

Acute toxic gas poisoning cases treated from 2004 through 2007 at a regional hospital emergency department: an epidemiologic study

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None

Objective: To determine changes in agents causing cases of chemical poisoning treated in the emergency department of Hospital San Agustín de Avilés in the period 2004 through 2007.

Methods: Descriptive observational study. The target population consisted of patients suffering from chemical poisoning who required emergency care at our hospital from 2004 through 2007. Data were extracted from the records of the clinical toxicology section of the Spanish Toxicology Association (AETOX).

Results: Two hundred four patients were treated for chemical poisoning during the study period. The agents involved were irritant gases (32.8%), caustic substances (25%), toxic gases (21.6%), solvents (11.3%), pesticides (4.4%), detergents (2.9%), and metals (2%). Toxic gas poisoning accounted for 68.2% of cases in the first 2 years (2004-2005) and 31.8% of cases in the second 2 years (2006-2007) ($P < .001$). Carbon monoxide (CO) was involved in 93.2% of the toxic gas cases. CO poisoning was mild-moderate in 78%, with carboxyhemoglobin levels between 11% and 30%. Most patients suffered from neurologic symptoms (95.%); the episode was resolved in the emergency department in 77.3% of the cases, usually by applying high-flow oxygen. The local media reported on the large number of poisonings treated in 2004 and 2005, at the same time publicizing measures for preventing CO poisoning.

Conclusions: The number of cases of toxic gas poisoning has decreased significantly, mainly due to a lower incidence of CO poisoning in the last 2 years. Efforts made to record cases and publicize information about how to maintain household heaters and heating systems have had a positive influence. [Emergencias 2009;21:350-353]

Key words: Acute intoxication. Toxic gases. Carbon monoxide. Epidemiology. Emergency health services.

Introduction

In 2004 the emergency department (ED) of Hospital San Agustín de Avilés (HSA) initiated a registry of acute poisoning by chemical products as part of the Toxicological Surveillance program of the Section of Toxicology Clinic (STC) of the Spanish Association of Toxicology (AETOX)¹. In this program, ED-treated cases of poisoning by household, agricultural or industrial chemical products are reported annually. Previously, no data on these cases were available in the ED so the focus was on cooperation with the STC and also on analyzing the characteristics of local poisoning. The purpose of the register was to obtain infor-

mation that could be used to develop control measures and reduce the risks of the most prevalent types of poisoning recorded in the population served by the HSA. The objective of this work was to study variations in the causal agent in acute poisoning by chemical agents during the period 2004-2007.

Method

We performed an observational, descriptive study. The study population was patients with chemical intoxication treated at our ED during the study period. We recorded the following variables

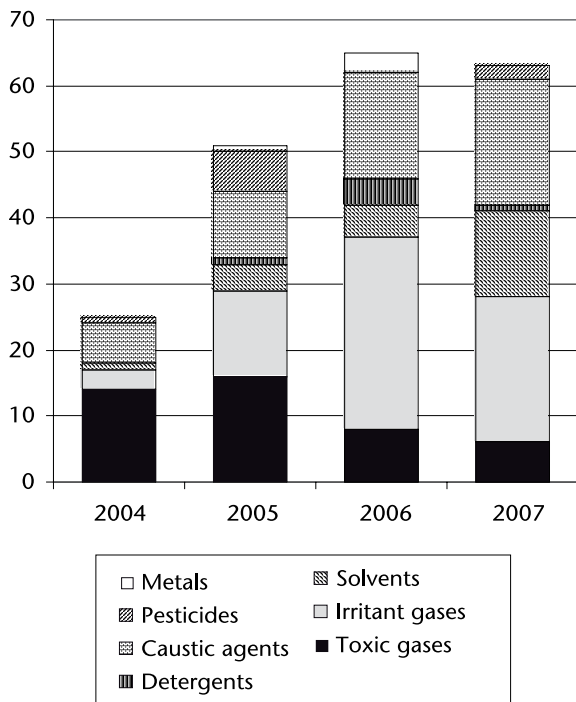


Figure 1. Distribution of types of poisoning from 2004 to 2007.

from the AETOX STC data base: age, sex, point of care, date and time of the poisoning and arrival at hospital, time to receiving attention, causes of poisoning (suicide, domestic work, murder), the type of toxin involved (toxic gases, irritant gases, solvents, detergents, caustics, pesticides, metals), place of poisoning, the route of poisoning, initial symptoms, sample collection (levels of carboxyhemoglobin (COHb) were determined in the ED laboratory as from the 2nd half of 2005, previously by another hospital), treatment administered, the length of hospital stay, admission or discharge and the reasons for discharge.

