Acute coronary syndrome and cocaine use: 8-year prevalence and inhospital outcomes

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Aims
The use of cocaine as a recreational drug has increased in recent years. The aims of this study were to analyse the prevalence and inhospital evolution of acute coronary syndrome (ACS) associated with cocaine consumption (ACS-ACC).

Methods and results
Prospective analysis of ACS patients admitted to a coronary care unit from January 2001 to December 2008. During the study period, 2752 patients were admitted for ACS, and among these 479 were ≤50 years of age. Fifty-six (11.7%) patients had a medical history of cocaine use with an increase in prevalence from 6.8% in 2001 to 21.7% in 2008 (P = 0.035). Among patients younger than 30 years of age, 25% admitted to being users compared with 5.5% of those aged 45–50 years (P = 0.007). Similarly, the prevalence of positive urine tests for cocaine was four times higher in the younger patients (18.2 vs. 4.1%, P = 0.035). Acute coronary syndrome associated with cocaine consumption patients (n = 24; those who had a positive urine test for cocaine or who admitted to being users upon admission) had larger myocardial infarcts as indicated by troponin I levels (52.9 vs. 23.4 ng/mL, P < 0.001), lower the left ventricular ejection fraction (44.5 vs. 52.2%, P = 0.049), and increased inhospital mortality (8.3 vs. 0.8%, P = 0.030).

Conclusions
The association between cocaine use and ACS has increased significantly over the past few years. Young adults with ACS-ACC that require admission to the coronary care unit have greater myocardial damage and more frequent complications.

Keywords
Cocaine • Acute coronary syndrome • Young

Introduction
The use of cocaine as a recreational drug is growing around the world, with 13 million Europeans estimated to have used cocaine at some point in their lives, representing 3.9% of adults between the ages of 15 and 64. The highest prevalence of cocaine use in Europe is found in Spain (8.3%), with an annual incidence of 3.1%.2 A parallel increase in medical complications associated with cocaine use has been observed. The number of treatments initiated for addiction in 2007 was 61 000 in Europe,1 and the number of emergency room visits caused by cocaine use represented between 12 and 41 cases for every 100 000 emergencies, of which between 6.7 and 25% were for cardiovascular complications.2,3 Cocaine causes more cardiovascular complications than any other illegal drug.4

Both acute and chronic cocaine use may cause arterial hypertension, aortic dissection, arrhythmias, acute pulmonary oedema, cardiomyopathy, and sudden death.5,6 The association between acute coronary syndrome (ACS) and cocaine consumption was first described by Coleman et al.7 in the early 1980s and later by Mittleman et al.8 in a large series. Cocaine use causes coronary vasospasm through a direct effect on the α-adrenergic receptors9 and increases thrombogenicity through procoagulant factors released by platelets and endothelial cells, with the development of intracoronary thrombosis.10 Increased coronary artery disease (CAD) burden has been reported in chronic cocaine users both by autopsy and angiography.11–13

This study aimed to evaluate the prevalence and inhospital evolution of ACS associated with cocaine consumption (ACS-ACC) in
young adults at a university hospital in Barcelona metro area between 2001 and 2008.

Patients and methods

We carried out a transversal study at the coronary care unit (CCU) of a university hospital in the northern metropolitan area of Barcelona that provides health coverage to ~800 000 inhabitants. The study population included consecutive ACS patients aged ≤50 years who were admitted to the CCU between 1 January 2001 and 31 December 2008. The CCU admission protocol for ACS patients under the age of 50 included a questionnaire about cocaine use and frequency of use as well as a urine test for cocaine within 48–72 h of admission. This study complies with the Helsinki Declaration, was evaluated by the local Ethics Committee, and all patients provided consent to participate.

Clinical variables, sociodemographic characteristics, medical history, and cardiovascular risk factors were recorded, as well as the evolution and development of complications during hospital stay. Mortality was defined as death of any cause occurring during hospital admission.

