

Is there an association between depressive symptoms and coronary artery disease in the Polish adult population?

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Abstract

Background: Evidence-based medicine has confirmed the role of psychosocial factors in the pathogenesis of many diseases, both cardiovascular (CVD) and metabolic. On the other hand, CVD patients often suffer from concomitant diseases. Depression was found to be an independent predictor of coronary artery disease (CAD) in many populations.

Aim: To evaluate the association between depressive symptoms (DS) and CAD in the Polish adult population.

Methods: A random sample of the Polish population (6392 men and 7153 women), aged 20–74 years, was examined in 2003–2005 for the presence of DS using the Beck Depression Inventory.

Results: In the examined population, CAD was found in 12.1% of men and 11.0% of women. Persons with CAD were older, more often finished their education at the level of primary school and lived in large communes, and more often had obesity, hypertension, diabetes and hyperlipidaemia compared to those without CAD. DS were found twice more often in persons with CAD compared to those without CAD, both in men and women. Subjects with DS were twice more likely to have CAD (men: odds ratio [OR] 2.14, 95% confidence interval [CI] 1.78–2.56; women: OR 2.03, 95% CI 1.70–2.43) and arrhythmia (women), and 1.5-fold more likely to report myocardial infarction and arrhythmia.

Conclusions: A significant association between DS and CAD, myocardial infarction and arrhythmia independent of CVD risk factors was found in the Polish adult population.

Key words: coronary artery disease, depressive symptoms, national survey

Kardiol Pol 2014; 72, 1: 50–55

INTRODUCTION

Depression, with its varied aetiology and clinical manifestation, has been an important and established factor in the studies on the epidemiology of coronary artery disease (CAD). In 1994, Ford et al. [1] reported results of a 35-year follow-up of 1,198 medicine graduates at the Johns Hopkins University. Depression was found to be an independent predictor of CAD. Pratt et al. [2] showed that at least one episode of major depression during lifetime was associated with 4-fold increased risk of myocardial infarction (MI). In a cohort study that included more than 5,600 patients aged 65 or more years, depressive symptoms were associated with an increased risk of depression among men [3]. Not only depression may be a predictor of CAD, but it may also have an effect on established CAD.

It has been confirmed that psychosocial risk factors including depression are independent cardiovascular (CV) risk factors [4]. In addition to an increased risk of first coronary event and worse outcomes in established CAD, psychosocial factors may also affect compliance to drug therapy and lifestyle modifications, as well as health promotion at both patient and population level [5, 6].

Among various psychosocial risk factors, chronic stress, low social support and depression play the most important role in the pathophysiology of CV disease (CVD). No large population studies have been undertaken on the association between depression and CAD in the Polish population.

The aim of the present study was to evaluate whether depression is independently associated with CAD in the Polish adult population.

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Received: 14.01.2013

Accepted: 28.05.2013

Available as AOP: 21.06.2013

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METHODS

Study population

The prevalence of depressive symptoms was evaluated in a survey of a randomly chosen sample of the Polish adult population aged 20–74 years that was studied in a multi-centre Polish population health status study, the WOBASZ study (Wieloośrodkowe Ogólnopolskie Badanie Stanu Zdrowia Ludności), in 2003–2005. The study protocol including methods and details of population sampling was published previously [6]. Briefly, 2 small communes (< 8,000 inhabitants), 2 medium communes (8,000–40,000 inhabitants), and 2 large communes (> 40,000 inhabitants) were randomly chosen in each of 16 voivodeships in Poland, and 100 men and 100 women were randomly chosen in each of the communes (overall 19,200 persons). This sample included 4 large communes within voivodeship capital cities, and additionally 1 commune was randomly chosen from each of the remaining voivodeship capital cities, with 100 women and 100 men selected from each of these 12 communes. Ultimately, 6,977 men and 7,792 women were evaluated. The study included questionnaire, physical examination including measurements of anthropometric parameters, and laboratory tests. The study was approved by an ethics committee, and the studied subjects gave their consent to questionnaire, physical examination, and laboratory tests.

