Kardiologia Polska 2013; 71, 11: 1168–1173; DOI: 10.5603/KP.2013.0297 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The prevalence of sexual dysfunction before myocardial infarction in population of Polish men: a retrospective pilot study

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Abstract

Background and aim: To assess the frequency of sexual dysfunction in men before myocardial infarction (MI).

Methods: Sixty-two men with cardiovascular disease risk factors who were admitted to the hospital because of a first MI, were asked to fill the IIEF-15 questionnaire to assess sexual dysfunction before MI.

Results: Erectile dysfunction (ED), decreased orgasmic function, decreased sexual desire, decreased intercourse satisfaction, and decreased overall satisfaction were reported by 51.6%, 14.5%, 50%, 69.4%, and 48.4% of men, respectively. Men with ED had significantly higher serum C-reactive protein (CRP) levels than men without ED (5.8 mg/L, 95% confidence interval [CI] 8.3–21.7) vs. 4.6 mg/L, 95% CI 3.0–11.3; p = 0.01). Men with decreased orgasmic function had significantly higher serum triglyceride levels (259.5 mg/dL, 95% CI 176.9–362.1 vs. 150 mg/dL, 95% CI 146.8–187.4; p = 0.01), and men with normal sexual desire had significantly higher serum high-density lipoprotein (HDL) cholesterol levels than men with decreased sexual desire (41 mg/dL, 95% CI 39.9–47.8 vs. 36 mg/dL, 95% CI 34.1–40.5; p = 0.01). Men with decreased sexual desire had significantly higher serum CRP levels (7 mg/L, 95% CI 7.7–21.4 vs. 5 mg/L, 95% CI 3.6–12.1; p = 0.03).

Conclusions: 1. ED was present in more than half of men before MI and it may be the first symptom of coronary artery disease. 2. Men with ED and decreased sexual desire have higher serum CRP levels in the acute peri-infarction period. 3. Serum triglyceride level is a factor that significantly affects orgasmic function, and serum HDL cholesterol level is a factor that significantly affects sexual desire.

Key words: myocardial infarction, erectile dysfunction, C- reactive protein, HDL cholesterol, triglycerides

Kardiol Pol 2013; 71, 11: 1168–1173

INTRODUCTION

Epidemiology of sexual dysfunction, erectile dysfunction and coronary artery disease

Erectile dysfunction (ED) is one of the most common health problems in middle-aged men. ED is currently present in about 11% of men aged 50–59 years, 4% of men aged 30–49 years, and 3% of men aged 18–24 years [1]. In other countries, the estimated rates are about 18% (18 million men) in the United States and 19.2% in Germany [2]. It has been predicted that globally, sexual dysfunction will be present in about 322 million men by 2025 [3]. The most recent 2011 report on sexuality in Poland (Raport Seksualności Polaków) indicates that 68% of Poles are happy with their sexual life, including 68% of men and 67% of women. About 23% of the surveyed subjects were unable to determine their level of sexual satisfaction. Only 8% of the surveyed subjects rated their sexual life as unsatisfactory. About 10% of men with ED are under specialist care, and about 20% use various kinds of aphrodisiacs [4]. These data indicated that sexual issues are often neglected during patient-physician encounters due to embarrassment. Most men with ED cease sexual activity and wait until the problem resolves spontaneously. According to the report on sexuality in Poland, men seek help mostly in the internet (about 33.6% of subjects), and from primary care physicians (about 12.8%), cardiologists (5.7%), sexuologists (4.6%), and psychologists (2.3%).

