Erectile Dysfunction Associated with Scleroderma: A Case-Control Study of Men with Scleroderma and Rheumatoid Arthritis

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ABSTRACT. ***Objective.*** To determine if men with systemic sclerosis (SSc) are at increased risk of developing erectile dysfunction (ED) compared to men with rheumatoid arthritis (RA), and to investigate the temporal relationship of ED related to rheumatologic disease.

Methods. Men with SSc identified from the practices of 2 rheumatologists were age matched to men with RA and were sent a standardized, validated questionnaire (SHIM IIEF-5) to assess ED and related factors. The questionnaire also addressed information on the subject's overall health and rheumatic disease status.

Results. The response rate was 50% (48% in SSc and 55% in RA), thus 43 with SSc and 23 with RA were included. The mean age of respondents was 53 yrs ± 1.34 (SEM), (range 34 to 83). No statistical differences were found for marital status, alcohol or drug use, or past/present smoking. Men with scleroderma weighed less than men with RA ($p < 0.004$) and were more likely to have Raynaud's phenomenon ($p < 0.0001$), and to have fewer biological children (2.0 ± 0.2 vs 2.7 ± 0.2, $p < 0.01$). The prevalence of erectile dysfunction was 81% (SSc) and 48% (RA), (relative risk for SSc vs RA: 4.77; 95% CI: 1.55, 14.66; $p < 0.005$). In subjects who had ED, 78% (both SSc and RA) reported it occurring after disease onset. Men with SSc noted their ED began 2.7 ± 1.2 (mean ± SEM) years after their disease was diagnosed, and similarly, men with RA noted their ED began 3.3 ± 2.2 years after disease diagnosis, $p = 0.82$. Eighty-six percent of patients with SSc had Raynaud's phenomenon (RP) compared to 19% RA, $p < 0.0001$. Eighty percent of subjects with RP (SSc + RA) had ED versus 50% of men without RP, $p < 0.01$. In RA subjects with RP ($n = 4$), 75% had experienced ED, versus 39% of RA without RP, $p = 0.18$. Possible confounding factors for ED were examined including smoking, hypertension, diabetes, and steroid use; all except self-reported history of nerve damage ($p < 0.0005$) and diabetes ($p < 0.02$) were insignificant for predicting the likelihood of increased ED. Patients with SSc were not more likely than RA to have experienced nerve damage ($p = 0.25$), or diabetes ($p = 0.19$).

Conclusion. ED occurs frequently in SSc, is more common than in RA, and occurs on average 3 years after disease onset. RP appears to be associated with ED in both SSc and RA, but is not necessarily an independent risk factor for ED in SSc alone. (J Rheumatol 2004;31:508–13)

Key Indexing Terms:
ERECTILE DYSFUNCTION SYSTEMIC SCLEROSIS RHEUMATOID ARTHRITIS

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Scleroderma (SSc) is a chronic autoimmune disease associated with abnormalities in the blood vessels and fibrosis often with vasculopathy of multiple organs. Erectile dysfunction (ED) may be quite prevalent among men with chronic illness, but it is often difficult to ascertain a direct pathologic causal relationship due to the many psychosocial and comorbid factors (commonly found with chronic illness) that can also contribute to ED. However, ED may occur more frequently in men with SSc than in other chronic diseases and is likely to have an organic basis in SSc.

Studies have reported the prevalence of ED in SSc as ranging from 12% to 80%[1-6]. The exact frequency of ED in men with SSc compared to men with other chronic disease and timing of ED in the disease course have not been studied in a large series of patients. One report that assessed sexual

function in patients with chronic illness (diabetes, angina pectoris, glaucoma) compared to a random population sample found that ED was increased among patients with diabetes (30%) and angina pectoris (29%) compared to the population controls, $p < 0.001$[7]. The time between the onset of ED with respect to disease symptoms was not assessed[7–9]. Several other studies of ED in SSc have examined the temporal relationship between the onset of erectile problems and diagnosis (or disease symptoms), but these were small studies or case histories, many of which found that ED is common in SSc, especially early in the disease[1-5].

Researchers have shown that ED in SSc patients is organic and not psychogenic[2-4,6]. Several vascular, fibrotic, or neurogenic factors have been suggested in the pathogenesis of ED in SSc, but the pathophysiologic mechanisms have not been clearly defined[1,2,10]. Some studies have reported that total testosterone and prolactin levels in blood are correlated with ED in men with SSc[1,2]. However others have not found this[1-5,10,11]. Nowlin, *et al* suggested that in some SSc patients, small artery lesions may be responsible for ED secondary to SSc[1]. We postulate that ED may also be associated with other vascular phenomena, such as Raynaud's phenomenon (RP), independent of vascular disease.

