Alleviating Effect of Antidepressant Treatment on Psychiatric Symptoms and Cardiac Conditions in a Patient with Coronary Slow Flow Comorbid with Depression and Anxiety

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Patient: Female, 52
Final Diagnosis: Coronary slow flow
Symptoms: Frequent chest pain • serious TIMI grade 2 flow without any coronary stenosis • comorbid with depression and anxiety
Medication: —
Clinical Procedure: —
Specialty: Psychiatry

Objective: Rare co-existence of disease or pathology
Background: Depressive patients are considerably more likely to suffer cardiovascular disease (CVD), and in patients with CVD, depression is a predictor of poor outcome. Recent findings suggest higher rates of depression and anxiety in patients with coronary slow flow (CSF). However, there is no research investigating whether the antidepressant treatment can mitigate psychiatric symptoms and cardiac conditions in CSF patients comorbid with depression.

Case Report: The patient was a 52-year-old Chinese female with frequent chest pain. The patient had serious TIMI (thrombolysis in myocardial infarction) grade 2 flow without any coronary stenosis, but comorbid with depression and anxiety. The CSF was very likely associated with her mental health condition, given that the chest distress and intermittent chest pain followed psychological stress and disturbed sleep. Therefore, paroxetine was used under the circumstances of the poor effect of cardiovascular active drugs. We found that the adjunctive use of paroxetine not only improved the psychiatric symptoms, but also alleviated the cardiac conditions.

Conclusions: Our findings strengthen the importance of the treatment of psychiatric symptoms in patients with CSF and this finding should promote randomized controlled trials in a larger population to confirm the beneficial effects of antidepressant treatment on psychiatric symptoms and cardiac conditions in CSF patients with psycho-cardiac conditions.

MeSH Keywords: Antidepressive Agents • Anxiety • Cardiovascular Diseases • Depression

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Background

The phenomenon of coronary slow flow (CSF) is characterized by delayed opacification of the distal coronary vessels without any evidence of obstructive epicardial coronary disease, which was first described by Tambe et al. in 6 patients with angina pectoris [1]. Although several mechanisms, such as endothelial and microvascular dysfunction, diffuse atherosclerosis, inflammation and small vessel disease, have been indicated to be responsible in the pathogenesis of CSF, the exact processes remain elusive [2]. Accumulating evidence reveals a tight connection between depression and cardiovascular diseases (CVD). Higher rates of depression and anxiety were repeatedly found in patients with CSF [3,4]. Although the comorbidity and bidirectional relationship between depression and CVD has been extensively described, whether antidepressant treatment can mitigate CSF in patients comorbid with depression and anxiety is unknown.

Case Report

A 52-year-old Chinese female with frequent chest distress and 7 hours of severe retrosternal chest pain was referred to our hospital in December 2016. Her blood pressure was 123/73 mm Hg, her heart rate was 72 beats/min and her cardiac and pulmonary auscultatory results were normal. The patient did not have any alcohol or tobacco addiction. The circulating concentrations of troponin T, creatine kinase (CK), and CK-MB, as well as B-type natriuretic peptide (BNP, or its N-terminal proform NT-proBNP) and D-dimer were within normal limits, ruling out acute myocardial infarction. The fasting lipid panel and other routine blood tests were also within normal limits. The electrocardiogram showed inhibited sinus rhythm and non-specific ST-T changes with normal QTc.

The patient was taken to the cardiac catheterization laboratory for coronary angiography that showed no significant epicardial coronary stenosis. The thrombolysis in myocardial infarction (TIMI) frame-count method was used to evaluate the degree of the slow antegrade filling. The patient had serious TIMI 2 flow and the corrected TIMI frame counts were observed to be 59 frames for the left anterior descending coronary artery. The TIMI frame count for the right coronary artery was 45 and the left circumflex artery was 61. The patient was initially treated with oral nitrates, nicorandil, diltiazem, and low molecular heparin. After 3 days of treatment, the severe chest pain was alleviated. However, the patient still complained about chest distress and intermittent chest pain, especially when she did not sleep well or experienced psychological stress such as family issues. On inquiry, she elaborated that she recently had felt anxious for her son’s college entrance examination and her sleep was disturbed. She also complained of irritability, loss of interest in all works, tiredness throughout the day, and burning sensation in her abdomen. Therefore, the mental health condition of the patient was evaluated by an expert psychiatrist by using the Generalized Anxiety Disorder (GAD-7) scale, the Patient Health Questionnaire (PHQ)-9 and the Hospital Anxiety and Depression (HAD) scale. As shown in Table 1, the patient presented with depressive and anxious symptoms. Based on these conditions, the selective serotonin reuptake inhibitor (SSRI), paroxetine was used in addition to the cardiovascular active drugs. In 7 weeks of follow-up, as illustrated in Table 1, the adjunctive use of paroxetine not only improved the psychiatric symptoms, but also alleviated the cardiac conditions, with significant improvement in the 5 dimensions of the Seattle Angina Questionnaire (SAQ), including anginal frequency, physical limitation, anginal stability, disease perception, and treatment satisfaction.