For the purpose of study group characterisation regarding the presence of depression and major CV risk factors in subjects with or without CAD, we identified subjects with depressive symptoms, hypertension, diabetes, hypercholesterolaemia, and obesity. CAD was identified based on a self-reported history of hospitalisation due to an acute coronary syndrome including MI, percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), or a history of MI or the diagnosis of CAD without hospitalisation. Arrhythmia was identified based on a self-reported history of arrhythmia, hospitalisation due to arrhythmia, or medical records indicating arrhythmia. Hypertension was defined as blood pressure $\geq 140/90$ mm Hg (mean from the second and third blood pressure measurement) or antihypertensive drug use. Hypercholesterolaemia was defined as total cholesterol level ≥ 5 mmol/L or low-density lipoprotein cholesterol level ≥ 3 mmol/L or the use of lipid-lowering drugs. Diabetes was defined as fasting blood glucose level ≥ 7.0 mmol/L or a self-reported history of diabetes, obesity as body mass index (BMI) ≥ 30 kg/m², and current smoking as smoking at least one cigarette per day.

The WOBASZ study was supported by the Polish Ministry of Health and Social Welfare (the POLKARD project 2003–2005).

Evaluation of depressive symptoms

The Beck Depression Inventory (BDI) was used to evaluate the presence and severity of depressive symptoms. The

presence of depressive symptoms was defined as the BDI score of at least 10 [7]. The BDI included 21 items scored 0 to 3, with questions assessing mood, pessimism, sense of failure, lack of satisfaction, guilty feelings, expectations of punishment, self-dislike, desire to die, crying, irritability, social withdrawal, body image, problems at work, sleeplessness, fatigue, loss of appetite, body weight reduction, and somatic complaints. Overall, the presence of depressive symptoms was evaluated in 6,164 men and 6,915 women. The methodology of psychological evaluation and its initial results were previously reported in a study by Piwonski et al. [8].

Statistical analyses

To perform all planned analyses, we used data collected in 5,874 men and 6,640 women for whom complete data were available. Analyses were performed separately for men and women. Results were presented as mean values \pm standard deviation (age) or proportions. The χ^2 test was used to compare frequencies of various risk factors, including depressive symptoms, in subjects with or without CAD. $P < 0.05$ was considered statistically significant. The relation between CAD and depressive symptoms was evaluated using multivariate logistic regression analysis that included conventional CVD risk factors (age, hypertension, smoking, hyperlipidaemia, diabetes, BMI), education, and municipality size. All analyses were performed using the Statistical Analysis System (SAS v 9.2) software.

RESULTS

Among participants of the WOBASZ study, a history of CAD was reported by 640 (10.9%) men and 659 (9.9%) women. Subjects with CAD more often lived in large municipalities and finished their education at the level of primary school. CVD risk factors including hypertension, diabetes, and hyperlipidaemia were significantly more frequent among those with CAD compared to those without CAD, both in men and women. Among subjects with CAD, the prevalence of hypertension and obesity was increased 2-fold, and the prevalence of diabetes was increased 3-fold (Table 1).

When we analysed the presence of depressive symptoms, we found that they were twice more prevalent in both men and women with CAD compared to those without CAD (Table 1) and also about twice more prevalent compared to the mean prevalence in the general population. Subjects with depression were more than twice as likely to report previous MI and arrhythmia, and 3 times as likely to report CAD (Fig. 1).