ED, defined as the inability to achieve or maintain erection sufficient for a satisfactory intercourse, may be a result

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Received: 12.11.2012 **Accepted:** 16.01.2013

and an early sign of endothelial dysfunction. The latter is caused by oxidative stress, with increased production of reactive oxygen species leading to activation of an inflammatory process in the vascular wall, which contributes to endothelial dysfunction and impaired nitric oxide synthesis. This was confirmed in a group of 30 men with ED free from cardiovascular (CV) disease and without CV risk factors, in whom a significantly reduced endothelial-dependent vasodilatation in the brachial artery was found compared to the control group without sexual dysfunction [5]. ED may be an early sign of coronary artery disease (CAD) and often coexist with other cardiovascular risk factor such as obstructive sleep apnea [6, 7]. This is related to the fact that penile arteries are smaller (diameter 1–2 mm) and develop obstructive lesions earlier that in larger coronary arteries (diameter 3–4 mm) or carotid arteries (diameter 5–7 mm). ED may also precede a CV event such as myocardial infarction (MI) or ischaemic stroke. Pritzker [8] studied 50 men aged 40–60 years with ED and CV disease risk factors but without symptoms of CAD. Electrocardiographic exercise test was abnormal in 56% of subjects (28 men), and CAD was identified by coronary angiography in 40% of subjects (20 men). In men with ED evaluated for CAD, electrocardiographic exercise test is usually abnormal in 5–56% men [8]. Thompson et al. [9] evaluated the relationship between ED and CAD in a population of 9457 men aged 55 or more years, including 4247 subjects without ED at baseline. During 5-year follow-up, patients were evaluated at 3-month intervals for ED and symptoms of CAD. ED developed during the follow-up in 57% of men. Men with ED had a significantly higher MI risk compared to men without ED [9]. In another study, angiographically documented CAD was found 1 in 5 men with ED of vascular aetiology [10]. Kawanishi et al. [11] studied 58 men with ED and identified CAD in 24.1% of them (13 subjects). These findings prompted us to evaluate the presence of ED before MI in Polish men.

METHODS

In this retrospective pilot study, we evaluated 62 men aged 40–75 years hospitalised in the First Chair and Department of Cardiology at the Medical University of Warsaw (WUM) due to a first MI. On average, 1 in 3 patients consented for participation in our study, more frequently younger subjects and patients with higher education. The study protocol was approved by the Regional Ethics Committee. Informed consent was obtained from every patient before enrolling in the study. We excluded patients with chronic kidney disease (glomerular filtration rate < 35 mL/min/1.7 m^2), severe chronic heart failure (New York Heart Association class III–IV), previous MI, psychiatric conditions, and hormonal abnormalities. The study group consisted of 53 patients with ST segment elevation MI and 9 patients with non-ST elevation MI, including 6 patients with diabetes. All patients were smokers. Baseline characteristics of the study group are shown in Table 1.

Table 1. Characteristics of the study group

Parametres	Mean ± SD/range/median OR number of subjects (%)
Patient age [years]	54.5 ± 6.4/41–70/55
Height [cm]	176 ± 7.1/155–188/176
Weight [kg]	89.9 ± 15.2/52–121/91
BMI [kg/m^2]	28.9 ± 4.3/19.3–41.7/29.1
No. of cigarettes smoked daily	24.3 ± 9.9/4–40/20
Type of MI:	
STEMI	53 (85.5%)
NSTEMI	9 (14.5%)
Education:	
Vocational	23 (37.1%)
Secondary	7 (11.3%)
Higher	32 (51.6%)
Marital status:	
Divorced	5 (8.1%)
Married	52 (83.9%)
Widowed	2 (3.2%)
Bachelors	3 (4.8%)
Sleeplessness:	
No	48 (77.4%)
Sometimes	7 (11.3%)
Yes	7 (11.3%)
Stress:	
No stress	6 (9.7%)
Moderate level	34 (54.8%)
High level	22 (35.5%)
Hypertension	32 (51.6%)
Diabetes:	
No	55 (88.7%)
Yes	6 (9.7%)
Impaired fasting glucose	1 (1.6%)
WBC count [10^3/μL]	11.1 ± 2.8/4.9–17.7/10.7
MPV [fL]	10.1 ± 1.2/7.4–13.5/10.1
Haemoglobin [g/dL]	15.8 ± 3.5/12.6–19.5/14.4
Total cholesterol [mg/dL]	199.4 ± 38.1/133–313/200.5
HDL cholesterol [mg/dL]	40.6 ± 10.3/24–77/38.5
LDL cholesterol [mg/dL]	123.6 ± 30.3/0–193/122.5
Triglycerides [mg/dL]	180.5 ± 85.7/73–434/160
TSH [μIU/mL]	1.7 ± 2.2/0.33–17.6/1.2
Creatinine [mg/dL]	0.9 ± 0.2/0.6–1.3/0.9
C-reactive protein [mg/L]	11.2 ± 15.8/0.5–69.8/5.2
Prolactin [ng/mL]	7.9 ± 3.5/0.9–18.2/7.6
DHEAS [μg/dL]	207.5 ± 89.9/37.03–440.6/202
LVEF (at 2–5 days of MI)	0.5 ± 0.1/0.22–0.6/0.49
BNP [pg/mL]	150 ± 164.4/10.4–920/99.1
Testosterone [nmol/L]	12.3 ± 6.5/1.6–37.1/10.5
No. of intercourses per month	5.3 ± 3.2/1.5–11/3.5

