Cannabinoids in Postmortem Toxicology

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Abstract

Cannabinoids are often excluded from postmortem toxicology screens due to their ubiquitous nature, interpretative difficulties and unanswered questions regarding their postmortem redistribution. In this study, we review 30 postmortem cases where a drug screen gave a positive cannabinoids result and a confirmation identified ∆9-tetrahydrocannabinol (THC), 11-hydroxy-∆9-tetrahydrocannabinol (11-OH-THC), and/or 11-nor-9-carboxy-∆9-tetrahydrocannabinol (THC-COOH) in peripheral (BL-P) or cardiac/central blood (BL-C) and/or urine (UR). Had cannabinoids not been included in these toxicologic evaluations, incomplete or erroneous inferences would have been drawn in a substantial number of cases regarding cause/manner of death. THC was detected in 28 BL-C and in all 30 BL-P. THC and THC-COOH were confirmed present in 2 and 23 UR, respectively. 11-OH-THC was detected in 4 BL-C, 6 BL-P, and 0 UR. The mean THC concentrations in BL-C and BL-P were 8.0 and 15.8 ng/mL, respectively. The mean THC-COOH concentrations in BL-C and BL-P were 55.2 and 60.6 ng/mL, respectively. The mean 11-OH-THC concentrations in BL-C and BL-P were 17.0 and 12.5 ng/mL, respectively. Postmortem interval (PMI) for each case was determined and evaluated in relation to BL-C/BL-P concentration ratios with THC-COOH exhibiting a possible trend. This study is the first of its kind and demonstrates the usefulness of cannabinoid analyses as part of death investigations. Furthermore, it provides distribution data that will improve the ability of toxicologists and pathologists to evaluate cannabinoid concentrations in human postmortem specimens.

Introduction

Cannabis and marijuana are terms associated with the plant Cannabis sativa. The plant and its derivatives are cultivated and used all over the world for their ability to produce a plethora of effects including euphoria, sedation, and hallucinations (1). The most recent report on cannabis production by the United Nations Office on Drugs and Crime reported that, in 2009, cannabis herb production ranged from 13,300 to 66,100 metric tons, and the production of cannabis resin ranged from 2200 to 9900 metric tons (2). The large worldwide supply and demand of cannabis explains its almost ubiquitous presence in 2009 with over 16.5 million Americans over the age of 12 self-reporting that they have used the drug at least once in the month prior to being surveyed (3). Also in 2009, the Drug Abuse Warning Network estimated that cannabis caused 376,467 emergency department visits in the United States of America (4). But despite such pronounced frequency of exposure, cannabinoids and their analogues only ranked 23rd in a recent list of the top 25 substance categories associated with the largest number of fatalities published by the American Association of Poison Control Centers’ National Poison Data System (5). In the postmortem toxicology arena, the drug is not part of the routine screening protocol, but notable exceptions are death investigations of drivers as well as deaths thought to be accidental where physical evidence or eyewitness testimony suggest the possibility of cannabis’ involvement. Among the reasons for the scarcity of cannabis measurements in postmortem specimens remain the persistent attitude by the medical community that cannabis “doesn’t kill” and therefore should not be part of routine death investigations as well as the significant interpretative challenges and limited knowledge of the postmortem redistribution behavior exhibited by the various cannabinoids. Reports of potentially toxic effects of cannabis have appeared in the literature and describe increases in heart rate and changes in blood pressure (6–8), atrial fibrillation (9,10), acute coronary syndrome, vascular complications, and even congenital heart defects (11). A series of six case reports declared as acute cardiovascular fatalities following cannabis use has also been described in the literature (12). In an attempt to offer a first study of the postmortem concentrations of common cannabinoids in typical human autopsy specimens, to evaluate their distribution patterns, and to report their involvement in cause and manner of death and thus to contribute some postmortem cannabis data beyond what is already in the literature pertaining to cannabinoid presence in...
Materials and Methods