A urine test for cocaine was performed qualitatively using immunoenzyme analysis (Dimension Flex Reagent Cartridge; Siemens Healthcare Diagnostics Ltd, Frimley, Camberley, UK). The extent of infarct was assessed by the peak of MB fraction of creatine phosphokinase (CPK-MB) and troponin I. Significant coronary disease was considered when stenosis was equal to or >70% in the right and left major coronary arteries and 50% in the left main coronary artery. The left ventricular ejection fraction was assessed by transthoracic echocardiography (Simpson biplane method) during hospital stay.

Definitions

Acute coronary syndrome was defined as a set of symptoms and clinical signs consistent with myocardial ischaemia with compatible electrocardiographic changes and/or an elevation of markers of myocardial damage.\(^{14,15}\)

According to patient’s medical history, cocaine use was classified into four levels:
- non-users, those who had never used cocaine;
- former users, those who reported to having used cocaine in the past, independent of the route of administration, but who were not current users in the month previous to hospitalization;
- occasional users, those who, despite admitting cocaine use upon admission, did not use it on a daily or weekly basis; and,
- current users, those who admitted daily use of cocaine, independent of the dose and route of administration.

For the objectives of this study, patients were also classified into the following two groups:
- acute coronary syndrome associated with cocaine consumption (ACS-ACC): those who had a positive cocaine urine test or who admitted to being current users upon admission.
- acute coronary syndrome not associated with cocaine consumption (ACS-NACC): patients who had a negative urine test for cocaine and who said they were non-users, former users, or occasional users.

Statistical analysis

Statistical analysis was performed by selecting index cases and excluding readmissions during the study period. Continuous variables were presented as a mean ± standard deviation or median and inter-quartile range (IQR), and categorical variables were presented as percentages. Differences between ACS-ACC and ACS-NACC were examined with Student’s \(t\)-test for continuous variables and \(\chi^2\) test for dichotomous variables, using Fisher’s exact test when needed. Logarithmic transformations were used for quantitative variables with non-normal distribution. The statistical software package SPSS version 15 was used for analyses (SPSS, Inc., Chicago, IL, USA). A \(P\)-value of <0.05 was considered significant.

Results

During the study period, 3970 patients were admitted to the CCU, of which 2752 were admitted for ACS. Among these patients, 479 were ≤50 years of age (17.4%), and a cocaine urine test was obtained in 403 patients (84.1%) (Figure 1). The prevalence of

Figure 1 Patients admitted to the coronary care unit between 1 January 2001 and 31 December 2008. ACS-ACC, acute coronary syndrome associated with cocaine consumption; ACS-NACC, acute coronary syndrome not associated with cocaine consumption.
ACS in patients aged ≤ 50 years upon admission remained stable throughout the study period. The clinical characteristics of the patients are shown in Table 1. The main diagnosis was Q-wave acute myocardial infarction (AMI) in 58.9% of the cases. Infarct location was anterolateral in 45.5% of cases, inferoposterior in 45.3% of cases, and undetermined in 9.2% of cases.

Complications during the patients’ stay in the CCU included sustained ventricular tachycardia in 28 (5.8%) patients, ventricular fibrillation in 34 (7.1%) patients, atrial fibrillation in 12 (2.5%) patients, complete atrioventricular block in 6 (1.3%) patients, acute ventricular conduction block in 6 (1.3%) patients, and pericarditis in 11 (2.3%) patients. We also identified 87.8% of patients as being Killip-Kimball class I, 5.1% class II, 3.6% class III, and 3.4% class IV.