Quantitative variables are described as arithmetic means with standard deviation and range, and qualitative variables as absolute and relative frequencies. We used chi square test to compare the distribution of the type of toxin by periods. For statistical analysis of results and associations between different variables, we used the statistical package SPSS for Windows, version 12.

Results

During the period 2004-2007, 204 patients were treated for chemical intoxication. The types of poisoning involved were: irritant gases in 67 cases (32.8%), caustic products 51 cases (25%),

toxic gases 44 cases (21.6%), solvents 23 cases (11.3%), pesticides 9 cases (4.4%), detergents 6 cases (2.9%) and metal 4 cases (2%). Yearly distribution is presented in Figure 1.

There were 30 cases (68.2%) of toxic gas poisoning (TGP) in the period 2004-2005 and 14 cases (31.8%) in the period 2006-2007, ($p < 0.001$) (Table 1). The great majority (41 cases, 93.2%) of TGP were caused by carbon monoxide (CO) poisoning; the others being due to butane and propane gases from a sewage tank, with one case each. No difference was found in relation to sex. Mean age was 32.4 ± 17.9 years (range 1-65). The place of poisoning was the home in 39 cases (88.6%), workplace in 4 (9.1%) and suicide in 1 case (2.3%), the latter by butane gas. The source of CO corresponded mainly to incomplete combustion in hot water heaters and heating systems in homes, which meant that most patients affected were cohabiting members of a family group at the time of the poisoning. The route of poisoning was respiratory in all cases (100%). CO poisoning according to levels of CO-Hb was mild-moderate in 32 cases (78%) with values of COHb between 11-30%, severe in 7 (17.1%) with 31-40% COHb and very severe in 2 (4.9%) with COHb over 40%. Mean oxygen saturation by pulse oximetry was 96.8% (SD: 1.3) (range 93%-99%). Symptoms were predominantly neurological (95.5%) and gastrointestinal (36.4%). Time to ED attention was 4.8 ± 5.6 hours (range 10 minutes-24 hours). Treatment was based on high-flow oxygen therapy and symptomatic measures such as analgesics and antiemetics. One case of CO poisoning required mechanical ventilation but died at 12 hours, and one case required hyperbaric chamber treatment. The mean stay was 16.8 ± 27.1 hours (range 1 hour-7 days); 77.3% (34 cases) of TGP were resolved in the ED, with 65.9% (29 cases) requiring ED bay observation, 13.6% (6 cases) were observed in the paediatric day hospital, 6.8% (3 cases) were admitted to hospital and there was one death due to CO poisoning.

Discussion

In the latest epidemiological surveillance study of chemical poisoning treated at EDs, caustics appear as the main group of toxins, followed by toxic fumes and irritant gases². In our study, irritant gases occupy the first place, followed by caustics and then toxic gases. In the group of toxic gases, CO dominated almost exclusively, the

Table 1. Distribution of types of poisoning by periods ($p < 0.001$)

| Type of poisoning | Period | | Total |
|-------------------|------------------|-------------------|------------|
| | 2004-2005 | 2006-2007 | |
| Toxic gases | 30 (68.2%) | 14 (31.8%) | 44 |
| Others | 46 (28.8%) | 114 (71.3%) | 160 |
| Total | 76 (100%) | 128 (100%) | 204 |

source being incomplete combustion in hot-water boiler and home heating systems.

The competitive binding of CO to the heme group of hemoglobin displaces oxygen giving rise to the formation of COHb, which reduces its oxygen-transporting capacity. This pathophysiological mechanism together with CO binding to myoglobin and to mitochondrial cytochrome oxidase, blocking tissue respiration, produces generalized hypoxia which affects multiple organs^{3,4}. The measure of mitochondrial dysfunction does not form part of attending practice^{4,5}.

