International Journal of Impotence Research (2006) 18, 111–114

0955-9930/06 $30.00
www.nature.com/ijir

BRIEF REPORT

Prevalence of erectile dysfunction in thyroid disorders: comparison with control subjects and with obese and diabetic patients

A Veronelli, A Masu, R Ranieri, C Rognoni, M Laneri and AE Pontiroli

Medicina interna, Università degli studi di Milano, Ospedale San Paolo, Milano, Italy

Diagnosis of erectile dysfunction (ED) requires anamnestic investigation, being rarely spontaneously declared by patients. ED occurs frequently in diabetes mellitus, and anecdotal evidence suggests that ED occurs in obesity and in hypothyroidism. The aim of this study was to evaluate the prevalence of ED in patients affected by thyroid disorders (hypothyroidism and hyperthyroidism), in comparison with control subjects and with patients at risk for ED, such as patients with obesity and with type II diabetes mellitus, and the role of age. Spontaneous deposition and International Index of Erectile Dysfunction (IIEF)-5 questionnaire were considered for control subjects and for all patients. Spontaneous deposition of ED occurred for three diabetic patients, never for obese patients, thyroid patients and controls, confirming the value of IIEF-5 in detecting ED. ED was more frequent in obese subjects (42%), and in patients affected by thyroid diseases (59%), than in controls (30%), although less frequent than in type II diabetes mellitus (81%). Both below and above the age of 50 years, ED score was worse in thyroid patients than in control subjects, while ED was more frequent in obese patients than in control subjects only below the age of 50 years.
International Journal of Impotence Research (2006) **18,** 111–114. doi:10.1038/sj.ijir.3901364; published online 4 August 2005

Keywords: erectile dysfunction; thyroid; diabetes mellitus; obesity; age; quality of life

Introduction

Erectile dysfunction (ED) is defined as the inability to achieve and maintain an erection sufficient to permit satisfactory sexual intercourse.[1] ED may be related to age,[2–7] to a series of systemic diseases such as diabetes mellitus, atherosclerosis, cardiac ischemic disease, arterial hypertension,[5,8–12] central and autonomic neuropathy, kidney and liver failure, anxiety and depression, or may be determined by drugs, surgical intervention and traumatisms, alcoholism and tabagism.[13] The prevalence of ED in diabetic patients has been exhaustively investigated and has been reported to be from 25 to 75%.[14–18] In Italy in a 2.8 years follow-up study, the incidence was 68 new cases/1000 patients/year towards 25.9 new cases/1000 patients/years found in the general population.[12,19] Fewer data are available about the relationship between ED and endocrine diseases. Androgen deficiency decreases nocturnal erections and libido,[20] and hyperprolactinemia causes ED.[21] Obesity is also associated with high risk of ED.[22] Up to date, there are no epidemiological data on ED in thyroid diseases, even though anecdotal evidence suggests a possible link between hypothyroidism and ED.[23,24]

Diagnosis of ED requires an accurate anamnestic investigation, being rarely spontaneously declared by patients, and is usually detected through *ad hoc* questionnaires (as the International Index of Erectile Dysfunction, IIEF-5);[25] IIEF-5 also allows categorization of the degree of ED.[7]

The aim of this study was to evaluate the prevalence of ED in patients affected by thyroid disorders (hypothyroidism and hyperthyroidism) in comparison with control subjects and with patients at risk for ED, such as patients with obesity and with diabetes mellitus, and the role of age; to do so, spontaneous deposition and a IIEF-5 questionnaire were considered for control subjects and for all patients.

Correspondence: Professor AE Pontiroli, Medicina interna, Università degli studi di Milano, Via a di rudini'8, H San Paolo, Milano 20142, Italy.
E-mail: antonio.pontiroli@unimi.it
Received 2 November 2004; revised 16 May 2005; accepted 22 May 2005; published online 4 August 2005

Materials and methods

The protocol of the study was approved by the local Ethics Committee. For each group of patients, who were on a regular follow-up, spontaneous deposition of ED was recorded during outpatient visits, that is, during history collection. Then, an IIEF-5[25] was administered to all subjects accepting to do so. After completion of the questionnaire, ED was scored as follows: no ED for score above 20; mild ED for score 16–20; moderate ED for score 11–15; and severe ED for score 10 and less.[7] Controls were from the medical staff and the administrative staff of Ospedale San Paolo, in good health, and free from medications for any current medical condition. Table 1 shows the clinical and metabolic details investigated for each group of subjects, the mean value and the relevant limits observed.

