Acute aortic dissection associated with use of cocaine

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Purpose: **Cocaine use can result in a variety of cardiovascular complications, including myocardial infarction, arterial thrombosis, coronary dissection, and cardiomyopathy. Cocaine-induced aortic dissection is uncommon and has been described largely in case reports. The purpose of this study was to review our experience with aortic dissection associated with cocaine abuse.**

Methods: **A retrospective chart review was performed of all hospital records during a 15-year period in patients diagnosed with aortic dissection. Among the 164 cases of acute aortic dissection, 16 patients (9.8%) had used cocaine or its derivative, crack cocaine, within 24 hours prior to the onset of symptoms. The remaining 148 patients (90.2%) had no history of cocaine usage. Clinical features, management, and outcome in these two groups were compared.**

Results: **In the cocaine group, powder cocaine was inhaled intranasally in 11 patients (69%) and crack cocaine was smoked in five cases (31%). The mean duration between cocaine use and the onset of aortic dissection was 12.8 hours (range, 4 to 24 hours). Patients in the cocaine group were younger in age and more likely to have a history of polysubstance abuse than the non-cocaine cohort. In the cocaine group, the incidence of DeBakey dissection type I, II, IIIa, and IIIb was 19%, 25%, 38%, and 19%, respectively. In the group without cocaine use, the incidence of DeBakey dissection type I, II, IIIa, and IIIb was 18%, 23%, 39%, and 20%, respectively. Surgical intervention for aortic dissection was performed in 50% of the cocaine group and 45% of the non-cocaine group. In patients who underwent surgical repair, greater pulmonary complications occurred in the cocaine group than the non-cocaine group ($n = 0.02$). No difference was noted in the hospital length of stay or 30-day operative mortality among the two groups.**

Conclusions: **Cocaine-associated aortic dissection occurs in predominantly male patients with illicit drug abuse who were younger than patients with aortic dissection without cocaine use. Greater pulmonary complications can occur in patients with cocaine-related aortic dissection following surgical interventions. (J Vasc Surg 2007;46:427-33.)**

The use of coca leaves by South Americans has been practiced since 2000 BC and was thought to arouse physical energy, fight pain, and diminish fatigue.[1] Chewing coca releases the alkaloid cocaine when contact with saliva is made, resulting in local anesthesia and slight euphoria. Green and brown in color, these leafy products are treated with alkali, hydrochloric acid, ammonia, and various organic solvents to produce cocaine hydrochloride.[2] Over the past century, coca leaves have been routinely processed to produce cocaine powder, which is a highly-addictive and commonly-used illicit drug in this country. It is estimated that more than four million Americans use cocaine regularly, of which one million individuals are clinically addicted to cocaine or its derivate crack cocaine. A recent study highlights the frequent problem of this illicit drug as cocaine-associated medical complaints accounts for 20% of all drug-related emergency department visits in the United States.[3]

Cocaine consumption can lead to serious health hazards, particularly involving the cardiovascular system. In fact, approximately one fifth of individuals who seek emergency room care due to cocaine overdose experienced symptoms relating to cardiovascular systems.[4] Cocaine-induced cardiovascular consequences are well described, which include myocardial ischemia, cardiac arrhythmia, congestive heart failure, and endocarditis.[5-7] Aortic dissection is another reported consequence of cocaine use that is less common.[8] Though millions of people are known to abuse cocaine, only a small minority develops aortic dissection, and this is likely related to a combination of predisposing factors and acute contributions from cocaine. Previous reports of cocaine induced aortic dissection have been largely limited to isolated case reports. The purpose of this study is to report our experience in patients with acute aortic dissection associated with the consumption of cocaine or its derivative crack cocaine. The presenting symptoms, management, and outcomes of these patients are described.

MATERIALS AND METHODS

The hospital charts of all patients diagnosed with acute aortic dissection based on ICD-9 code were reviewed from July 1990 to August 2006. For the purpose of study comparison, patients were divided into two groups. Group I was comprised of patients who presented with recent

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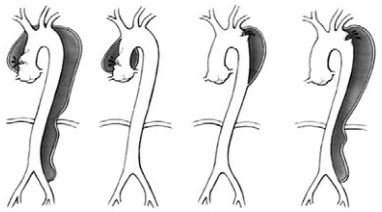

DeBakey Dissection Classification	I	II	IIIa	IIIb
Group I (n=16), (+) cocaine	3 (19%)	4 (25%)	6 (38%)	3 (19%)
Group II (n=148), (-) cocaine	26 (18%)	34 (23%)	58 (39%)	30 (20%)

