**Original article**

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# THE IMPACT OF DEPRESSION ON THERAPY OF ACUTE CORONARY SYNDROME

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Epidemiological studies suggest that besides classic factors for the development and prognosis of acute coronary syndromes, depression and anxiety are important as well. Depression and coronary heart disease occur simultaneously in the same person due to a common pathophysiological mechanism, possible genetic dysfunction of serotonin receptors. The aim of the paper was to examine the incidence and severity of the depression and anxiety in acute coronary syndrome patients and to examine whether the presence of depression had an impact on the decision for invasive or non-invasive approach.

We included 38 patients (23 males and 15 females, aged 63.5 ± 10years) hospitalized in the Clinic for cardiology Clinical Centre Niš, with the acute coronary syndrome (ACS). The patients were divided according to the type of ACS therapy: the invasive group (28 patients) with percutaneous coronary intervention and stenting and the second group of patients who underwent angiography without indication for stenting (non- invasive group of 10 patients). The anamnestic and clinical data, biomarkers of cardiac necrosis, standard laboratory, lipid profiles, and markers of inflammation were done. To examine the levels of depression and anxiety we used different questionnaires: The general questionnaire for socio-demographic data and the data about the disease progression, Beck Depression Inventory – questionnaire to measure the intensity of the depressive symptoms and State and Trait Anxiety Inventory (STAI) questionnaire to measure the intensity of actual anxiety (state anxiety), and anxiety as a personal characteristic (trait anxiety), Health Locus of Control – the questionnaire which measures where a patient puts the centre of the control of the disease. According to the level of depression, we formed four groups - 19 patients without depression (50 %), mild: 10 patients (26.3 %), moderate: 8 patients (21 %), and with severe depression: 1 patient (2.7 %).

The most common risk factors were: hypertension with 81.6 %, lipid disorders 68.4% and family history of cardiovascular disease in 52.6 %. Cardiovascular risk factors did not differ significantly between genders. Previous coronary artery disease (CAD) had 42.1 % hospitalised patients with ACS with a similar proportion of patients of both genders. The STEMI was the most common clinical presentation of ACS in 47.4 % of patients. The stenting procedures were performed in 28 (73.6 %) patients and medicamentous therapy in 10(26.4 %). The patients in non- invasive group were significantly older, more commonly obese, hyperlipidemic, with positive family history for CAD, with anamnestic data about previous AP and heart failure, and with higher heart rate. The presence of depression, especially a moderate level of depression was more common in non -invasive group (90.0 % and 50.0 % prospective) than in invasively treated group (35.7 % and 10.7 %) ($p < 0.01$ and $p < 0.05$). The correlation was found between the duration of hospitalization and the degree of hyperglycemia and depression and anxiety that exacerbate the progression ofCAD.

Increased depression leads to increased anxiety and higher blood glucose levels - both additional risk factors for the progression of CAD. Patients with depression more likely had actual anxiety and will be treated by non–invasive medicamentous therapy for ACS. Increase in anxiety in patients diagnosed with the CAD increases the risk of MI, lethal outcome of coronary disease.

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***Key words:*** *acute coronary syndrome, depression*

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## Introduction

According to studies, it has been suggested that depressive symptomes occur in 15 to 30 % of patients with coronary artery disease (CAD). Risk ratios for the first and recurrent cardiac events related to depression are comparable to well-established CAD risk factors and range from 2 to 7. During the

last decades, there is increasing evidence that psychological factors, including depression, lack of social support, anger, job- related stresses could have an effect on prognosis in cardiovascular patients. Recently a number of studies pointed out that depression is a big psychosocial problem (1, 2) .It is important to note that a certain amount of negative emotional reaction should be expected in acute coronary syndrome. A physician should expect everyday anxiety from the patient, irritability, and sadness. A physician should respond to those reactions with support, warmth and to give hope for recovery.

