G61 Aortic Dissection Due to Cocaine Use: A Case Series

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After attending this presentation, attendees will gain an understanding of the clinicopathology and the importance of aortic dissection due to cocaine use.

This presentation will impact the forensic science community by discussing the epidemiology, pharmacology, and pathophysiology of cocaine-induced aortic dissection. This topic is important for the forensic science community, but statistics on this occurrence are not well documented. Data accumulated by the Wayne County Medical Examiner's Office on the incidence of cocaine-induced aortic dissection will be reviewed.

Cocaine is a controlled Drug Enforcement Administration (DEA) Schedule II drug, with reliable lab detection. Cocaine is rapidly metabolized to the inactive compounds benzoylecgonine and ecgonine methyl ester. Ecgonine methyl ester is further hydrolyzed to ecgonine. Cocaine can be metabolized to benzoylecgonine even after death and sample collection. Active metabolites include cocaethylene (cocaine-ethanol byproduct) and norcocaine.

Cocaine is a naturally-occurring stimulant that affects the central nervous system by increasing sympathetic nerve activity, thus increasing norepinephrine release. This leads to sensitization of the adrenergic receptors to catecholamines, which increases sympathetic tone. Increased sympathetic tone leads to increased cardiac activity, vasoconstriction, and hypertension. The induction of rapidly transient hypertension causes shear stress on the aorta, which can then disrupt the aortic wall architecture and potentially result in rupture. Aortic dissection is characterized by longitudinal cleavage of the aortic media by a dissecting column of blood and is initiated by transverse tears through the intima and at least halfway through the media of the aortic wall. The sudden and violent severe elevation of blood pressure induced by cocaine and its active metabolites produces increased shear stress on the aorta. Factors known to predispose to aortic dissection include hypertension, arteriosclerosis, and chronic cocaine use that distort the medial architecture and contributes to the development of cystic medial degeneration.

Between 2002 and 2013, the Wayne County Medical Examiner's Office in Detroit, Michigan, investigated 94 cases of aortic dissection. Eighteen (19.1%) cases were due to cocaine use. There was a 15:3 male:female ratio. Of the eighteen decedents, sixteen were Black and two were White. Mean age was 51.9 years (range 31 to 67), mean body mass index was 27.9kg/m^2 (range 20.6 to 38.2kg/m^2), and mean heart weight was 549.4 grams (range 325 to 825 grams). Of the decedents, 72.2% had hypertension and arteriosclerotic cardiovascular disease, 16.7% had hypertension alone, 11.1% had no evidence of hypertension or arteriosclerotic cardiovascular disease, and 100% had a history of drug use. The mean level of cocaine and benzoylecgonine in the peripheral blood was 149.5ng/mL (range 33 to 410ng/mL) and 1,222.4ng/mL (range 52 to 4800ng/mL), respectively. Additionally, 58.8% had ecgonine methyl ester, 52.9% had methylecgonidine, and 23.5% had cocaethylene detected in the peripheral blood. The types of aortic dissection were as follows: 61.1% Debakey II/Stanford A; 27.8% DeBakey I/Stanford A; and, 11.1% DeBakey III/Stanford B. The sites of rupture were as follows: 72.2% in the proximal ascending arch of the aorta resulting in a hemopericardium; 11.1% in the arch of the aorta and proximal descending arch resulting in hemothoraces/left hemothorax; and, 16.7% confined to the aorta without identifiable rupture site.

This is the first large case series of aortic dissections due to cocaine use conducted at a medical examiner's office.

Cocaine, Aortic Dissection, Toxicology

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