The diagnosis of CO poisoning is based on clinical suspicion; it is helped by observing the same symptoms in cohabitants all with COHb levels above 10% (in the general population this ranges from 2-3% in non-smokers to 5-8% in smokers)^{6,7}. COHb is a good marker of exposure to CO, but there is no correlation between clinical symptoms and levels^{8,9}, which means that in practice oxygen therapy is initiated without waiting for the test results. In most cases the symptoms were non-specific, predominantly neurological (headache, dizziness and drowsiness) and gastrointestinal (nausea and vomiting), and which favours infra-diagnosis of this type of poisoning.

The goal of treatment is to reverse the acute picture acute and avoid sequelae, especially neurological, which can occur up to two months after exposure. High-flow oxygen therapy with high-dose oxygen accelerates the separation of CO from haemoglobin, and decreases its half life to an hour (compared to 4.5 hours without supplemental oxygen)¹⁰. The half-life is further decreased to 20 minutes with hyperbaric oxygen. This could also accelerate the removal of CO bound to cytochrome oxidase, which improves the metabolic situation and perhaps helps to decrease neurological sequelae. The problem is that, although HBO is indicated for the treatment of poisoned patients with loss of consciousness, advanced age, advanced pregnancy (foetal haemoglobin has greater affinity for CO than maternal haemoglobin) and for those with COHb > 40-50% COHb, there is no consensus on who else should receive

hyperbaric oxygen, since no clinical variable or COHb level has been established to identify them as likely to benefit from this therapy^{11,12}. In our study HBO was only used in one patient with loss of consciousness and COHb of 15%; however, we have no record of any neurological after effects, probably due to the rapid initiation of high-flow of oxygen administration.

The high number of cases of CO poisoning recorded in the period 2004-2005 led us to publicity about this in the local media and the implementation of preventive measures in relation to sources of CO^{5,13}. Through the press, we informed the public of this health area about the risks involved in using deficient combustion gas stoves, central heating water heaters, especially in cramped and poorly ventilated spaces. In addition, the necessary measures to avoid this type of poisoning were widely publicised (Table 2). After this campaign, there was a significant decrease in toxic gas poisoning, mainly CO, in the following two years, dropping from first place in the type of chemical poisoning to third in our health area. The information obtained through the toxicological surveillance registry and subsequent publicity given to preventive measures and maintenance of combustion heating and heaters in homes seems to have had a positive influence, thus validating the argument that toxicological surveillance is a useful tool for health promotion¹⁴.

References

- Objetivos de la STC de la Asociación Española de Toxicología (Consultado 3 Diciembre 2008). Disponible en: <http://wzar.unizar.es/stc/objetivos/objetivos.html>
- Ferrer Dufol A, Nogué Xarau S. Estudio de vigilancia epidemiológica en los servicios de urgencias de las intoxicaciones causadas por productos químicos. (Consultado 3 Diciembre 2008). Disponible en: <http://wzar.unizar.es/stc/toxicovigilancia/informes/Informe anual 2006.pdf>
- Varon J, Marik PE, Fromm RE Jr, Gueler A. Carbon monoxide poisoning: a review for clinicians. *J Emerg Med.* 1999;17:87-93.
- Miró O, Alonso JR, López S, Beato A, Casademont J, Cardellach F. Análisis ex vivo de la función mitocondrial en pacientes intoxicados por monóxido de carbono atendidos en urgencias. *Med Clin (Barc).* 2004;122:401-6.
- Nogué Xarau S, Dueñas Laita A. Monóxido de carbono: un homicida invisible y silencioso. *Med clin (Barc).* 2005;124:300-1.
- Piantadosi CA. Carbon monoxide poisoning. *N Engl J Med.* 2002;347:1054-5.
- Dueñas-Laita A, Ruiz-Mambrilla M, Gandía F, Cerdá R, Martín-Escudero JC, Pérez-Castrillón JL, Díaz G. Epidemiology of acute carbon monoxide poisoning in a Spanish region. *J Toxicol Clin Toxicol.* 2001;39:53-7.
- Santiago-Aguinaga I, Frauca Sagastibelza C, Bardón Ranz A, Encina Aguirre Y, Pinillos Echevarria MA. Intoxicaciones por monóxido de carbón. Utilidad de los niveles de carboxihemoglobina. *Emergencias.* 2004; 16 Extraordinario: 74.
- Hampson NB, Hauff NM. Carboxyhemoglobin levels in carbon monoxide poisoning: do they correlate with the clinical picture? *Am J Emerg Med.* 2008;26:665-9.
- Weaver LK, Howe S, Hopkins R, Chan KJ. Carboxyhemoglobin half-life in carbon monoxide-poisoned patients treated with 100% oxygen at atmospheric pressure. *Chest.* 2000;117:801-8.