These basic human emotions have a positive influence on the patients' recovery and progress. For example, the fear of the heart attack in the future could stimulate the acceptation of the proposed therapy. However, for some patients the fear is disproportional in comparison to the treatment and it is not adaptive such as the case with the intensive fear which leads to insomnia, atypical chest pain and avoiding of low-risk activities (3). Unlike the sudden fear which is a short-term reaction, the behaviour caused by serious illness is not seen immediately. Some patients are better at dealing with the changes in their lifestyles, including the stopping of some activities, while some others have difficulties with acceptance of their state and the emotional healing process is slow like curing of the infected wound. This unresolved grief could be the first sign of depression. Long stay in an intensive care unit or numerous re-hospitalizations could also trigger the depression. Epidemiological data suggest that on the medical ward cardiologist would probably see 16 % of the patients with major depression and 20 % a with minor depression form. Numbers are similar for hospitalized patients with myocardial infarction, unstable angina pectoris, and coronary bypass, angioplasty or heart failure (4, 5).

In the last 10 years, evidence has suggested that various psychological factors, including depression, lack of social support, anger, stress at work, can affect the prognosis of cardiac patients. More recently, the increasing number of well-designed studies published in medical journals and cardiology, has drawn attention to the great depression as a psychosocial risk.

Thrombus formation is a key factor in the rapid progression of CAD and in the occurrence of acute MI and unstable angina. The hypothesis of an association between depression and increased platelet activation, in particular, appears to explain the link between depression and mortality from heart diseases. Although serotonin itself is a weak agonist for platelet aggregation, serotonin can potentiate the effect of other agonists in the induction of platelet aggregation. Serotonin may further encourage the formation of thrombus by induction of coronary vasoconstriction of damaged vessels. During many years, platelets were used in psychiatric researches as a model of serotonin pre-and post-synaptic function in the brain (1, 2).

**Aims**

The aim of the study was to determine the incidence and severity of the depression and anxiety in acute coronary syndrome patients, as well as to examine its influence on the type of in-hospital therapy.

**Methods**

We included in the study 38 patients who were hospitalized in the Clinic for the cardiology Clinical Centre Niš with the acute coronary syndrome (unstable angina pectoris – UAP, myocardial infarction without ST - segment elevation – NSTEMI and myocardial infarction with ST - segment elevation – STEMI). The patients were divided into two groups according to tthe type of ACS therapy: the first group (invasive group 28 patients) has a percutaneous coronary intervention with stenting and second group has angiography without indication for stenting (non -invasive group 10 patients).

In all patients besides anamnestic data, routine laboratory examinations were done in Central laboratory Clinical Centre Niš. From the markers of necrosis, we followed troponin I (TnI), creatinine kinase isoform MB (CK-MB), from inflammation markers high sensistive C reactive protein (CRP) and white blood cells count were followed and lipid profile – triglycerides, cholesterol, LDL and HDL fractions. Standard 3 channel ECG, echocardiography and invasive examinations were done in all patients.

The patients with chronic non -cardiovascular diseases, chronic or acute inflammatory states, rheumatologic and systemic diseases were excluded from the analysis. The patients with an indication for further cardio-surgical revascularisation were also excluded from the study.

To obtain additional data about socio-demographic characteristics and the level of depression and anxiety we used different questionnaires:

1. The general questionnaire to obtain socio-demographic data and the data about the disease progression the (length of illness, number of hospitalizations), hereditary diseases, the association with psychological or stress disorders.

2. Beck Depression Inventory – the questionnaire to measure the intensity of the depressive symptoms. It has 21 questions and offered answers are 4-scaled (0-3). The final score is the sum of all the answers. According to the score, we could divide depression into 4 groups – without, mild, moderate and severe depression.

3. State and Trait Anxiety Inventory (STAI) – the questionnaire has 40 questions which measure the intensity of two anxiety forms – actual anxiety (state anxiety) and anxiety as personal characteristics (trait anxiety). According to the score, both types of anxiety could be divided into mild, moderate and intensive. With special tables, we could transform primary scores into Z- scores (0-100) which allow comparing those two forms of anxiety.

4. Health Locus of Control – questionnaire which measure where a patient puts centre of the control of the disease – internal (he thinks that he is dominantly responsible for his health and disease progression) and external (he thinks that the external factors determine if he will be sick or he will be healed as fate, by a physician and other people). With processing the results we get the intensity scores for (a) internal control centre, (b) control centre associated with the fate/coincidence, (c) doctors, and (d) other persons.

The data were processed in SPSS 16 program, including T test, Chi-square test, ANOVA and correlation analysis. The significance level was set to $< 0.05$.