Logistic regression analysis showed a significant association between depressive symptoms and CAD, previous MI, arrhythmia and previous revascularisation procedures (PCI or CABG) which was independent from age and major CVD risk factors. A subject with depressive symptoms was more than twice as likely to have CAD compared to a subject

Table 1. Characteristics of subjects with or without coronary artery disease (CAD)

| Variable | Men | | | Women | | |
|---|------------|-------------|----------|------------|-------------|----------|
| | CAD (+) | CAD (-) | P | CAD (+) | CAD (-) | P |
| Number of subjects | 640 | 5,234 | | 659 | 5,981 | |
| Age [years] | 60.6 ± 9.2 | 43.7 ± 14.5 | < 0.0001 | 61.3 ± 8.9 | 43.2 ± 14.5 | < 0.0001 |
| Commune size: | | | | | | |
| Small (< 8,000 inhabitants) | 29.4% | 34.8% | | 33.1% | 34.6% | |
| Medium (8,000–40,000 inhabitants) | 29.4% | 31.6% | 0.0004 | 28.1% | 32.5% | 0.0060 |
| Large (> 40,000 inhabitants) | 41.2% | 33.6% | | 38.8% | 32.9% | |
| Education: | | | | | | |
| Primary | 62.6% | 59.9% | | 67.2% | 46.1% | |
| Secondary | 28.0% | 29.6% | NS | 26.9% | 41.1% | < 0.0001 |
| Higher | 9.4% | 10.5% | | 5.9% | 12.8% | |
| Obesity (BMI ≥ 30 kg/m ²) | 35.0% | 18.8% | < 0.0001 | 47.3% | 19.5% | < 0.0001 |
| Current smoking (at least 1 cigarette/24 h) | 29.8% | 39.6% | < 0.0001 | 12.1% | 25.3% | < 0.0001 |
| Hypertension (≥ 140/90 mm Hg or drug treatment) | 69.8% | 37.0% | < 0.0001 | 72.8% | 27.8% | < 0.0001 |
| Hyperlipidaemia (TC ≥ 5.0 mmol/L or LDL-C ≥ 3.0 mmol/L or drug treatment) | 79.4% | 67.9% | < 0.0001 | 84.8% | 62.7% | < 0.0001 |
| Diabetes (blood glucose ≥ 7.0 mmol/L or history of diabetes) | 22.0% | 5.7% | < 0.0001 | 18.5% | 4.9% | < 0.0001 |
| Depression (BDI ≥ 10) | 46.3% | 21.2% | < 0.0001 | 60.2% | 30.7% | < 0.0001 |

BDI — Beck Depression Inventory; BMI — body mass index; LDL-C — low-density lipoprotein cholesterol; TC — total cholesterol

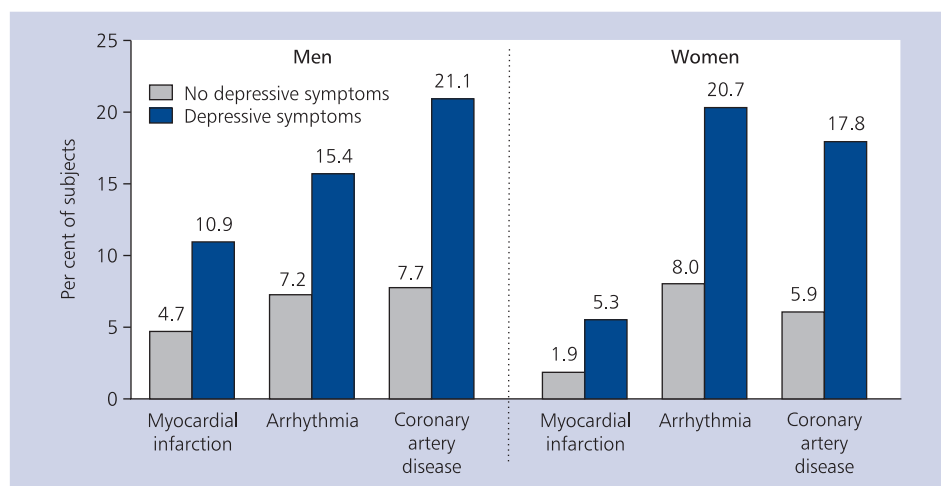


Figure 1. Proportion of subjects with coronary artery disease, previous myocardial infarction and arrhythmia with or without depressive symptoms

