—	—	—	—
8	M	68	12	—	—	+	—	—	+	—
9*	F	75	12	—	+	—	+	—	—	—
10*	F	64	6	—	—	+	—	—	—	—
11	F	59	8	+	—	—	—	+	—	—
12	F	61	12	+	+	+	—	—	—	—
13*	F	52	24	—	+	—	—	—	+	—
14	F	63	60	+	—	—	—	—	+	—
15	M	58	18	—	—	—	—	—	+	—
16*	F	65	84	—	+	+	+	—	—	—
17*	F	71	36	—	—	—	+	—	+	—
18*	F	66	6	—	—	—	—	—	+	—
19*	F	55	60	—	—	—	—	—	—	—
20*	M	70	12	—	—	—	—	—	—	+
21*	M	54	36	+	+	—	—	—	—	—
22	F	82	3	+	+	—	—	—	—	—
23*	M	62	3	+	+	—	—	—	—	—
24*	F	61	1	—	+	+	—	—	—	—
25*	F	48	8	+	—	+	—	—	—	—
26*	F	73	6	+	—	—	—	—	—	—
27*	M	50	120	—	—	—	—	—	—	—
28*	M	53	2	+	—	+	—	—	—	—
29*	F	40	12	+	—	—	—	+	—	—
30	M	49	1	+	—	—	—	—	—	—
31*	F	63	6	+	—	—	—	—	—	—
32*	F	36	9	+	—	—	—	+	—	—
33	F	87	18	—	—	—	—	—	+	—
34*	F	52	48	+	—	+	—	—	—	—
35*	F	75	2	+	+	+	—	—	—	—
36*	F	58	120	—	+	+	+	—	—	—
37*	F	68	36	—	+	+	—	—	—	—
38*	M	59	36	+	—	+	—	—	—	—
39*	F	44	84	+	—	+	—	—	—	—
40*	F	54	3	+	—	+	+	—	—	—
41*	M	57	1	+	+	—	—	—	—	—
42*	M	55	48	+	+	+	—	—	—	—
43*	F	64	12	+	—	+	—	—	+	—
44*	F	54	60	+	+	—	—	—	—	—
45*	F	57	18	—	—	—	—	—	+	—
46	F	66	12	—	+	—	—	—	—	—
47*	M	66	12	—	—	+	—	—	—	—
48	M	34	18	—	—	—	—	—	+	—
49*	F	59	6	—	+	+	—	—	—	—
50*	F	35	3	+	—	—	—	—	+	+
51*	F	63	12	+	—	—	—	—	+	—
52*	F	33	24	+	—	—	—	—	+	—
53*	F	72	24	—	+	+	—	—	—	—
54	F	70	36	—	+	+	—	—	+	—
55*	F	63	30	—	+	—	—	—	+	—
56	F	19	24	+	—	—	—	+	—	—
57	F	52	10	—	—	—	—	—	—	—
58*	F	56	36	—	—	—	—	—	—	—
59	F	66	12	—	+	—	—	—	—	—
60*	F	55	120	—	—	—	—	—	—	+
61*	M	44	6	+	+	+	+	—	—	—

Table 1 continues on the next page.

TABLE 1
Continued

Case[a]	Sex	Age	Symptom duration	Trauma history	HTN	OB	DM	SURG	STER	Other
62*	F	61	13	—	+	+	—	—	—	—
63*	M	52	6	—	—	+	—	—	—	—
64*	F	36	36	—	—	—	—	+	—	—
65*	F	52	6	—	—	—	—	—	+	—
66*	F	71	30	—	—	—	—	—	+	—
67*	F	68	24	—	—	—	—	—	—	—

[a] HTN, hypertension; OB, obesity; DM, diabetes mellitus; SURG, surgery; STER, steroid.
[b] Asterisks indicate patients with diagnosis confirmed at time of surgery.

TABLE 2
Summary of Diagnoses of Patients

Diagnosis	Patients (N)	Patients (%)
Hypertension	23	34
Obesity	23	34
Diabetes mellitus	6	9
Steroids[a]	13	19
Surgery/trauma	7	10

[a] Oral or intramuscular corticosteroids.