## Results

In the examined group we followed 38 patients, 23 men (60.5 %) and 15 women (39.5 %) aged 63.5 ± 10.0 years. The average age of male subjects was 65.2 ± 10.0 years and 61.1 ± 10.14 years for female, the difference was not statistically signi-ficant ($t = 1.2$, $p > 0.05$) (Table 1).

**Table 1.** Characteristics of the study population by age and gender

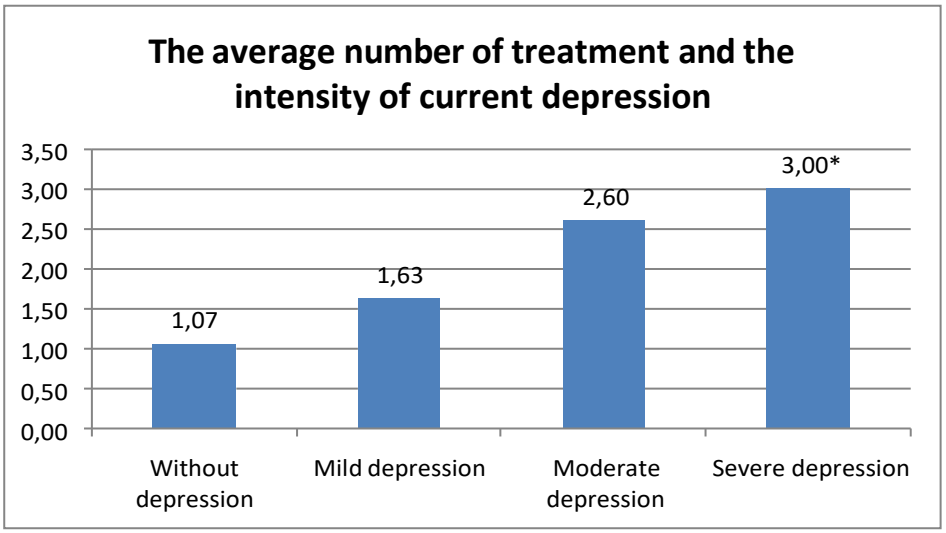
| | Male | Female | Total |
|---|---|---|---|
| n/% | 23 / 60.5 | 15 / 39.5 | 38 / 100.0 |
| Age mean±SD (y) | 65.2±10.0 | 61.1±9.8 | 63.5±10.0 |
| Age min - max (y) | 40-79 | 41-76 | 40 - 79 |
| Cardiovascular risc factors | | | |
| Hypertension | 20 / 87.0 | 11 / 73.3 | 31 / 81.6 |
| Diabetes melitus | 8 / 34.8 | 4 / 26.7 | 12 / 31.6 |
| Smoking | 5 / 21.7 | 3 / 20.0 | 8 / 21.1 |
| Lipid disorders | 17 / 73.9 | 9 / 60.0 | 26 / 68.4 |
| Obesity | 5 / 21.7 | 8 / 53.3* | 13 / 34.2 |
| Family history of CVD | 11 / 47.8 | 9 / 60.0 | 20 / 52.6 |
| Hystory of IM | 3 / 13.0 | 3 / 20.0 | 6 / 15.8 |
| Hystory of AP | 5 / 21.7 | 2 / 13.3 | 7 / 18.4 |
| Hystory of heart failure | 2 / 8.1 | 1 / 6.7 | 3 / 7.9 |
| Hystory of CABG | 2 / 8.6 | 1 / 6.6 | 3 / 7.9 |
| Cardiovascular parameters | | | |
| Heart rate | 76.2±15.1 | 84.1±17.6* | 79.3±18.3 |
| Systolic pressure | 147.1±22.8 | 140.7±32.8 | 144.5±30.0 |
| Diastolic pressure | 87.1±19.4 | 82.3±17.8 | 85.2±19.9 |
| Laboratory findings | | | |
| Total cholesterol | 6.2±1.8 | 6.4±1.8 | 6.3±1.7 |
| LDL cholesterol | 4.1±2.1 | 4.9±2.8 | 4.4±2.7 |
| HDL cholesterol | 1.1±0.4 | 1.2±0.4 | 1.1±0.4 |
| Tryglicerides | 2.5±1.1 | 2.8±0.9 | 2.6±1.2 |
| Clinical presentations of ACS | | | |
| UAP | 1 / 4.3 | 3 / 20 | 4 / 10.5 |
| NSTEMI | 9 / 39.1 | 7 / 46.7 | 16 / 42.1 |
| STEMI | 13 / 56.6 | 5 / 33.3 | 18 / 47.4 |
| Depresion | | | |
| with depression | 8 / 34.8 | 11 / 73.3* | 19 / 50.0 |
| mild depression | 4 / 17.4 | 6 / 40.0 | 10 / 26.3 |
| moderate depression | 4 / 17.4 | 4 / 26.7 | 8 / 21.0 |
| severe depression | 0 / 0.0 | 1 / 6.6 | 1 / 2.7 |