Calculations and statistical analysis

ED degrees were expressed as absolute frequencies, and compared among all groups and subgroups by χ^2 test. Age was expressed as mean $\pm$ s.e. Subjects and controls were subdivided according to age into two groups (<50 and >50 years). Pairwise regression analysis was performed between continuous variables and ED score. To further characterize the independent contribution of age and kind of disease, a multiple regression analysis was performed with variables statistically significant at univariate regression, plus age and kind of disease.

Results

Spontaneous deposition of ED occurred for three diabetic patients, never for obese patients, thyroid patients and controls. Therefore, the value of IIEF-5 was highly significant for both controls and obese patients, thyroid patients and diabetic patients (Figure 1). Figure 1 also shows that ED score (all ages) was better in controls than in thyroid patients and in diabetic patients, in obese patients than in diabetic patients and in thyroid patients than in diabetic patients, with no other significant differences. Figure 2a shows that in the age group 23–50 years, ED score was worse in obese patients than in

Table 1 Clinical and metabolic details of patients and controls in the study (absolute numbers, means $\pm$ s.e.)

Subjects	*N*	*Age (<50 years)*	*Age (>50 years)*	*Subgroups*	*Age limits*	*Relevant details and limits*
Controls	109	40.5 $\pm$ 1.19	61.9 $\pm$ 0.75 b		30–76	
Obese	45	36.5 $\pm$ 1.24 a	60.2 $\pm$ 1.66 c		23–74	BMI 31.6 (25.3–47)
Thyroid	68	41.6 $\pm$ 1.22	66.5 $\pm$ 1.02 d	Hypothyroid (55)	35–81	TSH 13.7 (0.01–106), ab 26 neg., 27 pos.
				Hyperthyroid (13)	36–78	TSH 0.5 (0.01–3.06), ab 6 neg., 7 pos.
Type II DM	52	43.8 $\pm$ 2.51	62.2 $\pm$ 0.89		35–75	$Hba1_c$ 7.5 (5.9–11.0), 6 DR, 1 CHD, 1 DN

DR = retinopathy (background); CHD = coronary heart disease; DN = diabetic neuropathy.
a = $P<0.05$ vs diabetes mellitus; b = $P<0.01$ vs thyroid; c = $P<0.05$ vs thyroid; d = $P<0.05$ vs diabetes.

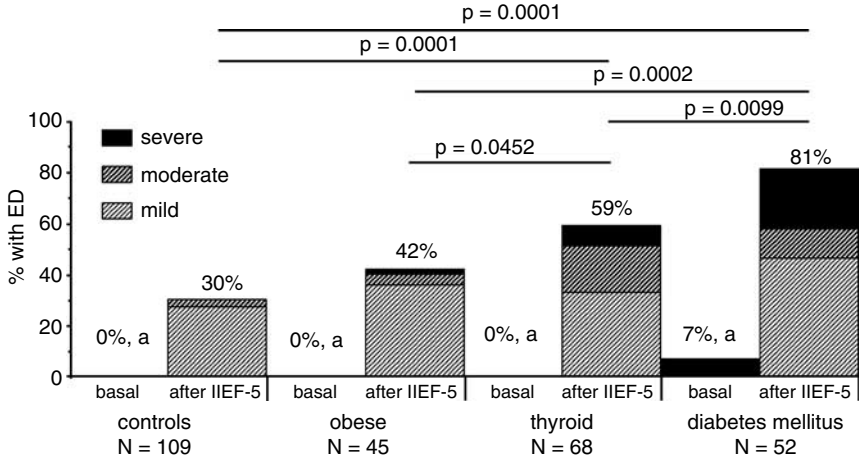

Figure 1 Prevalence of ED in control subjects and in patients with obesity, thyroid disorders and diabetes mellitus at spontaneous declaration (basal) and after filling an IIEF-5 questionnaire (after IIEF-5). Numbers on top of columns indicate the total prevalence of ED (any degree). a = $P = 0.0001$ basal vs after IIEF-5.