TABLE 3
Summary of Data of Patients with a Positive History of Corticosteroids

Case	Sex	Age	History
6	F	39	Crush injury of the ankle followed by six corticosteroid injections around the ankle
8	M	68	Prednisone therapy for 15 yrs for chronic bronchitis
13	F	52	Estrogen replacement for 5 yrs; multiple corticosteroid injections about the P.T.T., shoulder, and neck
15	M	58	Several corticosteroid injections around the P.T.T.
17	F	71	Oral corticosteroids for 38 yrs for endocrine disorder
18	F	66	Premarin replacement for 15 yrs; single corticosteroid injection about the tendon sheath
43	F	64	Prednisone therapy for >3 yrs for polymyalgia rheumatica
45	F	57	Premarin (>3 yrs), Provera, and prednisone therapy for greater than 3 yrs
48	M	34	Indeterminant number of injections
50	F	35	Indeterminant number of years of oral steroids for rheumatoid arthritis
52	F	33	Multiple steroid injections after ankle fracture
54	F	70	Dexamethasone injections (×3) about P.T.T.
66	F	71	Premarin replacement for >10 yrs; S/P at least one cortisone injection

of bilateral moderate pes planus and bilateral accessory navicular bones.

The criteria for the diagnoses of hypertension, obesity, and diabetes were delineated thoroughly in Materials and Methods. The data collected for patients with a history of steroid exposure are displayed in Table 3. Thirteen of 67 patients had a positive history of steroids. All but two of these patients were women. Three patients with rheumatoid arthritis, bronchitis, and polymyalgia rheumatica, respectively, had taken oral corticosteroids for a number of years for the control of their disease. One other patient received oral corticosteroids intermittently over a period of 38 years for an ill-defined endocrine disorder. The five remaining patients had several corticosteroid injections about the ankle and, frequently, adjacent to the posterior tibial tendon itself. One of these patients, case 6, had a total of six injections around the ankle after a crush injury to the ankle. Case 54 had three injections of dexamethasone about the posterior tibial tendon for presumed tendinitis.

Seven patients had a history of major trauma or surgery about the medial aspect of the foot remote and prior to the development of rupture of the posterior tibial tendon (see Table 4). Patients 32 and 64 had undergone excision of an accessory navicular bone (Kidner procedure) a few years prior to rupture of the posterior tibial tendon.[47] In one instance, a 36-year-old woman had undergone excision of an accessory navicular 10 years prior to the diagnosis of rupture. The second case was that of a 36-year-old woman who had an accessory navicular bone removed 4 years before her rupture on the same side. Other procedures in this group included excision of bone chips from the medial aspect of the foot and removal of bone spurs. One patient had a severe crush injury to the ankle that was followed by a series of cortisone injections. Finally, one patient had trauma with an associated metatarsocuneiform dislocation.

Patients were then assigned to one of two groups based upon their age. There were 14 patients between the ages of 19 and 50 years. Between the ages of 51 and 87, there were 53 patients. Within each group, patients were arranged by the diagnoses used for the analysis of the entire patient population. The number and percentages of patients by diagnosis for patients aged 19 to 50 years is represented in Table 5. In this group, only one patient had a diagnosis of diabetes

mellitus. No patient had hypertension. Three patients were obese by Craddock's criteria. Four patients (cases 6, 48, 50, and 52) had a history of corticosteroid therapy. Five of 14 patients (cases 6, 29, 32, 56, and 64) had a history of previous major trauma or surgery about the medial aspect of the foot.

The grouping by diagnosis for patients aged 51 to 87 years is outlined in Table 6. Fifty-three patients were included in this group. Twenty-three patients had hypertension. Twenty patients were obese. A history of steroid intake or injections was elicited in nine patients.

TABLE 4
Summary of Data of Patients with a Positive History of Trauma or Surgery

Case	Sex	Age	History
6	F	39	A major crush injury was sustained to the ankle 4 yrs prior to rupture; the patient also received six cortisone injections about the ankle
11	F	59	Bone chips were removed from the medial aspect of the ankle a few years prior to the onset of symptoms
29	F	40	History of multiple sprains; S/P Watson Jones procedure and S/P removal of heel spur from medial approach
32	F	36	S/P excision of accessory navicular some 10 yrs prior to onset of symptoms
35	F	75	Heel spur removed prior to the onset of symptoms
56	F	19	S/P metatarsocuneiform dislocation
64	F	36	S/P excision of accessory navicular 4 yrs before the diagnosis of rupture

TABLE 5
Diagnoses of Patients, Aged 19 to 50 years

Diagnosis	Patients (N)	Patients (%)
Hypertension	1/14	7
Obesity	3/14	21
Diabetes mellitus	1/14	7
Steroids	4/14	28
Surgery/trauma	5/14	36

TABLE 6
Diagnoses of Patients Aged 51 to 87 years

Diagnosis	Patients (N)	Patients (%)
Hypertension	23/53	43
Obesity	20/53	38
Diabetes mellitus	5/53	9
Steroids	9/53	17
Surgery/trauma	2/53	4

Five patients were diabetic. Only two patients had a history of previous surgery or major trauma to the foot.

