

Depression and coronary heart disease

relation to omega-3 fatty acids, homocysteine, platelet reactivity and coagulation

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Abstract

There is compelling evidence that depression is associated with elevated rates of cardiovascular morbidity and mortality. This elevation seems to be due to a significantly increased risk of coronary heart disease and myocardial infarction in depressed patients. There possibly related factors that have been recently highly concerned as having causal relation with both depression and IHD are an omega-3 fatty acids (omega-3 FAs) deficiency and elevated homocysteine level and exaggerated platelet reactivity. Hypercoagulable state is another variable that has been suggested to increase risk of IHD in depressed patients. In the current study we are trying to investigate the link between IHD and depression in relation to these factors, as well as to study whether or not the antidepressants has a role in ameliorating this risk. We studied 40 depressed patients and 20 control subjects. Both groups were submitted to determination of polyunsaturated fatty acids pattern, Measurement of homocysteine level, Assessment of platelet reactivity and coagulation. The patient group then received antidepressant therapy (paroxetine 20-40 mg/day) for 4-6 weeks and they were further subdivided into two subgroups according to their response to antidepressant therapy (improved and non improved patients) and a comparative study was done. Our results showed that depressed patients got significantly lower level of omega-3 FAs, higher level of homocysteine, higher levels of platelet reactivity indicators (platelet count, B-thromboglobulin level and platelet factor 4), higher levels of fibrinogen concentration and lower levels of tissue type thromboplasmin activator concentration compared to control group. After a course of antidepressant therapy improved depressed patients showed significant correction of most of these factors. Our finding that these risk factors including hypercoagulable state can be reduced by antidepressant medication may highlight the great importance of early and effective management of depression to reduce the risk of IHD in depressed patients.

Introduction

There is compelling evidence that depression is associated with elevated rates of cardiovascular morbidity and mortality. According to a group of UK researchers the risk of heart disease is doubled in people with depression (Musselman, 1996). The elevation in cardiovascular morbidity and mortality in depressed patients seems to be due to a significantly increased risk of coronary heart disease and myocardial infarction and once ischemic heart disease (IHD) is established, sudden cardiac death (Ford et al, 1998). Recent data suggest that the increased rates of IHD and myocardial infarction in patients with depression may be the result of one or more still unrecognized underlying physiological factors that predispose the patients to both depression and cardiovascular disease. Two possibly related factors that may have causal relation with both depression and IHD are an omega-3 fatty acids (omega-3 FAs) deficiency and elevated homocysteine level (Severus and Littman, 2001). Many studies suggested that long chain omega-3 polyunsaturated fatty acids (omega-3 PUFA), e.g. docosahexanoic acid (DHA) and eicosapentaenoic acid (EPA) derived mainly from

seafood -fish consumption- help to protect against both IHD (Kris-Etherton, 2002) and depression (Nemets et al 2002). In major depression some recent studies revealed significant depletion of omega-3 PUFAs and/or increase of the omega-6/omega-3 ratio in plasma, this was suggested as an important risk factor for IHD in depression (Mamalakis et al 2002). Homocysteine is another factor that have been recently linked to both depression and IHD. Homocysteine is an amino acid believed to be toxic to blood vessels, some recent studies have linked high levels of homocysteine to increased heart attack risk (Bottiglieri et al, 2000). A relationship between hyperhomocysteinemia (elevated plasma level of homocysteine) and depression has been also recently reported (Bottiglieri et al, 2001). Exaggerated platelet reactivity is a third factor that may have causal relation with both depressive disorders and cardiovascular disease. Depressed patients may display heightened platelet reactivity to mild cardiovascular challenge in comparison with normal subjects.