Fig. Disease distribution of acute aortic dissection based on the DeBakey classification in patients with or without cocaine usage.

cocaine abuse prior to aortic dissection, which was defined by cocaine consumption within 24 hours prior to the onset of symptoms. In contrast, group II was comprised of all remaining patients diagnosed with acute aortic dissection without recent history of cocaine usage. Patients diagnosed with chronic aortic dissection, dissecting aneurysm, or symptoms suggestive of chronic dissection were excluded from the study. The determination of cocaine use was based on either self-reporting history or positive urine toxicology. Data were collected with respect to (1) timing and route of cocaine usage, if applicable; (2) clinical presentations and diagnostic findings of aortic dissection; and (3) intervention and treatment outcome. Long-term data were obtained from clinical or hospital records or by telephone interview with the patient or the primary physician. Aortic dissection was assessed using the DeBakey dissection classification.[9] Briefly, they include (1) type I: dissection involving the ascending aorta, aortic arch, and descending aorta, (2) type II: dissection involving the ascending aorta, (3) type III: dissection involving the descending aorta distal to the left subclavian artery. Additionally, type III dissection is further divided to either type IIIa, which refers to dissections that originate distal to the left subclavian artery but extend distally above the diaphragm, or type IIIb, which refers to dissections that originate distal to the left subclavian artery and extend distally below the diaphragm (Fig). Relevant variables that may influence the clinical outcome were compared between the two groups. Statistical analyses were performed to compare clinical features, risk factors, and treatment outcome between the two groups using the χ^2 test with Yates correction for continuity and the pooled Student *t* test. In all analyses, we only considered variables with at least 80% of the data present or recorded. The test results were considered significant at a *P* value of less than .05. Statistical analysis was performed using a statistical software program (SAS Institute, Cary, NC).

RESULTS

A total of 164 patients with acute aortic dissection were identified during the study period. The presumptive diagnosis of cocaine-related aortic dissection was made in 16 (9.8%) patients (group I, 12 men and 4 women; overall mean age 47 ± 6.8 years, range 36 to 60 years) who used cocaine or its derivative, crack cocaine within 24 hours prior to the onset of symptoms. The remaining 148 patients (group II, 90.2%) had no history of cocaine usage. Disease pattern between these two groups was shown (Fig). Similar incidence of DeBakey type I, II, and III were noted between the two groups. Type IIIa remained the most common type of acute aortic dissection in group I and II, which occurred in 38% and 39%, respectively. Chest pain was the most common presenting symptoms, which occurred in 135 patients (82%). Specifically, the incidence of chest pain in group I and II was similar, which was 81% and 82%, respectively. No difference was noted with regards to the presenting symptoms between the two groups based on their respective dissection classifications.

In the cocaine group, powder cocaine was inhaled intranasally in 11 patients (69%) and crack cocaine was smoked in five cases (31%). The mean duration between cocaine use and the onset of aortic dissection was 12.8 hours (range, 4 to 24 hours). The mean history of cocaine usage was 4.6 years (range, 0.3 to 6.8 years). Besides using cocaine or its derivatives, all 16 patients had a prior history of other illicit narcotic drug usage. Clinical summary of 16 patients in group I was listed in Table I. All patients except one (patient 9) diagnosed with either type I or II dissection underwent surgical intervention. The only patient (patient 9) with a type II dissection who was treated medically was due to his refusal to undergo surgical intervention, and he expired on hospital day (HD) no. 1. In patients with type I or II dissection, standard surgical therapy with prosthetic graft replacement of the ascending aorta and proximal aortic arch was performed via a median sternotomy approach. This reconstructive strategy was performed in patient nos. 1, 2, 3, 5, 12, and 15 (Table I). In patients with type IIIa or IIIb dissection, aggressive medical therapy with blood pressure and heart rate control was initiated. Two patients (patients 4 and 11) failed to respond to medical therapy due to persistent visceral and lower extremity ischemia. Both patients underwent successful surgical intervention, which was thoracoabdominal aortic replacement. There were overall eight patients (50%) in the cocaine group who underwent surgical reconstruction of their acute aortic dissection. Numerous postoperative complications occurred in patients who underwent surgical intervention. Notably, postoperative pulmonary complications, which were defined by postoperative pneumonia or prolonged mechanical ventilation (>5 days), were common among all cocaine patient cohorts who underwent surgical repair. When analyzing a possible association between the time of the most recent cocaine consumption vs pulmonary complications, no temporal association was found between these two variables.

