Cognitive impairment in patients after myocardial infarction

Dominika Kasprzak[1*], Teresa Ganowicz-Kaatz[1*], Janusz Rzeźniczak[1*], Marek Słomczyński[1], Katarzyna Kaczmarek-Majer[2, 3], Jan Budzianowski[4], Konrad Pieszko[4], Jarosław Hiczkiewicz[4], Andrzej Tykarski[5], Paweł Burchardt[1, 5]

[1]Department of Cardiology, J. Struś Hospital, Poznań, Poland
[2]Department of Stochastic Methods, System Research Institute, Polish Academy of Sciences, Warszawa, Poland
[3]Information Technologies for Psychiatry Foundation, Poznań, Poland
[4]University of Zielona Góra, Zielona Góra, Poland
[5]Department of Hypertension, Angiology, and Internal Medicine, Poznan University of Medical Sciences, Poznań, Poland
*These authors equally contributed to the study

Correspondence to:
Dominika Kasprzak,
Department of Cardiology,
J. Struś Hospital,
Szwajcarska 3, 61–285 Poznań,
Poland,
phone: +48 618 739 395,
e-mail:
domi.kasprzak@gmail.com

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INTRODUCTION

Both cognitive impairment (CI) and dementia as well as cardiovascular disease (CVD), including myocardial infarction (MI), are a significant burden on the health and social care systems. Currently, 50 million people worldwide suffer from dementia, while CVDs are still the leading cause of death [1]. Unfortunately, assessment of cognitive function is not part of routine clinical practice, especially in cardiac patients. Nevertheless, a growing body of evidence points to an association between CVD, including ischemic heart disease, and the occurrence of CI. The mechanisms responsible for this remain largely unknown. The problem of the prevalence of psychiatric disorders in cardiac patients is also highlighted by the latest guidelines on cardiovascular prevention. They indicate that all mental disorders are associated with the development of CVD while the onset of CVD is associated with a 2–3 times higher risk of mental disorders. It is estimated that the annual prevalence of psychiatric disorders in patients with CVD is around 40%, leading to a significantly worse prognosis [2]. Given these clinical implications, we have undertaken an assessment of cognitive functioning in people after MI and 6 months later and attempted to identify factors that may influence it.

METHODS

Study design and patient population

This is a pilot study and precedes a larger prospective study. This prospective study was conducted at the Cardiology Department of J. Struś Hospital in Poznań, and its protocol was approved by the Local Bioethics Committee (approval no. 1201/16). Two hundred and twenty patients hospitalized for MI treated by percutaneous coronary intervention (PCI) participated in this pilot study. All participants were clinically assessed on two occasions: during the first MI-related hospitalization on days 2–3 following PCI, and 6 months later. Available medical records including a health history questionnaire, laboratory tests, and echocardiography were collected, and mental state was assessed with the Mini-Mental State Examination (MMSE), Schulman's clock-drawing test (CDT), Beck Depression Inventory (BDI), Athens Insomnia Scale (AIS), and Insomnia Severity Index (ISI) (Table 1 presents the main statistical characteristics of all variables). At baseline, the MMSE scores were corrected according to age and education. Due to the comparable results, the absolute values were used for further analysis. CI was defined as MMSE <27 points or CDT level ≥1. Depression was defined as BDI ≥12, and insomnia was defined as ISI ≥ 15. All tests used in the study have been adapted and validated for the Polish setting.

We distinguished 4 groups of patients depending on the changes in their mental status: (1) permanent CI — presented both at baseline and after 6 months; (2) transient — with deficits at baseline but with a normal test result after 6 months; (3) new onset CI — only after 6 months; and (4) without CI during follow-up.