Data are presented as mean±SD or n / group %; CVD- cardiovascular disease, IM-infarctus myocardi, AP-angina pectoris; UAP-unstable angina pectoris, NSTEMI-myocardial infarction without ST segment elevation, STEMI-myocardial infarction with ST segment elevation; *$p < 0.05$ vs. male

**Table 2.** Characteristics of the examined groups

| | Invasive | Non invasive |
|---|---|---|
| n/% | 28 / 73.6 | 10 / 26.4 |
| Age mean±SD (y) | 60.1±9.5 | 67.8±9.8* |
| Cardiovascular risc factors | | |
| Hypertension | 21 / 75.0 | 10 / 100.0 |
| Diabetes melitus | 8 / 28.6 | 4 / 40.0 |
| Smoking | 7 / 25.0 | 1 / 10.0 |
| Lipid disorders | 17 / 60.7 | 9 / 90.0* |
| Obesity | 4 /14.3 | 9 / 90.0** |
| Family history of CVD | 12 / 42.9 | 8 / 80.0* |
| Hystory of IM | 4 /14.3 | 2 / 20 |
| Hystory of AP | 2 / 7.1 | 5 / 50.0** |
| Hystory of heart failure | 0 / 0.0 | 3 / 30.0* |
| Hystory of CABG | 1 / 3.5 | 2 / 20.0 |
| Cardiovascular parameters | | |
| Heart rate | 75.1±14.2 | 88.8±19.6* |
| Systolic pressure | 140.3±20.7 | 148.1±31.8 |
| Diastolic pressure | 86.1±18.3 | 88.3±16.8 |
| Clinical presentations of ACS | | |
| UAP | 0 / 0.0 | 4 / 40.0 |
| NSTEMI | 10 / 35.7 | 6 / 60.0 |
| STEMI | 18 / 64.3 | 0 / 0.0 |
| Depresion | | |
| with depression | 10 / 35.7 | 9 / 90.0** |
| mild depression | 7 / 25.0 | 3 / 30.0 |
| moderate depression | 3 / 10.7 | 5 / 50.0* |
| severe depression | 0 / 0.0 | 1 / 10.0 |

Data are presented as mean±SD or n/%; CVD- cardiovascular disease, IM-infarctus myocardi, AP-angina pectoris; UAP-unstable angina pectoris, NSTEMI-myocardial infarction without ST segment elevation, STEMI-myocardial infarction with ST segment elevation; *p<0.05 vs. invasive, **p < 0.01 vs. invasive group




*p < 0.05 vs. without and mild depression, Students t test

**Graph 1.** The average number of treatment and the intensity of the current depression

The most common risk factors were: hypertension with 81.6 %, lipid disorders 68.4 % and family history of cardiovascular disease in 52.6 %. Cardiovascular risk factors did not differ significantly between genders. Previous coronary artery disease had 34.2 % hospitalised patients with ACS with similar proportion of patients of both genders. The STEMI was the most common clinical presentation of ACS. The patients with NSTEMI and UAP were somewhat more frequent women while STEMI were frequently men, but without significant difference between genders.

Depression was presented in 50 % of patients, significantly more in females 73.3 % than males 34.8 % ($p < 0.05$) with similar distribution of depression grades among gender (Table 1).

The stenting procedures were performed in 28 (73.6 %) patients and medicamentous therapy in 10(26.4 %) (Table 2). The patients in the non–invasive group were significantly older, more commonly obese, hyperlipidemic, with positive family history for CAD, with anamnestic data about previous AP and heart failure, and with higher heartrate.

T5he presence of depression, especially moderate level of depression was more common in the non-invasive group (90.0 % and 50.0 % prospective) than in the invasively treated group (35.7 % and 10.7 %) ($p < 0.01$ and $p < 0.05$). Characteristics of invasively and noninvasively treated groups were presented in Table 2.