Table 1  Characteristics of patients aged ≤50 years and hospitalized for acute coronary syndrome (n = 479 patients)

<table>
<thead>
<tr>
<th>Age (years), mean ± SD</th>
<th>43.8 ± 5.3</th>
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<tr>
<td>Men</td>
<td>406 (84.8)</td>
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</table>

Medical background

- Tobacco use
  - Former smoker: 73 (15.2)
  - Current smoker: 337 (70.4)
  - Non-smoker: 69 (14.4)
- Arterial hypertension: 140 (29.2)
- Dyslipidaemia: 272 (56.8)
- Diabetes mellitus: 64 (13.4)
- Peripheral vascular disease: 24 (5.0)
- Ischaemic heart disease
  - Angina: 92 (19.2)
  - Myocardial infarction: 58 (12.1)
  - Previous revascularization procedures: 22 (4.6)

Characteristics of ACS

- Diagnosis of ACS
  - Unstable angina: 68 (14.2)
  - Non-Q-wave AMI: 129 (26.9)
  - Q-wave AMI: 282 (58.9)
- CPK-MB (ng/mL), median (IQR): 130 (40.7–276.7)
- Troponin I (ng/mL), median (IQR): 22.9 (8.1–52.9)

Cocaine consumption

- According to patient’s medical history
  - Former user: 20 (4.2)
  - Occasional user: 21 (4.4)
  - Current user: 15 (3.1)
  - Non-user: 423 (88.3)
  - Positive cocaine urine test (n = 403): 21 (5.2)

Data are given as number of patients (%) unless otherwise noted. ACS, acute coronary syndrome; AMI, acute myocardial infarction; CPK, creatine phosphokinase; CPK-MB, MB fraction of creatine phosphokinase; IQR, inter-quartile range.

Prevalence of cocaine use

Fifty-six (11.7%) patients had a medical history of cocaine use, and 21 (5.2%) had a positive urine test for cocaine on admission. Figure 2 shows the annual distribution. The number of patients admitting cocaine consumption increased from 6.8% in 2001 to 21.7% in 2008 (P = 0.035), and the number of positive cocaine urine tests also increased, from 2.9 to 4.9%, but the difference was not significant. Figure 3 shows the age-adjusted prevalence of cocaine consumption, with 25% of patients younger than 30 years of age admitting to being users compared with 5.5% of those aged 45–50 years (P = 0.007). Similarly, the prevalence of positive cocaine urine tests was four times higher in the younger patient group (18.2 vs. 4.1%, P = 0.035).

Cocaine consumption based on the patients’ medical history identified 15 (3.1%) current users, 21 (4.4%) occasional users, and 20 (4.2%) former users (Table 1). A positive cocaine urine test in those admitting cocaine use was found in 73% of current users, 24% of occasional users, and none of former users. Remarkably, four patients who did not admit cocaine use upon interrogation had a positive urine test.

Acute coronary syndrome associated with cocaine consumption vs. acute coronary syndrome not associated with cocaine consumption

The combined variable of cocaine use based on the medical history and a positive cocaine urine test identified 24 cases (6%) with ACS-ACC. Table 2 shows a comparative analysis between ACS-ACC and ACS-NACC. No significant demographic differences were found between groups except for a lower proportion of arterial hypertension (P = 0.041) and more cigarette smoking (P = 0.003) among ACS-ACC patients. With regards to clinical manifestations or the location of ACS a tendency towards a greater proportion of Q-wave AMI was found in patients with ACS-ACC (P = 0.062). Markers of myocardial damage were higher (P = 0.038 and P < 0.001 for CPK-MB and troponin I, respectively) and the left ventricular ejection fraction was lower (P = 0.049) in patients with ACS-ACC compared with ACS-NACC patients. A comparative analysis of CAD severity showed a tendency for greater multivessel disease in ACS-ACC (59.7 vs. 38.5%; P = 0.096; Table 2). Only two patients in the ACS-ACC group were classified as having coronary vasospasm (defined as normal coronary arteries on angiography in the context of ACS and cocaine use), and no coronary dissections were identified in this cohort.