In order to assess the significance of these various diagnoses with respect to the occurrence of rupture of the posterior tibial tendon, it was necessary to define the incidence of these disorders for both age groups in the population at large. Statistical analysis was then used to compare the incidence of these disorders in the normal population with that in the population of patients with rupture of the posterior tibial tendon. In order to show a significant correlation between rupture of the posterior tibial tendon and any one of these various disorders, the statistical analysis had to show that the occurrence of a particular condition was higher in patients with rupture than would be expected in the population at large for the same age group. This goal was achieved by acquiring estimations of various disorders in the population at large and comparing these prevalence rates with those of the patient group using the Chi-square test for goodness of fit.[15] The prevalence of the various disorders in this study was established by the data in published population data. Another alternative would have been to determine the prevalence of these disorders for various specific subsets of populations, such as for patients for routine examination or orthopaedic trauma patients. The selection of the correct subset in itself may have introduced an additional factor of bias into the so-called control population.

In the general population, the estimation of prevalence of those patients with diabetes mellitus, hypertension, obesity, steroid use, and surgery or trauma is listed in Table 7.[3,7,10,26,28] The expected incidence of diabetes mellitus in people aged 19 to 50 years was estimated to be 2%. This prevalence rate was based on the rates for the general population which included such populations as blacks, Hispanics, and American Indians, in which there was a higher incidence of diabetes mellitus than in the Caucasian population. Since there were no blacks or American Indians and only one Hispanic patient in this series, the estimation used here would actually make any statistical significance slightly more valid for the population of this study. Comparing

TABLE 7
Expected Frequencies*

Diagnosis	19–50 Yrs (%)	51–87 Yrs (%)
Hypertension	14	35
Obesity	13	30
Diabetes mellitus	2	6
Steroids	*	*
Surgery/trauma	*	*

* Asterisks indicate that no expected frequencies are available.

this prevalence with that of diabetes mellitus in the 19 to 51 age group, the Chi-square goodness of fit method showed no statistically significant difference.

For patients aged 19 to 50 years, the estimated prevalence of hypertension was 14%. As is the case for diabetes mellitus, the Chi-square method indicated no statistical significance for hypertension. In a similar fashion, no significance was found between obesity and rupture of the posterior tibial tendon.

No accurate data could be found for the prevalence of steroid use, orally or by injection, in either age group. Therefore, Chi-square analysis could not be calculated for steroid exposure and rupture of the posterior tibial tendon. The data for steroid exposure could only be analyzed from a descriptive standpoint. Intuitively, a percentage of about 20% of patients in this group with a steroid history merits some interest as a factor suspicious in its relation to posterior tibial tendon ruptures. This statement is further supported by numerous investigations indicating the deleterious effects of steroids on tendon collagen.

A similar situation exists for an analysis of the significance of surgery or trauma. No accurate data could be determined for the incidence of trauma about the foot or ankle for the population at large between the ages of 19 and 50 years. Again, it is not probable that 36% of people under the age of 50 have had trauma or surgery about the medial aspect of the foot. The size of the population for this age in this study limits any more formal in-depth analysis of the role of trauma and surgery as a predisposing factor to rupture of the posterior tibial tendon.

The estimation of expected occurrences of hypertension, obesity, and diabetes mellitus in the population group of 51 to 86 years is indicated in Table 7. No significance between diabetes mellitus and rupture of the posterior tibial tendon could be determined by the Chi-square goodness of fit test. For obesity, an association with tendon rupture was determined at a confidence level of $P = .005$. A P-value of .025 was found for the association between rupture and hypertension. A very small number of patients had surgery or major trauma. In descriptive terms, trauma and surgery did not seem to play a role in tendon ruptures in this older population. Thirteen of the patients with rupture had histories of steroid exposure. The lack of an accurate incidence of steroid use in the population nullifies a closer statistical analysis.

DISCUSSION

An understanding of the epidemiological factors related to rupture of the posterior tibial tendon has not yet been clearly delineated within the orthopaedic literature. Several studies have identified an increased prevalence of rupture of the posterior tibial tendon in middle-aged women.[19,33,44] Our analysis confirms a predominance of late middle-aged women in the total group of patients in this study. There were also more middle-aged men than young or older men in the study's group of male patients. However, the largest absolute number of patients and percentage of patients were late middle-aged women.

