Fatal Strychnine Poisoning: Application of Gas Chromatography and Tandem Mass Spectrometry

Thomas G. Rosano[1,*], Jeffrey D. Hubbard[2], John M. Meola[1], and Thomas A. Swift[1]

[1]Department of Pathology and Laboratory Medicine, Albany Medical Center, 43 New Scotland Avenue, Albany, New York 12208 and [2]Bender-St. Peter's Laboratory, Albany, New York 12208

Abstract

The history and toxicological findings in a case of suicidal fatal strychnine poisoning are presented along with a description of the analytical methods. Detection and quantitation of strychnine in body fluids and tissues was performed by gas chromatography (GC) with nitrogen-phosphorus detection, using organic extraction and calibration by a standard addition method. Strychnine concentrations in subclavian blood (1.82 mg/mL), inferior vena cava blood (3.32 mg/mL), urine (3.35 mg/mL), bile (11.4 mg/mL), liver (98.6 mg/kg), lung (12.3 mg/kg), spleen (11.8 mg/kg), brain (2.42 mg/kg), and skeletal muscle (2.32 mg/kg) were determined. Confirmation of strychnine in blood and tissue was performed by GC with detection by tandem ion-trap mass spectrometry (MS). GC–MS–MS analysis, employing electron ionization followed by unit mass resolution and collision-induced dissociation of strychnine, resulted in confirmatory ions with mass-to-charge ratios of 334 (parent ion), 319, 306, 277, 261, 246, 233, and 220. Additional confirmation was provided by GC–MS–MS–MS analysis of each confirmatory ion, revealing an ion fragmentation pathway consistent with the molecular structure of strychnine. The case demonstrates body tissue and fluid distribution of strychnine in a fatal poisoning and the application of tandem MS in medical examiner casework.

Introduction

Strychnine is an odorless, bitter-tasting alkaloid originally isolated from the St. Ignatius plant (*Strychnos ignatii*) by Pelletier and Caventon. It is the principal alkaloid of certain members of the Strychnos family of plants and is normally obtained for commercial use from ripe seeds of *Strychnos nux-vomica,* which is native to India (1). Strychnine was used medically as early as 1540 and, until the early 1960s, was a component of various over-the-counter tonics and laxatives in the United States (2,3). Therapeutic use was discontinued in the United States with a 1962 amendment to the Federal Food, Drug and Cosmetic Act. Although there is no current medical use for strychnine, it remains available in this country for laboratory, research, and restricted pesticide use. The Environmental Protection Agency currently lists 38 actively registered strychnine-containing pesticides, including 21 restricted products (4). In a number of individual states, the use of all strychnine-containing pesticides has been restricted to certified pest exterminators (5).

Strychnine availability is restricted because of the agent's potent convulsant and stimulant effects on motor neurons of the central nervous system. The mechanism of neuroexcitation does not involve direct neuronal stimulation but occurs through competitive antagonism of the inhibitory neurotransmitter glycine (2). The inhibition of glycine receptors, which are located primarily in the spinal cord but also in the brain stem and higher centers (6,7), produces increased activity of motor neurons. Motor stimulation results in well-documented symptoms, including a variable prodromal state of agitation followed by severe, life-threatening muscle spasms (1,8,9). The characteristic pattern of muscle contractions include flexor and extensor spasms of the upper limbs and lower limbs, respectively, as well as opisthotonos and facial muscle spasms that are referred to as risus sardonicus. Motor convulsions are often sensory elicited. The usual cause of an acute death is respiratory arrest resulting from severe spasms of respiratory muscles. The minimum lethal dose of strychnine, although reported to be 1 to 2 mg/kg, varies with deaths resulting from doses as low as 5 to 10 mg in children to a reported adult survival following ingestion of 3750 mg (8). Prior to restriction of use in therapeutics, strychnine was a major cause of accidental poisoning in children (10,11). Even with restricted availability, strychnine poisonings classified as accidental (12–18), suicidal (1,19–30), homicidal (28,31–32), and undetermined (33–35) continue to be reported in the literature. American Association of Poison Control Centers data also indicate continuing accidental and intentional poisoning with strychnine and list 40 human exposures in the most recent report (36).

Detection and quantitation of strychnine in body fluids and tissue by valid analytical methods is important in the forensic investigation of deaths from strychnine poisoning. Postmortem measurements in fatal cases and serial measurements in survivors or delayed deaths form the basis for our understanding of strychnine distribution and toxicokinetics in humans.

