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Paper

Cocaine: Recent trends in Northern Ireland

James R Lyness

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SUMMARY

A review of autopsy reports in cases in which cocaine featured in the cause of death in Northern Ireland revealed that there were 18 deaths between 1st January 1999 and 31st December 2007. Analysis revealed an increasing incidence of these deaths during the study period and this is compared to national statistics and those published by local drug addiction services and police.

INTRODUCTION

In the autumn of 2008, cocaine made headline news following a record seizure of the drug off the coast of Ireland, with an estimated value of £403 million. This valuable cargo was destined for the streets of the United Kingdom and Ireland[1]. Just over a week following this seizure about £150,000 worth of cocaine was seized from a property in south Belfast[2]. It would appear that this drug, once thought to be the drug of choice for more affluent sections of society, has become increasingly available in Northern Ireland (figure 1).

Cocaine is a stimulant drug and has been used by the Peruvian Indians for centuries for the well-being and increased endurance it produces after sucking on the leaves of the plant *Erythroxylon coca* and related species[3]. It produces an increased euphoria and arousal with an elevation in alertness, mood and vigour, and it is these properties which have made it a drug of abuse in modern society. In the United Kingdom it is considered a Class A drug under the Misuse of Drugs Act 1971[4].

In Northern Ireland 28% of the population aged 15-64 years reported taking illegal drugs at some point in their life. Twenty five percent of the population reported the use of Cannabis, making it the most common drug of abuse. Poppers, which contain amyl nitrate, and Ecstasy, which contains methylenedioxymethamphetamine (MDMA), were the second most prevalent drugs of abuse with 8% of the population reporting their use. Despite cocaine's relatively lower rate of usage within this population (5%) it has become increasingly prevalent in Northern Ireland society in recent years[5]. This increase in cocaine use is further demonstrated by an increased referral rate of cocaine users to services for problem drug use and an increase in the annual seizure rate of cocaine by the Police Service of Northern Ireland (PSNI)[6,7]. This review compares the experience of the State Pathologist's Department for Northern Ireland with the data collected by the above agencies.

Fig 1. Line of cocaine.

METHODS

This review examined fatalities in which cocaine featured in the cause of death in Northern Ireland over a nine year period (1999 – 2007) using retrospective review of autopsy reports from the Northern Ireland State Pathologist's Department. Pathologists of this department undertake postmortem examinations in almost all cases of sudden unnatural death instructed by Coroners in the province.

The search term 'cocaine' was entered into the electronic register of the State Pathologist's Department. This allowed interrogation of cases between 1982 and 2007. Following case identification, the autopsy report for each fatality was reviewed and the relevant details recorded.

RESULTS

Eighteen cases where the cause of death was directly associated with the use of cocaine were identified in Northern Ireland between 1st January 1999 and 31st December 2007. The date of death and final cause of death of each case are shown in Table I. During the period 1999 to 2002 there were a total of five deaths associated with cocaine use. Of note however in only one of these deaths was cocaine the solitary drug detected on toxicological screening of post mortem samples. In the remaining four cases, death was attributed to multiple drug use including cocaine combined with amphetamines and/or opiates. During the period from 2005 to 2007 there were a total of thirteen deaths of which nine

State Pathologist's Department, Institute of Forensic Medicine, The Queen's University of Belfast, Belfast, BT12 6BS, United Kingdom.

Correspondence to Dr Lyness

James.Lyness@statepathni.org.uk

TABLE I

Date of Death and final cause of death on report of autopsy

Date of Death	Cause of Death
7/1999	1. Heroin intoxication combined with cocaine and methylenedioxymethylamphetamine.
9/2000	1. Combined heroin, cocaine, thioridazine, diazepam and alcohol intoxication.
9/2000	1. Cocaine intoxication
12/2001	1. Methylenedioxymethylamphetamine and cocaine intoxication.
11/2002	1. Heroin and cocaine intoxication combined with diazepam.
1/2005	1. Poisoning by heroin, amphetamine and cocaine.
6/2005	1. Cocaine intoxication.
12/2005	1. Poisoning by cocaine. 2. Alcohol intoxication.
12/2005	1. Carbon monoxide poisoning 2. Intoxication by alcohol and cocaine.
7/2006	1. Poisoning by cocaine and methylenedioxymethylamphetamine.
8/2006	1. Poisoning by cocaine.
10/2006	1. Poisoning by cocaine.
5/2007	1. Poisoning by cocaine.
5/2007	1. Myocardial necrosis and fibrosis due to cocaine abuse 2. Self Starvation.
7/2007	1. Poisoning by cocaine.
8/2007	1. Cocaine toxicity.
8/2007	1. Poisoning by cocaine.
8/2007	1. Poisoning by cocaine 2. Alcohol intoxication.

