Psychosis, depression, and high risk for sudden cardiac death: time for co-operation between psychiatrists and cardiologists

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This editorial refers to ‘Psychotropic medications and the risk of sudden cardiac death during an acute coronary event’¹, by J. Honkola et al., on page 745

Psychiatric patients have an excess rate of unexplained sudden death.¹ Some studies have linked this to the disease itself and to an increased prevalence of other risk factors such as dyslipidaemia and diabetes in this population.² However, a number of observations have suggested that the use of antipsychotic and antidepressant drugs might be at the root of these deaths.³

The work coming from the University of Oulu⁴ can bring some clarity to this debate. Is this a population at higher risk for sudden cardiac death who should be screened and treated accordingly, or is the treatment they receive the reason for the excess mortality, and thus the drugs administered to them should be carefully selected based on the probability of a proarrhythmic effect of the therapy? The study by Honkola and co-authors⁴ focuses on the incidence of sudden death related to an acute myocardial infarction (MI). Patients dying suddenly in the acute phase of an MI are compared with a cohort of patients surviving the MI. Comparison of different parameters, including previous use of antipsychotics and antidepressant drugs, is carried out in both groups. Such a study can only be performed if a very well designed long-term strategy has been implemented. The authors, and the University of Oulu, should be congratulated on such a large cohort of patients being extremely well analysed and classified. In total almost 3000 patients are included, 1800 of them with an acute MI proven at autopsy as the cause of sudden death and the remainder being survivors of a clinically proven MI. Sudden death victims during an acute MI were more frequently consumers of antipsychotic drugs than survivors [odds ratio (OR) 3.8], particularly malignant being the combination of phenothiazine and any antidepressant drug (OR 18.3).

In a very large previous study² it was shown that the use of antipsychotic drugs increases (up to three times) the risk of sudden death in a dose-dependent fashion. In the study of Honkola et al., there was no information on the final cause of death, but presumably many of the subjects could have had an MI as a precipitating factor. Interestingly, in another study with a very long-term follow-up (up to 26 years), the high incidence of diabetes and dyslipidaemia (and, thus, a higher incidence of coronary events) in the psychiatric population was considered the cause of the excess mortality.² Combining these two observations, it is quite possible that psychotropic drug users in fact represent a high-risk population for coronary events due to increased presence of classical risk factors. Use of drugs with a potential proarrhythmic effect can be very deleterious in the case of occurrence of an acute coronary event. Many studies have linked the use of different drugs to the presence of changes in the electrophysiological properties of the heart, especially the QT interval in basal conditions.⁵–⁷ The possibility of changes in the QT interval and its relationship with the occurrence of torsade de pointes is a mandatory investigation in any study of a new drug. However, all this new evidence requires that research should be expanded to look at the triggering effect that coronary events might represent, and how the use of drugs in ischaemic conditions can be a precipitating factor for sudden death. It is probably not enough to know whether the drug does or does not modify the ECG parameters (the QT interval in particular), we need to know also whether these drugs have some unwanted added effect in cells under extreme conditions, especially ischaemia. If this is the case, then users of these drugs should be carefully selected, and a clear cardiovascular risk profile should be obtained in each one of them before initiating any pharmacological therapy. Those showing a high-risk profile for cardiac events should be carefully evaluated for other options or for psychotropic drugs with a safer cardiovascular profile.

We are probably facing a new challenge: patients with psychiatric disorders are at risk of coronary events; drugs administered to them can be proarrhythmic, especially during ischaemic conditions. Careful selection of patients who are candidates to receive psychotropic drugs, according to their cardiovascular risk profile, is mandatory if we do not want to harm them. Cardiologists and psychiatrists should start thinking of the best way to establish
good and reliable links between the two specialities in order to best treat what in fact are common patients.

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References