without depression regardless of age, smoking, presence of hyperlipidaemia, hypertension and diabetes, BMI, education, and the place of residence. Similarly strong relationships were observed in men (odds ratio [OR] 2.15, 95% confidence interval [CI] 1.78–2.59) and women (OR 1.96, 95% CI 1.63–2.36) (Table 2). The strength of association between depression and CAD was similar to that between hyperten-

sion and CAD (data not shown). Similar relationships were found between depressive symptoms and a history of MI and arrhythmia (Table 2).

DISCUSSION

Our analysis of the data from the WOBASZ study showed a significant association between depressive symptoms and

Table 2. Odds ratios of coronary artery disease (OR_{CAD}), previous myocardial infarction (OR_{MI}), arrhythmia (OR_{arrhythmia}), and previous coronary artery bypass grafting or percutaneous coronary intervention (OR_{revasc}) depending on the presence or absence of depressive symptoms

| | OR _{CAD} (95% CI)*; p | OR _{MI} (95% CI)*; p | OR _{revasc.} (95% CI)*; p | OR _{arrhythmia} (95% CI)#; p |
|----------------------|--------------------------------|-------------------------------|------------------------------------|---------------------------------------|
| Men | | | | |
| Depressive symptoms: | | | | |
| No | 1.00 | 1.00 | 1.00 | 1.00 |
| Yes | 2.15 (1.78–2.59) < 0.0001 | 1.58 (1.25–2.00) 0.0001 | 2.19 (1.41–3.42) 0.0005 | 1.52 (1.23–1.88) < 0.0001 |
| Women | | | | |
| Depressive symptoms: | | | | |
| No | 1.00 | 1.00 | 1.00 | 1.00 |
| Yes | 1.96 (1.63–2.36) < 0.0001 | 1.55 (1.15–2.09) 0.0042 | 1.70 (0.63–4.61) NS | 2.09 (1.77–2.46) < 0.0001 |

*Adjusted for age, smoking, hypertension, hypercholesterolaemia, body mass index, diabetes, education, and municipality size; #adjusted for age, smoking, hypertension, hypercholesterolaemia, body mass index, diabetes, education, municipality size, and a history of coronary artery disease (CAD); CI — confidence interval; MI — myocardial infarction; OR — odds ratio

CAD, previous MI, and arrhythmia which was independent from conventional CVD risk factors.

Depression, which is probably the most common mental health condition, continues to be underrecognised in the clinical practice. However, its prevalence in aging populations rises and it is 2 to 3 times more frequent among patients with chronic diseases such as CVD or CAD [9]. In the WOBASZ study, the prevalence of depressive symptoms in the general population in Poland in 2003–2005 was 24% among men and 34% among women [8].

Multiple studies indicate that depression frequently coexists with CVD [9, 10]. Depression is considered an important CV risk factor, both in healthy subjects and in patients with CVD [11], and the risk of CAD associated with depression is independent from conventional CVD risk factors such as hypertension, elevated cholesterol level, and elevated BMI [9], as also shown in our study.

In a 40-year follow-up of 1,190 men, Ford et al. [12] showed that depression was an independent predictor of CAD events. In a study by Marzari et al. [3] in a cohort of more than 5,600 subjects aged 65 or more years, the presence of depressive symptoms was associated with an increased risk of CAD in men.

Specific neurophysiological and behavioural abnormalities present in patients with depressive symptoms may be responsible for the observed association between depression and CVD, including abnormalities of the hypothalamic-pituitary-adrenal axis, sympathetic dysfunction, platelet activation, and unhealthy lifestyle [11].

