ARRHYTHMIAS IN ACUTE AND PERSISTENT DEPRESSION IN PATIENTS WITH MYOCARDIAL INFARCTION

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ABSTRACT. Acute and persistent depression is recognized as a risk factor for coronary artery disease. Significant depressive symptoms are found in 40-65 percent of patients with myocardial infarction. Depression is often persistent and exacerbates cardiac symptoms. It increases the frequency of arrhythmias, angina, morbidity and mortality (through sudden cardiac death). The therapy with serotonin reuptake inhibitor antidepressants improves depression and has favorable effects on the severity of cardiovascular symptoms, on morbidity and mortality.

Keywords: acute and persistent depression, arrhythmias, myocardial infarction.

1. INTRODUCTION

Coronary artery disease and depression are in close relation and several pathophysiological mechanisms are proposed:

1) Depression induces hyperactivity of the hypothalamic-pituitary-adrenocortical axis with hypercortisolism. Corticosteroids have atherogenic effects, including the worsening of hypertension and the elevation of cholesterol and probably influence arterial endothelial function. Depressed patients show hyper secretion of norepinephrine. Increased plasma catecholamine’s stimulate heart rate, rise blood pressure and myocardial oxygen consumption and are proarrhythmic. The incidence of ventricular tachyarrhythmias is increased in depressed patients with coronary artery disease (responsible for the excess cardiac mortality).

2) These patients have diminished heart rate variability, resulting from a relative increase of sympathetic tone and/or a relative decrease of parasympathetic tone, which increases the risk of fatal arrhythmias.

3) Depression may be accompanied by changes in platelet aggregability. Serotonin secretion plays a major role in depression and is also known to influence thrombogenesis and enhance platelet activation and responsiveness to other thrombogenic agents.

2. METHODOLOGY

The study group consisted from 50 patients admitted in the clinic of cardiology between June-December 2009 with MI and depression. They were assessed for depressive symptoms according to Beck Depression Inventory-BDI (a self-report instrument consisting of 21 questions, total score ranging from 0 to 63) recorded in hospital and at 3, 6, 9, and 12 months post-MI. Patients with BDI score 10 were assessed for the presence of depressive disorder according to International Classification of Diseases, 10th revision (ICD-10). All patients had an echocardiographic examination with determination of left ventricular ejection fraction (LVEF) and performed a stress test in the first month after infarction and, at 3 and 6 months. 30 patients had Holter monitoring at the same intervals.

Patients were divided into 4 categories according to their LVEF during hospitalization, i.e. LVEF
<30%, LVEF 30–45%, LVEF 45–60%, and LVEF 60%. During hospitalization, presence of depressive symptoms was higher in patients with LV disfunction.

Regarding antidepressive therapy the patients were divided in 2 groups: the first group received antidepressant therapy, but those from the second refused it or had a very low compliance. Frequency and severity of angina attacks, complications, morbidity and hospitalization rate were noted. The first includes 31 patients with MI and depression (7 with mild depression, 13 with moderate and 11 with severe form), 26 men and 5 women, aged between 43 and 74 years (mean age 58,4 ± 8,2 years). All patients received conventional therapy for myocardial infarction (beta-blockers, aspirin, statins and nitrates) associated with antidepressant therapy (23 with sertraline and 9 with thianeptine). 11 Patients abandoned this therapy after different time intervals (3 and 6 months) and were added to the next group. The second group includes 19 patients MI and depression (3 with mild form, 8 with moderate and 8 with severe depression), 16 men and 3 women, aged between 51 and 72 years (mean age 61,2 + 9,2 years). All patients received conventional therapy for myocardial infarction but they refused antidepressant therapy.

3. RESULTS AND DISCUSSIONS

The results concerning left ventricular performance (LVEF), tolerance to effort and Holter monitoring and quality of life questionaires obtained in the two groups of patients, in the first month after infarction and at 3 and 6 month are presented in the following tables. The patients treated with antidepressants had better results as those who refused this medication or those who abandoned therapy. The first group had fewer anginas and the duration of the attacks was reduced. The incidence of arrhythmias was smaller and their severity was reduced. Morbidity and quality of life was better as in the second group. A relationship was found between LVEF and ICD-10 depressive disorder, i.e. a lower LVEF was associated with a higher rate of depression from 3–12 months post-MI. Levels of LVEF inversely correlated with the BDI score at 3 months post-MI, see table 1.

<table>
<thead>
<tr>
<th>LVEF</th>
<th>Acute D</th>
<th>Persistent D</th>
<th>Severe D</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF 60%</td>
<td>BDI–10 P</td>
<td>BDI–21 P</td>
<td>BDI–19 P</td>
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<tr>
<td></td>
<td>3</td>
<td>10</td>
<td>6</td>
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<tr>
<td>LVEF 45–60%</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>LVEF 30–45%</td>
<td>2</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>LVEF &lt;30%</td>
<td>2</td>
<td>1</td>
<td>4</td>
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</tbody>
</table>

D = depression

The patients with depression had more anginas during stress test and terminate the test sooner. On the Holter monitoring they had a higher incidence of arrhythmias and a poorer quality of life following myocardial infarction. Depression affects compliance with medication and risk reduction measures and rehabilitation. Those patients smoke and drink more and are more likely to drop out exercise programs.

The patients with MI, treated for depression, had a better tolerance to stress test, reflected through a longer duration of stress, a higher double product, lesser ischemic changes and fewer arrhythmias comparative with those that refused antidepressant therapy or abandoned it after some months.

The patients with MI treated for depression had a lesser incidence of arrhythmias, expressed especially through isolated ventricular beats, there were few malignant ventricular arrhythmias and the severity of those was reduced after 6 months of therapy. The incidence of ischemic changes was reduced comparative with those found by patients that refused antidepressant therapy or abandoned it after some months. In the last group the incidence and severity of arrhythmias was higher. There was evidence of premature ventricular beats class Lown III and IV and also ventricular tachycardia. In some patients conduction disturbances (synusal pauses or episodes of atrio-ventricular blocks) were found. The patients from the treated group reported fewer episodes of angina pain, of palpitations and anxiety comparative with the other group.

4. CONCLUSIONS

1. Depression is a negative prognostic indicator for patients with chronic myocardial infarction. It is associated with increased morbidity, mortality and impaired quality of life.

2. In MI patients, the rate of depression and the severity of depressive symptoms are significantly related to the severity of LV dysfunction. The association between depression and LV dysfunction must be acknowledged when evaluating the prognostic effects of depression in cardiac patients.

3. Patients with MI and depression have more anginas during stress test and on Holter monitoring there is evidence of more arrhythmias.

4. Depressed patients with MI have a poorer adherence to the medical regimen, to the exercise program and to risk reduction measures.

5. Administration of antidepressant agents to patients with MI and depression should be considered as an additional therapy with the purpose to improve the quality of life, to increase adherence to the medical regimen, rehabilitation and risk reduction measures.
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6. This therapy seems to reduce the frequency of angina, disability and mortality (due to sudden cardiac arrest).

REFERENCES