SD — standard deviation; BMI — body mass index; MI — myocardial infarction; STEMI — ST segment elevation MI; NSTEMI — non-ST segment elevation MI; WBC — white blood cells; MPV — mean platelet volume; HDL — high-density lipoprotein; LDL — low-density lipoprotein; TSH — thyroid-stimulating hormone; DHEAS — dehydroepiandrosterone sulphate, LVEF — left ventricular ejection fraction; BNP — B-type natriuretic peptide

Table 2. Scoring of the International Index of Erectile Function-15 questionnaire

	Abnormal result
Erectile dysfunction	< 25 points
Decreased orgasmic function	< 9 points
Decreased sexual desire	< 9 points
Decreased intercourse satisfaction	< 13 points
Decreased overall satisfaction	< 9 points

Table 3. Sexual dysfunction before myocardial infarction in the study group

	Study group
Erectile dysfunction	32 (51.6%)
Decreased orgasmic function	9 (14.5%)
Decreased sexual desire	31 (50%)
Decreased intercourse satisfaction	43 (69.4%)
Decreased overall satisfaction	30 (48.4%)

After obtaining a written informed consent for participation in the study, detailed history was taken and complete physical examination was performed to identify CV disease risk factors. All patients underwent retrospective evaluation of sexual function before MI using the International Index of Erectile Function-15 (IIEF-15) questionnaire [12]. We evaluated the following components of sexual function: erectile function, orgasmic function, sexual desire, intercourse satisfaction, and overall satisfaction from sexual activity. Erectile function was evaluated based on 6 questions, yielding a maximum score of 30 points. Results below 25 points were considered abnormal (i.e., indicating ED). Orgasmic function was evaluated using 2 questions, yielding a maximum score of 10 points. Results below 9 points were considered abnormal. Sexual desire was also evaluated using 2 questions, yielding a maximum score of 10 points, and results below 9 points were considered abnormal. Intercourse satisfaction was evaluated based on 3 questions, yielding a maximum score of 15 points, and the threshold value was 13 points. Overall satisfaction from sexual activity was evaluated using 2 questions, yielding a maximum score of 10 points, and results below 9 points were considered abnormal (Table 2).

Between 2 and 5 days of MI, B-type natriuretic peptide (BNP) and free testosterone levels were determined. BNP level was determined in venous blood using a rapid immunofluorometric test BNP Triage (Biosite). Serum free testosterone level was determined in venous blood between 6 and 8 AM in the central laboratory of our hospital. We also evaluated other hormonal parameters and basic CV risk biomarkers: lipid profile, glycaemia, full blood count, creatinine, thyroid-stimulating hormone, prolactin, and C-reactive protein (CRP). Obtained data were subjected to a statistical analysis.

Statistical analysis

Statistical analyses were performed using the STATISTICA 2011 software (version 10.0., StatSoft, Inc., www.statsoft.com) and the Excel spreadsheet. Quantitative variables were characterised by arithmetic mean, standard deviation, median, and minimum and maximum values (range). Qualitative variables were characterised by numbers and percentages. Variable distribution was evaluated using the Shapiro-Wilk W test, and equal variances were tested using the Levene (Brown-Forsythe) test.