(69.2%) were attributed to cocaine exclusively, including cases in which alcohol was also detected.

Despite no cocaine associated deaths being recorded in 2003 and 2004, during the review period there was an overall trend of increasing annual number of deaths associated with cocaine as reported by the State Pathologist's Department for Northern Ireland. Figure 2 shows the annual number of deaths in which cocaine is included in the final cause of death following full autopsy and toxicological examination. Although a relatively low number of cases, there is a definite upward trend, particularly during the period 2005-2007. There were no cocaine associated deaths recorded prior to 1999.

The average age of the deceased at the time of death was 30.27 years (range 18-51 years). Sixteen (89%) of the 18 deaths were male and two (11%) were female. Eight of the 18 (44%) individuals were unemployed at the time of death

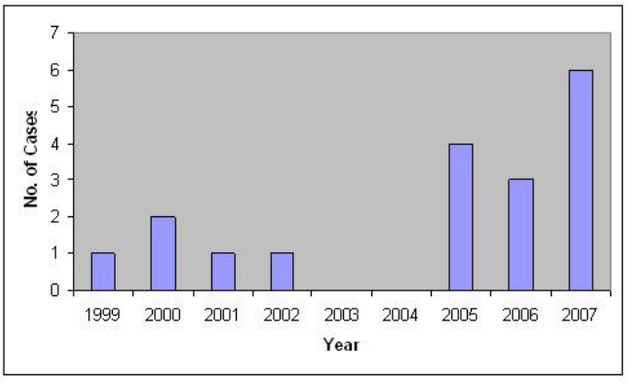

Fig. 2. Annual number of cocaine associated deaths recorded by the State Pathologist's Department for Northern Ireland.

and 10 (56%) employed. Five of the 18 (28%) individuals had no significant medical history, four (22%) had a history of previous drug abuse, five (28%) had a history of depression and two (11%) had a history of cardiovascular disease.

At autopsy there were minimal external findings and the majority of these were non specific. Two cases (11%) showed petechial haemorrhages in the conjunctival lining of the eyelids and six (33%) showed facial congestion. Two (11%) were associated with minor self inflicted or accidental trauma. In five (28%), needle puncture marks were detected, of these, three were associated with cocaine use combined with opiate use, one involved resuscitation attempts and in another, the needle mark was not thought to have been related to cocaine use.

On internal examination, eight cases (44%) showed cerebral swelling and the average brain weight was 1524g (range 1340 – 1870g). In three cases (17%) there was evidence of tongue biting, which may indicate seizure activity or simply be associated with a terminal cardio-respiratory arrest. The average heart weight was 383g (range 225 – 496g) and nine cases (50%) were over 400g in weight reflecting a degree of myocardial hypertrophy. Fourteen cases (78%) had normal coronary arteries, two (11%) demonstrated moderate narrowing, and in two cases (11%) there was severe narrowing. Two of the four cases with coronary artery disease had a previous history of cardiovascular disease. Myocardial fibrosis was detected on microscopy in five cases (28%).

Alcohol analysis of blood samples taken at post mortem revealed concurrent alcohol consumption in eight cases (44%). There was an overall average blood alcohol concentration of 61.6 mg per 100mls (range 0 – 231mg per 100mls).