* Author to whom correspondence should be addressed.

Classical methods of strychnine identification are based upon nonspecific color reactions or thin-layer chromatography with a number of approaches to quantitation employing organic-solvent extraction and ultraviolet measurement (29,37,38). Although earlier methods may still be useful in initial screening protocols, analytical confirmation and quantitation of strychnine in casework has been performed primarily by gas chromatographic methods employing flame ionization detection (GC–FID) (22,25,28), nitrogen-phosphorus detection (GC–NPD) (24), or mass spectrometry (GC–MS) (19,31,34,39) detection methods. In this report we describe a GC–NPD method for detection and quantitation of strychnine in a wide range of body fluids and tissues and also report a new GC–MS method employing tandem MS.

Case History

A white male in his fourth decade was found deceased in his bed in the morning. The deceased was found by his male roommate, along with a note expressing love for his girlfriend. The roommate described noises "like someone having a bad dream" coming from the decedent's room on the night preceding the discovery of the body. A small bottle labeled "strychnine sulphate" and a drink glass with particulate matter on the inner surface of the glass were found at the scene. Statements by a brother of the decedent indicated his prior knowledge of the bottle, apparently acquired by the victim as a gift some years previously, but denied knowledge of the suicidal intent. An autopsy revealed marked congestion of several organs. The lungs were dark red-purple and weighed 1540 g, 1.5 times the upper limit of normal. The stomach, which contained a few milliliters of murky brown fluid, was microscopically normal. There were no other abnormalities at autopsy. The bottle and drink container, along with samples of inferior vena cava blood, subclavian blood, urine, gastric contents, bile, liver, kidney, lung, brain, skeletal muscle, and spleen, were submitted for toxicological analysis.

Materials and Methods

Specimens

Toxicological analyses were performed on liver, lung, brain, spleen, skeletal muscle, bile, urine, and blood collected from both inferior vena cava and subclavian vein. All specimens were stored at –10°C prior to analysis. Qualitative toxicology testing was also performed on particulate matter obtained from the drink glass and on a sample of the white powder obtained from the inside surface of the bottle submitted in the case.

GC–NPD basic drug screen

Tissue samples of liver, lung, brain, spleen, and skeletal muscle were prepared for chromatographic analysis by homogenizing 3 to 5 g of tissue diluted with an equal volume of deionized water. A variable speed Tissue-Tearer (model 985-370, Fisher Scientific, Pittsburgh, PA) was used for homogenization. For extraction, 2.0 mL of a sodium chloride saturated Tris buffer (pH 9.3) and 0.200 mL of methapyrilene (1 mg/L) were added to 2.0 mL of homogenate, blood, or body fluid. The homogenate was adjusted to pH 9.3 and extracted with 10 mL of *n*-butyl chloride. The organic layer was back-extracted with 3.0 mL of 0.25N sulfuric acid. The aqueous phase was then alkalinized with sodium hydroxide and re-extracted with 5.0 mL of *n*-butyl chloride. The dried extract was reconstituted with ethyl acetate and injected into an Autosystem GC (Perkin Elmer, Norwalk, CT) equipped with an autosampler, an NPD, model 1022 data integrator, and a DB-5MS capillary column

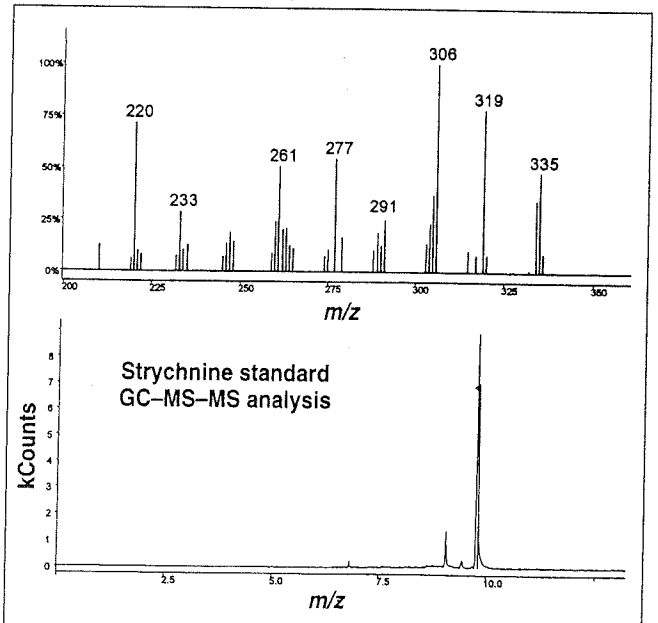

Figure 1. Reconstructed ion chromatogram and mass spectrum obtained by GC–MS–MS analysis of a blood-based control pool containing strychnine.

