H99 Aortic Dissection in Cocaine Abuse: A Fatal Case

Stefano D'Errico, MD, Department of Legal Medicine, Azienda USL Nordovest, Cittadella della Salute, Lucca 55100, ITALY; Sara Niballi, MD, Department of Legal Medicine, Azienda USL Toscana Nordovest, Cittadella della Salute, Lucca, ITALY; Benedetta Guidi, MD, Via Santa Dorotea 1, Pescia, Pistoia 51017, ITALY; Marco Conti, MD, Department of Legal Medicine, AUSL2, Lucca, AB, ITALY; Diana Bonuccelli, MD, Department of Legal Medicine, Cittadella della Salute, Lucca, ITALY; and Anna Talarico, University of Ferrara, Via fossato di mortara 70, FERRARA 44121, ITALY*

The goal of this presentation is to introduce a fatal case of aortic dissection after cocaine abuse. A complete postmortem examination and toxicological analysis revealed the cause of death.

This presentation will impact the forensic community by examining the rarity of fatal cases of aortic dissection in cocaine abusers and by emphasizing the importance for complete postmortem examinations in these cases.

Aortic dissection is generally thought to require a pathogenic co-factor, usually severe arterial hypertension or a connective tissue disorder and a predisposition to weakness in the aortic media and sudden changes in hemodynamic shear stress. The association between cocaine abuse and aortic dissection is well documented in the literature, but few fatal cases are reported. Presumably, the mechanisms relate first to an underlying process that has weakened the elastic media of the aorta and, second, to the severe sheer forces that result from the sudden and profound hypertension and tachycardia that accompany cocaine (particularly crack) use. Cocaine, by inhibiting the reuptake of both epinephrine and norepinephrine at the neural synapses, leads to profound sympathetic stimulation that presumably causes such sheer stress on the aorta's intima that a small "nick" or tear occurs. This physiology is particularly acute with the use of crack cocaine, after which the onset of systemic effects is almost immediate. With the use of cocaine, such tears may occur most often at the ligamentum arteriosum because this region of the aorta is relatively fixed anatomically and is less able to withstand the accelerating aortic pressure wave that speeds down the aorta after ventricular contraction. Once such an intimal tear has occurred, the weakened aortic wall allows entry of luminal blood, followed by propagation of the dissecting hematoma down (and/or up) the aorta. A second possible mechanism is that chronic cocaine use itself may lead to premature atherosclerosis. It has been postulated that recurrent cocaine exposure makes the endothelium more permeable to atherogenic low-density lipoprotein and may accelerate the migration of leukocytes to the aortic wall. Thus, predisposition to aortic dissection could include not only the impact of hypertension, but chronic cocaine's effects on the aorta as well.

Case Report: This study presents the case of a 50-year-old man found lifeless in his car that was parked in front of a disco. Medical history was unremarkable for acute cardiac problems, but he was known as a cocaine abuser. A complete postmortem examination was performed the day after death; type A (DeBakey type II) aortic dissection was detected and cardiac tamponade recorded as the cause of death. Samples of organs were collected for a complete histopathological study. Toxicological examination was performed and confirmed the suspicion of cocaine abuse.

Aortic Dissection, Cocaine, Death

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*Presenting Author