Table 1. Statistical characteristics of all variables considered in this pilot study were collected during the first MI-related hospitalization on days 2–3 following PCI, and 6 months later (follow-up)

Variable	First hospitalization	Follow-up (after 6 months)
	Mean (SD)/median (IQR)[a]	Mean (SD)/median (IQR)[a]
Age, years	60.1 (9.3)	Not collected
Hgb, g/dl	13.7 (1.6)	14.4 (13.6–15.2)
Hct, %	40.2 (4.2)	42.9 (41–44.9)
RBC, million/µl	4.5 (4.2–4.9)	4.8 (0.4)
WBC, thousand/µl	9.6 (7.8–11.7)	7.6 (6.3–8.8)
PLT, thousand/µl	221 (189–258)	235 (202.2–265.5)
Na, mmol/l	140.5 (139–142)	141 (140–143)
K, mmol/l	4.3 (4–4.5)	4.6 (4.4–4.9)
Creatinine, µmol/l	79 (69–91)	81 (69–91)
Urea, mmol/l	5.3 (4.4–6.3)	5.7 (4.9–6.7)
TN, ng/l	740 (86.5–4153)	9 (9–12)
ALAT, U/l	36 (24–51.5)	24 (18–32)
HDL-C, mmol/l	1.2 (0.9–1.4)	1.3 (1.1–1.5)
LDL-C, mmol/l	3.2 (2.4–3.8)	1.9 (1.6–2.4)
TG, mmol/l	1.4 (1.1–1.9)	1.3 (1–1.7)
TSH, µlU/ml	1 (0.6–1.7)	1.1 (0.8–1.6)
CK, IU/l	51.5 (24.8–128.2)	108 (83–158)
BNP, pg/ml	110.3 (54–199.8)	Not collected
SYNTAX, points	9 (6–14)	Not collected
EF, %	50 (40–50)	50 (50–60)
BDI, points	9 (5–14)	8 (4–13)
Absolute MMSE score, points	27 (25–29)	28 (26–29)
MMSE adjusted score, points	27 (25–28)	28 (26–29)
ISI, points	8 (4–13)	6 (3–11)
CDT, level	0 (0–1)	0 (0–1)
AIS, points	6 (4–9)	5 (3–8)

[a]Mean (SD) is reported if normal distribution was confirmed by the Shapiro-Wilk test. Otherwise, median (IQR) is reported. As a result, mean (SD) are reported for age, Hgb, and Hct variables for the first hospitalization and for RBC for a follow-up visit

Abbreviations: AIS, Athens Insomnia Scale; ALAT, alanine transaminase; BDI, Beck depression inventory; BNP, brain natriuretic peptide; CDT, clock-drawing test; CK, creatine kinase; EF, ejection fraction; Hct, hematocrit; HDL-C, high-density lipoprotein cholesterol; Hgb, hemoglobin; ISI, Insomnia Severity Index; K, potassium; LDL-C, low-density lipoprotein cholesterol; MMSE, Mini-Mental State Examination; Na. sodium; PLT, platelet count; RBC, red blood cells; TG, triglycerides; Tn, troponin; TSH, thyroid stimulating hormone; WBC, white blood cells

Statistical analysis

Continuous variables were reported as means (standard deviation [SD]) or medians (interquartile ranges [IQR]), as appropriate. Normal distribution was tested using the Shapiro-Wilk test. The differences in the numerical variables were tested using the Kruskal-Wallis test. Next, the Mann-Whitney test was performed as a post-hoc test sequentially for 2 groups, and the Bonferroni correction was applied. All analyses were done using programming language R and STATISTICA 10 (StatSoft). Two-sided *P*-values <0.05 were considered to be statistically significant.

RESULTS AND DISCUSSION

The main characteristics of the pilot study sample are presented in the Supplementary material, *Table S1*. At baseline, we identified CI in 40.5% (n = 89) of patients according to the MMSE, and in 34.5% (n = 76) using the CDT. In the follow-up, CI was observed in 33.6% (n = 74) of patients using MMSE and 26.8% (n = 59) using the CDT. Statistical characteristics of age (years), ISI (points), ejection fraction (%), brain natriuretic peptide (BNP, pg/ml), SYNTAX (points), troponin (ng/l), and BDI (points) in the identified four groups are presented in the Supplementary material, *Table S2*.

Patients with permanent deficits (Group 1) in the CDT compared to those without CI (Group 4) had lower peri-infarction ejection fraction (50% [40%–50%] vs. 50% [50%–60%]; $P = 0.006$) and a higher level of peri-infarction BNP (149.6% [91.3%–242.8%] vs. 87.7% [46%–140.8%]; $P = 0.003$)

The prevalence of previously undiagnosed CI in patients hospitalized for MI was high (nearly 40%). These disorders can be either temporary or permanent. Currently, we do not know the specific factors that would allow us to predict these cognitive disorders. However, we can hypothesize that there are different underlying causes of CI following MI. Permanent deficits may be involved in neurodegeneration but so can a higher burden of vascular risk factors. Therefore, the etiology is most likely mixed.

