Use of Selective Serotonin Reuptake Inhibitors and Myocardial Infarction

To the Editor:

In a recent study, Sauer et al. showed that the use of selective serotonin reuptake inhibitor antidepressants (SSRIs) may confer a protective effect against myocardial infarction (MI) in smokers. They hypothesized that this beneficial effect of SSRIs in reducing the risk of MI could mainly be attributed to the inhibitory effect on serotonin-mediated platelet activation in addition to treatment of depression itself. In contrast to these results, in a recent larger population-based case-control study, Meier et al. found that SSRI exposure adjusted for age, sex, geography, calendar time, smoking status, and body mass index was not associated with altered risk of developing first-time MI. MI risk estimates were also not influenced by timing, duration, and dosing of SSRIs. In addition, this analysis yielded no difference between patients using SSRIs or non-SSRIs, including tricyclic antidepressants (TCAs), with regard to MI. The reason for the discrepancy between results of the two aforementioned studies is not clear.

The proposed mechanism underlying the putative cardioprotective effect of SSRIs is an interference with serotonin handling decreasing platelet aggregability. Indeed, there are case reports extant associating use of SSRIs with abnormal platelet aggregation. Although inhibition of platelet aggregation may also result in bleeding disorders, a recent study provided weak, if any, evidence for a link between SSRIs and precipitation of bleeding events at a population level.

However, in addition to their putative effect on platelet function, SSRIs are thought to have clinically important cardiac and vascular effects as well. Surprisingly, in a recently study, the use of SSRIs similarly to TCAs was associated with cases of first-degree atrioventricular block, prolonged QTc interval and orthostatic hypotension in patients lacking cardiovascular disorders, raising new concerns about the putative cardiovascular safety of these compounds. Importantly, SSRI antidepressants, similarly to TCAs, also showed cardiovascular depressant effects in different isolated cardiovascular preparation by inhibiting cardiac and vascular ion channels. To date, it is not known whether SSR-induced cardiodepressant effects will promote arrhythmias and decompensation in patients with myocardial infarction. Thus, we should remain cautious in drawing too early conclusions of such large significance about beneficial effects of SSRIs in patients with risk for MI until further information is available on larger population based studies. Whether SSRIs exert any beneficial effect against the development of MI is an important issue that requires further evaluation.

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