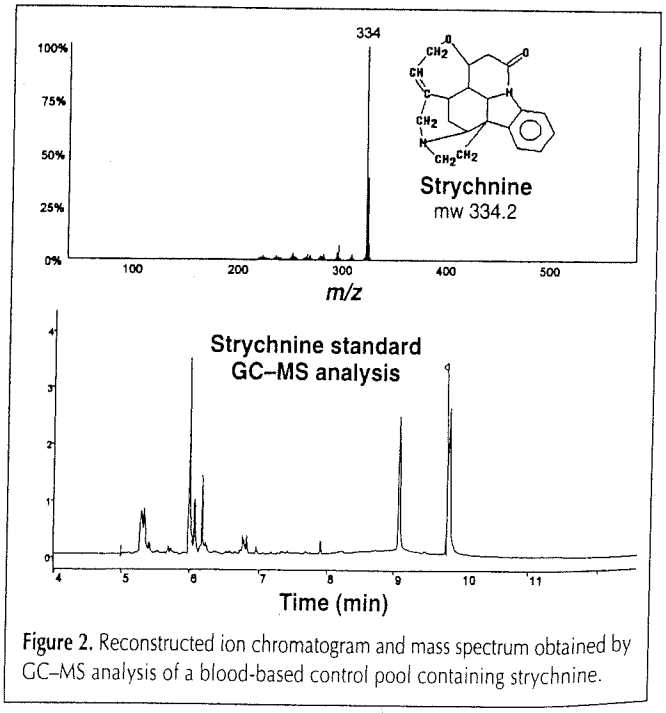

Figure 2. Reconstructed ion chromatogram and mass spectrum obtained by GC–MS analysis of a blood-based control pool containing strychnine.

(15 m × 0.25-mm i.d., 0.25-µm film thickness, J&W Scientific, Folsom, CA). Helium was used as the carrier gas with a flow rate of 8 mL/min. A sample injection volume of 1 µL was used in splitless mode. A column temperature program with 100°C for 1 min, a 100–300°C ramp (10°C/min) and a final hold at 300°C for 10 min was used. Injector port and detector temperatures were 250°C. Retention time and sensitivity for each analytical run was verified with injection of extracted calibrators containing amphetamine, methamphetamine, phencyclidine, dextromethorphan, amitriptyline, nortriptyline, maprotiline, clomipramine, flurazepam, alprazolam, thioridazine, trazodone, meperidine, normeperidine, diphenhydramine, methadone, cocaine, doxepin, nordoxepin, codeine, chlorpromazine, hydroxyzine, verapamil, norverapamil, pheniramine, lidocaine, chlorpheniramine, propoxyphene, imipramine, desipramine, pentazocine, diazepam, nordiazepam, loxapine, amoxapine, haloperidol, and strychnine at concentrations ranging from 0.1 to 0.3 mg/L. The retention time of the internal standard (methapyrilene) and strychnine was 8.5 and 16.4 min, respectively. Presumptive positive identification of drugs was based upon peak retention time within 2% of calibrator retention time and a signal-to-noise ratio of greater than 3. Presumptive positives and extracts with additional chromatographic peaks with a signal-to-noise ratio greater than 3 were subjected to GC–MS or GC–MS–MS analysis. All reagents and solvents were analytical grade. Methapyrilene was obtained from Alltech Applied Science Labs (State College, PA), and strychnine was purchased from Sigma (St. Louis, MO). All other calibrators were obtained from Alltech Applied Science Labs.

Strychnine quantitation by GC–NPD

Samples were extracted as described in the GC–NPD basic drug-screening procedure. Methapyrilene was used as an internal standard and calibration was performed by a standard addition method with an added strychnine concentration range up to 16 mg/L. The *x* intercept was determinated by regression analysis. Tissue homogenates and fluids with high concentration of strychnine were diluted with deionized water prior to extraction in order to accurately quantitate strychnine within the analytical range of the assay.