The discussion and conclusions to follow are based upon the analysis of data based on the diagnosis of tendon rupture in the 67 patients in this series. Of this group, the diagnosis was confirmed at the time of surgery in 51 patients. The sole reliance on history, physical examination, and radiographic studies for the remaining patients may cast some doubt on the diagnosis for the remaining nonoperative patients. It may, in fact, be argued that, indeed, some of the patients treated nonoperatively may have other causes of their acquired flatfoot deformity. These could include severe posterior tibial tendinitis or dysfunction of the medial ligaments of the longitudinal arch. However, the percentages of the various diagnoses calculated for those patients who underwent surgical exploration were almost identical to those for the group at large (Table 8).

Several investigations have indicated that trauma does not play a significant role in rupture of the posterior tibial tendon.[19,44] Funk et al.,[19] in a study of 19 patients with rupture of the posterior tibial tendon, found only four of 19 patients with a specific episode of trauma. Only 29 of 67 patients in this series could recall an acute injury prior to the onset of pain or loss of the longitudinal arch. When trauma could be recalled by patients, it usually consisted of a mild sprain about the foot and ankle. The majority of patients could not recall a specific traumatic incidence. The lack of the proximity of major trauma suggested that rupture of the posterior tibial tendon was more likely related to an intrinsic abnormality or biomechanical failure, rather than to an extrinsic traumatic factor. The only exceptions were

TABLE 8
Comparison of Surgically Confirmed Cases versus the Total Patient Group

	Surgical group (N = 51)		Total group (N = 67)	
	19–50 (N = 11)/(%)	51–87 (N = 40)/(%)	19–50 (N = 14)/(%)	51–87 (N = 53)/(%)
HTN*	9	43	7	43
OB	27	43	21	38
DM	9	13	7	9
ST	27	15	28	16
TX	36	3	36	4

* HTN, hypertension; OB, obesity; DM, diabetes mellitus; ST, steroids; TX, trauma/surgery.

found in the younger patients in this study. Remote prior major trauma, either by injury or surgery about the medial aspect of the foot, was noted in nearly ½ of the younger patients. This major event preceded the rupture by at least a few years. Attention was drawn to the possible importance of antecedent trauma by the relatively large number of patients in the younger group with major trauma about the medial foot and ankle. This finding was not seen in the older group of patients. Two patients specifically had the removal of an accessory navicular bone prior to their ruptures. Procedures for excision of an accessory navicular may place at risk both the attachment of the posterior tibial tendon and its adjacent blood supply.

Anzel et al.[1] has suggested that rupture of the posterior tibial tendon was likely to result in patients with rheumatoid arthritis. A review of other series did not support a strong relationship between rheumatoid arthritis and rupture of the posterior tibial tendon.[19,22,44] Funk et al.[19] had only one of 19 patients who had rheumatoid arthritis. Griffiths[22] reported one of four patients with rheumatoid arthritis. Only one patient in our study had a diagnosis of rheumatoid arthritis.

Age appeared to be a factor related to rupture of the posterior tibial tendon. Young patients with ruptures did not have a particularly high incidence of obesity, hypertension, or diabetes mellitus. Probably of most significance was a history of previous trauma or surgery in the younger group of patients. The small number of patients and the lack of statistics on the overall incidence of foot trauma and surgery for this age group made it impossible to draw specific statistical conclusions.

For patients 51 to 87 years old, obesity and hypertension appeared to be more significantly correlated as a potential factor associated with rupture of the posterior tibial tendon. This finding was noted in spite of the increased prevalence of hypertension and obesity in patients of advanced age. No specific significance could be derived for patients with ruptures and diabetes mellitus. Due to the lack of data for the use of corticosteroids in patients over the age of 50 years, no statistical significance could be calculated for these patients. However, from a descriptive standpoint, most patients in this series had numerous injections of more than just a trivial history of oral use of corticosteroids.

One potential area of concern in the design of this study was the reliance on published prevalence data of various disorders to calculate statistical analysis. It could be argued that the national proportions used for the incidence of diabetes, hypertension, and obesity could differ from those proportions specific to the population base from which the patients were drawn who had ruptures of the posterior tibial tendon. The population pool for this study tended to be somewhat more affluent and less diverse than the national population baseline figures. However, the estimates used were quite conservative in order to compensate for this potential bias. Alternatively, a regional population pool could have selected as the control. However, this would have introduced other biases into the analysis.

