

Acute cocaine intoxication in a body packer

Mee-Jung Park, Mi-Ae Lim and Hee-Sun Chung

*Dept of forensic science, National Institute of Scientific Investigation, 331-1,
Shinwol-7-dong, Yangcheon-gu, Seoul, 158-707, Korea*

(Received November 11, 2005, Accepted December 16, 2005)

Abstract : A 35-year-old Peruvian who suffered from grand mal seizures died in the aircraft on his way from the United States to Hongkong via Incheon international airport of Korea. While performing the autopsy, 115 packs made with double layer of transparent film and black plastic bag containing cocaine were found in the ileum and large intestine. Among of them, 3 packs were ruptured. To determine the concentration of cocaine and its metabolites, blood, urine, bile, liver, spleen, heart, kidney, brain and gastric contents were taken and toxicological analysis was performed. Gas chromatography/mass spectrometry (GC/MS) following liquid-phase extraction using chloroform:isopropanol (=9:1) and derivatization with bis(trimethylsilyl)-trifluoroacetamide (contains 1% trimethylchlorosilane) was performed. High levels of cocaine, benzoylecgonine (BE) and ecgonine methylester (EME) were found in the postmortem blood (0.96, 3.09 and 5.59 $\mu\text{g/mL}$) and urine (32.85, 145.35 and 53.17 $\mu\text{g/mL}$), respectively. Cocaine and its metabolites were also detected in all other biological specimen.

Key words : Cocaine, Body Packer, Biological specimen, GC/MS

1. Introduction

Body packers, also known as 'stuffers', 'swallowers' or 'mules' are persons who conceal narcotics for transport in capsules, condoms, balloons, plastic bags or fingers of latex gloves in various anatomical cavities or body orifices.^{1,2} The drug containers endanger the carrier because of the risk of leakage or rupture with subsequent absorption.³

A man suffered from hyperthermia, dizziness, abdominal pain, agitation, convulsion and grand mal seizure died in the aircraft of Korean Airlines. As he was dead in the aircraft of Korean Airlines designated to Incheon international airport, autopsy was performed

in National Institute of Scientific Investigation in Seoul, Korea. While performing the autopsy, 115 packs made with double layer of transparent film and black plastic bag containing cocaine were found in the gastrointestinal tract especially in the ileum and large intestine (*Fig. 1*). Each size of these packs was about 1.5~4 cm of length and 1.5 cm of diameter (*Fig. 2*). Among 115, three packs were ruptured and rapid absorption of cocaine from the gastrointestinal tract resulted in acute cocaine poisoning to death.

Cocaine is rapidly metabolized to EME by serum cholinesterase and liver esterase, and to BE by chemical hydrolysis (*Fig. 3*).^{4,5} This paper describes the identification and quantification of cocaine in a

★ Corresponding author

Phone : +82-(0)2-2600-4928 Fax : +82+(0)2-2600-4919

E-mail: meejung@nisi.go.kr



Fig. 1. Cocaine packs found in postmortem gastrointestinal tract especially in ileum and large intestine.



Fig. 2. Cocaine packs found in ileum and large intestine. Each size of these packs was about 1.5~4 cm of length and 1.5 cm of diameter (left). These were made with double layer of transparent film and black plastic bag (right).

body packer's postmortem specimen using GC/MS.

2. Materials and Methods

2.1. Reagents and standards

1 mg/mL methanol solution of cocaine, BE and EME were purchased from Cerilliant Co. (Round Rock, Texas, USA). Dextromethorphan (DM), bis(trimethylsilyl)trifluoroacetamide (contains 1% trimethylchlorosilane) (BSTFA/TMCS) were purchased from Sigma-Aldrich Co.(St. Louis, MO, USA).

2.2. Standard solution & calibration curves

1.0 mg/mL methanol solution of cocaine, BE and

EME were used for analysis. Stock solutions of 0.1, 1 and 10 $\mu\text{g/mL}$ of each drugs were made by serial dilution. Calibration curves for cocaine, BE and EME over range of 0.1~10 $\mu\text{g/mL}$ were determined with 10 $\mu\text{g/mL}$ DM (internal standard) solution. The ratios of the peak areas of cocaine, BE and EME to those of DM were utilized to calculate the concentration of these analytes in postmortem specimen.

2.3. Specimen

Postmortem blood, urine, bile, liver, spleen, heart, kidney, brain and gastric contents were obtained during autopsy and stored at -70°C until the toxicological analysis.