Strychnine confirmation by GC–MS–MS

Samples were extracted as described in the GC–NPD procedures. Analysis was performed with a Varian 3800 GC (Varian Chromatography Systems, Walnut Creek, CA) equipped with a Varian 1079 injector, Rtx-5MS capillary column (15 m × 0.25-mm i.d., 0.25-µm film thickness, Restek, Bellefonte, PA), and a Varian Saturn 2000 MS fitted with SilChrom ion trap, SilQuartz spacers, and wave board function. Helium was used as the carrier gas with a flow rate of 1 mL/min. A 1-µL sample was injection in splitless mode. A column temperature program (150–300°C, 20°C/min) was used with injector and ion-trap temperatures of 250°C and 200°C, respectively. For GC–MS–MS analysis, the ion preparation method involved initial electron ionization of strychnine with 10 µamps filament current and a maximum ionization time of 25 ms. Following ionization, the molecular ion (*m/z* 334) was isolated and fragmented by collision-induced dissociation under resonant waveform conditions with 0.60 V excitation amplitude. Full mass spectrum analysis was then performed. For additional confirmation of strychnine, the fragmentation pathway of strychnine was assessed by GC–MS–MS–MS analysis. GC–MS–MS analysis was modified for each fragment ion to allow assessment of first, second, and third generation product ions observed in the GC–MS–MS mass spectra. The GC–MS–MS–MS analysis was

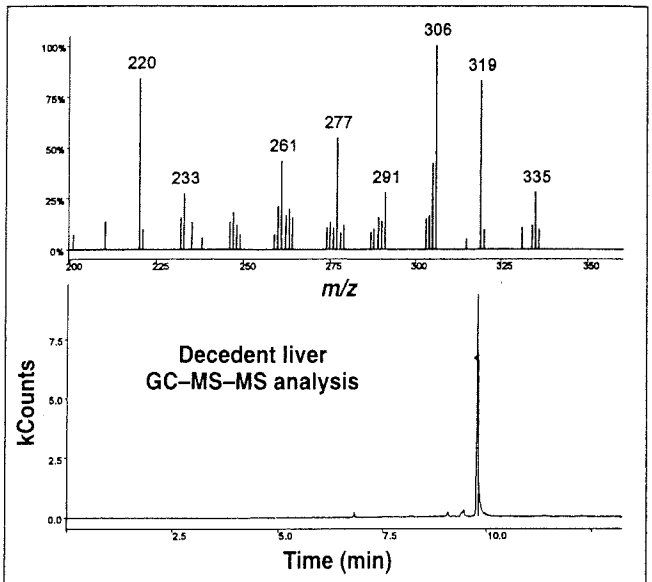

Figure 3. Reconstructed ion chromatogram and mass spectrum obtained by GC–MS–MS analysis of an extracted homogenate of liver from the decedent.

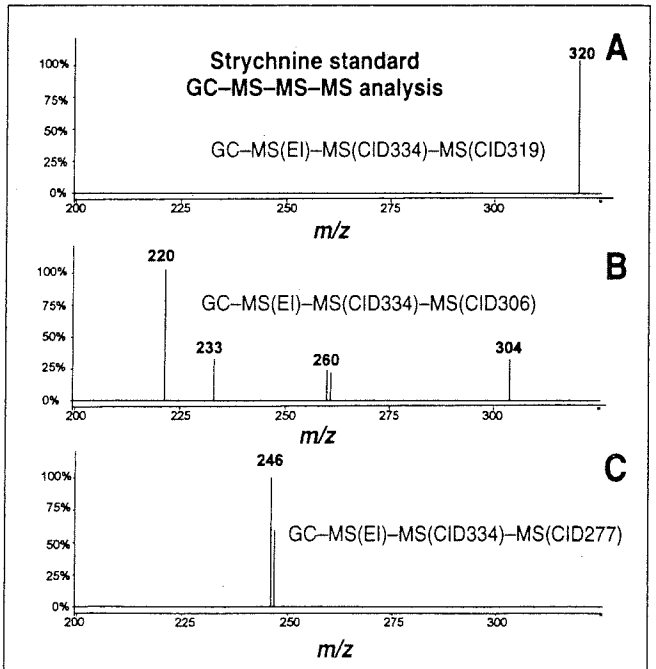

Figure 4. Mass spectra obtained from GC–MS–MS–MS analysis of the first-generation product ions generated by GC–MS–MS analysis of strychnine. In three separate studies, ion fragments with *m/z* 319 (A), *m/z* 306 (B), and *m/z* 277 (C) were isolated in the ion trap and then subjected to CID and mass analysis as described in the Methods section.

performed for product ions with *m/z* 319, 306, 277, 261, 246, 233, and 220. The ion preparation method employed 10 µamps filament current, a maximum ionization time of 25 ms, and a resonant waveform method with excitation amplitude optimized for each product ion. Full spectrum analysis of the product ions allowed a direct determination of fragmentation pathways. System control and data management was performed by Saturn GC–MS Workstation software version 5.2.