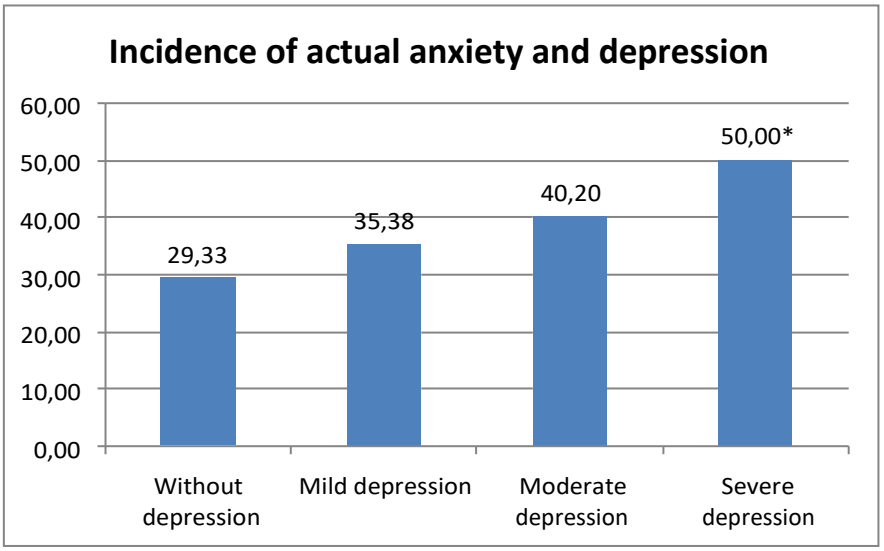
In the study population, with the number of hospitalizations, the severity of depression increased, and an average number of hospitalisations was significantly higher in severe depression compared to mild and no depression patients ($p < 0.05$) (Graph 1).

Actual anxiety was a very common finding in the observed coronary heart disease patients and strongly associated with level of depression. Its incidence was the lowest in patients without depression and the highest in patients with severe depression. Half of the examined patients (50 %) with severe depression had actual anxiety state (Graph 2).

Patients who had severe anxiety traits had the lowest values of internal locus of control, and differed from the others in a statistically significant level ($F = 5379$, $P = 0.011$) (Graph 3).

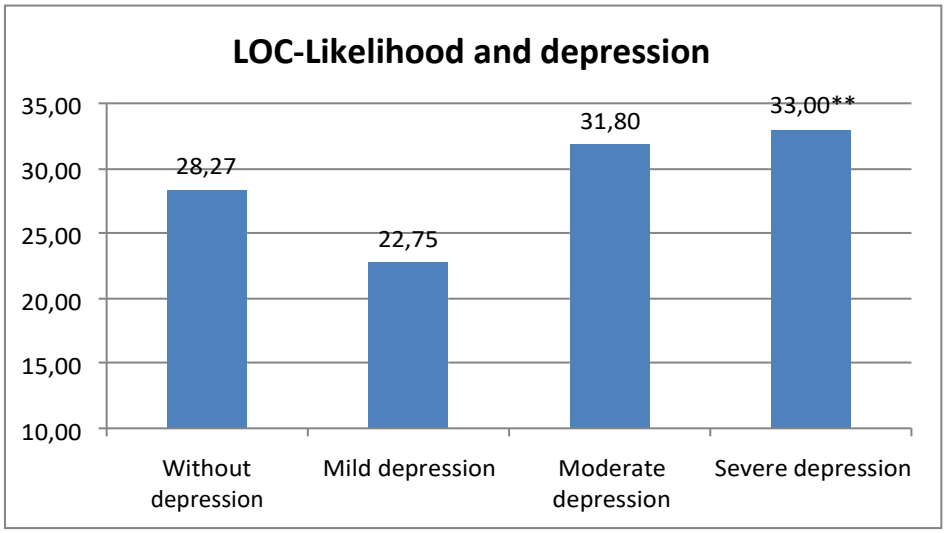
The Annalys of severity of depression according to the severity of current anxiety is shown in Graph 4. There is a significantly higer proprtion of patients with mild anxiety among patients without depression (66.5 %) compared to severe depression, where mild anxiety was not presented, only moderate in 35.6 %. Correlation analysis revealed a statistically significant positive correlation of severity of depression and severity of current anxiety ($C = 0.48$, $p < 0.05$) (Figure 4).

