Cocaine-Related Aortic Dissection in Perspective

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Cardiovascular complications of cocaine use have been ever more widely recognized and include the acceleration of atherosclerosis, coronary artery spasm, acute myocardial infarction, myocarditis, dilated cardiomyopathies, and cardiac arrhythmias. Less well known is the potentially lethal complication of aortic dissection. In the present issue of Circulation, Hsue and colleagues report on their 20-year experience with acute aortic dissection at an inner-city hospital. Remarkably, their findings indicate that 14 (37%) of 38 patients treated for acute dissection reported having used cocaine in the minutes or hours preceding their presentation. Cocaine, particularly crack cocaine, seemed to have played a significant role in precipitating aortic dissection among this cohort of young (age 41 ± 8.8 years), predominantly black (11 of 14; 79%), and hypertensive (11 of 14; 79%) individuals. This study represents the largest cohort of cocaine-related dissection ever reported. Its findings provoke a number of questions for those of us who study or manage this rare but highly lethal condition.

How common is cocaine-related aortic dissection? Previous reports have described a single patient or a summary of individual case reports. The presumption has been that cocaine is a very rare cause of a very rare condition. The report by Hsue et al would seem to challenge that logic, but the authors freely admit that the inner-city population served by their hospital likely is responsible for this. In fact, because they accumulated only 14 patients over 20 years at their hospital, a cocaine-related dissection was encountered less than once per year.

The International Registry for Aortic Dissection (IRAD) represents a unique effort by 17 aortic centers around the world to characterize the current status of acute aortic dissection, including its predisposing conditions. We were able to work with IRAD's coordinating center to characterize cocaine-related dissection as part of our response to the article by Hsue and colleagues.

The Table shows that among 921 cases of acute aortic dissection presenting to IRAD centers from 1996 through 2000, only 5 (0.5%) were associated with cocaine. This would strongly suggest that cocaine is not likely to be responsible for >1% of aortic dissections and that for any single hospital, identification of more than a patient every few years with cocaine-related dissection would be unusual. Moreover, although hundreds of thousands of people abuse cocaine, clearly only a small minority develops aortic dissections. It may be that among cocaine users, it is the presence of pre-existent aortic disease that allows a cocaine-induced intimal injury to precipitate the aortic dissection process. In fact, 11 of 14 patients in the series by Hsue et al were hypertensive, and all were smokers—both of which are major risk factors for disease of the aortic media.

What are the features of cocaine-related aortic dissection? The article by Hsue and colleagues showed that younger age, pre-existing (and untreated) hypertension, and black race were more common among the dissections caused by cocaine. Interestingly, more than half (8 of 14) were Type B dissections:

- **Type A** is the more common type, accounting for 60% to 70% of all aortic dissections.**
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In addition, among young patients with aortic dissection, Type A generally is even more common.

The IRAD experience, although limited to 5 patients, seems to confirm the observations of Hsue et al. Cocaine seems to provoke dissection very rarely, but when it does, it strikes in the descending aorta and afflicts predominantly young, black, hypertensive individuals (Table). Four of these 5 IRAD patients came from the 389 patients enrolled in the US sites.

What causes aortic dissection among cocaine users? Presumably, the mechanisms relate first to an underlying process that has weakened the elastic media of the aorta and, second, to the severe shearing forces that result from the sudden and profound hypertension and tachycardia that accompany cocaine (particularly crack) use. Cocaine, by inhibiting the re-uptake of both epinephrine and norepinephrine at the...
neural synapses, leads to profound sympathetic stimulation that presumably causes such shear 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. In the setting of cocaine use, 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. On the basis of the data presented by Hsue and colleagues, hypertension seems to be the underlying process predisposing patients to this possibility. Chronic smoking may also be a contributing factor.

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. Information about the chronicity of cocaine use among patients described in the report by Hsue et al was not available, nor was any pathology data, so conclusions with regard to this possibility cannot be established.

How can we recognize aortic dissection among cocaine users presenting to our emergency departments? This task is truly daunting. It is estimated that 25 million Americans have used cocaine, and >50,000 cocaine users present to an emergency room with complaints of chest discomfort each year. Of these, an incredibly small percentage will have an aortic dissection. Far more common will be myocardial ischemia or infarction, myocarditis, cardiac arrhythmias, and other cocaine-related conditions. On the basis of the IRAD experience, the sudden onset of chest or back pain (especially when severe and sharp or stabbing) is suggestive of aortic dissection. Certainly, any time such pain is accompanied by aortic blood pressure <120/80 mm Hg. The patient with cocaine-related chest pain is particularly challenging when myocardial ischemia is either suspected or definitely present. It has been argued that use of a β-blocker without simultaneous α-blockade in such a patient can promote worsening of myocardial ischemia due to arterial vasoconstriction caused by relatively unopposed α-adrenergic stimulation. For this reason, one should consider the use of labetolol, which has both α- and β-blocking properties, first intravenously for short-term and then orally for long-term use. Another therapeutic option is the use intravenous nitroglycerin or verapamil because they reverse both the vasoconstriction and hypertension caused by acute cocaine exposure. Combination therapy thus might include labetolol with a dihydropyridine calcium-channel blocker or verapamil with nitroglycerin, with additional agents directed at systemic hypertension (nitropusside) if necessary. In terms of long-term management of such a patient, the most important element must be a full-scale effort to eliminate cocaine use in the future. Aggressive control of blood pressure and heart rate is a given, as is careful surveillance of the aorta. This is performed serially with noninvasive imaging to watch for aneurysm formation or extension.

The article by Hsue and colleagues is an important contribution. It firmly connects cocaine use and aortic dissection, taking us from case reports to a cohort that exhibited certain features that should be committed to memory. Aortic dissection remains a rare but highly lethal condition. Cocaine is a rare cause of aortic dissection, but it does occur and we must maintain a high index of suspicion for it when we encounter cocaine users presenting to our emergency departments. In addition, further insights into the interactive effects of hypertension, smoking, and cocaine on the aorta may shed light not only on the mechanism of dissection among cocaine users, but also on acute and chronic underpinnings of aortic dissection in general.

References

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