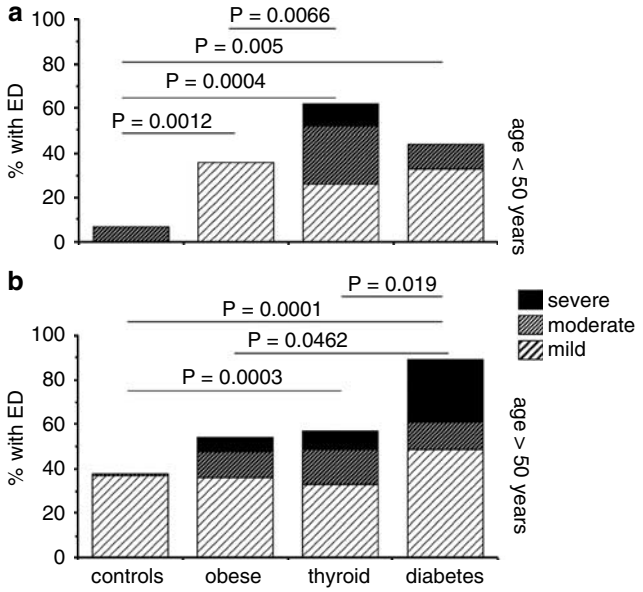

Figure 2 Prevalence of ED in control subjects and in patients with obesity, thyroid disorders, and diabetes mellitus (mild, moderate and severe ED) according to age (**a**: age <50 years; **b**: age >50 years). Subjects without ED are not shown.

controls, and better than in thyroid patients; in addition, ED score was worse in thyroid patients and in diabetic patients than in controls. It can be concluded that in this age group, obese patients are particularly at risk of ED. Figure 2b shows that in the age group 51–80 years, ED score was significantly better in controls than in thyroid patients and in diabetic patients; thyroid patients and obese patients also differed from diabetic patients. Table 2 shows that both kinds of thyroid disease (hypothyroidism and hyperthyroidism) are accompanied by a significant difference of ED score as compared to controls.

ED score was not associated, in diabetic patients, with the kind of treatment, and metabolic control (not shown); similarly, in thyroid patients, ED score was not associated with duration of thyroid disease, presence of thyroid antibodies, TSH levels, kind of treatment and other medical conditions (not shown). In controls and in diabetic patients, age (<50 vs >50 years) was associated with worsening of ED score, not in obese patients or in thyroid patients; at multiple regression analysis, ED score was associated with the presence of thyroid disease, obesity and diabetes mellitus ($F = 62.44$), and with age of patients ($F = 7.74$).

Discussion

Our data confirm that the use of an IIEF-5 questionnaire[24] allows detection of ED in a significant proportion of control subjects as well as of diabetic patients, obese patients and patients with thyroid disorders; without the use of a questionnaire, the great majority of these subjects would not spontaneously declare ED.

Table 2 ED score in controls and in thyroid patients according to the kind of thyroid disease, all ages (and age groups <50 years plus >50 years)

Score	*Controls*	*Hypothyroid*	*Hyperthyroid*
Severe	0 (0 + 0)	4 (0 + 4)	2 (2 + 0)
Moderate	3 (3 + 2)	11 (4 + 7)	2 (1 + 1)
Mild	30 (0 + 30)	15 (3 + 12)	6 (2 + 4)
No	76 (26 + 50)	25 (5 + 20)	3 (2 + 1)

Comparisons are between patients of all ages (in parentheses between patients of the two age groups).
Controls vs hypothyroid $\chi^2 = 25.79$, $P = 0.0001$ ($\chi^2 = 13.68$, $P = 0.0001$; $\chi^2 = 19.23$, $P = 0.0011$).
Controls vs hyperthyroid $\chi^2 = 10.84$, $P = 0.0043$ ($\chi^2 = 19.23$, $P = 0.0002$; $\chi^2 = 8.98$, $P = 0.0112$).