Correlation analysis revealed a significant po-sitive correlation among anxiety and fasting glicemia with strenght of depresion ($C = 0.59$ and $C = 0.45$, prospective), while this correlation was inverse with cholesterol values, Table 3.



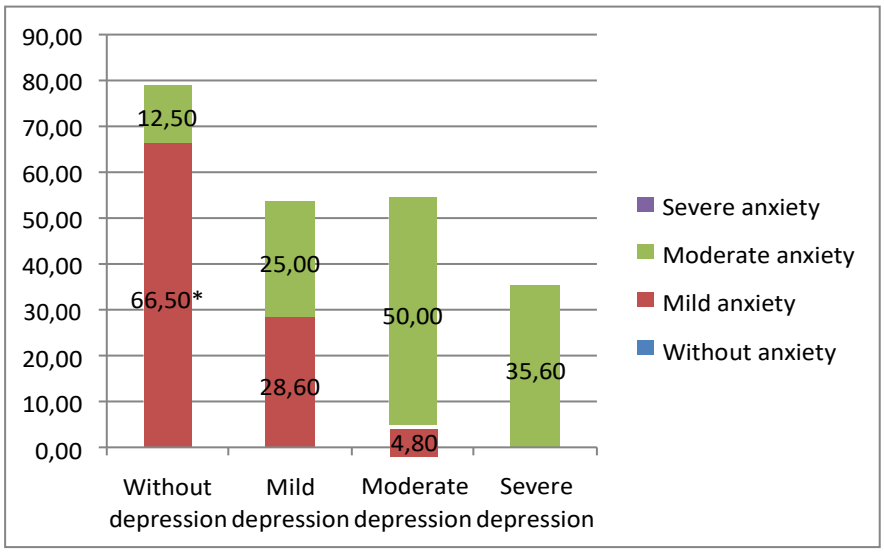

*$p < 0.05$ vs. without and mild depression, Hi square test

**Graph 2.** Incidence of actual anxiety and depression




**p < 0.01 vs. others

**Graph 3.** Average score of internal health locus of control and anxious personality traits




*p < 0.05 vs. moderate and severe depression, Hi square test

**Graph 4.** The current anxiety and depression

**Table 3.** Correlations of anxiety level and bichemical analyisis with depression

| | Pearson correlation coefficient | P value |
|---|---|---|
| Actual anxiety | 0.592 | 0.001 |
| Cholesterol | -0.382 | 0.045 |
| Glycemia | 0.452 | 0.014 |

## Discussion

To understand the occurrence of depression in patients with coronary artery disease it is required to understand that depression is neither the cause nor the result of coronary artery disease, but rather that it exists together in the same patient due to the patient having the same pathophysiological mechanism. It is possible that genetic dysfunction of serotonin receptors induces depression development and in platelets it could induce the risk of thrombotic events (1, 4, 5).

The relation between depression and coronary artery disease may be viewed through three different types of relationships. Firstly, depression may directly cause cardiac mortality through biological or behavioural mechanisms, as well as the type of ACS in-hospital and home therapy. Secondly, depression may be a consequence of the systemic complications of cardiovascular disease or its treatment. Finally, both depression and heart disease may share the common genetic and pathophysiological cause and have no causal correlation between each other (6).

Two other common pathophysiological mechanisms are low - intensity chronic inflammation and low intake of omega-3 fatty acids. Low - intensity chronic inflammation is an integral part of coronary artery disease. In patients with coronary artery disease markers of inflammation as CRP, IL6 or soluble intracellular adhesion molecules are high and are associated with a worse prognosis. They predict the development of CAD in a healthy population.

There is an interesting relation between depression and regulation of the immune function and inflammation. Meta-analyses of studies on patients without CAD concluded that major depression is associated with high leucocytes count, high CD4/CD8 ratio, and an increase of haptoglobin, prostaglandin E2, IL 6, lower natural killer cell cytotoxicity and low response on mitogens. Authors concluded that there is evidence that major depression is associated with one immune activation of reminiscent acute phase in inflammation response (5, 6).