The temporal relationship between patients who presented with aortic dissection based on the DeBakey classification was compared using four successive time periods in our study (Table II). No specific temporal pattern was noted with regards to the incidence of aortic dissection

Table I. Summary characteristics of patients with cocaine-related acute aortic dissection

Pt. no.	*Age (year)*	*Sex*	*DeBakey dissection type*	*Interval between cocaine usage and onset of symptom (hours)*	*Prior illicit drug usage*	*Treatment (medical vs surgical)*	*Postoperative complication*	*Outcome and follow-up*
1	29	Male	I	4	AP, CN, CC, LSD	Surgical	PC	Discharged home on POD# 18. The patient was lost to follow-up after 9 months.
2	38	Male	II	5	CO, HR, LSD	Surgical	PC, RI	Discharged to rehabilitation unit on POD# 26. Died of drug overdose at 8 months.
3	49	Female	I	12	CC, PCP, CN, AP	Surgical	PC	The patient died on POD #2 due to coagulopathy.
4	36	Male	IIIb	16	CO, CN, LSD	Surgical	NA	Failed to respond to initial medical therapy. Discharged home on HD #10, with uneventful recovery.
5	45	Male	I	23	CC, CN	Surgical	ST, PC	Discharged home on POD #15, with uneventful recovery. The patient was lost to follow-up after 10 months.
6	49	Female	IIIa	16	CO, HR, AP, CN	Medical	NA	Discharged home on HD #13, with uneventful recovery. The patient was lost to follow-up after 18 months.
7	36	Male	IIIa	24	CO, PCP, LSD, HR	Medical	NA	Discharged home on HD #16, with uneventful recovery.
8	52	Female	IIIb	7	CC, PCP, CN	Medical	NA	Discharged home on POD #21. The patient died of MI at 24 months.
9	53	Male	II	9	CO, HR, AP, CN	Medical	MI	The patient declined surgical treatment and died on HD #2.
10	56	Female	IIIa	13	CO, PCP, LSD, HR	Medical	NA	Discharged home on HD #13. The patient had an uneventful recovery.
11	53	Male	IIIb	16	CC, PCP, CN	Surgical	NA	Failed to respond to initial medical therapy. Discharged home on HD #18. The patient was lost to follow-up after 16 months.
12	51	Male	II	21	CO, CC, CN	Surgical	WC, PC	Patient died on POD #9.
13	49	Male	IIIa	21	AP, CN, CC, LSD	Medical	NA	Discharged home on HD #12. The patient was lost to follow-up after 35 months.
14	59	Male	IIIa	8	CO, HR, LSD	Medical	NA	Discharged home on HD #17. The patient died of stroke at 32 months.
15	60	Male	II	4	CO, CC, CN, AP	Surgical	MI, PC	Discharged home on HD #17. The patient died of MI at 20 months.
16	49	Male	IIIa	6	CO, HR, AP, CN	Medical	NA	Discharged home on POD #15. The patient died of drug overdose at 14 months.

CO, Cocaine; *CC,* crack cocaine; *CN,* cannabis; *LSD,* lysergic acid diethylamide; *HR,* heroin; *AP,* amphetamine; *PCP,* phencyclidine; *MI,* myocardial infarction; *ST,* stroke; *PC,* pulmonary complication (including pneumonia, prolonged intubation); *WC,* wound complication; *RI,* renal insufficiency; *NA,* not applicable; *POD,* postoperative day; *HD,* hospital day.

when compared with these four successive time intervals, reflecting a homogenous pattern in the occurrence of aortic dissection throughout the study period. Relevant clinical variables between the two groups are shown in Table III. The mean age of the cocaine group was significantly younger than the control group (47 vs 62 years, $P = .001$). Patients with cocaine-related aortic dissection were more likely to have other illicit drug usage when compared with the non-cocaine patient cohort (100% vs 7%, $P = .0001$). There were 67 patients (45%) in group II who underwent surgical repair because of their acute aortic dissection. No difference was noted when analyzing presenting hemodynamic variables or relevant risk factors between the two groups. When analyzing treatment variables among the two

Table II. Incidence of aortic dissection among patients with or without cocaine-related etiological factors in relation to four successive time periods

