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Anhedonia predicts adverse cardiac events in people with acute coronary syndromes

**QUESTION**

**Question:** Does anhedonia and/or depressed mood predict recurrent major adverse cardiac events (MACEs) in people with acute coronary syndromes (ACS)?

**Population:** Consecutive sample of 453 people (58% male, mean age 61 years, range 25–93 years) hospitalised for ACS: 21% with acute myocardial infarction with ST-segment elevation, 33% with acute myocardial infarction without ST-segment elevation and 46% with unstable angina. To be included, participants had to either be not depressed (scoring 0–4 on the Beck Depression Inventory, BDI) or have at least mild depression (scoring ≥10 on the BDI). People scoring 5–9 on the BDI were excluded to either be not depressed (scoring 0–4 on the Beck Depression Inventory, BDI) or have at least mild depression (scoring ≥10 on the BDI). People scoring 5–9 on the BDI were excluded to either be not depressed (scoring 0–4 on the Beck Depression Inventory, BDI) or have at least mild depression (scoring ≥10 on the BDI). People scoring 5–9 on the BDI were excluded to either be not depressed (scoring 0–4 on the Beck Depression Inventory, BDI) or have at least mild depression (scoring ≥10 on the BDI). People scoring 5–9 on the BDI were excluded to either be not depressed (scoring 0–4 on the Beck Depression Inventory, BDI) or have at least mild depression (scoring ≥10 on the BDI).

**Setting:** Cardiac units in three university hospitals, New York and Connecticut, USA; recruitment May 2003 to June 2005.

**Prognostic factors:** Anhedonia and depressed mood (clinical or patient assessed), depressive symptoms severity (BDI score <5 vs ≥10), major depressive episodes (MDEs). Participants had a semistructured diagnostic interview 1 week after admission for ACS. The interview assessed anhedonia, depressed mood and Diagnostic and Statistical Manual, Fourth Edition MDEs. Symptoms had to be present for at least 2 weeks and clinically impairing for clinician-rated anhedonia or depressed mood to be diagnosed. Depression symptom severity was assessed with the BDI; and patient-reported anhedonia and depressed mood were assessed using the BDI items for sadness and crying for depressed mood and loss of enjoyment and loss of interest for anhedonia. The scores for these items were summed to create a score of 0–6 for each of these areas and used to place participants in a low or medium group (scores 0–3), and a high group (scored 4–6) which indicated severe depressed mood or anhedonia. Medical records and patient histories were used to provide data for medical covariates including the Global Registry of Acute Coronary Events risk score (range 1–265 points – higher scores having higher mortality risk), Charlson comorbidity index, left ventricular ejection fraction and anti-depressant use. Cox proportional hazard models were used to estimate the HR for MACEs and all-cause mortality. Analyses were adjusted for age, sex and medical covariates.

**Outcomes:** MACEs (urgent or emergency coronary revascularisation, hospitalisation for unstable angina, myocardial infarction) or all-cause mortality. Participants were contacted at 1, 3, 6 and 18 months after the index ACS event by telephone or in person. Hospital records were used to provide supporting documentation of outcomes.

**METHODS**

**Design:** Cohort study.

**Follow-up period:** 12 months.

**MAIN RESULTS**

Clinician-rated anhedonia was diagnosed in 24% (n=108), depressed mood in 17% (n=77) and MDE in 11% (n=48) at baseline. During follow-up, there were 67 events (14.8%) comprising 17 deaths and 50 MACEs. MDE was a significant predictor of MACE and all-cause mortality in adjusted analyses, but depressive symptom severity was not (see table 1). Both anhedonia and depressed mood predicted age-adjusted MACE or all-cause mortality, but when medical covariates, age and sex were controlled for, only anhedonia predicted these outcomes (anhedonia: HR 1.58, 95% CI 1.16 to 2.14, p<0.01; depressed mood: HR 1.28, 95% CI 0.96 to 1.71, p=0.09; see table 1). Further adjusting the depressed mood analysis for anhedonia and vice versa produced similar findings (see table 1). Anhedonia also remained a significant predictor after adjustment for MDE or depressive symptom severity (see table 1). Results for patient-rated symptoms was similar with patient-reported anhedonia significantly predicting MACE or all-cause mortality (HR 2.26, 95% CI 1.33 to 3.82, p=0.002) but patient-reported depressed mood not a significant predictor (HR 0.86, 95% CI 0.51 to 1.43; p=0.55).

**CONCLUSIONS**

Anhedonia is a risk factor for recurrent adverse coronary events in people with ACS independent of other prognostic factors including major depressive episode and depressive symptom severity. Depressed mood does not appear to be an independent risk factor.

**ABSTRACTED FROM**


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**COMMENTARY**

Davidson and colleagues build on recent research which tries to disentangle the association between symptoms of depression and cardiovascular prognosis. For example, de Jonge and colleagues showed that somatic/affective depressive symptoms predicted cardiovascular mortality, but that cognitive/affective symptoms did not. Davidson and colleagues’ results are more specific in that they concentrated on two core diagnostic symptoms – anhedonia and depressed mood – with only anhedonia being a consistent predictor of prognosis. The results require replication, as other recent findings are somewhat contradictory. The generalisability of the results may be affected by the lower participation rates of Hispanic and older patients, while the use of all-cause mortality as an end point could be a further biasing factor. Elimination of those with intermediate levels of depressive symptoms is another acknowledged limitation. Innovatively, the authors showed that anhedonia remained predictive when controlling for the effects of major depression but also somatic depressive symptoms. While screening for anhedonia could easily be included in clinical practice, it is perhaps unlikely to be so, given the relatively ineffective interventions for depression available in this population. Furthermore, as the authors point out, it is unknown if these treatments affect anhedonia levels specifically. The results highlight the probability that the heterogeneity of major depression criteria has hindered research into this area. Re-categorisation of the diagnostic criteria into more specific subsets may aid future research, for example, the use of categories such as ‘anhedonic depression’. Overall, information from such studies will likely inform more sophisticated, and hopefully successful, intervention trials in cardiac patients in the future.