Autopsies normally take place within 24 h of the time of death, but that time frame may be significantly extended based on scheduling requirements and/or the need for a more extensive case investigation. Blood specimens collected at autopsy are normally refrigerated at 4°C overnight, and the Laboratory Division receives them on the morning of the business day following the autopsy for accessioning, analyses, storage and eventual disposal. Blood specimens are submitted in grey-top test tubes containing potassium oxalate and sodium fluoride, and urine specimens are submitted in red-top test tubes. Blood specimens submitted to the laboratory are normally labeled as either BL-P or BL-C by the collecting physician. BL-P specimens are received in small, 10-mL grey-top test tubes and allow for the assumption that blood contained therein does not include blood from the inferior vena cava or from the larger central area of the body. The laboratory, however, has no means of independently verifying that a proper specimen collection including ligation of the vessel has taken place at autopsy. Specimens are stored at 4°C from the time of accessioning through the time of analyses (a few days to several weeks later) and until the time of disposal (a year or more later). Specifically for cannabinoids, ELISA kits by Venture Labs (Redwood City, CA) are employed to screen BL-C and UR. The ELISA cutoffs used for BL and UR are 5 and 50 ng/mL, respectively. Following a positive ELISA result in BL-C and/or UR, confirmation and/or quantitation takes place in BL-P or a new aliquot of UR. Specimens are then shipped via overnight unrefrigerated transport to a contracted reference laboratory that confirms and quantifies cannabinoids by gas chromatography–mass spectrometry (GC–MS) with a limit of quantitation of 1 ng/mL for Δ9-tetrahydrocannabinol (THC) and 5 ng/mL for 11-hydroxy-Δ9-tetrahydrocannabinol (11-OH-THC) and 11-nor-9-carboxy-Δ9-tetrahydrocannabinol (THC-COOH) for both blood and urine. The deuterated compounds THC-d3 and THC-COOH-d3 are used as internal standards. For THC-d3, the target (underlined) and qualifier ions are 389 and 374. For THC-COOH-d3, the target (underlined) and two qualifier ions are 374, 476, and 491. For THC, the target (underlined) and qualifier ions are 386 and 371. For THC-COOH, the target (underlined) and two qualifier ions are 371, 473, and 488. Finally, for THC-OH, the target (underlined) and two qualifier ions are 371, 459, and 474.

For the purposes of the present study, the in-house computerized database was interrogated, and 30 postmortem cases in which the laboratory had reported cannabinoids in blood specimens were identified. Further review of the case histories and autopsy reports allowed for the collection of information pertaining to the reported date/time of death and the reported date/time of specimen collection at autopsy. Case files were manually reviewed, and all results related to cannabinoid analysis in BL-C, BL-P, and UR were tabulated using commercially available spreadsheet software. This review identified cases in which the initial ELISA drug screen had produced positive cannabinoid results in BL-C and confirmatory analyses had resulted in one or more cannabinoids identified in the case BL-P. In cases where confirmations/quantitations took place in the case BL-P per our laboratory’s analytical protocol, additional confirmatory/quantitative analyses took place subsequently in BL-C. In cases where UR was collected at autopsy, a similar analytical protocol was followed with UR screened by ELISA for cannabinoids (a few days later) and any positive results subjected to confirmatory analyses without quantitation (several days to several weeks later).

Results

The 30 cases in this study pertained to 28 male and 2 female decedents. The mean and median ages of our subjects were 38.2 and 35.0 years, respectively, and ranged from 19 to 65 years. Specimen collection at autopsy took place on average 2393 min (i.e., 1.7 days) after death occurred. Twelve of our 30 cases had a time interval of less than 24 h, 8 cases had a time interval between 24 and 48 h, 3 cases had a time interval between 48 and 72 h, and 7 cases had a time interval of greater than 72 h. Time intervals ranged between 374 min (i.e., 6 h) to 5055 min (i.e., 3.5 days) with a median of 1973 min (i.e., 1.3 days).

Table I presents the demographic data of the decedents included in this study, tabulates the analytical findings pertaining to THC, THC-COOH, and THC-OH in their BL-C, BL-P, and UR, lists the PMI for each case, and summarizes any other compounds detected in the decedents’ blood or urine specimens.