	DeBakey dissection type I		*DeBakey dissection type II*		*DeBakey dissection type IIIa*		*DeBakey dissection type IIIb*	
	(+) cocaine	*(−) cocaine*	*(+) cocaine*	*(−) cocaine*	*(+) cocaine*	*(−) cocaine*	*(+) cocaine*	*(−) cocaine*
Time period	(n = 3)	(n = 26)	(n = 4)	(n = 34)	(n = 6)	(n = 58)	(n = 3)	(n = 30)
1990-1994	1 (33%)	6 (23%)	0	9 (26%)	2 (33%)	14 (24%)	1 (33%)	8 (27%)
1995-1998	0	5 (19%)	1 (25%)	7 (21%)	1 (17%)	13 (22%)	1 (33%)	7 (23%)
1999-2002	1 (33%)	8 (31%)	2 (50%)	8 (24%)	1 (17%)	16 (28%)	1 (33%)	9 (30%)
2003-2006	1 (33%)	6 (23%)	1 (25%)	10 (29%)	2 (33%)	15 (26%)	0	6 (20%)

Table III. Comparison of clinical features between patients with cocaine or non-cocaine related aortic dissection

	Group I (+) cocaine; n = 16	*Group II (−) cocaine; n = 148*	P *value*
Mean age (years)	47 ± 6.8	62 ± 9.8	.001
Male sex	12/16 (75%)	101/148 (68%)	.64
Cigarette smoking	16/16 (100%)	89/148 (60%)	.005
History of illicit drug usage	16/16 (100%)	9/128 (7%)	.0001
Presenting hemodynamic variables			
Systolic blood pressure (mm Hg)	185 ± 35	192 ± 41	.56
Diastolic blood pressure (mm Hg)	105 ± 27	98 ± 36	.43
Heart rate (bpm)	92 ± 24	86 ± 31	.68
Respiratory rate (per min)	18 ± 6	21 ± 9	.98
Associated risk factors			
Coronary artery disease	3/16 (19%)	34/148 (23%)	.89
Hypertension	11/16 (69%)	105/148 (71%)	.67
Diabetes	2/16 (13%)	22/148 (15%)	.82
Aortic insufficiency	4/16 (25%)	33/148 (22%)	.48
Treatment variable	(n = 8)	(n = 67)	
Postoperative ICU length of stay (days)	9 ± 3	5 ± 4	.06
Postoperative hospital length of stay (days)	13 ± 5	10 ± 4	.21
Postoperative complication	5/8 (63%)	37/67 (55%)	.52
30-day operative mortality	2/8 (25%)	14/67 (21%)	.69
Postoperative complication			
Cardiac complication	2/8 (25%)	12/67 (18%)	.68
Pulmonary complication	5/8 (63%)	14/67 (21%)	.02
Renal complication	1/8 (13%)	7/67 (10%)	.64
Bleeding complication	1/8 (13%)	4/67 (5%)	.52

patients groups who received surgical interventions, patients in group I showed a trend toward prolonged ICU length of stay compared with non-cocaine patient cohort (9 ± 3 vs 5 ± 4 day; $P = .06$). No differences were noted in the overall postoperative length of stay or in-hospital surgical mortality rates between the two groups. Similar postoperative complications were noted between the two groups. However, a greater incidence of pulmonary complications was noted in group I compared with group II (63% vs 21%, $P = .02$). No differences in other specific categories of postoperative complications were noted between the two groups (Table III).

Twelve patients (75%) in the cocaine group were available for follow-up following discharge (mean, 26 months; range, 6 to 75 months). Two patients died of drug overdose at 8 and 14 months later. There were two deaths due to myocardial infarction at 20 and 24 months. One patient died due to stoke at 32 months. Among the remaining seven surviving patients, one was able to quit all illicit drug usage while the remaining five patients admitted their habitual consumptions of cocaine or crack cocaine. The 30-day mortality in group I and II was 13% and 10%, respectively (non-significant). In contrast, significantly higher mortality was noted in group I when compared with group II at 1 year, which was 31% and 22%, respectively ($P < .05$).

DISCUSSION

Our study highlights the importance of cocaine consumption as an etiological factor of aortic dissection, particularly in young patients with a history of illicit drug usage. This report is also notable because it represents the largest series of patients with cocaine-induced aortic dissection. Although patients with cocaine-related aortic dissection were typically younger than those with non-cocaine related dissection, these patients were more likely to experience pulmonary complications following surgical intervention. These findings underscore the morbidity of this

condition as well as the potential pulmonary frailty in patients with habitual cocaine consumption.