Our objectives were (1) to assess the prevalence of ED in a relatively large SSc sample compared to a control population of men with rheumatoid arthritis (RA); (2) to investigate the possible relationship of ED to other comorbidity (or demographic factors); and (3) to examine the temporal relationship between SSc diagnosis/symptom onset and the occurrence of ED. We hypothesized that there would be a stronger association between SSc and ED compared to RA and that ED may be a presenting (or early) symptom of SSc.

MATERIALS AND METHODS

Approval for the study was obtained from both the University of Western Ontario Health Sciences Research Ethics Board and the University of Medicine and Dentistry of New Jersey - Robert Wood Johnson Medical School Institutional Review Board. Men with a diagnosis of systemic sclerosis (n = 16), who met the American College of Rheumatology (ACR) preliminary criteria for SSc[12] were identified from the outpatient practice database at St. Joseph's Health Care London, a referral center for SSc. An additional cohort was used to identify cases with SSc that met the study criteria (n = 73) from an American SSc referral center. Controls included men diagnosed with RA, meeting ACR criteria[13] and age matched within 2 years of the SSc patients (cases). They were selected from the Canadian practice. We chose RA as a chronic autoimmune disease for the control group, as there is pain, loss of mobility, possible employment loss, and the need to take medications. We thought men with chronic inflammatory arthritis would serve as an adequate comparison group. Demographic and clinical variables were analyzed for differences between the American and Canadian SSc cohorts to ensure that the Canadian RA population could be used as a representative control group for both American and Canadian men. Clinical variables were analyzed between respondents versus non-respondents and cases (SSc) versus controls (RA) to ensure that disease severity was comparable.

All patients with SSc and RA were mailed a confidential questionnaire designed to assess ED and related factors, as well as the subject's overall health and factors related to the rheumatic disease. The questions to assess ED were derived from a standardized, validated, 5-item questionnaire, the Sexual Health Inventory for Men, also known as the International Index of Erectile Function (SHIM IIEF-5)[14]. ED was defined as the inability to achieve and maintain an erection (a standardized definition). The 3-page questionnaire (32 items) included current medications, followed by a brief demographics section assessing items such as smoking, drug and alcohol use, number of children, height, weight, disease onset, and comorbidity. Subjects were also asked to answer questions about their employment status, and relationship with their partner. Subsequent items addressed possible factors associated with ED, including fainting episodes, nerve damage (as defined in the questionnaire as numbness, tingling, or reduction of feeling in hands and/or feet), pelvic injury or radiation, and bike riding. Subjects were then asked about their difficulty achieving erections, how much they thought about ED, whether the change in their sexual function had been gradual or rapid, and the temporal relationship with respect to their disease onset. The final section addressed descriptive aspects of subjects' ED including any physical or sensation changes to the penis or testes. Some items required subjects to indicate which statement best described their confidence in maintaining erections as well as the difficulty and quality of intercourse. The non-respondents were re-sent the questionnaire. A chart review was conducted for all non-respondents to obtain information on baseline demographics as well as date of disease onset (diagnosis) and the presence of RP. Although not systematically collected, testosterone and prolactin levels were recorded from chart review where available.

Data collected from respondents were entered and analyzed in London, Ontario using JMP statistical software (SAS Institute, Cary, NC, USA). Descriptive and comparative statistics were done using chi-squared tests and t tests.

Demographic and clinical variables were not statistically different between the US and Canadian SSc cohorts, with the following exceptions: (1) 54% of American men had diffuse disease, compared to 89% of Canadian men ($p = 0.06$); (2) 80% of American men with SSc had RP versus 100% of Canadians ($p < 0.04$); and (3) Americans were also more likely to rate their relationship with their partner as good or fair (versus poor) than Canadians (100% vs 85%, $p < 0.03$). As the US and Canadian men with SSc seemed comparable, their data were pooled.