Toxicological analysis of post mortem blood samples detected unmetabolised cocaine in 14 cases (78%). Of these 14 cases only 10 specified the concentration of the drug detected and the average concentration was 1.22µg per ml (range 0.03-5.9 µg per ml). The major cocaine metabolite, benzoylecgonine, was detected in 17 cases (94.4%) including the four cases in which no unmetabolised cocaine was reported. The cutting agent lignocaine was detected in nine cases (50%). Amphetamines were detected in five cases (28%) and opiates in four cases (22%). Furthermore, in three of the cases in which opiate was detected the heroin metabolite

6-monoacetylmorhine was also reported. Nasal swabs were taken in seven cases (39%) and all of these were positive for cocaine, indicating nasal insufflation. Three (17%) of the seven cases in which stomach contents were analysed showed the presence of cocaine, consistent with oral intake, however, the possibility of drug swallowed following nasal insufflation cannot be excluded.

DISCUSSION

The incidence of deaths attributed to cocaine in Northern Ireland remains low but there has been an increase in the number of cocaine related deaths during the review period, 1999 to 2007. Of particular note there was an increase in the number of deaths attributable to cocaine as the single drug of use since 2005. Prior to this cocaine was largely associated with multiple drug use. Statistics published by the Office for National Statistics for England and Wales show a similar increase in the annual numbers of deaths where cocaine was mentioned on death certification between 1998 and 2006[8,9] (figure 3).

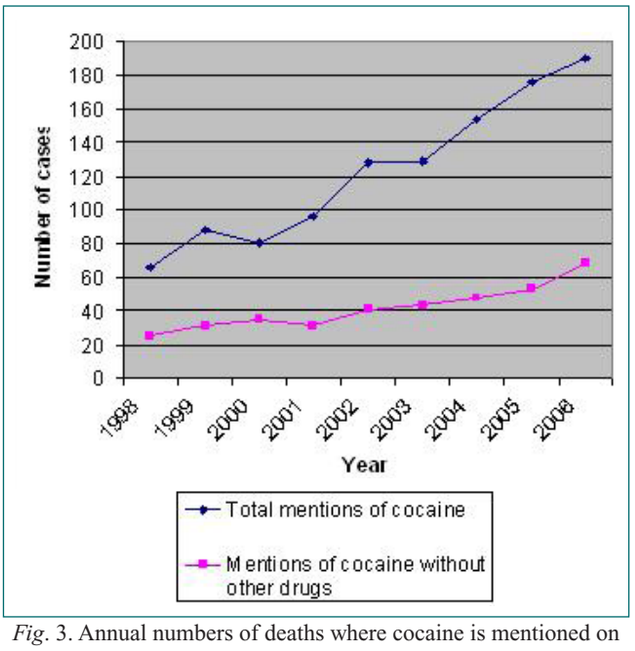

Fig. 3. Annual numbers of deaths where cocaine is mentioned on the death certificate in England and Wales, 1998 to 2006.

Drug addiction services and the PSNI have reported similar trends within Northern Ireland during this time. Figure 4 shows the breakdown of drug use in individuals presenting to drug addiction services for problem drug misuse in Northern Ireland during the 12 month period ending 31st March 2007. Only slightly more individuals reported cocaine (147) than heroin (145) as their primary problem drug, making cocaine the third most reported main problem drug of abuse. The proportion of individuals reporting cocaine use increased to 30% when all subsidiary abused drugs are included, making it the second most common drug of misuse behind cannabis. There was also an increase in the proportion of clients between the periods 2005/06 and 2006/07 who reported cocaine as their main problem drug, from 5% to 11% respectively[10]. Of note, these figures do not include alcohol which is undoubtedly the most common substance of abuse.

There has been an increase in the number of cocaine seizures

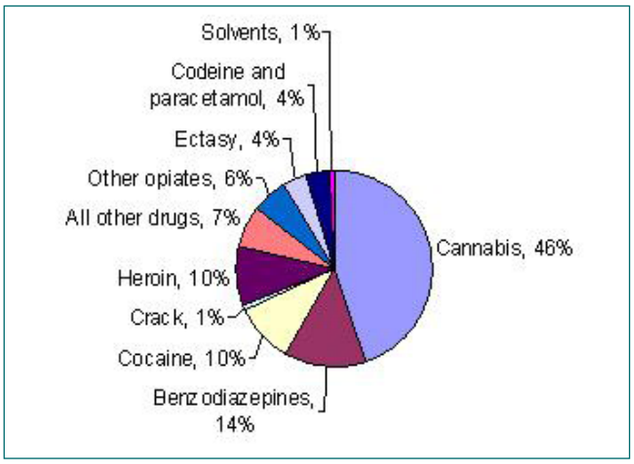

Fig. 4. Main drug of abuse reported by individuals presenting to services for problem drug misuse in Northern Ireland between March 2006 and March 2007.

recorded by the Police Service for Northern Ireland. In the year 2005/06 the PSNI recorded 168 individual seizures of cocaine - this increased to 405 individual seizures in the year 2007/08[7].

