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Case Report

Alimemazine poisoning as evidence of Munchausen syndrome by proxy: A pediatric case report

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ABSTRACT

Munchausen syndrome by proxy (MSBP), also known as fabricated or induced illness in a child by a caretaker, is a form of abuse where a caregiver deliberately produces or feigns illness in a person under his or her care, so that the proxy will receive medical care that gratifies the caregiver. The affected children are often hospitalized for long periods and endure repetitive, painful and expensive diagnostic attempts. We present an analytically confirmed case of MSBP by alimemazine. A 3-year-old boy was brought repetitively to a Pediatric Emergency Department by his mother because he presented limb tremors, dysarthria, obnubilation, and ataxia and generalized tonic–clonic seizures coinciding with intermittent fever. Neither the rest of the physical examination nor the complementary tests showed any significant alterations. MSBP was suspected and a routine systematic toxicological analysis in urine and blood was requested. Alimemazine was detected in all biological samples. The administration of this drug was never mentioned by the mother and the subsequent interview with her corroborated the suspicion of MSBP. Clinically, after separation from the mother, the child's neurological symptoms gradually improved until the complete disappearance of the cerebellar symptoms. Alimemazine was quantified in serum, urine, gastric content and cerebrospinal fluid samples by gas chromatography–mass spectrometry (maximum serum level was 0.42 μg/ml). Hair quantification of alimemazine was performed by ultra-performance liquid chromatography–tandem mass spectrometry in different segments of hair. The results confirmed regular substance use during the at least eight last months (8.8, 14.7, 19.7 and 4.6 ng/mg hair starting from most proximal segment). This patient represents the first case published with analytical data of alimemazine in blood, urine, gastric content, cerebrospinal fluid and hair, which allowed us to prove an acute and repetitive poisoning with alimemazine as evidence of MSBP.

1. Introduction

Munchausen syndrome by proxy (MSBP), also known as fabricated or induced illness in a child by a caretaker [1,2], is a form of abuse where a caregiver deliberately produces or feigns illness in a person under his or her care, frequently a child, so that the proxy will receive medical care that gratifies the caregiver. The most common methods of inflicting harm are poisoning and suffocation. Neurological manifestations are common with this type of maltreatment. Overall mortality is 6% to 9%. The diagnosis of MSBP in a child can be difficult [1,2]. Blood and urine are the conventional specimens to document drug exposure. However, in cases of drug-facilitated crime or MSBP, hair sampling is a necessary complement to these analyses as segmentation of the hair permits the differentiation of a single exposure from chronic

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or repeated administration of a drug. Alimemazine or trimeprazine (N,N,2-trimethyl-3-phenothiazin-10-ylpropan-1-amine) is a phenothiazine derivative related to chlorpromazine; it has the properties of antihistamines. It has been available on the market in Spain since 1964 under the name of Variargil (Logogen Laboratories) in oral drops. The systemic antihistamine alimemazine is licensed in Spain for use as a symptomatic treatment of allergic manifestations such as seasonal or perennial allergic rhinitis, allergic conjunctivitis, angioedema and mild urticarial [3]. However, off-label use of alimemazine for a long time has been relatively frequently prescribed for the treatment of infant sleep disturbance [4]. Alimemazine is not licensed or recommended for use in children less than 2 years old because of indications of highly variable pharmacokinetics [5], heavy hangovers as a commonly reported side effect [6], and a few reports of serious or fatal adverse reactions such as malignant hyperpyrexia [7], neuroleptic malignant syndrome [8], respiratory depression [9], convulsions [10] and sudden infant death [11].

Here, we report a case of MSBP by poisoning a child with alimemazine.

2. Case report

A 3-year-old boy was brought to a Pediatric Emergency Department (ED) by his mother with various neurological symptoms (limb tremors, dysarthria, ataxia and generalized tonic–clonic seizures, GTCS). His mother described fluctuating and self-limiting symptoms over two weeks, coinciding with intermittent fever attributed to acute respiratory infectious process. Blood tests for neurotropic virus serology, cerebral computerized tomography (CT), electroencephalogram (EEG) and cerebral magnetic resonance imaging (MRI) were performed without objectifying any abnormalities. He was discharged after three days of remaining asymptomatic.

Three months later, he returned to our hospital because he had had about five episodes of GTCS over a week coexisting with fever. During the last 24 h, he also presented dysarthria, ataxia and limb loss. Complementary tests showed a mild elevation of liver enzymes, qualitative screening of benzodiazepines and opiates in urine were negative, cerebrospinal fluid (CSF) analysis was normal, cerebral CT scan showed an occipital fracture secondary to fall to the ground in a seizure, without intracranial lesions and EEG showed an overall slowing of brain activity.