Our data show that ED is a frequent finding in patients affected by thyroid diseases, be it patients with hypothyroidism or hyperthyroidism. The prevalence is, however, in general lower than in diabetic patients. The nature of the association between thyroid disorders and ED is a matter of speculation, as we found no association between ED and kind of disease, duration of disease, presence or absence of thyroid antibodies, kind of treatment and TSH levels. Therefore, we cannot put forward any interpretation, and we cannot exclude a mere psychological reason, justified by concern of patients for their own thyroid disease. Of interest is also the fact that aging was not associated, in these patients, with a significant worsening of ED score, indicating that thyroid problems are more important than the aging process in determining the appearance of ED. One limit to this study is that it was cross-sectional, and therefore prospective studies are required to assess the effect of diseases themselves and of substitution therapy in hypothyroid patients as well as the effect of thionamides and of other drugs in hyperthyroid patients.

In diabetic patients, we found a high prevalence of ED; this agrees with previous data indicating that diabetes mellitus carries a very high risk of ED.[12–19] However, probably due to the small sample size of our diabetic patients, all with type II diabetes, we could find no relationship between ED, $HbA1_c$ levels, presence of complications, treatment with oral agents or with insulin, although ED score worsened with age, while it is known, from larger studies, that ED is more frequent in patients with longer duration of diabetes, and with poor metabolic control.[12,19,26]

Obesity, as already reported,[22] is frequently associated with ED; it has to be underlined, however, that ED was mainly of mild degree, and this

has never been reported so far; an additional novel finding is that ED appears quite early in life, as before the age of 50 years, it carries a high risk of ED, higher than in control subjects, although lower than in thyroid patients. The reason for this early appearance of ED is a matter for conjecture; we would only like to remember that obesity, similar to diabetes, is characterized by endothelial dysfunction,[27] a basic mechanism for ED.[9,10] Recently, it was shown that weight loss is accompanied by an improvement of endothelial dysfunction, and by improvement of ED.[28]

In conclusion, this paper emphasizes the utility of structured interviews for detection of ED in normal subjects and in various kinds of patients, confirming the frequent occurrence of ED in diabetic and obese patients, and indicates that ED is a frequent finding in patients with thyroid disorders. Further research is required to understand the nature of the link between thyroid disorders and ED, for instance, by looking at endothelial function in thyroid disorders.