These figures may reflect increases in the supply of cocaine within Northern Ireland, changes in the demand, trends within the local drug culture, or reporting of use. As a consequence it seems probable that cocaine will become an increasing problem in Northern Ireland and as a result there will be a corresponding increase in the incidence of cocaine related health problems.

Cocaine use can affect all of the body systems and death can result from a single dose or from the chronic effects due to prolonged use. Table II shows a summary of the major cocaine related diseases. Many of the pathological changes associated with cocaine use appear to be catecholamine mediated. Cocaine use disrupts catecholamine metabolism and it has been shown that cocaine abusers have elevated circulating levels of catecholamines[11]. The most commonly involved system is the cardiovascular due to the cocaine associated elevation in blood pressure, heart rate and vascular tone. Potentially lethal myocardial alterations include hypertrophy, fibrosis and microangiopathy[12-14]. As indicated in Table II, cocaine use is also associated with accelerated atherosclerosis and an increased risk of thrombosis. The risk of sudden death is further increased if an individual has pre-existing coronary artery disease. Similarly, coronary artery spasm (which is commonly quoted by medical students in relation to cocaine use) is unlikely to cause episodes of myocardial infarction unless it occurs at the site of pre-existing coronary atheroma[11]. In this series, 22% of the cases had evidence of coronary atheroma and 28% showed evidence of chronic myocardial damage on microscopy by the detection of myocardial fibrosis. The fibrosis in each case was patchy or multi-focal, which is consistent with catecholamine toxicity rather than the typical, more confluent, pattern seen in coronary atheromatous disease. Chronic cocaine users have an increased incidence of cardiac hypertrophy, which further increases the risk of sudden cardiac death, and 50% of the hearts examined were over 400g in weight, in keeping with a degree of myocardial hypertrophy.

TABLE II.

Major Cocaine associated diseases

Cardiovascular System	• Myocardial infarction • Arrhythmia • Accelerated atherosclerosis • Thrombosis • Coronary artery spasm • Myocardial hypertrophy • Myocardial fibrosis • Aortic dissection
Central Nervous System	• Cerebral infarction • Subarachnoid haemorrhage • Intracranial haemorrhage • Seizures • Excited Delirium
Respiratory System	• Erosion and perforation of nasal septum • Bronchiolitis obliterans • Pulmonary oedema • Pulmonary hypertension
Gastrointestinal System	• Ischaemic colitis
Renal System	• Acute tubular necrosis due to Rhabdomyolysis • Accelerated arteriosclerosis • Infarction and thrombosis

The second major organ system associated with sudden death and cocaine use is the central nervous system. There were no definite cases within this series in which death was attributed to central nervous system sequelae of cocaine. However, it is well recognised that the incidence of intracerebral haemorrhage, cerebral ischaemia and subarachnoid haemorrhage following drug abuse is increasing[15]. Seventeen percent of the cases in this study showed signs of tongue biting, but this is a non specific finding and should not be taken as definitive evidence of a seizure.

Excited delirium is often associated with cocaine use and is widely reported in the literature following numerous controversial cases involving individuals, apparently suffering from this condition, who were in the custody of the police or who were being chased by the police at the time of death[16,17]. Excited delirium is characterised by hyperthermia, delirium, agitation, respiratory arrest and subsequent death. The precise mechanism of death in these cases is somewhat controversial, and a full discussion is beyond the scope of this review, but it is often associated with hyperthermia, rhabdomyolosis, hyperkalaemia and sudden cardio-respiratory arrest[18]. None of the cases in this series specifically recorded a diagnosis of excited delirium. However, there were two cases in which bizarre behaviour was noted prior to death. The first involved apparently uncontrolled physical activity, within the confines of a bedroom, which resulted in multiple injuries. The second reported that the deceased individual had forced a vacuum cleaner nozzle into his mouth, apparently to alleviate shortness of breath.