THC

Out of the 30 cases examined in this study, THC was identified in 28 BL-C and all 30 BL-P specimens. In the 26 of 30 cases that had sufficient UR volume for cannabinoid confirmations, THC was identified in 2 of them. The mean and median THC concentrations in BL-C were 8.0 and 4.2 ng/mL, respectively. The mean and median THC concentrations in BL-P were 16.1 and 11.5 ng/mL, respectively. For the 30 cases of the study, the BL-C: BL-P THC concentration ratios ranged from 0.00 to 2.60 (mean: 0.62; standard deviation: 0.61; median: 0.38).

THC-COOH

The inactive metabolite THC-COOH was identified in all 30 cases and in every available BL-C, BL-P, and UR specimen. The mean and median THC-COOH concentrations in BL-C were 57.0 and 37.5 ng/mL, respectively. The mean and median THC-COOH concentrations in BL-P were 61.2 and 32.0 ng/mL, respectively. For the 30 cases of the study that had this metabolite detected, the BL-C/BL-P concentration ratios ranged from 0.23 to 3.86 (mean: 1.07; standard deviation: 0.70; median: 0.89).
<table>
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<th>Case No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Specimens Submitted to the Lab</th>
<th>PMI (min)</th>
<th>THC</th>
<th>THC-COOH</th>
<th>THC-OH</th>
<th>Other Drugs Detected in Blood</th>
<th>Other Drugs Detected in Urine</th>
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<tr>
<td>1</td>
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<td>CIT metabolite (NIC, COT, CAF)</td>
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* PMI is defined as the time between date/time of death and date/time of specimen collection at autopsy. Incidental toxicology findings are in parentheses.

† Abbreviations: ND, none detected; VH, vitreous humor; CP, confirmed present; NO UR, urine specimen not available; PP-ISV, presumptive positive result by ELISA but with insufficient specimen volume for confirmation by GC–MS; COD, codeine; COT, cotinine; NIC, nicotine; CAF, caffeine; MOR, morphine; 6-MAM, 6-monoacetylmorphine; BZE, benzoylecgonine; MAMP, methamphetamine; AMP, amphetamine; DIAZ, diazepam; NDIAZ, nordiazepam; HYCOD, hydrocodone; DHC, dihydrocodeine; EDDP, 2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine; TRAZ, trazodone; DPH, diphenhydramine; and CIT, citalopram.
Table I (continued). Demographic Information, Specimen Types, Postmortem Interval (PMI), Cannabinoid Results in BL-C, BL-P, and UR, and Other Toxicology Findings for the 30 Cases Included in the Present Study*

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<th>Case No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Specimens Submitted to the Lab</th>
<th>PMI (min)</th>
<th>THC</th>
<th>THC-COOH</th>
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<td>MAMP, AMP, methadone, EDDP (COT)</td>
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* PMI is defined as the time between date/time of death and date/time of specimen collection at autopsy. Incidental toxicology findings are in parentheses.
† Abbreviations: ND, none detected; VH, vitreous humor; CP, confirmed present; NO UR, urine specimen not available; PP-ISV, presumptive positive result by ELISA but with insufficient specimen volume for confirmation by GC–MS; COD, codeine; COT, cotinine; NIC, nicotine; CAF, caffeine; MOR, morphine; 6-MAM, 6-monoacetylmorphine; BZE, benzoylecgonine; MAMP, methamphetamine; AMP, amphetamine; DIAZ, diazepam; NDIAZ, nordiazepam; HYCOD, hydrocodone; DHC, dihydrocodeine; EDDP, 2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine; TRAZ, trazodone; DPH, diphenhydramine; and CIT, citalopram.
11-OH-THC

11-OH-THC, the equipotent metabolite of THC, was found in four BL-C and six BL-P specimens. It was not found in any UR specimens. The mean and median 11-OH-THC concentrations in BL-C were 14.8 and 10.5 ng/mL, respectively. The mean and median 11-OH-THC concentrations in BL-P were 11.3 and 9.5 ng/mL, respectively. In those six cases with THC-OH, the BL-C/BL-P concentration ratios ranged from 0.00 to 1.64 (mean 0.90; standard deviation: 0.74; median: 1.17).