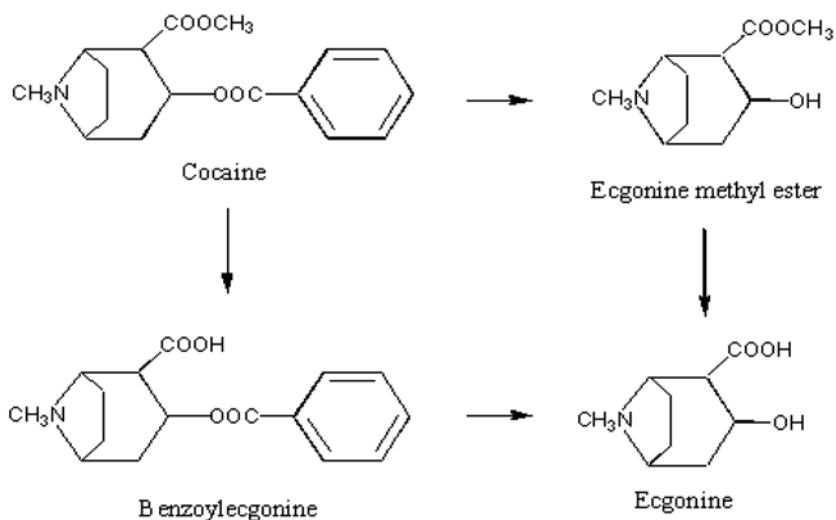


Fig. 3. Metabolic pathway of cocaine. It's major metabolites are benzoylecgonine (BE) and ecgonine methylester (EME).

2.4. Extraction and derivatization

Biological tissues were homogenized as previously described.⁶ Briefly, 1 g of samples was homogenized with 3 mL of 3% perchloric acid solution. After centrifugation, upper layer was used for extraction. Other non-tissue samples such as blood, urine, bile juice and gastric contents were taken 1 mL or 1 g. 500 ng of DM (internal standard) was added to samples and adjust pH=8 with sodium bicarbonate: potassium carbonate(=2:1). The drugs were extracted with 5 mL of chloroform:isopropanol(=9:1) thrice and organic layer was evaporated to dryness. To the dried extract, 30 μ l of ethyl acetate and 30 μ l of BSTFA (1% TMCS) was added and incubated at 90°C for 20 min. After cooling at room temperature, 1 μ l was injected into GC/MS. We used UNDCP guidelines for extraction and derivatization for cocaine and its metabolite in biological specimen.⁷

2.5. Instrumentation

Cocaine and its metabolite BE, EME were identified and quantified with GC/MS analysis with Agilent 6890 series GC, interfaced to Agilent 5973 MSD. A fused silica capillary HP-5 MS (30 m \times 0.25 mm, 0.25 μ m film thickness) column was used in the splitless injection mode. The oven temperature was initially 100°C for 1 min and then raised to 290 °C (10 min) at a 20°C/min. The temperature of the ion source and transfer line was 230 and 270°C, respectively. Mass spectrometer was set to monitor the molecular ions using electron impact mode at 70 eV. After the identification of cocaine, BE and EME with full scan mode, selective ion monitoring mode was used for quantification of the drugs. Following ions were selected for SIM mode: cocaine (82, 182, 303), BE-TMS (82, 240, 361), EME (82, 96, 168) and DM (271, 150). Underlined ions were used for quantification.

3. Results and Discussion

The smuggling of illicit drugs by concealing them within the human body has become a widespread practice.⁸ The body packer syndrome refers to

international smugglers who ingest or insert into body orifices small packages of drugs for the purposes of transport and subsequent retrieval of the drug in a foreign country.⁹ During their stay in the stomach, the packages may be damaged by mechanical movement or by chemical digestion of the binding by which the latex fingers/condoms are tied. Cocaine may diffuse, or fluid from the gastrointestinal tract may permeate the wall of the packet, drawn in by the hyperosmolaric cocaine salt and resulting in rupture or leakage of the package.¹⁰ Cocaine intoxication presents as a sympathomimetic syndrome with a constellation of tachycardia, hypertension, mild agitation, mydriasis and diaphoresis. More severe intoxications may produce seizures, hyperthermia and dysrhythmias.¹¹

Calibration curves for cocaine, BE and EME were linear from 0.1~10 μ g/mL ($r > 0.99$, respectively). Cocaine and EME were detected as low as 0.02 μ g/mL. In case of BE, it was detected as low as 0.01 μ g/mL. The toxicological data of our case are given in *Table 1*. Blood concentration of cocaine, BE and EME were 0.96, 3.09 and 5.59 μ g/mL, respectively. In case of urine, concentration of cocaine, BE and EME were 32.85, 145.35 and 53.17 μ g/mL, respectively. In 13 cases of cocaine intoxication at Maryland in US, the mean blood cocaine and BE concentration were 0.90 μ g/mL (range: 0.05~3.40 μ g/mL) and 3.96 μ g/mL (range: 0.30~9.10 μ g/mL), respectively. Jenkins *et al.* also reported that blood to urine cocaine ratios in cocaine-related deaths was ranged from 0.002~10