There has been considerable media attention surrounding cases of cocaine related nasal septum perforation. This is often due to celebrities using nasal insufflation of cocaine. Such is the increasing usage of cocaine worldwide that its' insufflation should be included in the differential diagnosis of destructive lesions of the mid face, along side more classical diagnoses such as Wegener's Granulomatosis[19]. In this series only 39% of cases record a nasal swab being taken at post mortem and all of these yielded a positive result for cocaine. None of these demonstrated any obvious nasal septum necrosis.

Additional common routes of administration of cocaine include intravenous injection and oral consumption. Twenty eight percent of cases recorded needle marks on external examination at post mortem. None of these are thought to have been caused by the solitary injection of cocaine and all were associated with either combined opiate use or medical intervention. Seventeen percent of the seven cases in which stomach contents were analysed were positive for cocaine. This may indicate oral consumption but it may also be accounted for by swallowing of traces cocaine following nasal insufflation or redistribution in the post mortem period. In cases of oral consumption it is recognised that dentists may encounter individuals with cocaine related oral disease[20]. Finally, crack cocaine, which is cocaine produced in its base form, can be smoked and the detection of the cocaine metabolite anhydroecgonine methyl ester, in toxicological specimens, would confirm this route of administration[21]. Routine toxicological analysis does not screen for this metabolite.

Analysis and interpretation of post-mortem toxicological specimens is complex and provides a number of very specific challenges. These include consideration of the unstable nature and degradation of the substance and redistribution of the substance from tissues containing a higher concentration than the blood during the post-mortem interval. Furthermore, with prolonged usage of some drugs, including cocaine, tolerance will undoubtedly develop to the desired effects[22]. Therefore, there is no well defined fatal range of blood levels for cocaine. Indeed, in cases in which toxicological analysis is negative for cocaine but at post mortem there is evidence of potentially fatal complications of the drug in a known user, it would not be unreasonable to include cocaine as a factor in death. Unmetabolised cocaine was detected in 14 cases (78%), indicating recent usage of the drug prior to death. The term 'recent usage' is often cited as it can be difficult, as with any drug, to give a dogmatic indication of the time prior to death following its administration The half life of cocaine is reported as being between 0.7 and 1.5 hours, depending on whether or not the individual is a chronic or naïve user. In the absence of alcohol the principle metabolite of cocaine is benzoylecgonine and this was detected in 17 of the reviewed cases, including the 4 cases in which no unmetabolised cocaine was detected. Benzoylecgonine has a much longer half-life than cocaine, approximately 4.5 hours, and is likely to be detectable in the plasma for up to 48hrs after ingestion[23]. The single case in which no benzoylecgonine was detected would indicate extremely rapid death following administration of cocaine.

CONCLUSION

The incidence of cocaine associated deaths recorded by the State Pathologist's Department has increased during the period 1999-2007. This upward trend appears to reflect similar trends in statistics published by other agencies which show increased availability and use of cocaine in Northern Ireland. As the use of cocaine continues to increase the medical profession will increasingly be exposed to cocaine related morbidity and mortality.

The author has no conflict of interest.

