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Follow Up platelet functional changes in Depression and Heart disease

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Comorbid depression in patients with cardiovascular disease carries higher rates of morbidity and mortality as has been previously documented. As such, several mechanisms have been proposed to account for this increased risk. We have examined whether platelet activation and in particular platelet serotonin signaling can play a part of this increased morbidity. Of particular importance is the timing of biological assessments in depression and heart disease. Many studies have assessed platelet function and other biological factors several months after the initial cardiac event. We have assessed platelet function in a cohort of both stable coronary artery disease and acute coronary syndrome patients (ACS) with and without depression within 72 hours after presentation with a repeat assessment at a one-month follow up period. We hypothesize that depressed ACS patients but not depressed stable CAD patients would have persistently increased platelet activation after a one month follow up period.

Methods: A total of 300 patients with CAD were enrolled (145 with acute coronary syndrome and 155 with stable coronary artery disease). Of these 262 returned for a second visit at a one-month follow up period. Symptoms of depression were assessed by the Beck Depression Inventory – II scale (BDI-II) and major depression was assessed using the Structured Clinical Interview for DSM III-R (SCID) at both visits. Platelet activation was measured by PAC1 expression. Platelet serotonin response was measured by serotonin augmented aggregation and direct platelet serotonin activation.

Results: ACS patients had a reduction in the rate for depressive symptoms (BDI $\geq$ 10) from 48% to 33.6%, p=0.0017 compared to the stable CAD group who had no significant reduction in the rate of depressive symptoms, 24.5% at first visit compared to 20.5% at follow up visit, p=0.18. The overall level of MDD did not change at the one month follow up in the overall group which was 14% in the entire group on the first visit and at the one month follow up it was 11.4%, p=0.13. Of note ACS patients had no difference in their level of MDD which was 18.7% on presentation and 17.6% at follow up. Depressed ACS (N=53) and depressed stable CAD (N=37) (BDI ≥ 10) patients have decreased platelet activation as measured by change in PAC1 and serotonin response from visit 1 to visit 2: PAC 1 change: -10.82%, p=0.0006 for ACS, -5.59%, p=0.03 stable CAD, Serotonin (0.03-30uM) change: -6.68 to -9.05%, p=0.001-0.01 for ACS, -4.71 to -7.15%, p=0.01-0.15 for stable CAD. Non-depressed (BDI<10) ACS patients had no difference in PAC 1 platelet activation p=0.06 but had decreased platelet serotonin response from visit 1 to visit 2: Serotonin (0.03-30uM) change: -4.97 to -6.83% p=0.01-0.03. Non-depressed stable CAD patients had no significant change in PAC-1 or platelet serotonin response (p=NS).

Conclusion: Persistent platelet activation is not evident by assessment of platelet response to serotonin in depressed patients with either stable or unstable CAD.

Additional platelet functional assessment may be warranted in comorbid depression and cardiovascular disease.

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