The following clinical measurements were recorded where available and compared between respondents and non-respondents: the Health Assessment Questionnaire (HAQ) score, number of disease modifying antirheumatic drugs (DMARD) currently taken, and current steroid dose. For respondents versus non-respondents in the subgroup of men with RA, HAQ scores (0.78 ± 0.2 vs 0.74 ± 0.2), current steroids (22% vs 7%), and mean number of DMARD (0.96 ± 0.2 vs 1.40 ± 0.2) were not different between groups. The same clinical measurements were compared between respondents versus non-respondents in the SSc subgroup. HAQ scores were only available for 3 non-respondents, but were not different (mean 1.38 ± 0.4) than the mean HAQ score for the respondents (0.94 ± 0.2), $p = 0.08$. The mean number of DMARD was not different in SSc respondents versus non-respondents ($p = 0.7$), and current steroids could not be compared due to low numbers in the non-respondents, resulting in an unstable p value. These results indicate that the respondents versus non-respondents and RA versus SSc patient samples used in this study were clinically comparable in their overall disease severity.

RESULTS

Eighty-nine men with SSc and 42 men with RA were mailed questionnaires. The overall response rate was 50%: 48% for SSc, with 13 (30%) of respondents from Canada, and 30 (70%) from the US; and 55% for RA (n = 23). Table 1 shows the comparison of baseline characteristics between the men with RA and those with SSc (US and Canadian pooled). As

Table 1. Demographic factors for subjects with scleroderma and RA (controls). Relative risk (RR) was calculated for men with SSc relative to men with RA.

Demographic Factors	Scleroderma (Cases)	RA (Controls)	p	RR	95% Confidence Interval for RR
Respondents, % (n)	48 (43)	55 (23)	0.45	0.76	(0.36, 1.58)
Age, mean ± SEM	52 ± 1.7	53 ± 2.3	0.89	—	—
Weight, kg, mean ± SEM (min, max)	79.0 ± 2.0 (59, 109)	89.4 ± 2.8 (66, 123)	0.004	—	—
Yrs of marriage, mean ± SEM (min, max)	24.2 ± 2.2 (0, 54)	24.2 ± 3.0 (0, 50)	0.99	—	—
Consider relationship to be fair or good, % (n/those in a relationship)	95 (37/39)	95 (19/20)	0.98	0.97	(0.83, 11.44)
Number of biological children, mean ± SEM (min, max)	2.0 ± 0.2 (0, 4)	2.7 ± 0.2 (0, 5)	0.01	—	—
Currently employed, %	69	65	0.75	1.20	(0.39, 3.71)
Drug plan, %	87	67	0.06	3.30	(0.89, 12.19)
Alcohol consumption, mean oz/wk (min, max)	10.4 (0, 120)	4.9 (0, 24)	0.23	—	—
Smoking history, % positive	14	27	0.19	0.43	(0.12, 1.55)
Disease duration, yrs, mean ± SEM (min, max)	6.8 ± 1.1 (0.5, 29)	8.4 ± 1.5 (1, 25)	0.40	—	—
Raynaud's phenomenon, %	86	19	0.0001	27.0	(6.75, 107.96)
Hypertension, % positive	21	26	0.63	0.75	(0.23, 2.45)
Diabetes, % positive	14	4	0.20	3.57	(0.40, 31.62)
Steroids ever, %	40	43	0.86	0.91	(0.31, 2.62)
Nerve damage*, %	67	52	0.25	1.83	(0.65, 5.18)
Pelvic injury, %	2	9	0.23	0.25	(0.02, 2.92)
Pelvic radiation, %	9	4	0.48	2.15	(0.22, 20.53)
Ride a bike, %	16	18	0.85	0.88	(0.23, 3.38)
Fainting episodes Hx, %	7	9	0.80	0.79	(0.12, 5.09)

* Nerve damage appears to have been over-estimated by respondents.

expected, men with SSc were more likely to have RP (86% vs 19%, $p < 0.0001$). Mean weight (kg) ± SEM, was lower in men with SSc than in men with RA (79.0 ± 2.0 vs 89.4 ± 2.8, $p < 0.0004$), as was the mean number of biological children (2.0 ± 0.2 vs 2.7 ± 0.2, $p < 0.01$). Those with SSc were slightly more likely to have a drug plan (87% vs 67%, $p < 0.05$).

