

CARDIOTOXICITY ASSOCIATED WITH METHAMPHETAMINE USE AND SIGNS OF CARDIOVASCULAR PATHOLOGY AMONG METHAMPHETAMINE USERS

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EXECUTIVE SUMMARY

BACKGROUND

The use of methamphetamine is widespread and, in many countries, is a major drug of abuse. As such, it is important to identify and understand the adverse health effects associated with methamphetamine use and consider the risk of such consequences for users. Although methamphetamine has effects on multiple organ systems, this report will focus on the cardiovascular effects of methamphetamine. Specifically, the aim of this report is to review the evidence for methamphetamine-related cardiovascular pathology and discuss the implications for methamphetamine users.

Methamphetamine cardiotoxicity

Methamphetamine increases catecholamine activity in the branch of the peripheral nervous system responsible for modulating heart rate and blood pressure. Excessive catecholamine activity is thought to be the primary mechanism underlying the cardiotoxic effects of methamphetamine. High catecholamine levels are known to be cardiotoxic, causing narrowing and spasm of the blood vessels, rapid heart rate (tachycardia), high blood pressure (hypertension), and possible death of the heart muscle. Other features of catecholamine toxicity include the formation of fibrous tissue and an increase in the size of heart muscle cells.

Evidence of cardiotoxicity among methamphetamine users

The most widely reported adverse cardiovascular effects of methamphetamine use are chest pain, tachycardia and other cardiac arrhythmias, shortness of breath and high blood pressure. The less frequently observed, but more severe, acute cardiovascular complications of methamphetamine use are acute myocardial infarction, acute aortic dissection, and sudden cardiac death. The medical literature contained several single case reports and case series reports of acute myocardial infarction. Acute myocardial infarction often occurred in the absence of identifiable coronary artery disease.

The forms of chronic cardiovascular disease that are most commonly associated with methamphetamine use are coronary artery disease and cardiomyopathy. Studies of methamphetamine-related fatalities have suggested that methamphetamine users are at risk of the premature and accelerated development of coronary artery disease. Clinical and experimental evidence alike suggest that the use of methamphetamine, particularly long-term use, can induce cardiomyopathy. As with acute myocardial infarction, cardiomyopathy has been associated with various routes of methamphetamine administration (e.g. oral, smoking and intravenous).

Factors influencing the cardiovascular effects of methamphetamine

The necessary and sufficient dose to produce serious cardiovascular complications or death - that is, the “toxic” dose - is unclear, as the response to a specific dose varies due to individual differences in responsiveness and variations in degree of tolerance. The literature indicates that cardiovascular complications associated with methamphetamine use can occur with all of the major routes of administration: that is, intranasal, oral, smoking, and injecting. While there is no evidence to suggest that any one route of methamphetamine administration should be more strongly associated with cardiotoxicity than another, the risk of complications may be higher with patterns of use that are associated with frequent use and taking higher doses, such as injecting and smoking crystalline methamphetamine. Previous research also suggests that the risk of cardiovascular problems among methamphetamine users is increased when the drug is combined with alcohol, cocaine or opiates. Of particular concern is the concomitant use of methamphetamine and other psychostimulant drugs, such as cocaine, due to their potential synergistic effect on catecholamine activity.

Conclusions and recommendations

Low level use of methamphetamine - for example, sporadic, low dosage use - does not appear to be associated with major acute complications, such as myocardial infarction, or chronic cardiovascular disease, in an otherwise healthy user. Methamphetamine may, however, exacerbate pre-existing underlying cardiac pathology, such as coronary atherosclerosis or cardiomyopathy, thereby increasing the risk of an acute event such as myocardial infarction or even sudden cardiac death. Long-term methamphetamine users appear to be most at risk of cardiovascular damage, such as premature, accelerated coronary artery disease. As such, methamphetamine toxicity is more likely to have a fatal outcome with chronic use.

Given their high levels of polydrug use, methamphetamine users should also be made aware of the increased risk of adverse cardiovascular effects when methamphetamine is used with other drugs, particularly other psychostimulant drugs. Because of the individual variation in sensitivity to methamphetamine’s cardiotoxic properties, treating methamphetamine toxicity should be based on the symptom presentation rather than the reported dose administered.

Further research is needed to establish the risk of serious cardiac events among methamphetamine users, whether there is evidence of a dose-response relationship between methamphetamine use and cardio toxicity in humans, and also the relative contribution of methamphetamine over other concurrent risk factors, such as tobacco smoking, alcohol and other drug use, obesity, and pre-existing cardiac pathology.