Discussion

Cannabinoids should be included in the routine toxicologic evaluation of all postmortem case investigations irrespective of suspected cause/manner of death. As Table I shows, had cannabinoids not been part of the toxicology protocol, 40% of the cases (12/30) would have been mistakenly evaluated assuming negative blood toxicology and more than half of the cases (15/27, 56%) would have been mistakenly evaluated assuming negative urine toxicology. Table II presents the cause and manner of death for each of the 30 cases comprising our study sample. In the 10 cases mannered as accidental deaths, cannabinoids were the only drugs detected in 30% of them, whereas urine analyses showed that cannabinoids were the only drugs detected in 67% of them. Among the seven cases mannered as suicides, cannabinoids were the only drugs detected in both blood and urine in 71% of them. Finally, in the three cases in our study sample that were eventually mannered as suicides, cannabinoids were never found alone in blood toxicology (0%) but were found alone in (33%) of these suicides based on urine toxicology. These results clearly suggest that in order to arrive to a more complete and accurate cause and manner of death, cannabinoids should be included in routine postmortem toxicology studies.

Although cannabinoid concentrations have been previously reported in human postmortem case reports (13–21) and their postmortem redistribution has recently been reported in animal models (22), to our knowledge, this is the first study examining and comparing the concentrations of cannabinoids in BL-C, BL-P, and UR specimens from human autopsies. From our findings, it is once again clear that psychoactive cannabinoids and metabolites are detectable in postmortem specimens, and in many cases, they are the only drugs found irrespective of manner of death (i.e., natural, accident, homicide, suicide). This finding strongly supports the notion that cannabinoids should be included in routine postmortem toxicology evaluations in order for such medicolegal death investigations to be as complete as possible.

Postmortem interval (PMI) is a parameter often outside the toxicologist's control that must be recognized as having the potential of being a significant factor adversely affecting measured drug concentrations (23). Unfortunately, PMI studies to date

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### Table I (continued). Demographic Information, Specimen Types, Postmortem Interval (PMI), Cannabinoid Results in BL-C, BL-P, and UR, and Other Toxicology Findings for the 30 Cases Included in the Present Study*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Specimens Submitted to the Lab</th>
<th>PMI (min)</th>
<th>THC in BL-C</th>
<th>THC-COOH in BL-C</th>
<th>THC-OH in BL-C</th>
<th>Other Drugs Detected in Blood</th>
<th>Other Drugs Detected in Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>35</td>
<td>m</td>
<td>BP-P, BL-C, UR, VH</td>
<td>3879</td>
<td>5.0</td>
<td>45.0</td>
<td>ND†</td>
<td>120</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>610</td>
<td>11.0</td>
</tr>
<tr>
<td>28</td>
<td>31</td>
<td>m</td>
<td>BP-P, BL-C, UR, VH</td>
<td>1270</td>
<td>26.0</td>
<td>21.0</td>
<td>ND†</td>
<td>420</td>
<td>330</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5100</td>
<td>30.0</td>
</tr>
<tr>
<td>29</td>
<td>65</td>
<td>m</td>
<td>BP-P, BL-C, UR, VH</td>
<td>374</td>
<td>7.0</td>
<td>21.0</td>
<td>ND†</td>
<td>35</td>
<td>110</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>58</td>
<td>17.0</td>
</tr>
<tr>
<td>30</td>
<td>22</td>
<td>m</td>
<td>BP-P, BL-C, UR, VH</td>
<td>955</td>
<td>27.0</td>
<td>24.0</td>
<td>ND†</td>
<td>110</td>
<td>78</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>530</td>
<td>8.2</td>
</tr>
</tbody>
</table>