Regarded as the most common cause of drug-related deaths in the United States, cocaine and its derivate crack cocaine have become the most widely used illicit drug among individuals seeking acute care in hospitals or drug-treatment centers.[6] It has been reported that cocaine was detectable in the urine sample of up to 17% of patients with chest pain who sought emergency room care.[3] In our series, chest pain was the most common presenting symptoms of acute aortic dissection, which was noted in 81% among the cocaine users. In a multicenter registry study, Hagan and associates noted that the diagnosis of aortic dissection can be missed in up to 38% of patients during initial evaluation and is often discovered only at postmortem examination.[10] This finding underscores the importance of recognizing any inciting etiological factors. Consequently, when treating young cocaine users who present with sudden onset of chest pain, clinicians must have a heightened level of suspicion and consider aortic dissection as a differential diagnosis.

The deleterious effects of cocaine on the cardiovascular system are well known and researchers have postulated various pathogenic mechanisms of cocaine induced cellular damage. Cocaine induced myocardial ischemia has been documented even in patients with normal coronary arteries and is unrelated to the dose, route, or frequency of use.[6,7,11] Vasoconstriction of the coronary arteries, increased myocardial oxygen demand, and acute arterial thrombosis from cocaine all contribute to ischemia.[2,5,12] Coronary ischemic symptoms related to thrombosis are seen relatively rapidly after cocaine use, while peripheral arterial thrombosis is seen hours after usage and likely a consequence of critical vasoconstriction.[2,13] Left ventricular hypertrophy, dilated cardiomyopathy, and dysrhythmias can occur with acute and chronic cocaine use and are a function of its sympathomimetic activity and effects on sodium and calcium channels.[5] In contrast to normal treatment algorithms for atherosclerosis-induced angina, pure β-blockers are contraindicated in cocaine-related ischemia due to the risk of unopposed α-receptor activity that is already upregulated by cocaine.[11] Based on this consideration, physicians should have a heightened level of awareness regarding the risk of β-blocker administration alone as a means of blood pressure control in patients who present with cocaine-induced aortic dissection.

The literature regarding cocaine-related aortic dissection, which consists primarily of case descriptions, is characterized by a number of recurring themes. In a compilation of 12 case reports, Rashid and colleagues noted similar findings as seen in our study, notably, younger age at presentation compared with non-cocaine related dissection, various routes of cocaine usage, no significant trend towards a particular dissection type, and a predominance of pre-existing hypertension.[8] Palmiere and associates reported a young male who developed acute chest pain following cocaine ingestion. Due in part to a pre-existing connective tissue disorder, this patient developed concomitant coronary dissection and suffered a fatal outcome.[14] Other case reports have described patients with habitual cocaine abuse who developed clinical sequelae of aortic dissection including false aneurysm with abdominal aortic rupture, ascending aortic rupture, and visceral organ ischemia.[15-17] Hsue and colleagues published the only prior single institution series on cocaine and aortic dissection; they reported 14 patients with cocaine-related acute aortic dissection during a 20-year period at an urban hospital.[18] The incidence of cocaine-related aortic dissection was 37% during their study period, which was similar to the 31% seen in our study. Consistent with our findings, these researchers observed a primarily younger patient cohort with a short onset of symptoms following cocaine consumption, as the mean interval between cocaine use and the onset of symptoms was 12 hours.[18]

Based on the available literature, it is difficult to ascertain an accurate prevalence of cocaine-related aortic dissection. In the International Registry for Aortic Dissection (IRAD) study, which represents a collective effort from 17 aortic centers around the world of the 921 cases of aortic dissection, only five patients (0.5%) were associated with cocaine usage.[10] Findings reported by Hue and our study clearly showed a significantly higher prevalence of cocaine-induced aortic dissection, which is likely attributable to the urban patient population served by these institutions. Despite the variability in the disease prevalence, all of these studies demonstrated no difference in the type or location of the dissection in patients with cocaine usage compared with those who did not use cocaine.[3,8,10,14,18]