With the clinical suspicion of viral encephalitis, the child was admitted with intravenous treatment with acyclovir and valproate acid (bolus of 20 mg/kg and subsequent infusion to 0.5 mg/kg/day) for seizures. Cerebellar symptoms initially remained, with a fluctuating course but without any fever or seizures. The following additional tests were performed: cerebral MRI, anti-neuronal antibodies, complete metabolic study, genetic study of febrile seizures plus and neurotropic virus serology. All of them were negative.

On the seventh day, coinciding with the withdrawal of acyclovir, the fever and seizures reappeared with an episode of stupor that required admission to the Pediatric Intensive Care Unit (PICU). A new cerebral CT and lumbar puncture was made but showed no signs of pathology.

The patient's symptoms gradually improved during the following week, without any new seizures and progressive improvement of ataxia, dysarthria and limb loss. Immediately after discharge, fever and repeated convulsions reappeared. Despite this, the mother did not return to the hospital and only three days after she telephoned the medical team to explain what had happened and accepted to return with the child. On arrival, the child was obnubilated and presented severe ataxia with failed to maintain seated. Given the bizarre, fluctuating and inconsistent clinical picture, the possibility of poisoning caused by the mother was suspected. At home, the mother explained that she only treated the boy with acetaminophen to try to relief the symptoms, and also with the therapeutically prescribed valproate. She denied that the boy could have taken any other medications, and stated that the only drugs that were discovered at home were: acetaminophen, diclofenac, tramadol, diazepam, alprazolam, gabapentin, baclofen, enalapril, simvastatin and hydrochlorothiazide. In the Pediatric ED, a routine systematic toxicological analysis (STA) was requested and the patient was transferred to the PICU for a close clinical monitoring.

STA indicated the presence of alimemazine, acetaminophen, chlorpheniramine and diphenhydramine. No other drugs that were discovered at child's home were detected using GC-MS library spectra of the National Institute of Standards and Technology (NIST Mass Spectral Library Revision January 2010).

After the positivity of alimemazine in urine, a substance that the mother had not mentioned as having at home, a gastric content aspiration was performed with the diagnostic purpose of recent ingestion. The interview with her showed contradictory statements that made us suspect MSBP. She recognized that two years ago his pediatrician had prescribed alimemazine to the child for three days to regulate sleep cycles. It was this finding that gave rise to the suspicion of a chronic intoxication, and so it was ordered a hair analysis to check if it had been produced. At the time the hair sample was taken, the mother completed a volunteer consent form.

We were able to recover CSF and serum samples collected 14 and 4 days before, respectively. Besides this, for the actual episode she stated that she had administered a dose of syrup with phenylephrine, diphenhydramine and chlorpheniramine. The case was reported to the Social Services and the Office of Children, which dictated a restraining order with regards to the child's mother, and the child was placed under the protection of the maternal aunt.

Clinically, after separation from the mother, the child's neurological symptoms improved progressively until the complete disappearance of the cerebellar symptoms, with no new seizures, allowing valproic acid withdrawal. The boy could be discharged eleven days later.

The highest serum concentration was reported during the first day after last admission into the PICU. At that time, the child was stable hemodynamic and respiratory unsupported. Neurologically he was stupor, with a Glasgow coma scale of 11–12, with severe ataxia and dysarthria, negative meningeal signs, mobilizing extremities spontaneously. Examination of the cranial nerves was not possible. Reactive pupils, sensitivity preserved and tendon reflexes not found at the time of the examination. The dose of valproic acid at admission was 35 mg/kg/day.

3. Material and methods

In our institution, STA included serum ethanol, that was undetectable (<0.10 g/l) by an enzymatic method (Abbott Diagnostics), a urine drug screening by immunoassay (DRI®, Abbott Diagnostics-Santa Clara, CA, USA) for cannabinoids, cocaine metabolite, opiates, benzodiazepines, ecstasy and amphetamines, that was also negative (below test cut-offs of 25 ng/ml for cannabinoids, 200 ng/ml for ecstasy, 500 ng/ml for amphetamines and 100 ng/ml for cocaine metabolite, opiates and benzodiazepines), followed a liquid–liquid extraction and derivatization for neutral and alkaline drug screen in urine by gas chromatography coupled to mass spectrometry (GC-MS) (Agilent 5975/6890®, Santa Clara, CA, USA). This screening indicated the presence of alimemazine, acetaminophen, chlorpheniramine and diphenhydramine.