During the acute phase, ACS-ACC patients received less beta-blockers (25 vs. 76.5%, P < 0.001), more calcium channel blockers (29.2 vs. 10.3%, P = 0.012), and more dobutamine (16.7 vs. 5.3%, P = 0.046). No significant differences were found between the two groups in terms of other treatments administered. Of the 255 patients with ACS and ST segment elevation, 187 (73.3%) underwent reperfusion therapy. Acute coronary syndrome associated with cocaine consumption and ACS-NACC patients were equally reperfused (66.7 vs. 73.9%, P = 0.471). No significant differences were found between groups in terms of the revascularization procedure (thrombolysis 38.1% in ACS-ACC vs. 38.0% in
ACS-NACC, \( P = 0.996 \); and primary coronary intervention 35.9 vs. 28.6%, \( P = 0.501 \), respectively). The interval from symptoms onset to revascularization was 200 min (112.5–338.25) in the ACS-ACC group and 165 min (120–274.5) in the ACS-NACC group (\( P = 0.328 \)).

The ACS-ACC patients had sustained ventricular tachycardia (20.8 vs. 5.0%, \( P = 0.01 \)) and second degree atrioventricular block (12.5 vs. 0.3%, \( P = 0.001 \)) more often than ACS-NACC patients. With respect to the development of heart failure during hospitalization, we observed a trend towards a higher percentage of Killip-Kimball class III–IV patients among those with ACS-ACC (17.4 vs. 5.8%, \( P = 0.053 \)). No differences were found between groups for ventricular fibrillation, atrial fibrillation, complete atrioventricular block, post-infarct angina, reinfarction, and need for coronary artery bypass graft. The ACS-ACC patients had 10-fold higher inhospital mortality (8.3 vs. 0.8%, \( P = 0.030 \)).

**Discussion**

The main two findings of this study are the identification of a steady increase in cocaine consumption among young patients with ACS and the adverse prognosis of these patients compared with patients with ACS-NACC.

**Prevalence of cocaine consumption**

Cocaine consumption in our cohort of patients hospitalized for ACS markedly increased from 6.8% in 2001 to 21.7% in 2008, which is probably a mirror of the general population. Indeed,
patients with a very significant increase over the last 8 years, and current cocaine users were prevalent among young ACS associated with acute cocaine use, we found that both occasional on more than 10 occasions. Although ACS has traditionally been found a 4.6-fold greater risk for patients who had used cocaine having a heart attack than non-users, and Aslibekyan et al. found that frequent cocaine users had a seven-fold higher risk of 1248pathogenesis of ACS. Six to 14% of patients with cocaine-related use. 16–18 Qureshi patients younger than 30 years of age having a history of cocaine reached 11.8%, in line with our findings with up to 25% of ACS the predominant risk factors, with males being at a higher risk and with values similar to those seen in our study. However, cocaine use is only very rarely recorded in large registries or multi-centre studies, and our data indicates that cocaine should be perceived as a cardiovascular risk factor in young patients with ACS.22

In this study, combined data from the patients’ medical history with the result of the cocaine urine test were used for detecting ACS patients with recent cocaine consumption. Cocaine was detected in the urine of up to 4.9% of patients, similar to that reported by other groups in patients admitted to the emergency room with chest pain.23

Data are given as number of patients (%) unless otherwise noted. AMI, acute myocardial infarction; CPK MB, MB fraction of creatine phosphokinase; IQR, inter-quartile range; LVEF, left ventricular ejection fraction.

*Mean difference of the variable using a logarithmic scale.