Parallels can be seen between clinical data on inflammation and data in experimental studies. Animal experimental studies suggest that immune activation can induce depression similar behaviour and, conversely, that chronic stress induces the release of pro-inflammatory cytokines in the brain with consequent systemic responses. Pro-inflammatory cytokine-1 and tumor necrosis factor- alpha produced by activated immune cells promote the production of interleukin-6 from leukocytes, adipocytes or endothelial cells. Interleukin-6 induces broad systemic effects, including liver production of acute phase proteins (such as C-reactive protein) inhibition lipoprotein lipase activity, and increased platelet aggregation in response to ADP and epinephrine. Interestingly, systemic pro-inflammatory cytokines circulating in the blood also induce interleukin-1 activity in the hippocampus and hypothalamus, which acts as a messenger and SRES stimulate serotonin and norepinephrine neurotransmission and the release of corticotropin-releasing factor.

A conclusion can be drawn that the prognostic impact and prevalence of depression in patients with coronary artery disease may be attributed to the immune activation associated with progression of coronary artery disease score inducing depressive episodes in susceptible patients. The psychological concept of "vital exhaustion", defined as a combination of excess fatigue, irritability, and low morale may be particularly relevant for understanding the relationship between immune activation and symptoms of depression. Big epidemiological study of the elderly without coronary disease was found, although the C-reactive protein was associated with symptoms of depression and fatigue symptoms, the relationship remains independent of cardiovascular covariates and measures of physical weakness for symptoms of fatigue. It happens that the patient communicating vital exhaustion symptoms may be more likely to have concomitant immune activation than more common symptoms of depression. Finally, there is a statement about the normalization of inflammatory markers after antidepressant treatment (6).

In the discussion, we expect that higher level of depression and anxiety lead to higher blood glucose levels - both additional risk factors for the progression of coronary artery disease with an increase in the anxiety in already diagnosed patients which increases the risk for myocardial infarction, lethal outcome, and sudden coronary death. In addition to the direct effects on myocardial vulnerability, anxiety can lead to diabetes, hypertension, and hyperlipidemia. Chronically increased catecholamine levels were shown to increase the level of lipoprotein lipase induced hyperglycemia and increase blood pressure.

Considerable epidemiological evidence supports a link between chronic emotional stress and coronary heart disease /CHD/. Emotional factors related to atherosclerosis and adverse cardiac events include primarily disorders such as depression, anxiety, anger, and hostility. It is now well established that depression is associated not only with the incidence of CHD but also with the prognosis of patients with the disease. In meta-analysis relating to the role of depression in the development of coronary artery disease, Regulies found that individuals with clinical depression have > 2.5 fold increased risk of myocardial infarction or coronary death than the general population. In the patients with established coronary heart disease, major depression is not only a significant predictor of mortality after acute myocardial infarction, but also the level of depressive symptoms has a dose-dependent relationship with cardiac mortality over several years of monitoring. There is no evidence that could recommend systematic screening for depression in CAD patients. Self-reported questionnaire similar to BDI is quick and easy but it is too sensitive, and it gives many false positive diagnoses. However, systematic screening can overcome already scarce mental health resources available for a patient seeking psychiatric help. Instead, we need cardiologists to ask a few questions about mental health problems during regular visits. It can also encourage the patient to accept the referral to a psychiatrist when necessary. Studies have demonstrated that patients with CAD experience depression at a higher rate than the general population. Because of this connection, it is

critical to recognize depression and manage it effectively for people with CAD. Studies have also provided evidence that identifying and treating depression in patients early after myocardial infarction improve clinical outcomes. In addition, a number of studies have discussed the negative effects that can occur from untreated depression in those patients. The cited negative effects include mortality, recurrent myocardial events, and a worse quality of life (7). Some simple questions can be used to open a chapter of emotional distress without using the word depression, which often leads to rejection (8). Based on all of this evidence, it is important to diagnose and manage depression in patients with coronary artery disease. As such, depression screenings in cardiovascular disease care should be performed more often, because currently, they are under-performed due to the issue of optimal screening tolls cut-off (9).

## Conclusion

In our study, increased depression leads to increased anxiety and higher blood glucose levels - both additional risk factors for the progression of CAD. Patients with depression more likely had actual anxiety and will be treated by non- invasive medicamentous therapy for ACS. Increase in anxiety in patients diagnosed with the CAD increases the risk of MI, lethal outcome of coronary disease and poor further outcome. Depression is associated with an increased incidence of cardiac death and re-hospitalization, as well as continues with chronic depression.