Discussion

Depressive patients are considerably more likely to suffer CVD, and in patients with CVD, depression is a predictor of poor outcomes [5]. The comorbidity of depression and CVD, such as CSF, is associated with hyperactivity of the hypothalamic-pituitary-adrenal axis, overactivation of the sympathetic nervous system, disturbances in platelet reactivity and inflammation [6,7]. Although the well-recognized comorbidity worsens
the cardiovascular prognosis, the beneficial effects of antidepressant treatment on cardiovascular outcomes are controversial [8]. Higher prevalence of depression and anxiety has been repeatedly observed in patients with CSF [3,4]. However, there have been no studies or case reports investigating the management of depression in patients with CSF. In this case, we presented a severely CSF patient with normal coronary arteries, and comorbid with depressive and anxious symptoms. The CSF in this case was very likely associated with her mental health condition, given her chest distress and intermittent chest pain following psychological stress and disturbed sleep. SAQ evaluation results have shown that the use of paroxetine improves the cardiac conditions, including anginal frequency, physical limitation, anginal stability, disease perception, and treatment satisfaction. Additionally, considering the intolerable pain in measuring the coronary flow by using TIMI, the degree of the coronary flow was not evaluated after the use of paroxetine. Significantly, SSRIs such as paroxetine have an anti-platelet factor which may be beneficial in patients with pain related to coronary ischemia [9], thus its alleviating effects on psychiatric symptoms and cardiac conditions in patients with CSF comorbid with depression and anxiety require further explanation.

Conclusions

We have shown the improvement of cardiac symptoms in a patient with CSF by the mid-term treatment with antidepressants. These findings strengthen the importance of treatment of psychiatric symptoms in patients with CSF. However, the beneficial effects of such treatment in patients with psychocardiac conditions should be confirmed by the randomized controlled trials in a larger population of patients.

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Conflict of interest

None.

References:

1. Tambe AA, Demany MA, Zimmerman HA, Mascarenhas E: Angina pectoris and slow flow velocity of dye in coronary arteries – a new angiographic finding. Am Heart J, 1972; 84: 66–71
2. Wang X, Nie SP: The coronary slow flow phenomenon: Characteristics, mechanisms and implications. Cardiovasc Diagn Ther, 2011; 1: 37–43
3. Durmaz T, Keles T, Erdogan KE et al: Coronary slow flow is associated with depression and anxiety. Acta Cardiol Sin, 2014; 30: 197–203
4. Yalvac D, Ozturk S, Sivri N et al: Effects of patients anxiety and depression scores on coronary flow in patients with normal coronary arteries. Int J Cardiol, 2014; 180: 55–57
5. Amare AT, Schubert KO, Klinglerhoffmann M et al: The genetic overlap between mood disorders and cardiometabolic diseases: A systematic review of genome wide and candidate gene studies. Transl Psychiatry, 2017; 7: e1007
6. Carney RM, Freedland KE: Depression and coronary heart disease. Nat Rev Cardiol, 2017; 14: 145–55
7. Carnevali L, Montano N, Statello R, Sgoifo A: Rodent models of depression-cardiovascular comorbidity: Bridging the known to the new. Neurosci Biobehav Rev, 2017; 76: 144–53
8. Shapiro PA: Management of depression after myocardial infarction. Curr Cardiol Rep, 2015; 17: 80
9. Hergovich N, Aigner M, Eichler HG et al: Paroxetine decreases platelet serotonin storage and platelet function in human beings. Clin Pharmacol Ther, 2000; 68: 435–42