ED occurred in 81% of SSc versus 48% of RA men (Table 2), $p < 0.005$ and a change in sexual function was noticed by 84% of men with SSc compared to 59% of men with RA ($p < 0.03$); 22% thought the sexual function change was rapid (as opposed to gradual) in SSc versus 46% in RA ($p = 0.1$). The mean time period of reporting ED following disease onset was brief in each group, 2.7 ± 1.2 years in SSc versus 3.3 ± 2.2 years in RA, $p = 0.82$. For the majority of both men with SSc (77%) and men with RA (82%), their ED began after the time of diagnosis (ED did not predate their disease), $p = 0.74$. Interestingly, RP was associated with ED, as 80% of men with RP also had ED, while 50% of men without RP had ED [relative risk (RR) = 4.0, $p < 0.01$]. Men with RP also had fewer biological children than men without RP [(mean ± SEM) 2.0 ± 0.2 vs 2.6 ± 0.2, $p < 0.02$). The majority of SSc patients (86%) had RP, and 71% of SSc men versus 14% of RA reported both RP and ED, $p < 0.0001$. However, in a subset analysis of only men with SSc, 81% of those with RP reported having ED, while 83% of those without RP reported ED ($p = 0.9$).

HAQ scores (SSc 0.94 ± 0.2 vs RA 0.78 ± 0.2, $p = 0.6$) and current steroids (SSc 23% vs RA 22%, $p = 0.1$) were not statistically different between groups, but higher HAQ scores were seen in the SSc group. Not surprisingly, mean number of DMARD was increased among RA compared to SSc patients (1.0 ± 0.1 vs 0.3 ± 0.2), $p < 0.006$.

Possible confounders, such as smoking, weight, hypertension and other vascular disorders, and diabetes were examined. ED occurred more commonly in men who (1) also had diabetes (compared to those without diabetes; 100% vs 66%, $p < 0.02$), and who (2) reported having sustained nerve damage (compared to those who had not; 85% vs 44%, RR = 7.21, $p < 0.0005$). Several other factors showed an increased RR for ED, including smoking, hypertension, steroid use, pelvic radiation, fainting, riding a bike, having a self-employed or supervisory occupation, and rating personal relationship as fair or good; however, these were not significant. Other factors (employment status and past/present pelvic injury) were less commonly associated with ED. Results are summarized in Table 3.

We adjusted for potential confounding variables (diabetes, RP, and nerve damage) to determine what was statistically significant using logistic regression. In the unadjusted model, the prevalence of ED in SSc was significantly different than the prevalence of ED in RA, $p < 0.003$. The model gained statistical significance when adjusted for

Table 2. Raynaud's phenomenon (RP), erectile dysfunction (ED), and associated variables for subjects with scleroderma and RA (controls). Relative risk (RR) was calculated for men with SSc relative to men with RA.

ED Related Factors	SSc	RA (Controls)	p	RR	95% CI for RR
n	43	23	—	—	
Erectile dysfunction, %	81	48	0.005	4.77	(1.55, 14.66)
Noticed a change in sexual function, %	84	59	0.03	3.56	(1.10, 11.52)
Change in sexual function was rapid (vs gradual), %	22	46	0.10	0.33	(0.90, 1.28)
ED began after disease diagnosis, %	77	82	0.74	0.75	(0.13, 4.20)
Time from disease onset until impotence, yrs, mean ± SEM (min, max)	2.7 ± 1.2 (–12, 24)	3.3 ± 2.2 (–9, 21)	0.82	—	—
RP*, %	86	19	0.0001	27.0	(6.75, 107.96)
Men with both ED and RP, %	71	14	0.0001	15.83	(3.94, 63.54)
ED among patients with RP, %	81	75	0.80	1.38	(0.12, 15.36)
Time from disease onset until RP, yrs, mean ± SEM (min, max)	–0.9 ± 0.7 (–19, 7)	–1.0 ± 2.5 (–3, 0)	0.97	—	—

* Values were significantly different between American and Canadian men with SSc.

Table 3. Possible confounding factors for ED in all subjects (SSc and RA). The percentage who reported ED and were positive for the trait (out of total men positive for the trait with or without ED) is indicated and similar for those negative for the trait. Relative risk was calculated for men with ED and positive for the trait relative to men with ED and negative for the trait.