The linkage of obesity, hypertension, and diabetes mellitus is key to the interpretation of the descriptive and statistical data presented thus far. Overall, 35 of 67 patients (52%) were either hypertensive, obese, or diabetic. There is ample evidence to support the singular association of these disorders. Defronzo and Ferrannini[12] have recently reviewed numerous studies that indicate that the common occurrence of the pentad of obesity, diabetes, hypertension, atherosclerotic coronary vascular disease (ASCVD), and dyslipidemia is more than a chance occurrence and may be related in part to a gene or set of genes. Hypertension is very commonly seen in obese patients and patients with diabetes.[8,27,32,46,51,54] Manicardi[43] has demonstrated a high prevalence of insulin resistance in obese, hypertensive patients. Numerous authors have further shown that obesity leads to insulin resistance.[5,6,11,13,14,16,20,37] Currently, endocrinologists believe that insulin resistance and hyperinsulinemia are the common links for hypertension, obesity, diabetes, ASCVD, and dyslipidemia.[12] The presence of hyperinsulinemia is thought to promote vascular hyperplasia and sclerosis, which further leads to arterial stiffness, luminal narrowing, and increased resistance to blood flow. Through this mechanism, hypertension, diabetes, and obesity lead to alterations in arterial vessel resistance, hypertrophy of vessel walls, and the resultant microvascular and macrovascular arterial disease.[4,17,38,57]

Rupture of the posterior tibial tendon may also be related to direct changes in the collagen as a result of the progression of age-related degenerative changes in the body. It has been demonstrated that these changes are further accelerated in patients with obesity, hypertension, and diabetes. Hamlin[25] has demonstrated that diabetes mellitus accelerates the aging process of collagen, as manifested by increased collagen stiffness, cross-linkage, and collagen stabilization.[23–25] There may, therefore, be a change in the ultrastructure of collagen as a direct result of the biochemical manifestations of aging and the acceleration of the aging process, as seen in hypertension, obesity, and diabetes.

The presence of local trauma and surgery about the foot and ankle on a descriptive level appear to be important factors associated with rupture in young patients. Twenty percent of patients had a history of multiple steroid injections or a history of oral corticosteroids. The question of whether this percentage is

unique to this population or can be found at the same level in the population at large is beyond the scope and focus of this investigation.

Surgery about the medial aspect of the foot can certainly place the local blood supply in this area at risk of injury and, ultimately, attenuation. Small and large vessel patency has been shown to be attenuated with age, hypertension, obesity, and diabetes.[4,17] It can be manifested in decreased capillary filling, renal dysfunction, and various other degenerative and age-related problems. Commonly, the endpoint is small and large vessel changes that ultimately lead to diminished small vessel patency and perfusion. Local microavascular attenuation has been shown to result from local corticosteroid injections.[34] Numerous cases in the literature indicate the cause and effect relationship of oral corticosteroids and peritendinous injection of corticosteroids to tendon rupture.[2,29,40,45,55,56] Avascular changes in bone are changes well documented to be related to the systemic intake of corticosteroids.[52,53,58] The common thread, then, for these processes is micro and macrovascular attenuation.

This model of the interrelatedness of aging, local trauma, obesity, and hypertension and, in a related manner, diabetes suggests that rupture of the posterior tibial tendon may be the result of more than just biomechanical factors. Ruptures have been documented to frequently occur distal to the medial malleolus. Ruptures strictly on an attritional basis would be more likely to occur immediately posterior to the medial malleolus. However, this is an infrequent site of rupture. McMaster[42] suggests that tendons with as much as half of their cross-sectional area removed still retain a considerable degree of their tensile strength. The conclusions of McMaster were: (1) normal tendons do not rupture even when subjected to severe strain; (2) spontaneous rupture does not occur following the cutting of three fourths of the fibers of a tendon under normal activity; and (3) obstruction of the blood supply can lead to rupture adjacent to the area of diminished blood supply.

In a report by Frey et al.[18] a modified Spalteholz technique was utilized to assess the vascularity of the posterior tibial tendon. An intervening zone of avascularity was noted to lie between the boy insertion on the navicular and the musculotendinous junction.[18]

An anatomic dissection by Lovell and Tanner[41] demonstrated that the posterior tibial tendon was without a mesotendon. Other tendons of the foot were found to be enveloped in a mesotendon. Microvascular injection studies by Smith[49,50] have confirmed the segmental vascular contribution of the mesotendon to tendons. Other investigators have been in agreement with this finding.[9] The mesotendon supplies blood to the tendon, in addition to that supplied at the bone insertion and the musculotendinous junction. The lack of a mesotendon, the zone of avascularity, and histologic findings of avascular changes all suggest at least a relative vascular susceptibility of the posterior tibial tendon to vascular compromise. Local vascular impairment could then lead to rupture under almost physiologic stress conditions.

