Available online at www.sciencedirect.com
SCIENCE DIRECT®
Forensic Science International 146S (2004) S79–S81
Forensic Science International
www.elsevier.com/locate/forsciint

Case report

Aldicarb poisoning: one case report

Paula Proença[a,*], Helena Teixeira[a,b], M.C. de Mendonça[a], Fernando Castanheira[a], Estela P. Marques[a,b], Francisco Corte-Real[a,b], Duarte Nuno Vieira[a,b]

[a]*Delegation of Coimbra, National Institute of Legal Medicine, Coimbra, Portugal*
[b]*Faculty of Medicine, University of Coimbra, Coimbra, Portugal*

Available online 18 October 2004

Abstract

The carbamate aldicarb (*Temik*®) is a highly toxic (LD50 = 0.9 mg/kg oral in rats) insecticide often used in agriculture.

The authors present a fatal intoxication case with aldicarb, in a 24-year-old male, under police custody in the island of S. Tome and Prince (STP), in Africa. He was found 3 h after his arrest, lying on the floor of his cell, unconscious, frothing at the mouth, and entered into the hospital already dead. Aldicarb analysis was carried out by high-performance liquid chromatography, with a post-column derivatization system (with *o*-phthaldialdehyde and 2-mercaptoethanol, under alkaline conditions at 80 °C) with fluorescence detection at λ_{ex} = 339 nm and λ_{em} = 445 nm.

The toxicological analysis revealed toxic concentrations of aldicarb in the post-mortem samples: blood (6.2 μg/ml), stomach (48.9 μg/g), liver (0.80 μg/g), kidney (8.10 μg/g), heart (6.70 μg/g) and urine (17.50 μg/ml). It was also detected to be the same substance in a grey granulate powder supplied by criminal authorities of that country, as a probable substance ingested by the victim.

The toxicological analysis results, the autopsy findings and the information given, allowed us to conclude that death occurred due to an aldicarb acute intoxication. Although the case suggested a suicide, particularly based on the known information and on the high aldicarb concentration found in the stomach, it was not possible to indicate precisely its etiology (suicide or homicide).

Keywords: Aldicarb; Fatal intoxication; HPLC

1. Introduction

Aldicarb (propanal, 2-methyl-2-(methylthio)-*O*-[(methyl-amino) carbonyl] oxime) is a systemic carbamate used in agriculture as insecticide and nematicide, is hazardous by dermal, oral or subcutaneous exposure routes. Aldicarb is known to be highly toxic (LD50 = 0.9 mg/kg orally for rat) [1], therefore is commercialised as a dust-free, granulated material (*Temik*®) [2].

Carbamates are potent cholinesterase inhibitors as organophosphates, although they are of shorter duration, with a reversible toxic action [3,4].

A few cases of aldicarb poisoning have been described, eight nonlethal [5–12] and one reporting a fatal intoxication case [13].

The authors present the first fatal case of aldicarb poisoning detected in the Forensic Toxicology Laboratory of Coimbra's Delegation of the National Institute of Legal Medicine (NILM).

Aldicarb was determined by liquid chromatography with post-column derivatization reaction and fluorescence detection [14].

2. Case report

A 24-year-old male, under police custody in the island of S. Tome and Prince (STP), in Africa, was found 3 h after his

* Corresponding author. Tel.: +351 2398 542 30; fax: +351 2398 205 49.
E-mail address: paulaproenca@dcinml.mj.pt (P. Proença).

0379-0738/$ – see front matter
doi:10.1016/j.forsciint.2004.09.028

arrest, lying on the floor of his cell, unconscious, frothing at the mouth, and was therefore submitted to the hospital where he arrived already as a cadaver. Autopsy was performed 3 days after death, by local experts, and one second autopsy which was carried out by a forensic expert from the NILM, eight days after death, due to a request by the Government of the Republic of STP. At autopsy, the external examination revealed marked cyanosis and absence of traumatic injuries. The internal examination revealed a small amount of grey pap with a sandy aspect and a suggestive smell, dark fluid blood and generalized visceral congestion. No signs of violence were observed. A grey granulate was supplied by the criminal authorities of STP, locally called as ''rats poison''. Being of easy acquisition, it could have been the eventual toxic taken by the victim.

Post-mortem samples submitted for toxicological analysis were blood, stomach, liver, kidney, heart and urine. A granulate powder was also submitted to analysis.

3. Materials and methods

3.1. Chemicals

Acetonitrile and methanol of HPLC-grade, concentrated hydrochloric acid and dichloromethane LiChrosolv® were obtained from Merck (Darmstadt, Germany). Water was purified by a Milli-Q system obtained from Millipore (Molsheim, France), ortho-phthaldialdehyde (OPA), 2-mercaptoethanol (MCE), sodium hydroxide and sodium sulphate anhydrous were supplied by Merck (Darmstadt, Germany). Sodium tetraborate decahydrate of analysis-grade and the aldicarb standard were obtained from Riedel-de Haën. Stock solution (1 mg/ml) was prepared in acetonitrile and stored at 4 °C.

Standard working solutions were prepared from stock solution at concentrations of 5–50 μg/ml diluted with water, which had been acidified to pH 3.0 with concentrated HCl.

The mobile phase was filtered with a 0.20 μm Schleicher & Schuell filter and degassed in an ultrasonic bath for 15 min just before use.