Table 1. Concentrations of cocaine and metabolites (μ g/mL or μ g/g) in biological specimen

Tissue	Cocaine	BE	EME
Blood	0.96	3.09	5.59
Urine	32.85	145.35	53.17
Bile	2.96	4.89	14.84
Liver	0.12	0.73	1.09
Spleen	2.90	0.28	0.18
Heart	3.54	0.32	0.31
Kidney	4.46	0.44	0.30
Brain	3.94	0.18	0.31
Lung	0.26	0.20	3.44
Gastric content	3.19	2.27	0.86

$\mu\text{g/mL}$ (mean: $0.12 \mu\text{g/mL}$) and blood cocaine to BE ratios ranged from $0.008\sim 0.063 \mu\text{g/mL}$ (mean: $0.21 \mu\text{g/mL}$).¹² In our cases, blood to urine cocaine ratio was 0.03 and cocaine to BE ratio in blood was 0.31 .

The estimated minimum lethal dose of cocaine is 1.2 g , but susceptible persons have died from doses as small as 30 mg when applied to mucous membranes. Generally, in body packer cases, large quantities of cocaine are not involved, but due to the poor wrapping lethal exposure may ensue.

Mean brain cocaine concentration in overdose fatalities are $13.3 \mu\text{g/g}$ (range: $0.17\sim 31 \mu\text{g/g}$), whereas brain levels reaching $30 \mu\text{g/g}$ are more likely to be found in those fatalities with suicidal intention.¹³ In our cases, brain concentration of cocaine, BE and EME were 3.94 , 0.18 and $0.31 \mu\text{g/g}$, respectively. Urine, bile, spleen, heart, kidney and brain had exceeded cocaine concentration compared to that of blood. These results are in agreement with previously reported tissue data and indicate that when urine is not available, kidney, spleen, heart, brain and bile should be the specimen of choice for cocaine detection.¹⁴ In blood, urine, bile and liver, concentration of BE and EME were higher than that of cocaine and opposite result were found in spleen, heart, kidney and brain.

In summary, cocaine and its metabolites, BE and EME were detected in blood, urine, bile, gastric content, liver, spleen, heart, kidney, and brain. Toxicological analysis revealed that acute cocaine poisoning was the cause of death of the body packer.

References

1. J. M. Glass and H. J. Scott, *J. R. Soc. Med.*, **88**, 450-453(1995).
2. A. Stewart, N. D. Heaton and B. Hogbin, *Postgrad. Med. K.*, **66**, 659-661(1990).
3. A. Heinemann, S. Miyaishi, A. Iwersen, A. Schmoldt and A. K. Puschel, *Forensic Sci. Int.*, **92**, 1-10(1998).
4. R. C. Baselt, *J. Chromatogr.*, **268**, 502-505(1983).
5. Y. Liu, R. D. Budd and E. C. Griesemer, *J. Chromatogr.*, **248**, 308-310(1982).
6. H. S. Chung, E. M. Kim, M. D. Kim and M. K. Kim, *Annual Report of N.I.S.I.*, **28**, 325-332(1992).
7. UNDCP guidelines, Recommended Methods for the Detection and assay of Heroin, Cannabinoids, cocaine, Amphetamine, Methamphetamine and Ring-substitute amphetamine derivatives in Biological Specimens: Manual for use by National Laboratories by UNDCP (1995).
8. T. Robinson, R. Birrer, N. Mandava and W. F. Pizzi, *Surgery*, **113**, 709-711(1993).
9. F. Patel, *Forensic Sci. Int.*, **108**, 61-66(2000).
10. N. E. Beck and J. E. Hale, *Br. J. Surg.*, **80**, 1513-1516 (1993).
11. K. A. Sporer and J. Firestone, *Ann. Emerg. Med.*, **29**, 596-601(1997).
12. A. J. Jenkins, B. Levine, J. Titus and J. E. Smialek, *Forensic Sci. Int.*, **101**, 17-25(1999).
13. V. R. Spiehlerand and D. Reed, *J. Forensic Sci.*, **30**, 1003-1011(1985).
14. A. Poklis, A. Maginn and J. L. Barr, *Forensic Sci. Int.*, **33**, 83-88(1987).