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Serum levels of acetaminophen and valproate were quantified by spectrophotometric and quimioluminiscence immunoassay methods, respectively (Abbott Diagnostics Santa Clara, CA, USA).

Alimemazine was quantified in serum, urine, gastric content and CSF samples by GC–MS. The GC was equipped with a fused-silica HP-5MS capillary column (30 m × 0.25 mm × 25 μm) from Agilent. The internal standard was Proadifen hydrochloride (SKF-525A, Sigma-Aldrich, St. Louis, USA). Calibration standards containing different ng amounts (limit of quantification 0.1 μg/ml for serum, 0.5 μg/ml for urine, 0.1 μg/ml for CSF and 1 μg/ml for gastric content) of analyte under investigation were prepared for each analytical batch by adding suitable amounts of standard stock solutions to 1 ml of pre-checked drug-free biological fluids. All samples (1 ml) were mixed with a phosphate buffer (100 mM, pH 6.0, 2 ml). The mixture was then applied onto a Bond Elut Certify solid-phase extraction (SPE) column (Agilent Technologies). Alimemazine was eluted from the column with a mix of methylene chloride/isopropyl alcohol/NH_4OH (78/20/2). The eluent was evaporated to dryness at 40 °C. The residue was dissolved in methanol and an aliquot of 1 μl was injected into the GC–MS system. GC–MS was operated in the selected ion monitoring (SIM) mode. The monitored ions used for quantitation were *m/z* 58, 298 and 198.

Concentrations are presented in Table 1.

Ultra-performance liquid chromatography–tandem mass spectrometric assays were used to identify and quantify the alimemazine in different segments of hair to confirm regular substance use. Hair strand was twice decontaminated using methylene chloride and then segmented (segments of 2 cm). Each segment was cut into small pieces (<1 mm). Calibration standards containing 1, 2, 5, 10, 20, 50 ng/mg hair were prepared daily for each analytical batch by adding nist amounts of methanol working solutions to 20 mg of pre-checked drug-free hair pool. Approximately 20 mg of each piece was incubated in 1 ml of phosphate buffer at pH 8.4 at 40 °C overnight. After a liquid–liquid extraction with organic solvent and evaporation to dryness of organic phase, the residue was reconstituted with ammonium formate 5 mM pH 3 and acetonitrile. A sample volume of 1 μl was injected into the liquid chromatograph.

Concentrations are presented in Table 2.

4. Method validation

Prior to application to real samples, both GC–MS and UPLC–MS were tested in a validation protocol following the accepted criteria for bioanalytical method validation. Selectivity, matrix effect, recovery, linearity, limit of detection (LOD) and quantification (LOQ), precision, accuracy and stability (freeze/thaw cycles and four-month mid-term stability) were assayed as we previously reported [12]. Linear calibration curves were obtained with a determination coefficient (r^2) higher than 0.99 for the different drugs. The analytical recoveries obtained at three quality control (QC) concentrations were always higher than 90% for analyte. Limits of detection and quantification were always below the first point of the calibration curves in all biological matrices and satisfactorily met the internationally established acceptance criteria [13]. Intra-assay and inter-assay precision and accuracy were always better than 15%. No relevant degradation was observed in QC samples after any of the three freeze/thaw cycles, with differences in the initial concentration of less than 10%. Similar results (differences to the initial concentration always lower than 10%) were obtained in the case of the mid-term stability test (QC samples analyzed once a month during a four-month period), assuring the feasibility of stored sample analysis.

Table 2
Alimemazine concentrations after segmental analyses of the hair.

Hair segment	Alimemazine (ng/mg)
0–2 cm (proximal segment corresponding to the last two months before the cut)	8.8
2.1–4 cm (central segment corresponding to a time period of 3–4 months before cutting)	14.7
4.1–6 cm (central segment corresponding to a time period of 5–6 months before cutting)	19.1
6.1–8 cm (central segment corresponding to a time period of 7–8 months before cutting)	4.6

5. Discussion

We report here a case of acute alimemazine toxicity and chronic administration of alimemazine, demonstrated by the presence of the drug in several biological samples in the context of a MSBP. According to the existing literature, blood level of alimemazine in children after recommended preanesthetic oral dose of 3 mg/kg is between 0.08 and 0.15 μg/ml [5], while adult therapeutic level is between 0.05 and 0.4 μg/ml [14]. In the current case, the blood levels of alimemazine are clearly above the pediatric reported range in all samples except one. Alimemazine is a poorly characterized drug and there is sparse information of the relation between dose and concentration in plasma in the living individuals, especially with regard to multiple dosing. In our case, the child showed many of the serious adverse effects and intoxication symptoms described. In the adult population, Druid et al. [15] compared several cases of alimemazine whole blood concentrations from suspected drugged drivers with other cases with fatal outcome. The average concentration in drivers and in post mortem associated with assumed therapeutic use was of 0.1 μg/ml, while in lethal intoxications involving alimemazine intake alone or in combination with other substances was of 1.6 and 0.9 μg/ml, respectively.