* PMI is defined as the time between date/time of death and date/time of specimen collection at autopsy. Incidental toxicology findings are in parentheses.
† Abbreviations: ND, none detected; VH, vitreous humor; CP, confirmed present; NO UR, urine specimen not available; PP-ISV, presumptive positive result by ELISA but with insufficient specimen volume for confirmation by GC–MS; COD, codeine; COT, cotinine; NIC, nicotine; CAF, caffeine; MOR, morphine; 6-MAM, 6-monoacetylmorphine; BZE, benzoylecgonine; MAMP, methamphetamine; AMP, amphetamine; DIAZ, diazepam; NDIAZ, nordiazepam; HYCOD, hydrocodone; DHC, dihydrocodeine; EDDP, 2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine; TRAZ, trazodone; DPH, diphenhydramine; and CIT, citalopram.
have excluded cannabinoids and there remains a void in the literature in this area. In our study of 30 cases where PMI was evaluated, there appears to be a limited relationship between PMI and BL-C/BL-P when it comes to THC-COOH but no relationship could be defined for the other two cannabinoids we evaluated in this study (i.e., THC and THC-OH) as shown in Figure 1.

Similarly, postmortem redistribution (PMR), although a well-publicized phenomenon, is not well characterized or understood. For example, THC appears on first glance to be a good candidate for PMR based on its relatively large volume of distribution of 10 L/kg and high plasma protein bound fraction (24). However, the distribution pattern observed in our 30 cases is clearly not consistent with that expectation. This