3.2. Instrumentation

The chromatographic system used was a Waters Carbamate Analysis System including a Model 600E Multisolvent Dellivery System Controller, Fluid Handling Unit with integral post-column reaction system at 80 °C and a 7125 Rheodyne injector with a 10 μl loop. The mobile phase was a nonlinear gradient of water/methanol/acetonitrile, with a flow rate of 1.5 ml/min. The separation was performed on a Waters carbamate analysis column, C_{18} (3.9 × 150 mm, 4 μm) at 25 °C. Scanning Fluorescence, model 470 (Waters), λ_{ex} = 339 nm and λ_{em} = 445 nm was used. Millennium Software version S/S 2010 ADD (Waters) was used to control the system, acquisition and data processing, by an interface SAT/IN.

3.3. Sample preparation

Control and calibration samples were prepared by spiking drug-free post-mortem blood samples with standard solutions.

Post-mortem samples were extracted three times with dichloromethane and filtered by sodium sulphate anhydrous. Supernatants were evaporated to dryness under a slow stream of nitrogen, at 40 °C. The dried extracts were reconstituted with 250 μl water (pH 3) and an aliquot (10 μl) was injected into the HPLC system.

4. Results and discussion

Calibration curve of aldicarb in blood samples was linear over a concentration range of 0–50 μg/ml, with a correlation coefficient of 0.9998. The detection limit (LOD) of aldicarb was 1 ng/ml.

The toxicological analysis of the post-mortem samples revealed toxic concentrations of aldicarb: blood (6.2 μg/ml), stomach (48.9 μg/g), liver (0.80 μg/g), kidney (8.10 μg/g), heart (6.70 μg/g) and urine (17.50 μg/ml). Aldicarb was also detected in the granulate powder. Sam Hoai Ngo [13] reported a fatal case with this carbamate with a post-mortem blood concentration of 11 μg/ml. Aldicarb concentrations obtained in our fatal case may reflect a phenomenon of post-mortem redistribution, reported by several authors [15–18], due to the range of time between death and autopsy sampling.

The forensic pathologist noticed that 8 days after death, the corpse was in excellent condition, without any sign of putrefaction although he has been submitted to a previous autopsy. It is also to emphasize that the weather conditions of the region, temperature and relative humidity favour the rapid growth of putrefactive phenomena in the corpse, which allow us to reinforce the conclusion that the high toxicity of aldicarb prevented a post-mortem bacterial proliferation.

5. Conclusion

The toxicological analysis results, the autopsy findings and the information obtained, allowed us to conclude that death occurred due to an aldicarb acute intoxication. Although the case suggested a suicide, particularly based on the known information and on the high aldicarb concentration found in the stomach, it was not possible to indicate, precisely, the etiology of death (suicide or homicide).

References

[1] R.H. Dreishach, W.O. Robertson, Handbook of Poisoning, Appleton and Large Publications, Los Atlos, 1982.
[2] Guia dos Produtos fitofarmacêuticos (Ed.) Direcção Geral de Protecção das Culturas-DGPC, 2, 2003.

[3] http://www.inchem.org/documents/pds/pds/pest53_e.htm.
[4] Matthew J. Ellenhorn, Ellenhorn's Medical Toxicology. Diagnosis and Treatment of Human Poisoning, 2a edição, 68, 1623–1625.
[5] A. Covaci, P. Manirakiza, et al. A case of aldicarbe poisoning: a possible murder attempt, J. Anal. Toxicol. 23 (4) (1999) 290–293.
[6] E. Lacassie, P. Marquet, et al. Sensitive and specific multi-residue methods for the determination of pesticides of various classes in clinical and forensic toxicology, Forensic Sci. Int. 121 (2001) 116–125.
[7] J.L. Burgess, J.N. Bernstein, et al. Aldicarbe poisoning: a case report with prolonged cholinesterase inhibition and improvement after pralidoxime therapy, Arch. Int. Med. 154 (1990) 141–147.
[8] M. Lifshitz, M. Rotenberg, et al. Carbamate poisoning and oxime treatment in children: a clinical and laboratory study, Pediatrics 93 (1994) 652–655.
[9] F. Moritz, J.M. Droy, et al. Acute pancreatitis after carbamate insecticide intoxication, Intensive Care Med. 20 (1994) 49–50.
[10] A. Tracqui, F. Flesch, et al. Repeated measurements of aldicarb in blood and urine in a case of nonfatal poisoning, Hum. Exp. Toxicol. 20 (2001) 657–660.
[11] P. Parts, J. Lipman, et al. Carbamate toxicology: a case report, S. Afr. Med. J. 72 (1987) 222–225.
[12] M. Garber, Carbamate poisoning: the ''other'' insecticide'', Pediatrics 79 (1987) 734–738.
[13] Sam Hoai Ngo, A fatal case involving aldicarbe, TIAFT Bulletin Cases Notes, 21(2) 1991 (http://www.tiaft.org/tmembers/cnr/1991/21_2_3.html).
[14] P. Proença, E.G.P. Marques, et al. *N*-Metilcarbamatos: determinação múltipla por cromatografia líquida de alta resolução com detecção por fluorescência, Boletim de Medicina Legal e Toxicologia Forense XII (1) (1998) 187–193.
[15] D.J. Pounder, G.R. Jones, Post-mortem drug redistribution: a toxicological nightmare, Forensic Sci. Int. 45 (1990) 253–263.
[16] A.L. Pelissier-Alicot, et al., Mécanismes de la redistribution post-mortem des xénobiotiques: le point sur l'état actual des connaissances, Annales de Toxicologie Analytique, vol. XIII, No. 1, 2001.
[17] D.J. Pounder, J.I. Davies, Zopiclone poisoning: tissue distribution and potencial for post-mortem diffusion, Forensic Sci. Int. 65 (1994) 177–183.
[18] D.J. Pounder, C. Fuke, et al. Post-mortem diffusion of drugs from gastric residue: an experimental study, Am. J. Forensic Med. Pathol. 17 (1) (1996) 1–7.