Other procedures

The Mandelin color test was performed on the organic extracts of blood, fluid, and tissue submitted in the case and also on particulate matter from the bottle and drink glass submitted in the case (37). Fluorescence polarization immunoassay (FPIA) screening for opiates, benzoylecgonine, benzodiazepine, amphetamines, cannabinoids, propoxyphene, tricyclic antidepressants, and phencyclidine was performed on postmortem blood using a methanolic pretreatment and modification of the Abbott Laboratories (North Chicago, IL) procedures (40). Urine was screened by FPIA using the manufacturer's procedures without modification. Screening for volatile alcohols was performed by GC–FID with headspace sampling and qualitative confirmation of 11-carboxy-tetrahydrocannabinol in urine was performed by GC–MS analysis based upon a positive threshold concentration of 25 ng/mL. GC–MS analysis of a blood-based control pool containing strychnine was also performed in order to compare conventional mass spectra with the tandem mass spectra.

Results and Discussion

The screening of the decedent's postmortem blood for opiates, benzolyecgonine, benzodiazepine, amphetamines, cannabinoids, propoxyphene, tricyclic antidepressants, and phencyclidine was negative. Volatile alcohols were not detected by GC–FID. Screening of the urine by FPIA methods was positive for cannabinoids and confirmed positive by a GC–MS procedure. Strychnine was detected in blood, tissue, and body fluids when analyzed by the GC–NPD screening procedure. Additional drugs were not detected in the chromatographic screening method. The Mandelin color reaction (37) was positive with organic extracts of tissue, blood, and fluids obtained from the decedent and also with particulate matter from the drink glass and bottle submitted in the case.

Confirmation of strychnine in postmortem blood, tissue, and fluids was performed by GC–MS–MS and GC–MS–MS–MS analysis. The tandem MS analyses revealed greater structural information than GC–MS. GC–MS–MS analysis, as shown in Figure 1, resulted in an efficient chromatography for strychnine (9.81 min) and a mass spectrum comprised of molecular ions (*m/z* 334), protonated molecular ions (*m/z* 335) and strychnine fragment ions with *m/z* 319, 306 (base peak), 277, 261, 246, 233, and 220. The tandem mass spectral analysis showed enhanced structural information when compared with GC–MS method reported previously (39). For direct comparison to GC–MS analysis, we reanalyzed the strychnine control pool under GC–MS conditions, and results are shown in Figure 2. The mass spectrum shows an abundance of molecular ion (*m/z* 334) with an absence of any significant qualifying ions. In contrast, GC–MS–MS analysis generates an abundance of fragment ions. The ratio of product ions to base peak ions was within 20% for replicate injections, indicating acceptable reproducibility of the mass spectra. A library spectrum file for strychnine by GC–MS–MS was established in-house and was used for strychnine identification in the forensic casework. Figure 3 shows a chromatogram and mass spectrum obtained by GC–MS–MS analysis of liver from the decedent. The analyses show a single prominent chromatographic peak with a mass spectrum and strychnine library fit of greater than 96%. GC–MS–MS confirmation of strychnine was also performed in blood, fluids, and other tissue from the decedent with similar results.