The highest reported lethal value was a 58-year-old male who intentionally ingested an unknown dose of alimemazine achieved a blood concentration of 6.52 μg/ml of alimemazine [16].

However, when comparing plasma concentrations from living patients with results from analysis done in postmortem blood, it

Table 1
Alimemazine concentrations in acute episode.

Specimen	Day; Time[a]				
	−14; 10:55	−4; 9:35	1; 12:24	1; 17:55	2; 10:05
Serum (μg/ml)	NA	0.12	0.29	0.42	0.17
Urine (μg/mg creatinine)	NA	NA	6.66	3.06	2.43
Gastric content (μg/ml)	NA	NA	794.82	NA	NA
Cerebrospinal fluid (μg/ml)	0.14	NA	NA	NA	NA

[a] Day 1: days after last PICU admission; NA: not available.

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should be considered that alimemazine is probably a drug with a large volume of distribution [17] and hence possibility for large redistribution post mortem.

Alimemazine was detected in hair in a range of 4.6 to 19.1 ng/mg. These concentrations are very high compared with the only published case of two children sedated with alimemazine, with levels of drug between 0.023 and 0.339 ng/mg [18]. The significance of these concentrations could not be determined due to a lack of more data in the literature. However, we must bear in mind that in our patient the clinical situation was very severe, with ataxia, dysarthria, dysmetry and recurrent episodes of seizures and stupor, while levels previously reported correspond to sedation status, without loss of consciousness or other clinical symptoms. Given the length of the boy's hair, exposure to alimemazine should have occurred at least during the previous 8 months [19], but it was not possible to put any quantitative interpretation on the dosage that was administered to the child. It is however, obvious that repetitive administrations have occurred but it is not possible to determine the number of exposures. All the symptoms of impairment disappeared after the child was taken away from his mother.

Systemic antihistamines are prescribed to many children; however adverse drug reactions are more common than suggested by pharmaceutical company studies [20]. According to the study performed by Finkelstein et al. the second, most common drug class leading to pediatric seizures was over-the-counter anticholinergics/antihistamines [21].

Cases of MSBP are so varied and bizarre that often diagnosis is very difficult to make. As with any condition, the first step in making the diagnosis of MSBP is to consider the possibility of MSBP in the context of a differential diagnosis. A wide variety of neurological manifestations such as weakness, ataxia and/or seizures, have been reported in cases of medical child abuse. The majority of medications used in intentional poisoning have been prescriptive medications with the most common being anticonvulsants and psychoactive neurological medications [22].

The length of time from onset of neurologic abnormalities to diagnosis was 5 months in this case. At our university hospital, there is an intensified interdisciplinary co-operation with clinical toxicology cases, where the Paediatrics Department is included. This circumstance could have been helpful in reaching a diagnosis in a under-average length of time [23].

Routine toxicology in urine and blood can target common drugs of abuse and high levels of a drug in the body, but this does not include all drugs or poisons. The major practical advantage of hair testing compared with urine or blood testing for drugs is that it has a larger surveillance window. For practical purposes, the two tests complement each other [24]. Urinalysis and blood analysis provide short-term information of an individual's drug exposure, whereas long-term histories are accessible through hair analysis. In addition, hair analysis may be especially useful when a history of drug use is difficult or impossible to obtain, as it obviously is in cases when a child or a baby has been poisoned [25]. The discrimination between a single exposure and long term use can be documented by multi-sectional analysis [19].

6. Conclusions

This patient represents the first case of MSBP with analytical confirmation of an acute and repetitive poisoning with alimemazine in a clinical setting. Acute and chronic administration of alimemazine has been demonstrated by the detection and quantification in serum, urine, cerebrospinal fluid and gastric content, and hair analysis, respectively. The detection of alimemazine confirmed the clinical suspicion of MSBP and allowed us to explain the child's complex symptoms.

The diagnosis and subsequent prosecution of MSBP cases requires the collaborative teamwork of health care teams, laboratory personnel, law enforcement, and social services, as illustrated in this case.

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Conflict of interest statement

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

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