Surgery or major local trauma can lead to the disruption of the local blood supply about the posterior tibial tendon. Two patients had undergone excision of an accessory navicular prior to rupture of the posterior tibial tendon. The excision of the accessory navicular and the mobilization and reattachment of the posterior tibial tendon are risks to the adjacent vascular supply of the tendon. Furthermore, the patients who underwent excision of the accessory navicular bone were 26 and 32 years old, respectively. Unlike preteens and teenagers, older patients who undergo excision may have a more vulnerable blood supply to the posterior tibial tendon and, therefore, may subsequently be placed at greater risk for rupture of the posterior tibial tendon.

SUMMARY

This study has attempted to analyze certain disorders with a known or suspected relationship to aging and degenerative processes in general. Posterior tibial tendon ruptures occur predominantly in the late middle-aged population (average age 57 years). Only 17 of the 67 patients (25%) did not have either hypertension, obesity, diabetes, corticosteroid exposure, or major surgery around the posterior tibial tendon. In other words, 75% of patients had a significant identifiable systemic or local vascular risk. The data presented here are of interest in light of: (1) the known vascular consequences of the triad of hypertension, diabetes, and obesity; (2) the consequences of aging on collagen ultrastructure and tensile characteristics; (3) the known local vascular anatomy of tendons and the posterior tibial tendon in particular; and (4) the known association of tendon ruptures with systemic and local corticosteroids.

Therefore, rupture of the posterior tibial tendon may be related to both local and systemic vascular impairment. Age, hypertension, obesity, traumatic disruption of local blood supply, and the administration of corticosteroids may lead to vascular compromise and subsequent tendon rupture. It is our hope that this epidemiological association will serve as an important adjunct to our understanding of rupture of the posterior tibial tendon.