Possible Confounding Factors for ED	Men with ED and Positive for Factor	Men with ED and Negative for Factor	p	Relative Risk (RR)	95% CI for RR
Smoking (current), %	83	68	0.27	2.36	(0.46, 11.98)
Weight, kg* (mean ± SEM)	82.0 ± 2.1	84.0 ± 3.2*	0.60	—	—
Hypertension, %	80	67	0.32	2.00	(0.50, 8.05)
Diabetes**, %	100	66	0.02	—	—
RP, %	80	50	0.01	4.00	(1.31, 12.18)
Steroids (ever taken), %	77	68	0.41	1.60	(0.51, 5.02)
Currently employed, %	67	80	0.28	0.50	(0.14, 1.78)
Sustained nerve damage (ever), %	85	44	0.0005	7.21	(2.23, 23.32)
Past/present pelvic injury, %	67	70	0.91	0.86	(0.07, 10.11)
Pelvic radiation (ever)***, %	80	68	0.57	1.85	(0.19, 17.72)
Ride a bike, %	73	68	0.78	1.22	(0.29, 5.20)
Suffered fainting episodes (ever), %	80	69	0.60	1.81	(0.19, 17.30)
Years of marriage or stable relationship* (mean ± SEM)	26.1 ± 2.1	20.2 ± 3.1*	0.12	—	—
Rated personal relationship as fair or good (vs poor), %	70	67	0.91	1.15	(0.10, 13.52)

n = n (positive for factor with ED)/n (respondents positive for factor). * Weight and years of marriage were continuous variables, so subjects could not be positive or negative. Logistic fit analysis produced a p indicating that both were insignificant predictors of ED. In these cases, only the mean ± SEM for men without ED is indicated in the "Negative for factor" column. ** RR could not be calculated because all men with diabetes also reported ED (denominator = 0). *** Including radiographs.

diabetes ($p < 0.002$), nerve damage ($p < 0.0001$), and the combination of diabetes, nerve damage, and RP ($p < 0.0002$). When adjusting for RP alone, statistical significance persisted ($p < 0.012$).

Where available, hormone levels were extracted from the medical records of men with SSc (n = 11). The mean testosterone and prolactin levels fell into the low normal range for men of the same age group, with an average testosterone level of 10.9 ± 0.98 nmol/l (mean ± SEM), with individual levels ranging from 6.5-16.3 nmol/l, compared to the normal range of 9.1-55.0 nmol/l in males aged 20-49 years, and 6.3-26.0 nmol/l in males aged > 50 years. Two of 11 men with SSc tested for hormone levels (18%) had testosterone levels below the reference range for their age. The mean prolactin level in our men with SSc was mid-range normal, 9.89 ± 2.39 µg/l (mean ± SEM), with range 4.97-33 µg/l, compared to the normal range of 2-18 µg/l. One of 11 men tested (9%) had an elevated prolactin level. Thus, although data were not available for all subjects, of those for whom laboratory results were available, problems with testosterone or prolactin in the men with SSc were uncommon and did not solely account for ED.

DISCUSSION

Our results support findings in the literature that the prevalence of ED associated with SSc is 80%[1-6]. Men with SSc were almost 5 times as likely to have experienced ED than age-matched controls with RA ($p < 0.005$). We found the high prevalence of ED among SSc patients well above the American population norm, which the 1992 National Institutes of Health Consensus Statement reported as 5% at age 40, increasing to 15-25% at age 65 and older[15]. With respect to onset of ED compared to disease symptoms, others have suggested that ED may be a very early manifestation of SSc[5,11]. However, we found that it occurred on average after disease symptoms and diagnosis, with a mean time from diagnosis of SSc to development of ED symptoms of 2.7 years ± 1.2 (SEM). Thus, our findings suggest that the presence of erectile problems would not be useful in most cases for diagnosis of SSc.

RP may be a risk factor for ED, as men with RP were more likely to have ED compared to men without RP ($p < 0.01$) and results remained unchanged when statistically adjusted for disease (SSc or RA). It certainly appears that ED is related to RP secondary to SSc. However, in a subset analysis of only men with SSc, 81% of those with RP reported having ED, while 83% of those without RP reported ED ($p = 0.9$), thus, indicating that RP is not necessarily an independent risk factor for the development of ED in SSc. The prevalence of ED in the SSc cohort was high (approximately 80%) in those with and without RP. As only 6 SSc patients had no RP, we cannot conclude the absence of an association between RP and ED with certainty.

Interestingly, the trend towards RP associated with ED was present in the RA cohort, although numbers in this study were too small to allow specific conclusions (only 4 RA patients also had RP). Another group, van Basten, *et al*, showed in a study of the prevalence of sexual dysfunction following chemotherapy treatment for nonseminomatous testicular germ cell tumor that men with chemotherapy-induced RP were more likely to suffer from ED than those without RP (RR = 4.41, $p < 0.002$). Those with acral (saddle) parathesia from tumor resection did not have an increased risk of ED[16]. Altogether, these findings suggest that ED associated with RP may be primarily a vasculopathy; however, fibrosis could also play a role. We propose that future studies addressing the prevalence of ED in men with primary Raynaud's disease are warranted, as they have a vasospastic disorder, not a chronic illness.