Significance of differences between two groups (unpaired sample model) was evaluated using the Student *t* test (or the Welch test in case of unequal variances) or the Mann-Whitney U test (for cardinal variables or if conditions for the use of the Student *t* test were met). Significance of differences between more than two groups was evaluated using the F-test (analysis of variance [ANOVA]) or the Kruskal-Wallis test (or if conditions for the use of ANOVA). If significant differences between groups were found, *post hoc* tests were also applied (the Tukey test for the F-test, and the Dunn test for the Kruskal-Wallis test).

For the paired sample model, the Student *t* test or the Wilcoxon signed-rank test was used. Significance of differences between more than two groups in the paired sample model was test using the repeated measures ANOVA or the Friedman test. The χ^2 test was used for qualitative variables. To determine associations with their strength and directions, correlation analysis was used by calculating the Pearson and/or Spearman correlation coefficients. $P = 0.05$ was considered significant.

RESULTS

Table 3 shows characteristics of sexual dysfunction in the study group at least 1 month before the occurrence of MI. ED was present before MI in nearly half of our patients. Decreased orgasmic function before MI was reported by nearly 15% of men, and decreased sexual desire, decreased intercourse satisfaction, and decreased overall satisfaction were reported by respectively half, more than two thirds, and half of men,

To evaluate the effect of various studied parameters on sexual function, men were divided into groups with impaired or normal sexual function before MI. Only significant results were presented in the Tables 4–6.

We found than with ED before MI had significantly higher plasma CRP level in the peri-infarction period compared to patients without erectile dysfunction ($p = 0.01$). The ED group included nearly 5-fold more patients with vocational education and fewer patients with higher education compared to patients without ED. Among patients with ED before MI, more men took no medications due to chronic diseases. In contrast, more men without ED were on drug therapy due to chronic diseases (Table 4). The remaining variables and evaluated associations, including those of testosterone and BNP levels, yielded insignificant results.

Table 4. Erectile dysfunction

	Erectile dysfunction	No erectile dysfunction	Overall	Statistical test
C-reactive protein:				
Mean ± SD	15.0 ± 18.6	7.2 ± 11.1	11.2 ± 15.8	U M-W = –2.45
Range	1.4–69.8	0.5–60.9	0.5–69.8	p = 0.01
Median	5.8	4.6	5.2	
95% CI	[8.3; 21.7]	[3.0; 11.3]	[7.2; 15.2]	
Education:				
Vocational	19/59.4%	4/13.3%	23/37.1%	χ^2 = 17.86
Secondary	0/0.0%	7/23.3%	7/11.3%	p = 0.0001
Higher	13/40.6%	19/63.3%	32/51.6%	
Medications before infarction:				p = 0.61
No medications	27/84.4%	18/60%	45/72.6%	χ^2 = 4.62
Medications*	5/15.6%	12/40%	17/27.4%	p = 0.03

*Defined as the use of any of the following: acetylsalicylic acid, beta-blocker, angiotensin-converting enzyme, angiotensin receptor antagonist, statin, metformin; SD — standard deviations; CI — confidence interval; U M-W — Mann-Whitney U test

Table 5. Decreased orgasmic function

	Decreased orgasmic function	No decreased orgasmic function	Overall	Statistical test
Triglicerides:				
Mean ± SD	269.5 ± 110.8	167.1 ± 73.6	180.5 ± 85.7	U M-W = –2.54
Range	107–434	73–417	73–434	p = 0.01
Median	259.5	155	160	
95% CI	[176.9; 362.1]	[146.8; 187.4]	[158.6; 202.4]	
Education:				
Vocational	7/77.8%	16/30.2%	23/37.1%	χ^2 = 7.65
Secondary	0/0.0%	7/13.2%	7/11.3%	p = 0.02
Higher	2/22.2%	30/56.6%	32/51.6%	