From the pathophysiologic point of view, depressive disorder largely resembles chronic stress, with its chronic hypercortisolaemia and activation of the autonomic nervous system, leading to both immunosuppression and increased

release of proinflammatory cytokines, along with elevated blood pressure and cholesterol levels. Subjects with depression are characterised by depressed mood which promotes unhealthy behaviours such as smoking, poor diet, and low or lacking physical activity, all contributing to the risk of CAD [8].

As already mentioned, not only depression may be a predictor of CAD but it may also modify the clinical course of established disease and is a predictor of worse outcomes after MI, similarly to such factors as smoking or previous MI [13–15]. In addition, depression is associated with a 4- to 5-fold increase in mortality at 18 months after the MI [13] and an increased rate of recurrent coronary events and hospitalisations [16].

It has been estimated that about 20% of patients with stable CAD fulfil the diagnostic criteria of major depression, and mood disorders are present in an even higher proportion of patients [17]. After an MI, major depression was found in about 15–23% of patients [17, 18]. In a retrospective study by Lesperance et al. [18], a history of depression was reported by 28% of patients with MI. In addition, nearly 48% of patients with MI reported clear depressive symptoms immediately before the infarction. It was confirmed that depressive symptoms often precede CAD, with higher risk of CAD and sudden cardiac death in those with more severe symptoms [19].

Not only patients with depression are more likely to develop CAD but also CAD is associated with an increased rate of depression. In our study, subjects with depression were about 3 times as likely to have CAD, and depressive symptoms were twice as likely in patients with CAD. Patients with an acute MI and concomitant depressive symptoms are at high risk of death or reinfarction. This elevated risk is present not only in the acute period but also later [20]. Thus, it is important to screen patients with CAD, especially those after

an MI, for depressive symptoms, and particularly for major depression which was reported in 15–30% of patients during 18-month follow-up after an MI [21]. Depressive mood is often considered a normal reaction to major disease, both by patients and doctors. Sleep disturbances and fatigue are also considered to be related to cardiological problems and often remain unreported to the physician. All these factors lead to a low rate of the diagnosis of depression in patients with CAD (about 10%). Unrecognised depression and associated mood disturbances result in patient isolation and poor adherence to both cardiac rehabilitation and drug therapy.

Depression is also an important independent predictor of mortality in patients after CABG or awaiting CABG, and in patient with unstable CAD. In these groups, depression is more prevalent than on average in patients with CVD. Low level of education is a risk factor for depression in the perioperative period [4]. Depression after CABG is associated with more than 2-fold increase in the readmission rate (OR 2.31) [22, 23]. In our study population, depressive symptoms were associated with previous CABG only in men.

One possible explanation of the observed association between depression and CVD, and particularly arrhythmia, may be autonomic dysfunction present in subjects with depression and manifesting by reduced heart rate variability, impaired baroreceptor function, and changes in heart rate [11]. In the WOBASZ study, depressive symptoms were associated with arrhythmia both in men and in women.

Association between depression and CVD may also be partially mediated by genetic factors, including serotonin transporter gene (serotonin-transporter-linked polymorphic region, 5HTTLPR) polymorphism [24], alleles of which are not only associated with increased vulnerability to stress and more frequent occurrence of depressive episodes, but also predict an increased risk of CV events [25]. Platelets and the effect of serotonin on platelets may be a factor linking this polymorphism, depressive symptoms, and CAD. Serotonin (5-hydroxytryptamine, 5-HT) was shown to affect platelet aggregation, vasoconstriction, and vascular smooth muscle cell hyperplasia which all lead to thrombus formation and may have a role in the pathogenesis of MI. Platelet aggregation and vasoconstriction are stimulated by binding of serotonin, transported by serotonin transporter (5-HTT), with serotonin receptor type 2A (5-HT_{2A}).