REFERENCES

1. Three held over half billion euro cocaine bust. *Belfast Telegraph*. 2008 Nov 7. Available from: http://www.belfasttelegraph.co.uk/news/local-national/three-held-over-half-billion-euro-cocaine-bust-14043626.html. Last accessed February 2009.
2. Cocaine seized in south Belfast. *BBC News.* 2008 Nov 15. Available from: http://news.bbc.co.uk/1/hi/northern_ireland/northern_ireland_politics/7730758.stm. Last accessed February 2009.
3. Drummer OH, Odell M. The Forensic Pharmacology of Drugs of Abuse. London: Arnold; 2001.
4. Home Office. Drugs and the law. Classes of drugs. Class A, B and C drugs. Available from: http://www.homeoffice.gov.uk/drugs/drugs-law/Class-a-b-c. Last accessed February 2009.
5. Drug Use in Ireland and Northern Ireland: first results from the 2006/2007 Drug Prevalence Survey. National Advisory Committee on Drugs (NACD) & Drug and Alcohol Information and Research Unit (DAIRU). Belfast: Department of Health, Social Services; January 2008. Available from: http://www.dhsspsni.gov.uk/first-results-200607-drug-prevalence-survey.pdf. Last accessed February 2009.
6. Department of Health, Social Services and Public Safety. Statistics from the Northern Ireland Drug Addicts Index 2007. Belfast: Northern Ireland Statistics and Research Agency, Health, Social Services and Public Safety: March 2008. Available from: http://www.dhsspsni.gov.uk/addicts-index-report-2007.pdf. Last accessed February 2009.
7. PSNI Statistics, Annual Statistical Report, Statistical Report No. 4. Drug seizures and Arrests, 1st April 2007 – 31st March 2008. Belfast: Police Service of Northern Ireland; 2008. Available from: http://www.psni.police.uk/4._drug_seizures_and_arrests_summary.pdf. Last accessed February 2009.
8. Office for National Statistics, Deaths related to drug poisoning: England and Wales, 1998-2002, *Health Statistics Quarterly.*2004;21(Spring):59-65. Available from: http://www.statistics.gov.uk/downloads/theme_health/HSQ21.pdf. Last accessed February 2009.
9. Office for National Statistics, Deaths related to drug poisoning in England and Wales, 2002-2006, *Health Statistics Quarterly* 2007;**36:**66-72. Available from: http://www.statistics.gov.uk/downloads/theme_health/HSQ36.pdf. Last accessed February 2009.
10. Department of Health, Social Services and Public Safety. Statistics from the Northern Ireland Drug Misuse Database: 1st April 2006 – 31st March 2007. Northern Ireland Statistics and Research Agency, Health, Social Services and Public Safety. Available from: http://www.dhsspsni.gov.uk/statistics-from-ni-drugs-misuse-database-01april06-31march07.pdf. Last accessed February 2009.
11. Karch SB. Karch's Pathology of drug abuse. 3rd ed. Boca Raton: CRC Press; 2002.
12. Lange RA, Cigarroa JE, Hillis LD. Theodore E. Woodward Award: cardiovascular complications of cocaine abuse. *Trans Am Clin Climatol Assoc* 2004;**115**:99-114.
13. Karch SB. Cocaine cardiovascular toxicity, *South Med J* 2005;**98(8):**794-9. Comment in: Bauman JL. Response to cardiovascular toxicity. 2006;**99(7)**:780. Author reply 2006;**99(7):**780.
14. Egred M, Davis GK. Cocaine and the heart. *Postgrad Med J* 2005; **81(959)**:568-71.
15. McEvoy AW, Kitchen ND, Thomas DG, Intracerebral haemorrhage and drug abuse in young adults. *Br J Neurosurg* 2000;**14(5)**:449-54.
16. Bunai Y, Akaza K, Jiang WX, Nagai A. Fatal hyperthermia associated with excited delirium during an arrest. *Leg Med (Tokyo)* 2008;**10(6)**:306-9.
17. Pollanen MS, Chiasson DA, Cairns JT,Young JG. Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. *Can Med Assoc J* 1998;1**58(12)**:1603-7.
18. Ruttenber AJ, McAnally HB, Wetli CV. Cocaine associated rhabdomyolyisis and excited delirium: different stages of the same syndrome. *Am J Forensic Med Pathol* 1999;**20(2)**:120-7.
19. Rachapalli SM, Kiely PD. Cocaine-induced midline destructive lesions mimicking ENT-limited Wegener's granulomatosis, *Scand J Rheumatol* 2008;**37(6):**477-80.
20. Brand HS, Gonggrijp S, Blanksma CJ. Cocaine and oral health. *Br Dent J* 2008;**204(7)**:365-9.
21. Jenkins AJ, Goldberger BA. Identification of unique cocaine metabolites and smoking by-products in postmortem blood and urine specimens, *J Forensic Sci* 1997;**42(5)**:824-7.
22. Drummer OH. Postmortem toxicology of drugs of abuse. *Forensic Sci Int* 2004;**142(2-3)**:101-13.
23. Baselt RC. Disposition of toxic drugs and chemicals in man. Foster City, CA; Chemical Toxicology Institute: 2000.