SD — standard deviations; CI — confidence interval; U M-W — Mann-Whitney U test

Table 6. Decreased sexual desire

	Decreased sexual desire	No decreased sexual desire	Overall	Statistical test
HDL cholesterol:				
Mean ± SD	37.3 ± 8.7	43.9 ± 10.8	40.6 ± 10.3	U M-W = –2.58
Range	24–64	28–77	24–77	p = 0.01
Median	36.0	41.0	38.5	
95% CI	[34.1; 40.5]	[39.9; 47.8]	[38.0; 43.2]	
C-reactive protein:				
Mean ± SD	14.5 ± 18.7	7.9 ± 11.6	11.2 ± 15.8	U M-W = 2.10
Range	1.4–69.8	0.5–60.9	0.5–69.8	p = 0.03
Median	7	5	5.2	
95% CI	[7.7; 21.4]	[3.6; 12.1]	[7.2; 15.2]	

HDL — high-density lipoprotein; CI — confidence interval; U M-W — Mann-Whitney U test; SD — standard deviation

Men with decreased orgasmic function before MI had significantly higher plasma triglyceride level. Among patients with normal orgasmic function (i.e. with no sexual dysfunction), a higher proportion of men with higher education was found (Table 5).

Men with normal sexual desire before MI had significantly higher plasma high-density lipoprotein (HDL) level compared to patients with decreased sexual desire. A statistically significant, 2-fold higher CRP level was found in patients with decreased sexual desire (Table 6).

DISCUSSION

ED and CAD often coexist, particularly in men above 40 years of age. For the clinical practice, it is important to estimate time from the development of ED to the occurrence of MI. Our retrospective survey indicates that is present in more than half of men at least 1 month before MI. In the study by Montorsi et al. [13] who also identified ED as a predictor of MI, ED was present about 3 years before MI in 67% of the study subjects. As reported in various studies, ED is present in 42–75% of patients with CAD and in more than a half of men after MI [14]. We also showed that HDL cholesterol and triglyceride levels significantly affect various components of the sexual function. Similar conclusions were arrived at in the study by Wei et al. [15] who demonstrated that ED was associated with high plasma total cholesterol level. Each increase in total cholesterol level by 1 mmol/L was associated with a 1.32-fold increase in the rate of ED. The risk of ED is lowest in subjects with HDL cholesterol level above 60 mg/dL. In contrast, patients with total cholesterol level above 240 mg/dL had a nearly 2-fold higher risk of ED compared to patients with total cholesterol level below 180 mg/dL [15]. High triglyceride level is often associated with endothelial dysfunction, and this affects some other components of sexual function then ED, as demonstrated in the present study. Perhaps this was related to overall poor health condition of the evaluated men.

Both ED and sexual desire closely correlate with CRP level in the peri-infarction period. This was also reported by Billups et al. [16] who showed higher plasma CRP levels in men with sexual dysfunction caused by atherosclerosis in the penile arteries [17]. In our study group, most men were obese. Obesity promotes inflammation, and CRP level was reported to be increased in subjects with the metabolic syndrome [18].

In our study, we also showed that patients with higher education were less likely to report sexual dysfunction. Higher education probably facilitates healthy behaviours. Untreated CV disease contributes to the development of ED, while adverse effects of medications generally do not significantly affect erectile function. This was confirmed in a study by Shiri et al. [19] who did not find an increased risk of ED in patients treated with angiotensin-converting enzyme inhibitors, cardioselective beta-blockers, and lipid-lowering drugs [18].

Previous surveys of ED were focused on the evaluation of this condition after MI, and not retrospective evaluation of ED before MI. In a similar survey performed in our centre, 72.2% of men reported having sexual intercourses less frequently than before MI, and only 27.8% reported the same frequency of having sexual intercourses as before MI. ED after MI was reported by 31.5% of respondents, while 68.5% reported no change. Overall, sexual activity significantly decreased in the study group. One third of men reported lower frequency of sexual intercourses after MI compared to the period before MI. About 33.3% of the surveyed men reported problems with erection at least once during the last 6 months, 29.6% reported less interest in sexual life, 31.5% were afraid to engage in sexual activity due to subjectively perceived reduced exercise capacity, and only 18.5% noticed no changes [20].