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Cause of Death</th>
<th>Manner of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Gunshot wounds</td>
<td>Homicide</td>
</tr>
<tr>
<td>2</td>
<td>Probable acute morphine intoxication. Other conditions: cannabinoids present</td>
<td>Accident</td>
</tr>
<tr>
<td>3</td>
<td>Multiple blunt force injuries. Other conditions: ethanol and cannabinoids present</td>
<td>Homicide</td>
</tr>
<tr>
<td>4</td>
<td>Acute polysubstance intoxication (ethanol, heroin, and diazepam)</td>
<td>Accident</td>
</tr>
<tr>
<td>5</td>
<td>Complications of severe hypotension due to cirrhosis with severe macrosteatosis of liver due to chronic ethanol with chronic Hepatitis C virus infection. Other conditions: ethanol, diphenhydramine, and cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>6</td>
<td>Multiple blunt force injuries. Other conditions: cannabinoids and diazepam present</td>
<td>Suicide</td>
</tr>
<tr>
<td>7</td>
<td>Atherosclerotic cardiovascular disease. Other conditions: cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>8</td>
<td>Hanging. Other conditions: ethanol and cannabinoids present</td>
<td>Suicide</td>
</tr>
<tr>
<td>9</td>
<td>Atherosclerotic cardiovascular disease with acute cannabinoid intoxication. Other conditions: history of chronic ethanolism</td>
<td>Accident</td>
</tr>
<tr>
<td>10</td>
<td>Complications of laryngeal angioedema due to histaminic anaphylactic complications of multiple bee stings. Other conditions: elevated tryptase, cannabinoids present</td>
<td>Accident</td>
</tr>
<tr>
<td>11</td>
<td>Atherosclerotic cardiovascular disease. Other conditions: cardiomyopathy not otherwise specified, cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>12</td>
<td>Gunshot wound of the head and neck. Other conditions: cannabinoids present</td>
<td>Homicide</td>
</tr>
<tr>
<td>13</td>
<td>Blunt force injuries of head and neck. Other conditions: polysubstances present</td>
<td>Suicide</td>
</tr>
<tr>
<td>14</td>
<td>Blunt force injuries of chest and abdomen. Other conditions: cannabinoids and cocaine metabolite present</td>
<td>Accident</td>
</tr>
<tr>
<td>15</td>
<td>Acute mixed drug intoxication (heroin and cocaine). Other conditions: cannabinoids present</td>
<td>Accident</td>
</tr>
<tr>
<td>16</td>
<td>Acute ecstasy intoxication. Other conditions: cannabinoids present</td>
<td>Accident</td>
</tr>
<tr>
<td>17</td>
<td>Complications of atherosclerotic cardiovascular disease. Other conditions: cirrhosis, cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>18</td>
<td>Gunshot wounds</td>
<td>Homicide</td>
</tr>
<tr>
<td>19</td>
<td>Gram-positive bacterial myocardial abscess due to complications of acute bacterial endocarditis due to complications of chronic injection drug abuse. Other conditions: polysubstances present</td>
<td>Natural</td>
</tr>
<tr>
<td>20</td>
<td>Gunshot wounds</td>
<td>Homicide</td>
</tr>
<tr>
<td>21</td>
<td>Hypertensive atherosclerotic cardiovascular disease. Other conditions: cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>22</td>
<td>Multiple blunt traumatic injuries</td>
<td>Homicide</td>
</tr>
<tr>
<td>23</td>
<td>Blunt force injuries of the neck, chest, and abdomen. Other conditions: cannabinoids present</td>
<td>Homicide</td>
</tr>
<tr>
<td>24</td>
<td>Blunt force injuries of the neck, chest, and abdomen. Other conditions: ethanol and cannabinoids present</td>
<td>Homicide</td>
</tr>
<tr>
<td>25</td>
<td>Multiple gunshot wounds</td>
<td>Homicide</td>
</tr>
<tr>
<td>26</td>
<td>Complications of gunshot wound of chest. Other conditions: polysubstances present</td>
<td>Homicide</td>
</tr>
<tr>
<td>27</td>
<td>Acute tetrahydrocannabinol intoxication with hypertensive heart disease. Other conditions: polypharmacy present, atherosclerotic cardiovascular disease</td>
<td>Accident</td>
</tr>
<tr>
<td>28</td>
<td>Asphyxia due to aspirated gastric contents including plastic fentanyl transdermal patch due to acute polysubstance intoxication (fentanyl and cannabinoids)</td>
<td>Accident</td>
</tr>
<tr>
<td>29</td>
<td>Probable hypertensive heart disease. Other conditions: cannabinoids present</td>
<td>Natural</td>
</tr>
<tr>
<td>30</td>
<td>Blunt force injuries of chest. Other conditions: cannabinoids present</td>
<td>Accident</td>
</tr>
</tbody>
</table>
apparent discrepancy could be due to further PMI dependence not characterized by our limited study sample, sampling differences at the time of autopsy, subtle delays in analytical time, or the relatively small number of cases reviewed in this study. However, one must also consider that the discrepancy may be due to the fact that THC quickly and broadly disperses and distributes away from the blood vessels within the human body because of its significantly high lipophilicity. Caution should, therefore, be exercised when attempting to understand and interpret the concentrations of the two common psychoactive cannabinoids, THC and 11-OH-THC, as the site of the blood specimen collection appears to have significant effects on their concentrations. The non-psychoactive metabolite THC-COOH appears, in this study, to be relative unaffected by site-specific variations in concentration but may be affected by PMI. However, one should always keep in mind that neither BL-C nor BL-P is as good as antemortem hospital blood specimens in giving a better impression of actual drug concentrations in the peri-mortem time interval and that both BL-P and BL-C drug concentrations may have already changed as a result of post-mortem redistribution that occurred well before they were ever sampled during autopsy.

Conclusions

It is of paramount importance that forensic and anatomic pathologists and autopsy technicians assist in addressing the interpretative needs of their cases, by performing proper specimen collections, including blood vessel ligation, and clearly identifying each specimen’s collection site. Forensic and analytical toxicologists should perform analyses in a timely fashion and offer interpretations and opinions only after realizing that postmortem interval and postmortem redistribution are factors that can and do have the potential to significantly alter drug concentrations for most drugs and poisons just as the present study demonstrates for THC and 11-OH-THC, two commonly encountered psychoactive cannabinoids. It is also important that cannabinoids are routinely included in postmortem toxicologic case evaluations as excluding them could lead forensic pathologists, coroners, and forensic and analytical toxicologists to incomplete case investigations and ultimately to erroneous and incomplete cause and manner of death determinations in all types of postmortem cases. Case reports and further studies similar to this one are needed (including investigative, anatomic and analytical findings) so that the necessary body of literature develops that will permit more accurate determinations of the role of cannabinoids in death investigations.

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References