**Table 2  Differential characteristics between patients with acute coronary syndrome associated with cocaine consumption (current users or positive urine test for cocaine) and acute coronary syndrome not associated with cocaine consumption (n = 403)**

<table>
<thead>
<tr>
<th></th>
<th>ACS-ACC (n = 24)</th>
<th>ACS-NACC (n = 379)</th>
<th>P-value</th>
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<tbody>
<tr>
<td>Medical history</td>
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<tr>
<td>Tobacco use</td>
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<tr>
<td>Former smoker/non-smoker</td>
<td>0 (0)</td>
<td>104 (29.4)</td>
<td>0.003</td>
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<tr>
<td>Current smoker</td>
<td>24 (100)</td>
<td>275 (72.6)</td>
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<tr>
<td>Arterial hypertension</td>
<td>2 (8.3)</td>
<td>103 (27.2)</td>
<td>0.041</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>9 (37.5)</td>
<td>211 (55.7)</td>
<td>0.083</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4 (16.7)</td>
<td>45 (11.9)</td>
<td>0.515</td>
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<tr>
<td>Peripheral vascular disease</td>
<td>2 (8.3)</td>
<td>16 (4.2)</td>
<td>0.291</td>
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<tr>
<td>Ischaemic heart disease</td>
<td></td>
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<tr>
<td>Angina pectoris</td>
<td>2 (8.3)</td>
<td>66 (17.4)</td>
<td>0.398</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2 (8.3)</td>
<td>40 (10.6)</td>
<td>1.000</td>
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<tr>
<td>Characteristics of ACS</td>
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<tr>
<td>Unstable angina</td>
<td>1 (4.2)</td>
<td>49 (12.9)</td>
<td>0.337</td>
</tr>
<tr>
<td>Myocardial infarction (n = 353)</td>
<td>23 (95.8)</td>
<td>330 (87.1)</td>
<td></td>
</tr>
<tr>
<td>Q-wave AMI</td>
<td>20 (87.0)</td>
<td>226 (68.5)</td>
<td>0.062</td>
</tr>
<tr>
<td>Non-Q-wave AMI</td>
<td>3 (13.0)</td>
<td>104 (31.5)</td>
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<tr>
<td>CPK-MB (U/L), median (IQR)</td>
<td>245 (75–468)</td>
<td>132 (40.7–280)</td>
<td>0.038*</td>
</tr>
<tr>
<td>Tropinin I (ng/mL), median (IQR)</td>
<td>52.9 (14–95.7)</td>
<td>23.4 (7.8–53.9)</td>
<td>&lt;0.001*</td>
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<tr>
<td>Coronary anatomy (n = 281)</td>
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<tr>
<td>0–1 vessel disease</td>
<td>8 (42.1)</td>
<td>161 (61.5)</td>
<td>0.966</td>
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<td>11 (59.7)</td>
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<td>LVEF (%), mean ± SD</td>
<td>44.5 ± 15.1</td>
<td>52.2 ± 12.5</td>
<td>0.049</td>
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especially since 2005. The baseline clinical characteristics of our study did not differ from other young ACS cohorts. If we compare with the Euro Heart Survey,21 we find that, in ACS patients under the age of 55, tobacco use and dyslipidaemia are the predominant risk factors, with males being at a higher risk and with values similar to those seen in our study. However, cocaine use is only very rarely recorded in large registries or multi-centre studies, and our data indicates that cocaine should be perceived as a cardiovascular risk factor in young patients with ACS.22

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<tr>
<td>Age (years), mean ± SD</td>
<td>42 ± 6.4</td>
<td>43.3 ± 5.2</td>
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- Data from the National Plan on Drugs collected using an at-home questionnaire on alcohol and drug use in Spain showed a progressive increase in cocaine use up to 8.3% in the general population in the year 2008. Among the younger population (35 years or younger), this increase in use in the general population reached 11.8%, in line with our findings with up to 25% of ACS patients younger than 30 years of age having a history of cocaine use.