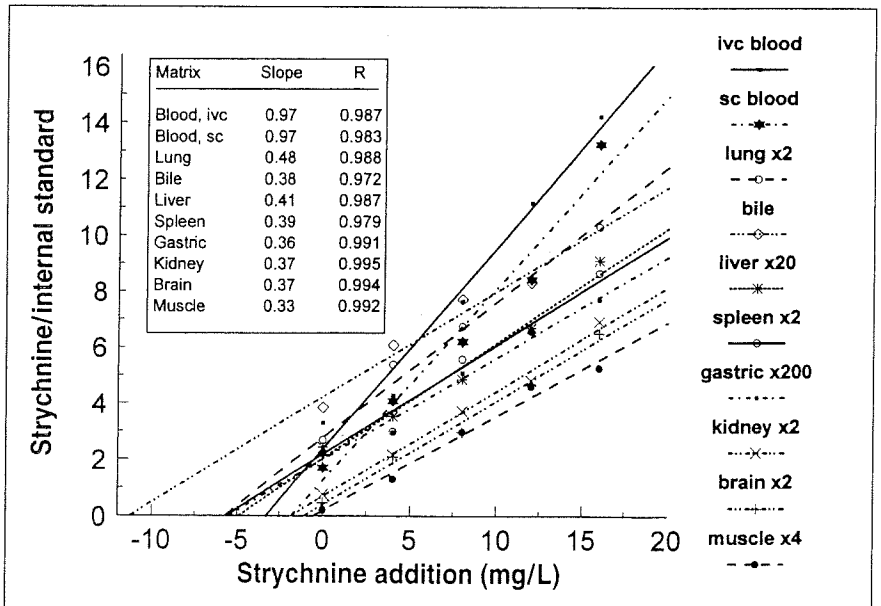

Figure 5. A representative standard addition study for quantitation of strychnine in blood, lung, bile, liver, spleen, gastric contents, kidney, brain, and skeletal muscle. The slope and correlation coefficient for the regression lines are shown in the insert for each fluid and tissue homogenate tested.

Further structural identification of strychnine in liver tissue from the decedent was revealed with fragmentation pathway analysis by tandem MS. Figure 4 shows mass spectra for GC–MS–MS–MS analysis of first-generation product ions observed in the GC–MS–MS spectrum for strychnine. After unit mass isolation and collision-induced dissociation of ionized strychnine, ion fragments with *m/z* 319 (Figure 4A), *m/z* 306 (Figure 4B), and *m/z* 277 (Figure 4C) were isolated in time within the ion trap and then subjected to collision induced dissociation and mass analysis. Analysis of both pure strychnine and liver extracts from the decedent showed that ions with *m/z* 261, 233, and 220 result from further fragmentation of *m/z* 306 ions, that *m/z* 246 ions are a product of ions with *m/z* 277, and

that *m/z* 319 ions do not undergo further fragmentation. Similar analysis of *m/z* 261 ions revealed fragmentation to *m/z* 233 ions, whereas ions with *m/z* of 246, 233, and 220 failed to show further fragmentation products. Based on these studies, the mass-to-charge ratios of first (319, 306, 277), second (261, 246, 220), and third (233) generation product ions were identified for collision-induced dissociation of pure strychnine as well as the strychnine extracted from the decedent's liver.

Strychnine concentrations in body fluids and tissue samples were determinated by GC–NPD analysis. Initial use of blood-based calibrators revealed matrix-dependent variability in the analytical recovery of strychnine from blood and tissue specimens. Quantitation of strychnine levels was therefore performed by a standard addition method. Figure 5 shows a representative standard addition study with a linear increase in the strychnine/internal standard ratio (peak area) in relation to amount of strychnine added. The slope and correlation coefficient for the regression lines are shown in the insert for each type of specimen tested. The difference in slope was greatest between blood and tissue specimens, consistent with the matrix-dependent differences in analytical recovery observed in the initial recovery studies.

Table I displays the distribution of strychnine in body fluids and tissue following the fatal poisoning. Consistent with prior reports, the highest tissue concentration was observed in liver. Parent drug was also present in bile at relatively high concentration, suggesting a direct route of parent drug elimination through the biliary canilicular system. A significant amount of strychnine was also measured in the gastric contents. Postmortem redistribution of strychnine has not been previously reported but may be due to the relatively high tissue content and a reported volume of distribution (13 L/kg) of strychnine (41). It was found that the strychnine level in postmortem blood obtained from the inferior vena cava was 1.8-fold higher than subclavian blood, suggesting that postmortem distribution does occur.

Table I. Strychnine Levels in Current Case Compared with Prior Reports*

Body fluid or tissue	Strychnine levels (mg/L or mg/kg)	
	Current case	Prior reports
Blood (subclavian)	1.82	0.4–61†
Blood (ivc)	3.32	
Muscle, skeletal	2.32	trace–0.5
Brain	2.42	0.47–66
Urine	3.35	0.5–33
Kidney	3.72	0.07–106
Bile	11.4	9.2
Spleen	11.8	not reported
Lung	12.3	10
Liver	98.6	0.27–257
Gastric contents	149	7.5–1000‡

* Collation of literature cited in reference 39.
† Site of collection not specified.
‡ Total content in milligrams.

Conclusions

Based on the circumstances of the case and the toxicological findings, the death was certified as suicide by strychnine ingestion. The case demonstrates that human strychnine poisoning still occurs despite restricted availability of the agent. The case also details current methods of analysis that allow specific identification and accurate quantitation of strychnine in a wide range of body fluids and tissues.

Acknowledgments

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