Table 2. Publicised measures for preventing carbon monoxide (CO) poisoning

1. Ensure taps are closed when not in use.
2. If there are changes in the colour of the gas flame (normal blue), notify technicians.
3. Ensure periodic check-ups in accordance with the manufacturer's instructions (it was publicised that CO can be disproportionately produced by old and poorly maintained equipment, and leak into the environment if there is inadequate disposal of combustion gases).
4. Have small fire extinguishers installed in homes.
5. Vents in kitchens and bathrooms must always remain open.
6. Install detectors of abnormal concentrations of CO in homes, as recommended by the experts.

- 11 Buckley NA, Isbister GK, Stokes B, Juurlink DN. Hyperbaric oxygen for carbon monoxide poisoning: a systematic review and critical analysis of the evidence. *Toxicol Rev.* 2005;24:75-92.
- 12 Wolf SJ, Lavonas EJ, Sloan EP, Jagoda AS. American College of Emergency Physicians Clinical. Clinical policy: critical issues in the management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. *Ann Emerg Med.* 2008;51:138-52.
- 13 Runyan CW, Johnson RM, Yang J, Waller AE, Perks D, Marshall SW, Coyne-Beasley T, McGee KS. Risk and protective factors for fires, burns, and carbon monoxide poisoning in U.S. households. *Am J Prev Med.* 2005;28:102-8.
- 14 Ferrer A, Nogué S, Vargas F, Castillo O. Toxicovigilancia: una herramienta útil para la salud pública. *Med Clin (Barc).* 2000;115:238.

Evolución epidemiológica de las intoxicaciones agudas por gases tóxicos atendidas durante el periodo de 2004 a 2007 en urgencias de un hospital comarcal

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Objetivo: Conocer las variaciones producidas en el agente causal en las intoxicaciones agudas por productos químicos atendidas en el servicio de urgencias (SU) del Hospital San Agustín de Avilés (HSA) durante el periodo 2004-2007.

Método: Se realizó un estudio observacional descriptivo. La población objetivo fueron los pacientes intoxicados por productos químicos atendidos en los años 2004-2007 en el SU del HSA. Se recogieron las variables de la ficha de intoxicaciones de la Sección de Toxicología Clínica (STC) de la Asociación Española de Toxicología (AETOX).

Resultados: Durante el periodo de 2004 a 2007 se atendieron 204 pacientes intoxicados por productos químicos: gases irritantes 32,8%, productos cáusticos 25%, gases tóxicos 21,6%, disolventes 11,3%, plaguicidas 4,4%, detergentes 2,9% y metales 2%. Las intoxicaciones por gases tóxicos (IGT) en 2004-2005 fueron el 68,2% del total y en 2006-2007 el 31,8% ($p < 0,001$). El grupo de IGT está constituido en el 93,18% por monóxido de carbono (CO). Las intoxicaciones por CO fueron leves-moderadas (78%) con carboxihemoglobina (COHb) del 11-30%. La clínica fue fundamentalmente neurológica (95,5%). El 77,3% se resolvieron en el SU. El tratamiento mayoritario fue oxígeno a alto flujo. El elevado número de intoxicaciones en 2004-2005 fue difundido a los medios de comunicación locales, así como las medidas preventivas en relación a las fuentes de CO.

Conclusiones: Se ha producido un descenso significativo de las intoxicaciones por gases tóxicos y de su mayor representante, el CO, en los últimos dos años. El esfuerzo del registro y la difusión de las medidas de mantenimiento de las instalaciones de calefacción y calentadores de los domicilios ha podido tener una influencia positiva. [Emergencias 2009;21:350-353]

Palabras clave: Intoxicaciones agudas. Gases tóxicos. Monóxido de carbono. Epidemiología. Urgencias hospitalarias.