In patients with transient deficits, the cause may be psychological stress after MI and acute phase of the disease but also appropriate treatment and vascular risk factors reduction.

On the other hand, new-onset CI may be connected with accumulating mental health disorders, such as sleep disturbances, and worse control of vascular risk factors. The latter is less likely because our participants had optimal treatment.

While analyzing variables that may affect cognitive function, it is also important to bear in mind depressive disorders, which often occur following MI. Thirty-three percent of the participants in our study presented them during their first hospitalization. This is consistent with previous data reporting depression in 20%–40% of MI patients [3]. CI is among the main symptoms of depression, and its presence is a predictor of dementia development [4].

It is also important to highlight the influence of age, which is a major risk factor for both CI and MI. In our project, patients with persistent CI were significantly older than those without CI during the study. Those included in our project represent a younger population than the average MI patient (60.1 vs. 65.1 for men and 72 for women) [5]. It can be, therefore, assumed that the prevalence of cognitive deficits is underestimated and is higher in clinical practice.

Little is also known about the impact of arrhythmias on cognitive function. Most researchers have focused on atrial fibrillation (AF), associating its presence with higher risk of CI and dementia [6]. Preliminary results of our project did not show that AF significantly affected CI in patients after MI, whereas in those with peri-infarct non-sustained ventricular tachycardia (NSVT), CI was significantly more frequent after 6 months of follow-up ($P = 0.02$). Chen et al. [7] also showed that NSVT was independently associated with CI occurrence and with impairment of executive function in particular. This may suggest that the occurrence of asymptomatic episodes of arrhythmia during follow-up in patients with peri-infarct NSVTs results in ischemic brain lesions. Therefore, they may represent a risk group and should be subject to more careful follow-up.

The results presented here are part of a pilot study. A larger population study is currently being conducted to analyze in detail the factors affecting cognitive function in patients with acute coronary syndrome. We are living longer, but longevity must be accompanied by the quality. CI significantly affects daily functioning not only of those affected but also carers. It is, therefore, important to proactively detect CI at an early stage and try to modify potentially reversible risk factors. If we detect changes in cognitive functioning early, we can implement appropriate management and have time to refer patients to other specialists such as psychologists or neurologists.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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REFERENCES

1. World Health Organization. Global action plan on the public health response to dementia 2017–2025. Geneva 2017.
2. Visseren FLJ, Mach F, Smulders YM, et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021; 42(34): 3227–3337, doi: 10.1093/eurheartj/ehab484, indexed in Pubmed: 34458905.
3. Saquib J, AlRomaih NA, Al-Mutairi HM, et al. Correlates of memory loss and depression among myocardial infarction patients in Al-Qassim, Saudi Arabia. J Saudi Heart Assoc. 2018; 30(3): 240–246, doi: 10.1016/j.jsha.2018.04.001, indexed in Pubmed: 29983498.
4. Sáez-Fonseca JA, Lee L, Walker Z. Long-term outcome of depressive pseudodementia in the elderly. J Affect Disord. 2007; 101(1-3): 123–129, doi: 10.1016/j.jad.2006.11.004, indexed in Pubmed: 17184844.
5. Benjamin EJ, Muntner P, Alonso A, et al. Heart Disease and Stroke Statistics-2019 Update: A Report From the American Heart Association. Circulation. 2019; 139(10): e56–e5e528, doi: 10.1161/CIR.0000000000000659, indexed in Pubmed: 30700139.
6. Sawicki S, Mierzyńska A, Krauze A, et al. Silent atrial fibrillation episodes and cognitive impairment in patients with cardiac implantable devices. Protocol of the SAFE-COG ongoing observational single-center non-experimental case-control clinical study. Kardiol Pol. 2021; 79(12): 1362–1364, doi: 10.33963/KP.a2021.0162, indexed in Pubmed: 34845712.
7. Chen L, Agarwal S, Lopez F, et al. Abstract 12946: Higher Burden of Atrial Fibrillation is Independently Associated With Lower Cognitive Function: The Atherosclerosis Risk in Communities (ARIC) Study. Circulation. 2014; 130(suppl_2), doi: 10.1161/circ.130.suppl_2.12946.