Limitations of the study

The WOBASZ study was an epidemiological investigation, so clinical verification of the diagnosis of depression made on single administration of BDI was not possible. We were also unable to determine whether depressive symptoms preceded the occurrence of CAD or developed afterwards. In addition, due to the fact that BDI includes questions on various nonspecific symptoms seen not only in depression but also various somatic disorders, some authors have suggested that

the diagnostic threshold should be higher than 10 points in studies on the association between depression and somatic problems. However, a threshold score of 10 was used in most published studies based on BDI.

CONCLUSIONS

In our study, a significant association between depressive symptoms and CAD, MI and arrhythmia independent of CVD risk factors was found in the Polish adult population.

Conflict of interest: none declared

References

1. Ford ES, Ahluwalia IB, Galuska DA. Social relationships and cardiovascular disease risk factors: Findings from the Third National Health and Nutrition Examination Survey. *Prev Med*, 2000; 30: 83–92.
2. Pratt LA, Ford DE, Crum RM et al. Depression, psychotropic medication and risk of myocardial infarction: prospective data from Baltimore ECA follow-up. *Circulation*, 1996; 94: 3123–3129.
3. Marzari C, Maggi S, Manzato E et al. Depressive symptoms and development of coronary heart disease events: the Italian longitudinal study of aging. *J Gerontol A Biol Sci Med Sci*, 2005; 60: 85–92.
4. Wilkowska A. Epizody depresyjne u pacjentów z chorobą niedokrwienną serca. *Psych Prakt Klin*, 2008; 1: 12–21.
5. Graham I, Atar D, Borch-Johnsen K et al. Europejskie wytyczne dotyczące prewencji chorób sercowo-naczyniowych w praktyce klinicznej: wersja skrócona. *Kardiol Pol*, 2008; 66: 4 (suppl. 1): S1–S48.
6. Piwoński J, Piwońska A, Sygnowska E. Do depressive symptoms adversely affect the lifestyle? Results of the WOBASZ study. *Kardiol Pol*, 2010; 68: 912–918.
7. Beck AT, Steer CH, Mendelson M et al. An inventory for measuring depression. *Arch Gen Psychiatry*, 1961; 4: 561–571.
8. Piwoński J, Piwońska A, Głuszek J et al. Ocena częstości występowania niskiego poziomu wsparcia społecznego oraz objawów depresji w populacji polskiej. Wyniki programu WOBASZ. *Kardiol Pol*, 2005; 63 (suppl. 4): S645–S648.
9. Barefoot JC, Schroll M. Symptoms of depression, acute myocardial infarction, and total mortality in a community sample. *Circulation*, 1996; 93: 1976–1980.
10. Rudisch B, Nemeroff CB. Epidemiology of comorbid coronary artery disease and depression. *Biol Psychiatry*, 2003; 54: 227–240.
11. Grippo AJ, Johnson AK. Biological mechanisms in the relationship between depression and heart disease. *Neurosci Biobehav Rev*, 2002; 26: 941–962.
12. Ford DE, Mead LA, Chang PP et al. Depression is a risk factor for coronary artery disease in men. *Arch Intern Med*, 1998; 158: 1422–1426.
13. Frasure-Smith N, Lesperance F, Talajic M. Depression and 18 months prognosis after myocardial infarction. *Circulation*, 1995; 91: 999–1005.
14. Glassman AH, Shapiro PA. Depression and the course of coronary artery disease. *Am J Psychiatry*, 1998; 155: 4–11.
15. Pawlak A, Krejca M, Janas-Kozik M et al. Ocena lęku i depresji w okresie okołoperacyjnym u pacjentów poddawanych rewaskularyzacji mięśnia sercowego. *Psychiatr Pol*, 2012; XLVI: 63–74.
16. Ladwig KH, Roll G, Breithardt G et al. Post-infarction depression and incomplete recovery 6 months after myocardial infarction. *Lancet*, 1994; 343: 20–23.
17. Parissis JT, Fountoulaki K, Fillipatos G et al. Depression in coronary artery disease: Novel pathophysiologic mechanisms and therapeutic implications. *Int J Cardiol*, 2007; 116: 153–160.