Limitations of the present study included small patient sample (62 men), its retrospective nature, and methodological issues (a subjective, declarative nature of the survey). In addition, penile vascular studies were not performed in the studied men, which would provide data on the aetiology of sexual dysfunction.

CONCLUSIONS

1. ED was present in more than half of men before MI and it may be the first symptom of CAD.
2. Men with ED and decreased sexual desire have higher serum CRP levels in the acute peri-infarction period.
3. Serum triglyceride level is a factor that significantly affects orgasmic function, and serum HDL cholesterol level is a factor that significantly affects sexual desire.

Conflict of interest: *none declared*

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Częstość występowania dysfunkcji seksualnych przed zawałem serca w populacji polskich mężczyzn: retrospektywne badanie pilotażowe

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Streszczenie

Wstęp i cel: Celem badania była ocena częstości występowania dysfunkcji seksualnych u mężczyzn przed zawałem serca (MI).

Metody: Grupę 62 mężczyzn z czynnikami ryzyka chorób sercowo-naczyniowych hospitalizowanych z powodu pierwszego w życiu MI poddano retrospektywnej ocenie zaburzeń funkcji seksualnych za pomocą kwestionariusza IIEF 15.

Wyniki: Zaburzenia erekcji (ED), upośledzone osiąganie orgazmu, obniżone pożądanie płciowe, obniżona satysfakcja ze stosunku płciowego, obniżone zadowolenie z życia seksualnego wystąpiły odpowiednio u: 51,6%, 14,5%, 50%, 69,4%, 48,4% mężczyzn przed MI. Wykazano, że mężczyźni z ED charakteryzowali się istotnie statystycznie wyższym osoczowym stężeniem białka C-reaktywnego (CRP) niż mężczyźni bez ED (5,8 mg/l; 95% CI 8,3–21,7 *vs*. 4,6 mg/l; 95% CI 3,0–11,3; p = 0,01). U mężczyzn ze stwierdzonym upośledzonym osiąganiem orgazmu przed wystąpieniem MI zanotowano istotnie statystycznie wyższe osoczowe stężenie triglicerydów (259,5 mg/dl; 95% CI 176,9–362,1 *vs*. 150 mg/dl; 95% CI 146,8–187,4; p = 0,01), z kolei pacjenci z prawidłowym pożądaniem płciowym (bez obniżenia pożądania płciowego) charakteryzowali się znamiennie statystycznie wyższym osoczowym stężeniem cholesterolu frakcji HDL w porównaniu z pacjentami z obniżonym pożądaniem płciowym (41 mg/dl; 95% CI 39,9–47,8 *vs*. 36 mg/dl; 95% CI 34,1–40,5; p = 0,01). U pacjentów z obniżonym pożądaniem płciowym stwierdzono natomiast istotnie statystycznie wyższe osoczowe stężenie CRP (7 mg/l; 95% CI 7,7–21,4 *vs*. 5 mg/l; 95% CI 3,6–12,1; p = 0,03).

Wnioski: 1. ED występują u ponad połowy mężczyzn przed MI i mogą być pierwszym objawem choroby wieńcowej. 2. Mężczyźni z ED oraz z obniżonym pożądaniem płciowym cechują się wyższym stężeniem CRP w okresie okołozawałowym. 3. Stężenie triglicerydów jest czynnikiem istotnie wpływającym na zdolność do osiągania orgazmu, z kolei stężenie cholesterolu HDL jest istotnym czynnikiem wpływającym na stopień pożądania płciowego.

Słowa kluczowe: zawał serca, zaburzenia erekcji, białko C-reaktywne, HDL cholesterol, triglicerydy

Kardiol Pol 2013; 71, 11: 1168–1173

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Praca wpłynęła: 12.11.2012 r. **Zaakceptowana do druku:** 16.01.2013 r.