References

1 NIH Consensus Development Panel on Impotence. NIH consensus conference: impotence. *JAMA* 1993; **270**: 83–90.
2 Bortolotti A, Perazzini F, Colli E, Landoni M. The epidemiology of erectile dysfunction and its risk factors. *Int J Androl* 1997; **20**: 323–334.
3 Benet AE, Melman A. The epidemiology of erectile dysfunction. *Urol Clin N Am* 1995; **22**: 699–709.
4 Mulligan T, Retchin SM, Chinchilli VM, Bettinger CB. The role of aging and chronic disease in sexual dysfunction. *J Am Geriatr Soc* 1988; **36**: 520–524.
5 Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: results of the Massachussetts Male Aging Study. *J Urol* 1994; **151**: 54–61.
6 Mak R, De Baker G, Kornitzer M, De Meyer JM. Prevalence and correlates of erectile dysfunction in a population-based study in Belgium. *Eur Urol* 2002; **41**: 132–138.
7 Giuliano F, Chevret-Measson M, Tsatsaris A, Reitz C, Murino M, Thonneau P. Prevalence of erectile dysfunction in France: results of an epidemiological survey of a representative sample of 1004 men. *Eur Urol* 2002; **42**: 382–389.
8 Furlow WL. Prevalence of impotence in the United States. *Med Asp Hum Sex* 1985; **19**: 13–16.
9 Virag R, Bouilly P, Frydman D. Is impotence an arterial disorder? A study of arterial risk factors in 440 impotent men. *Lancet* 1985; **26**: 181–184.
10 Wabrek AJ, Burchell RC. Male sexual dysfunction associated with coronary heart disease. *Arch Sex Behav* 1980; **9**: 69–75.
11 Bansal S. Sexual dysfunction in hypertensive men. *Hypertension* 1988; **12**: 1–10.
12 Fedele D, Bortolotti A, Coscelli C, Santeusanio F, Chatenoud L, Colli E *et al*. Gruppo Italiano Deficit Erettile Diabetici. Erectile dysfunction in type 1 and type 2 diabetics in Italy. *Int J Epidemiol* 2000; **29**: 524–531.
13 Lue TF. Erectile dysfunction. *N Engl J Med* 2000; **342**: 1802–1813.
14 McCulloch DK, Campbell IW, Wu FC, Prescott RJ, Clarke BF. The prevalence of diabetic impotence. *Diabetologia* 1980; **18**: 279–283.
15 Rubin A, Babbott B. Impotence and diabetes mellitus. *JAMA* 1958; **168**: 498–500.
16 Brunner GA, Pieber TR, Schattenberg S, Ressi G, Wieselmann G, Altziebler S *et al*. Erectile dysfunction in patients with type I diabetes mellitus. *Wien Med Wochenschr* 1995; **145**: 584–586.
17 Klein R, Klein BE, Lee KE, Moss SE, Cruickshanks KJ. Prevalence of self-reported erectile dysfunction in people with long term IDDM. *Diabetes Care* 1996; **19**: 135–141.
18 Johannes CB, Araujo AB, Feldman HA, Derby CA, Kleinman KP, McKinlay JB. Incidence of erectile dysfunction in men 40 to 69 years old: longitudinal results from the Massachusetts Male Aging Study. *J Urol* 2000; **163**: 460–463.
19 Fedele D, Coscelli C, Cucinotta D, Forti G, Santeusanio F, Viaggi S *et al*. Incidence of erectile dysfunction in Italian men with diabetes. *J Urol* 2001; **166**: 1368–1371.
20 Bancroft J, Wu FC. Changes in erectile responsiveness during androgen replacement therapy. *Arch Sex Behav* 1983; **12**: 59–66.
21 Buvat J, Lemaire A. Endocrine screening in 1,022 men with erectile dysfunction: clinical significance and cost-effective strategy. *J Urol* 1997; **158**: 1768–1769.
22 Bacon CG, Mittleman MA, Kawachi I, Giovannucci E, Glasser DB, Rimm EB. Sexual function in men older than 50 years of age: results from the health professionals follow-up study. *Ann Int Med* 2003; **139**: 161–168.
23 Baskin HJ. Endocrinologic evaluation of impotence. *South Med J* 1989; **82**: 446–449.
24 Bodie J, Lewis J, Schow D, Monga M. Laboratory evaluation of erectile dysfunction: an evidence based approach. *J Urol* 2003; **169**: 2262–2264.
25 Rosen RC, Riley A, Wagner G, Osterloh IH, Kirkpatrick J, Mishra A. The international index of erectile dysfunction (IIEF): A multidimensional scale for assessment of erectile dysfunction. *Urology* 1997; **49**: 822–830.
26 Fonseca V, Seftel A, Denne J, Fredlund P. Impact of diabetes mellitus on the severity of erectile dysfunction and response to treatment: analysis of data from tadalafil clinical trials. *Diabetologia* 2004; **47**: 1914–1923.
27 Pontiroli AE, Pizzocri P, Koprivec D, Vedani P, Marchi M, Arcelloni C *et al*. Body weight and glucose metabolism have a different effect on circulating levels of ICAM-1, E-selectin, and endothelin-1 in humans. *Eur J Endocrinol* 2004; **150**: 195–200.
28 Esposito K, Giugliano F, Di Palo C, Giugliano G, Marfella R, D'Andrea F *et al*. Effect of lifestyle changes on erectile dysfunction in obese men: a randomized controlled trial. *JAMA* 2004; **291**: 2978–2984.