REFERENCES

1. **Anzel, S.H., Covey, K.W., Weiner, A.D., and Lipscomb, P.R.:** Disruption of muscles and tendons. An analysis of 1,014 cases. Surgery, 45:406–414, 1959.
2. **Balasubramanian, P., and Prathap, K.:** The effect of injection of hydrocortisone into rabbit calcaneal tendons. J. Bone Joint Surg., 54(B):729–734, 1972.
3. **Beeson, P.B., and McDermott, W.:** Textbook of Medicine. Philadelphia, W.B. Saunders, pp. 981–994, 1375–1386, 1599–1618, 1975.
4. **Blumenthal, H.T., Goldenberg, S., and Berns, A.W.:** Pathology and pathogenesis of the disseminated angiopathy of diabetes mellitus: on the nature and treatment of diabetes. In International Congressional Series, no. 84. Leibel, B.S., and Wrenshall, G.A. (eds.), Amsterdam, Excerpta Medica, pp. 397–408, 1965.
5. **Bogardus, C., Lillioja, S., Mott, D., Reaven, G.R., Kashiwagi, A., and Foley, J.:** Relationship between obesity and maximal insulin-stimulated glucose uptake in vivo and in vitro in Pima Indians. J. Clin Invest., 73:800–805, 1984.
6. **Bonadonna, R., Groop, L., Kraemer, N., and DeFronzo, R.A.:** Obesity and insulin resistance in man: a dose response study. Metabolism, 39:452–459, 1990.
7. **Bray, G.A. (ed.):** Overview of obesity in America. Bethesda, MD, National Institutes of Health, 1980.
8. **Butler, W.J., Ostrander, L.D., Carman, W.J., and Lamphiear, D.E.:** Diabetes mellitus in Tecumseh, Michigan: prevalence, incidence and associated conditions. Am. J. Epidemiol., 116:971–980, 1982.
9. **Colville, J., Callison, J.R., and White, W.L.:** Role of mesotenon in blood supply. Plast. Reconstr. Surg., 43:53–60, 1969.
10. **Craddock, D.:** Obesity and its management, 3rd Ed. Edinburgh, Churchill Livingstone, pp. 2–3, 1970.
11. **DeFronzo, R.A.:** Lilly lecture 1987: The triumvirate: B-cell, muscle, liver: a collusion responsible for NIDDM. Diabetes, 37:667–687, 1988.
12. **DeFronzo, R.A., and Ferrannini, E.:** Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care, 14(3):173–194, 1991.
13. **DeFronzo, R.A., Jacot, E., Jequier, E., Maeder, E., Wahren, J., and Felber, J.P.:** The effect of insulin on the disposal of intravenous glucose: results of indirect calorimetry and hepatic and femoral venous catherization. Diabetes, 30:1000–1007, 1981.
14. **DeFronzo, R.A., Sherwin, R.S., Hendler, R., and Felig, P.:** Insulin binding to monocytes and insulin action in human obesity, starvation, and refeeding. J. Clin. Invest., 62:204–213, 1978.
15. **Dunn, O.J.:** Basic statistics: a primer for the biomedical sciences, 2nd Ed. New York, John Wiley & Sons, pp. 122–135, 1977.
16. **Felber, J-P., Ferrannini, E., Golay, A., Meyer, H.U., Theiband, D., Curchod, B., Maeder, E., Jequier, E., and DeFronzo, R.A.:** Role of lipid oxidation in pathogenesis of insulin resistance of obesity and type II diabetes. Diabetes, 36:1341–1350, 1987.
17. **Folkow, B.:** The hemodynamic consequences of adaptive structural changes of the resistance vessels in hypertension. Clin. Sci., 41:1–12, 1971.
18. **Frey, C.C., Greenidge, N.C., and Shereff, M.:** Vascularity of the posterior tibial tendon. J. Bone Joint Surg., 72(A):884–888, 1990.
19. **Funk, D.A., Cass, J.A., and Johnson, K.A.:** Acquired flat-foot secondary to posterior tibial tendon pathology. J. Bone Joint Surg., 68(A):95–102, 1986.
20. **Golay, A., Felber, J.P., Jequier, E., DeFronzo, R.A., and Ferrannini, E.:** Metabolic basis of obesity and noninsulin dependent diabetes mellitus. Diabetes Metab. Rev., 4:727–747, 1988.
21. **Goldner, J.L., Keates, P.K., Bassett, F.H., and Clippinger, F.W.:** Progressive talipes equinovarus due to trauma or degeneration of the posterior tibial tendon and medial plantar ligaments. Orthop. Clin. North Am., 5:39, 1974.
22. **Griffiths, J.C.:** Tendon injuries around the ankle. J. Bone Joint Surg., 47(B):686–689, 1965.
23. **Hamlin, C.R., and Kohn, R.R.:** Evidence for progressive, age-related structural changes in post-mature human collagen. Biochim. Biophys. Acta., 236:458–467, 1971.
24. **Hamlin, C.R., and Kohn, R.R.:** Determination of human chronological age by study of a collagen sample. Exp. Gerontol., 7:377–379, 1972.
25. **Hamlin, C.R., Kohn, R.R., and Luschin, J.H.:** Apparent accelerated aging of human collagen in diabetes mellitus. Diabetes, 24:902–904, 1975.
26. **Harris, M.I.:** Prevalence of noninsulin-dependent diabetes and impaired glucose tolerance in *Diabetes in America*. Bethesda, MD, National Diabetes Data Group, Ch. VI, pp. 1–31, 1985.
27. **Havlik, R.J., Hubert, H.B., Fabsitz, R.R., and Feinleib, M.:** Weight and hypertension. Ann. Intern. Med., 98:855–859, 1983.
28. **Horan, M.J.:** Diabetes and hypertension in *Diabetes in America*. Bethesda, MD, National Diabetes Data Group, Ch. XVII, pp. 1–22, 1985.
29. **Ismail, A.M., Balakrishnan, R., and Rajakumar, M.K.:** Rupture of patellar ligament after steroid infiltration. A report of a case. J. Bone Joint Surg., 51(B):503–505, 1969.
30. **Jahss, M.H.:** Spontaneous rupture of the tibialis posterior tendon: clinical findings. Tenographic studies and a new technique of repair (Abstract). Foot Ankle, 3:158–166, 1982.
31. **Jahss, M.H.:** The pathology, anatomy, and management of degenerative changes of the tibialis posterior tendon. Foot Ankle, 8:113, 1987.
32. **Jarrett, R.J., Keen, H., McCartney, M., Fuller, J.H., Hamilton, P.J.S., Reid, D.D., and Rose, G.:** Glucose tolerance and blood pressure in two population samples: their relation to diabetes mellitus and hypertension. Int. J. Epidemiol., 7:15–24, 1978.
33. **Johnson, K.A.:** Tibialis posterior tendon rupture. Clin. Orthop., 177:140–147, 1983.
34. **Kennedy, J.C., and Willis, R.B.:** The effects of local steroid injections on tendons: a biomechanical and microscopic correlative study. Am. J. Sports Med., 4(1):11–21, 1976.
35. **Kettlekamp, D.B., and Alexander, H.H.:** Spontaneous rupture of the posterior tibial tendon. J. Bone Joint Surg., 51(A):759–764, 1969.
36. **Key, J.A.:** Partial rupture of the tendon of the posterior tibial muscle. J. Bone Joint Surg., 35(A):1006–1008, 1953.
37. **Kolterman, O.G., Insel, J., Saekow, M., and Olefsky, J.M.:** Mechanism of insulin resistance in human obesity: evidence for receptor and post-receptor defects. J. Clin. Invest., 65:1273–1284, 1980.
38. **Kuller, L., and Tonascia, S.:** A follow-up study of the commission on chronic illness morbidity in Baltimore: IV. Factors influencing morbidity from stroke and arteriosclerotic heart disease (1954–1967). J. Chronic Dis., 24:111, 1971.
39. **Kulowski, J.:** Tenovaginitis (tenosynovitis): General discussion and report of one case involving the posterior tibial tendon. J. Missouri Med. Assoc., 33:135, 1936.
40. **Lee, H.B.:** Avulsion and rupture of the tendon calcaneus after injection of hydrocortisone. Br. Med. J., 2:395, 1957.