Multiple pathogenic mechanisms have been proposed with regards to cocaine-induced aortic dissection. Cocaine initially exerts its effects by blocking the reuptake of norepinephrine and dopamine at the presynaptic cleft. This profound release of catecholamines leads to the triad of profound hypertension, intense vasoconstriction and increased cardiac workload. The arterial walls are now subject to severe shearing forces from malignant hypertension and tachycardia. This physiological phenomenon can be especially profound with the use of crack cocaine, since the onset of systemic effects is nearly immediate. Under such a circumstance, an aortic tear may occur typically at the ligamentum arteriosum because it is less likely to tolerate the accelerating aortic pressure wave and shear stress generated by the ventricular contraction due to its fixed anatomy relative to the aorta.[5] At the cellular level, platelet aggregation and thrombus formation, upregulation of endothelin production and cessation of nitric oxide release all contribute to further ischemia, vasoconstriction and resulting injury.[12] Ultimately, cocaine-related acute dissection is the accumulation of multiple inciting factors. Most patients have pre-existing hypertension and a chronic smoking history. It appears that cocaine may accelerate the natural progression of aortic disease in patients who have an environmental and genetic predisposition.

Our study showed an increase in pulmonary complication in the cocaine users following surgical intervention. We postulated that this may be attributed to the

lung impairment caused by smoking crack cocaine. Additionally, many of these cocaine users are habitual cigarette smokers. Others have similarly reported increased pulmonary complications related to cocaine use which include pneumonia, pulmonary edema or hemorrhage, talcosis, pneumothorax, and emphysema.[19] Acute lung disease seen with cocaine smoking is known as "crack lung" and refers to development of respiratory failure with bilateral areas of increased opacity that appears shortly after crack use.[20] One study demonstrated short-term exposure to cocaine results in a burst of inflammatory activity by polymorphonuclear leukocytes and cytokines that could lead to acute lung injury.[21,22] One study examined lavage fluid from crack smokers and suggested a high proportion of asymptomatic users had chronic alveolar hemorrhage.[22] Cocaine is thought to decrease pulmonary diffusing capacity by direct damage to the alveoli and vascular bed and increased endothelial permeability leading to development of edema and effusion.[23,24] Thermal airway injury and tracheal stenosis, asthma exacerbation and pulmonary eosinophilia have also been reported after inhalation of cocaine. In the subset of patients who required surgery, increased pulmonary complications and length of ICU stay were seen with the cocaine-related group. Findings from our study suggest that prior to admission and surgery for aortic dissection, these patients with cocaine usage likely had suffered inflammatory consequences of chronic lung disease that elevated their odds of developing pneumonia, pleural effusion, and ventilator-associated complications, and decreased their ability to engage in adequate pulmonary toilet. While several anecdotal or case series have suggested that endovascular treatment of type III aortic dissection may be technically feasible with short term satisfactory results,[25-27] the current technology remains limited in this application due to lack of large scale and long-term data to validate the clinical durability of this treatment strategy. Undoubtedly, if endovascular treatment becomes a proven treatment modality, particularly in patients with cocaine-related aortic dissection, we postulate this treatment will result in significantly less posttreatment pulmonary complications due to avoidance of thoracotomy incision.

There are admittedly several limitations in our study. The retrospective nature of the study design may influence the study outcome due in part to a bias in patient selection and treatment approach. Additionally, the treatment strategy between those with or without cocaine usage was not based on an established protocol. Consequently, the clinical significance of increased pulmonary complications in the cocaine users following surgical repair may be debatable, particularly given the relatively small patient sample size in our study. Lastly, methods of determination of cocaine usage were largely based on self-reporting history, rather than uniform urine toxicology test. Despite these shortcomings, we believe our study is noteworthy because it represents a large clinical series of this uncommon clinical entity.

CONCLUSION

In conclusion, our study contrasts the unique population of patients with acute aortic dissection who have a recent history of cocaine use with those who have traditional factors leading to dissection. Cocaine-associated aortic dissection occurs in predominantly male patients with illicit drug abuse who were younger than patients with aortic dissection without cocaine use. This diagnosis should be suspected in young patients with chest pain and a recent history of cocaine abuse. Administration of isolated β-blocker should be used with caution due to potential risk of unopposed α-receptor activity which is likely upregulated by cocaine. Postoperative pulmonary complications occur more frequently in patients with cocaine-related aortic dissection. Physicians should be cognizant of this outcome so that appropriate treatment strategies can be implemented to reduce potential treatment-related pulmonary morbidity.

AUTHOR CONTRIBUTIONS

Conception and design: PL, JD
Analysis and interpretation: PL, JD, WZ, SL
Data collection: JD, PL, HS
Writing the article: PL, JD
Critical revision of the article: PL, JD, JC, JH, SL
Final approval of the article: PL, JD, WZ, JC, JH, PK, HS
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