- Both acute and chronic cocaine use seem to participate in the pathogenesis of ACS. Six to 14% of patients with cocaine-related chest pain develop a myocardial infarction. Qureshi et al. found that frequent cocaine users had a seven-fold higher risk of having a heart attack than non-users, and Aslibekyan et al. found a 4.6-fold greater risk for patients who had used cocaine on more than 10 occasions. Although ACS has traditionally been associated with acute cocaine use, we found that both occasional and current cocaine users were prevalent among young ACS patients with a very significant increase over the last 8 years,
taking and a systematic search for cocaine in urine should be recommended as this group of patients not always spontaneously admit their use. Indeed, in our cohort four patients who denied cocaine use had a positive urine test for cocaine. In sum, testing for cocaine in the urine of ACS patients younger than 50 years should be incorporated in routine clinical practice.

Clinical characteristics and evolution

The diagnostic classification of ACS between cocaine users and non-users was not significantly different, and only a tendency towards a greater percentage of Q-wave AMI was observed in ACS-ACC patients. These data are consistent with those reported by the Euro Heart Survey with the main diagnosis of ACS in young patients being predominately Q-wave AMI. Remarkably, we found that ACS-ACC patients had larger infarcts as assessed by both biochemical markers of myocardial damage and ventricular function, with a greater proportion of Killip class III–IV patients and more frequent development of complications leading to increased hospital mortality. Our interpretation for the larger infarct size in the ACS-ACC group is multifactorial. On the one hand, it may be due to the greater CAD burden and the higher prevalence of Q-wave myocardial infarcts in ACS-ACC, which often jeopardize a larger myocardial area than non-Q-wave infarcts. The underuse of beta-blockade and increased adrenergic drive in cocaine patients may increase oxygen demand in ischemic myocardium and likely increase infarct size. Finally, the presence of undetected (simultaneous) vasospasms in other arteries may also explain the larger infarcts in ACS-ACC patients. The inhospital mortality was similar to that seen in the large Euro Heart Survey (1.4%) but differs from the retrospective study by Hollander et al. (0%). These differences could be explained by the different inclusion criteria of the different studies. In the study by Hollander, patients were younger, with smaller infarcts and less heart failure, and therefore at a low risk for complications and mortality compared with our cohort that included higher risk patients desiring CCU admission. Of note, we found a significantly higher mortality rate in ACS-ACC patients (8.3%) compared to ACS-NACC patients (0.8%), despite both groups receiving similar reperfusion therapies upon admission.

In addition to increased inhospital mortality, ACS-ACC patients were more likely to have ventricular tachycardia during the hospital stay, mainly within the first hours after admission. This observation is probably due to adrenergic discharge caused by cocaine and the reduced use of beta-blockers in this group. These data are consistent with those obtained by others, in which up to 17.7% of cases had ventricular tachycardia, mainly within the first 12 h. Beta-blockers can exacerbate a pre-existing vasospasm mechanism, including that induced by cocaine. As such, these drugs are mainly contraindicated in the treatment of ACS-ACC. However, beta-blockers have anti-arrhythmic and anti-ischemic effects from which these patients do not benefit. Whether beta-blockade should be reconsidered in ACS-ACC patients, especially in those with overt CAD is beyond the scope of this study, but they are certainly powerful drugs to limit arrhythmic events and prolong survival in ischaemic patients.

This study has several limitations. First, this is a single-centre study with a limited number of patients and, as such, results have to be considered with caution; second, the urine test for cocaine was not performed in ~15% of patients due to unawareness of the physician in charge. Nevertheless, the findings are a reflection of a consecutive population over 8 years and representative of the metropolitan area of Barcelona. We must acknowledge that the sample size is small and the event rate low to adequately perform full multivariable analysis.

Conclusions

The association between cocaine use and ACS has grown exponentially in the metropolitan area of Barcelona, from 6.8% in 2001 to 21.7% in 2008, and may be a reflection of current trends in the metropolitan areas of big European cities. Young adults who were admitted to the CCU with ACS had greater myocardial damage and more frequent complications.

In view of the results, we think that a specific medical history on cocaine use and urine tests for cocaine at the time of admission should be incorporated into patient care protocols in young adults with suspected ACS.

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Conflict of interest: none declared.

References