The cause of ED associated with SSc does not appear to be associated with low testosterone or elevated prolactin levels. In our study, there was a very small proportion of men taking testosterone treatment, and in whom no improvement of ED was observed with testosterone replacement. An autonomic neuropathic process could be the causal factor for ED. However, we do not believe that to be the case, as the self-reports of nerve damage in our subjects were extremely high and probably overrated (the question regarding nerve damage asked if the patient had ever experienced a sensation of pins and needles with or without a diagnosis from a physician). In addition, compression neuropathies such as carpal tunnel syndrome are common in SSc and RA, but this is not necessarily true of peripheral neuropathy from other causes. Three other groups have reported isolated cases of ED occurring in conjunction with severe secondary RP[17-19]. The complaints of ED in SSc seem not to be psychogenic (69% of SSc subjects with ED also reported they did not have night or morning erections) and are well above the expected prevalence in another chronic disease (RA). It should be noted that of those men who had ED, a sizeable number of SSc subjects had also noticed a change in the appearance (26% of SSc vs 11% of RA also with ED, $p = 0.16$) or shape (26% vs 16%, $p = 0.4$) of the penis, and 23% of SSc subjects versus 10% with RA had noticed a reduction in testes size, $p = 0.2$. Such changes may have been a result of fibrosis of the corporal body and/or penile skin, and suggest the potential role for fibrosis secondary to SSc as a factor in ED. However, conclusions here are limited by the fact that changes were not validated by a complete urological examination as part of this study.

Interestingly, the number of biological children of subjects with SSc was fewer than for those with RA (2.0 ± 0.2 vs 2.7 ± 0.2, $p < 0.01$). This may or may not be related to ED; the study was not designed to address fertility or family size. Data on the occurrence of ED relative to the birth of offspring were not collected. The birth of subjects' children likely predated their diagnosis of SSc in many cases. More information on the time between disease onset, ED, and the birth dates of offspring would be required to further investigate fertility in men with SSc. This may be an interesting direction for future studies, as much of the literature on fertility among SSc patients focuses on pregnancy in women[20-22]. In an epidemiological survey of RP (n = 571 paired returns) conducted in the UK, a 6% increase in infertility was noted among cases compared to controls without RP, ($p < 0.02$)[23]. In our study, men with RP had fewer biological children than men without RP (2.0 ± 0.2 vs 2.6 ± 0.2, $p < 0.02$). However we were unable to draw firm conclusions about infertility in men with RP from the limited data as we were not testing this hypothesis.

We recognize that there are limitations to this study, especially the mediocre response rate. However, in sexual health questionnaires this is not atypical. A large epidemiological study of ED in rural New York state was conducted in which researchers mailed a survey to 5198 randomly selected men, of whom 32% participated in the study[24]. Similarly, in a recent survey of quality of female sexual function in SSc, response rate to questionnaires administered in personal interview was 93.8% whereas response to mailed surveys was 33.7%[25]. However, the response rates were similar in cases and controls. RP was identified by women with SSc as

a strong factor in diminished quality of sex along with vaginal dryness and dyspareunia[25]. In our study, the respondents may have been different from the non-respondents. The observations collected by questionnaire may be prone to subject bias or misinterpretation of the questions, especially considering the personal nature of the subject matter. Confounding factors not addressed in the questionnaire may influence the relationship between SSc and ED. The hormone measurements were available only for a subset of men with SSc and thus, should be considered anecdotal. Additionally, although the questionnaire is a standardized and validated instrument, it has not been specifically validated for use in men who suffer from rheumatic diseases. Thus, conclusions must be drawn bearing in mind the possibility of the questionnaire measuring the impact rheumatic disease (chronic illness) through decreased erectile function. However, we believe this is not the case, since men with SSc experienced more ED than matched controls with RA.

Our study confirms that ED is common in SSc, it may occur within 3 years of diagnosis and more commonly than in RA, and therefore is an important clinical symptom of which physicians need to be aware. ED is increased in SSc, in which the majority of individuals also have RP. However, as shown by the subset of men with SSc, the presence of absence of RP does not predict ED, and thus, RP is not necessarily an independent risk factor for the subsequent development of ED.

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