
Q Does depression after a myocardial infarction affect cardiovascular prognosis and survival?

METHODS

Main inclusion criteria: studies comparing cardiac prognosis in depressed versus non-depressed people admitted for a myocardial infarction; depression had to be diagnosed within three months of the initial myocardial infarction. Analysis: the random effects and fixed models were used to carry out meta-analyses.

Outcomes: All-cause and cardiac mortality; cardiovascular events.

MAIN RESULTS

Twenty two studies met inclusion criteria (n = 6367). All-cause and cardiac mortality were increased in depressed versus non-depressed people (all cause mortality: 96952 (10.1%) with depression vs 116/2130 (5.5%) with no depression, n = 3082, OR 2.38, 95% CI 1.76 to 3.22, p<0.00001; cardiac mortality: 62/1091 (5.8%) with depression vs 57/2252 (2.6%) with no depression, n = 3343, OR 2.59, 95% CI 1.77 to 3.77, p<0.00001). Cardiovascular events were increased in depressed versus non-depressed people (cardiovascular events: 325/1078 (30.2%) with depression vs 449/2323 (19.4%), n = 3401, OR 1.95, 95% CI 1.33 to 2.85).

CONCLUSIONS

All-cause and cardiac mortality plus cardiovascular events are more common in depressed versus non-depressed people after a myocardial infarction.

NOTES

The authors note there is some evidence for selective reporting of cardiovascular mortality results in the medical literature. The direction of the association between depression and outcome was almost invariably attenuated from the bivariate association. It is unfortunate that so few studies present multivariate analyses for consideration and in their absence, we should, as the authors state, “remain careful before making causal inferences”.

Whether or not depression following MI is an independent risk factor for prognosis is an important public health matter. To date, only one randomised controlled trial in this population has been published; cognitive behaviour therapy, supplemented when necessary with antidepressive medication, was associated with significant reductions in depressive symptomatology, but had no effect on all-cause mortality or recurrent non-fatal MI.5 However, outcomes other than mortality and cardiac morbidity need to be considered. The MIND-IT trial,6 in addition to investigating the impact of antidepressant medication on mortality following MI, also considers its impact on quality of life. The findings are due to be published soon. Even if it shows that treating depression affects quality of life in MI patients, but not mortality or morbidity, this will be an important result. Given the high prevalence and persistence of depression during the first 12 months following MI, as well as its detrimental impact on participation in cardiac rehabilitation and compliance with medication, reduced quality of life should be a sufficient imperative for treatment.

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Commentary

The precise nature of the relation between depression following myocardial infarction (MI) and prognosis has been the subject of much recent debate1–3 and the relevant prospective observational studies have now been meta-analysed by van Melle et al. The pooled odds ratios—2.38, 2.59, and 1.95—for all-cause mortality, cardiac mortality, and cardiovascular events respectively, indicated a significantly poorer prognosis for those who were depressed following MI. The means of assessing depression (depressive symptomatology or major depressive disorder) and the length of follow up (0–6 months and >6 months) did not influence the strength of the association between depression and outcome. However, as the authors readily concede, these conclusions are based on pooling bivariate analyses, not multivariate analyses. As has been argued previously,6 adjusting for potential confounders, such as disease severity, is necessary to determine whether depression following MI is an independent risk factor—that is, causative—in this context. van Melle et al note that where multivariate analyses were performed, the association between depression and outcome was almost invariably attenuated from the bivariate association. It is unfortunate that so few studies present multivariate analyses for consideration and in their absence, we should, as the authors state, “remain careful before making causal inferences”.


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