41. **Lovell, A.G.H., and Tanner, H.H.:** Synovial membranes, with special reference to those related to the tendons of the foot and ankle. J. Anat., 42:414–423, 1908.
42. **McMaster, P.E.:** Tendon and muscle ruptures. Clinical and experimental studies on the causes and location of subcutaneous ruptures. J. Bone Joint Surg., 15:705–722, 1933.
43. **Manicardi, V., Camellini, L., Bellodi, G., Coscelli, C., and Ferrannini, E.:** Evidence for an association of high blood pressure and hyperinsulinemia in obese man. J. Clin. Endocrinol. Metab., 62:1302–1304, 1986.
44. **Mann, R.A., and Thompson, F.M.:** Rupture of the posterior tibial tendon causing flat foot. Surgical treatment. J. Bone Joint Surg., 67(A):556–561, 1985.
45. **Meimed, E.P.:** Spontaneous bilateral rupture of the calcaneal tendon during steroid therapy. J. Bone Joint Surg., 47(B):104–105, 1965.
46. **Modan, M., Halkin, H., Almog, S., Lusky, A., Eskol, A., Shefi, M., Shitrit, A., and Fuchs, I.:** Hyperinsulinemia: a link between hypertension, obesity, and glucose intolerance. J. Clin. Invest., 75:809–817, 1985.
47. **Pfeffinger, L.L., and Mann, R.A.:** Sesamoid and Accessory Bones in Surgery of the Foot. 5th Ed. Mann, R.A. (ed.). St. Louis, C.V. Mosby, pp: 209–229, 1986.
48. **Scheller, A.D., Kasser, J.R., and Quigley, T.B.:** Tendon injuries about the ankle. Orthop. Clin. North. Am., 2:801–811, 1980.
49. **Smith, J.W.:** Blood supply of tendons. Am. J. Surg., 109:272–276, 1965.
50. **Smith, J.W., and Conway, H.:** La dynamique du glissement des tendons normaux et greffes. Rev. Chir. Orthop., 52:185–196, 1966.
51. **Stamler, J., Wentworth, D., and Neaton, J.D.:** Serum cholesterol: is the relationship between cholesterol and risk of premature death from coronary heart disease continuous and graded? J.A.M.A., 256:2835–2838, 1986.
52. **Streeten, D.H.P.:** Corticosteroid therapy. II. Complications and therapeutic indications. J.A.M.A., 232:1046–1049, 1975.
53. **Sutton, R.D., Benedeck, T.G., and Edwards, G.A.:** Aseptic bone necrosis and corticosteroid therapy. Arch. Int. Med., 112:594–602, 1963.
54. **Turner, R.C.:** United Kingdom prospective diabetes study. III. Prevalence of hypertension and hypotensive therapy in patients with newly diagnosed diabetes. Hypertension, 7:(Suppl. II)8–13, 1985.
55. **Unverferth, L.J., and Olix, M.L.:** The effect of local steroid injections on tendon. J. Sports Med., 4:31–37, 1973.
56. **Warrenfeltz, A., and Graham, W.P.:** Avulsion injuries in patients receiving corticosteroids. Am. Fam. Physician, 12:74, 1975.
57. **Weidmann, P., and Ferrari, P.:** Central role of sodium in hypertension in diabetic subjects. Diabetes Care, 14(3):220–232, 1991.
58. **Williams, P.L., and Corbett, M.:** Avascular necrosis complicating corticosteroid replacement therapy. Ann. Rheum. Dis., 42:276–279, 1983.