18. Lesperance F, Frasura-Smith N, Talajic M. Major depression before and after myocardial infarction: its nature and consequences. *Psychosom Med*, 1996; 58: 99–100.
19. Everson SA, Goldberg DE, Kaplan GA et al. Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosom Med*, 1996; 58: 113–121.
20. Zellweger J, Michael. Coronary artery disease and depression. *Eur Heart J*, 2004; 25: 3–7.
21. Strik JMH, Honig A, Maes M. Depression and myocardial infarction: relationship between heart and mind. *Prog Neuropsychopharmacol Biol Psychiatry*, 2001; 25: 879–892.
22. Connerney I, Shapiro PA, Mc Laughlin JS et al. Relationship between depression after coronary artery bypass surgery and 12-month outcome: a prospective study. *Lancet*, 2001; 358: 1766–1771.
23. Saur CD, Granger BB, Muhlbaier LH et al. Depressive symptoms and outcome of coronary artery bypass grafting. *Am J Crit Care*, 2001; 10: 4–10.
24. Capi A, Sudgen K, Moffitt TE et al. Influence of life stress on depression: moderation a polymorphism in the 5-HTT gene. *Science*, 2003; 301: 386–389.
25. Otte C, Neylan TC, Pipkin SS et al. Depressive symptoms and 24-hour urinary norepinephrine excretion levels in patients with coronary disease: findings from the Heart and Soul Study. *Am J Psychiatry*, 2002; 36: 173–182.

Czy istnieje związek między objawami depresji a chorobą wieńcową w populacji dorosłych Polaków?

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Streszczenie

Wstęp: Depresja jako zespół zaburzeń psychosomatycznych znalazła szczególne zastosowanie w badaniach nad epidemiologią choroby wieńcowej (CAD) i okazała się niezależnym jej predyktorem.

Cel: Celem niniejszej pracy było zbadanie, czy istnieje niezależny związek między objawami depresji a występowaniem CAD w populacji dorosłych Polaków.

Metody: Próba losowa populacji polskiej (6392 mężczyzn i 7153 kobiety), w wieku 20–74 lat, została przebadana w latach 2003–2005 w kierunku objawów depresji za pomocą kwestionariusza Becka.

Wyniki: Spośród osób zbadanych w badaniu WOBASZ, CAD w wywiadzie podawało 12,1% mężczyzn i 11,0% kobiet. Osoby z CAD były starsze, częściej miały wykształcenie podstawowe i mieszkały w dużych miastach oraz częściej stwierdzano u nich otyłość, nadciśnienie tętnicze, cukrzycę czy hiperlipidemię. Objawy depresji występowały 2-krotnie częściej u osób z CAD, zarówno wśród mężczyzn, jak i kobiet, niż u osób bez CAD. Zanotowano istotny statystycznie oraz niezależny od wieku i obecności głównych czynników ryzyka chorób układu sercowo-naczyniowego związek między występowaniem objawów depresji a CAD, zawałem serca, zaburzeniami rytmu serca w wywiadzie i pomostowaniem aortalno-wieńcowym. Zależność zaobserwowano w takim samym nasileniu u mężczyzn (OR = 2,14, 95% CI 1,78–2,56), jak i u kobiet (OR = 2,03, 95% CI 1,70–2,43).

Wnioski: Stwierdzono istotny i niezależny od klasycznych czynników ryzyka chorób układu sercowo-naczyniowego związek między objawami depresji a CAD, w tym zawałem serca oraz zaburzeniami rytmu serca w populacji dorosłych Polaków.

Słowa kluczowe: choroba wieńcowa, objawy depresji, badanie przekrojowe

Kardiol Pol 2014; 72, 1: 50–55

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Praca wpłynęła: 14.01.2013 r.

Zaakceptowana do druku: 28.05.2013 r.

Data publikacji AoP: 21.06.2013 r.