To prevent the adverse impact of depression on the prognosis of acute coronary syndrome there should be promptly implemented psychiatric and therapeutic support.

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# UTICAJ DEPRESIJE NA TERAPIJU AKUTNOG KORONARNOG SINDROMA

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Epidemiološke studije ukazuju da su pored uobičajenih faktora za razvoj i prognozu akut-nog koronarnog sindroma, depresija i anksioznost bitni faktori. Depresija i koronarna arte-rijska bolest pojavljuju se simultano kod bolesnika zbog zajedničkog patofiziološkog mehanizma, odnosno mogućih genetskih disfunkcija seratoninskih receptora. Cilj ovog rada je ispitati incidenciju i ozbiljnost depresije i anksioznosti kod bolesnika sa akutnim koronarnim sindromom i dati uvid u to da li prisustvo depresije ima uticaj na odluku o tome da li treba pri-meniti invazivni ili neinvazivni pristup. Ispitivano je 38 bolesnika (23 muškaraca i 15 žena, starosti 63,5 ± 10 godina) lečenih na Klinici za kardiologiju Kliničkog centra Niš, sa akutnim koronarnim sindromom (ACS). Bolesnici su bili odvojeni po tipu ACS terapije: invazivna grupa (28 bolesnika) sa perkutanom koronarnom intervencijom i stentiranjem i druga grupa koja je bila podvrgnuta angiografiji bez indikacija za stent (neinvazivna grupa, 10 bolesnika). Odra-đeni su anamnestički klinički podaci, biomarkeri miokardne nekroze, standardna laboratorija, profil lipida, marker zapaljenja. Da bi se ispitao nivo depresije i anksioznosti koristili smo razli-čite upitnike: generalni upitnik o socio-demografskim podacima i podacima o razvoju bolesti, Beck Depression Inventory – upitnik koji meri intenzitet depresivnih simptoma i State and Trait Anxiety Inventory (STAI)-upitnik koji meri intenzitet aktuelne anksioznosti (state an-xiety) i anksioznost kao karakternu crtu (trait anxiety). Health Locus of Control – upitnik koji meri gde bolesnik postavlja centar kontrole bolesti. Po ozbiljnosti depresije formirali smo četiri grupe – 19 bolesnika bez depresije (50 %), 10 sa blagom depresijom (26,3 %), 8 sa srednjom depresijom (21 %) i jedan bolesnik sa teškom depresijom (2,7 %).

Najčešći faktor rizika bili su hipertenzija (81,6 %), poremećaj lipida (68,4 %) i poro-dična istorija kardiovaskularnih bolesti (52,6 %). Kardiovaskularni faktori rizika nisu se bitno razlikovali između polova. Prethodnu koronarnu bolest (CAD) imalo je 42,1 % hospitalizovanih bolesnika sa ACS, sa proporcionalnim brojem bolesnika oba pola. STEMI je bio najčešća kli-nička prezentacija ACS u 47,4 % bolesnika. Implantacija stenta urađena je kod 28 (73.6 %) bolesnika i medikamentozna terapija kod 10 (26.4 %) bolesnika. Pacijenti u neinvazivnoj grupi značajno su stariji, češće gojazni, sa hiperlipoproteinemijom, sa porodičnom istorijom CAD, sa anamnestičkim podacima prethodne AP i srčane insuficijencije i sa većim brojem otkucaja srca. Prisustvo depresije, posebno srednjih nivoa depresije, češći su u neinvazivnoj grupi (90,0 % i 50,0 % prospektivno) nego kod invazivne grupe (35,7 % i 10,7 %) ($p < 0,01$ i $p < 0,05$). Korelacija je pronađena između dužine trajanja hospitalizacije i nivoa povišenog šećera i depresije i anksioznosti koji ubrzavaju progresiju CAD.

Teža depresija vodi ka težoj anksioznosti i višim vrednostima šećera u krvi – od kojih su oba faktora rizika za progresiju CAD. Pacijenti sa depresijom češće su imali aktuelnu an-ksioznost i bili su lečeni neinvazivnom medikamentnom terapijom za ACS. Porast anksiozno-sti kod pacijenata sa dijagnozom CAD povećava rizik za MI, smrtni ishod.

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***Ključne reči:*** *akutni koronarni sindrom, depresija*