

Case report: oral chloroform poisoning



The Poisoning Information Centre's objective is to provide adequate advice quickly so as to reduce the incidence of illness, damage to health and death as a result of severe cases of poisoning. Our centres are staffed with internationally trained and highly experienced doctors and nurses working in the fields of emergency medicine, anaesthesia and intensive care.

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Introduction

The Poisoning Information Centre (PIC) received a call from 90-year old man's relative. The patient had ingested mistakenly 10 minutes previously a sip of chloroform.

Chloroform, a halogenated hydrocarbon, is clear, colorless, and volatile liquid with radiopacity². Poisonings after oral chloroform ingestion, remains rare and information regarding the clinical course and management dilemmas of chloroform ingestions is limited. Chloroform acts mainly as a central nervous system (CNS) and cardiac depressant. As little as 10 mL in an acute ingestion may result in central nervous system depression and death¹.

Through active metabolites production of a toxic metabolite by the cytochrome P450 system, delayed hepatic and renal toxicity may occur with a peak elevation of liver enzymes on 6th³ and 5th day² after exposure.

Treatment with N-acetylcysteine (NAC) has been proposed to prevent liver injury.

The utilization of NAC for chloroform-induced hepatotoxicity has demonstrated successful outcomes in 2 cases:

1. ingestion of about 75ml of chloroform, treated with intravenously administered NAC 150 mg/kg over 1h, followed by 50mg/kg over 4h, after that regimen of 6.25mg/kg/h was continued for 6 days³
2. ingestion of about 100 ml of chloroform treated with NAC 600 mg/day for 11 days²

Objective

Aim of this study is to describe retrospectively the clinical findings, treatment and outcome of an accidental ingestion of liquid chloroform by a 90-year old man.

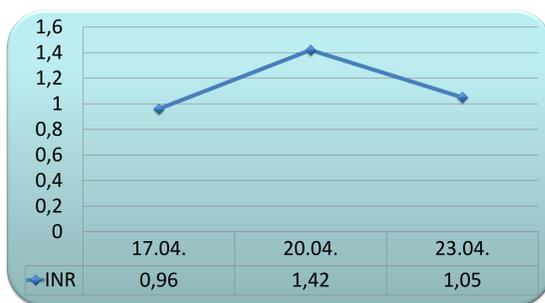


Fig.1 A peak elevation of INR after 72h from chloroform ingestion.

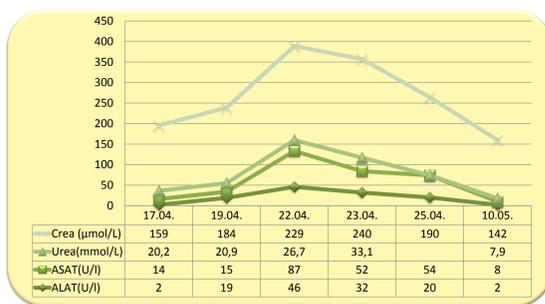
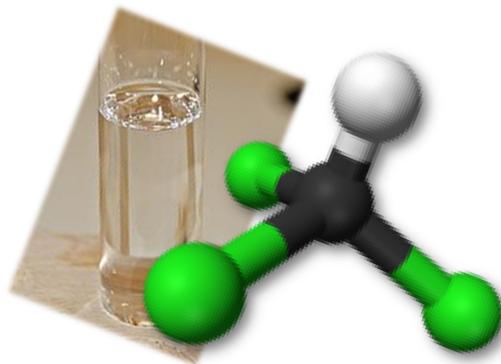


Fig.2 A peak elevation of liver enzymes after 120h from chloroform ingestion.



Case report

Drowsiness began shortly after ingestion, coma developed in **10 minutes**, bradycardia was detected in the ambulance, approximately on the **45th minute**. The patient was admitted to hospital comatose (GCS 3p, SpO2 68%, RR 80/50mmHg, wide QRS-complex 46x/min) on the **80th minute**.

Prior to arrival the emergency department was informed by the Poisoning Information Centre about the potential health effects of chloroform and hepatotoxicity prevention options. Administration of NAC i/v in loading dose (150 mg/kg) was initiated within one hour of admission. The patient was intubated and ventilated with 100% oxygen, diuresis was forced by furosemide, haemodynamics stabilized with infusion of vasopressors.

The patient was transferred after **4 hours and 15 minutes** from ingestion to the ICU. Within the **next 24-hours** his general condition stabilized with supportive care. The patient was extubated. Initially the patient remained oxygen dependent and needed small doses of vasopressors.

Slight hepatorenal damage developed (maximal values of AST 87U/l, ALT 46 U/l, Crea 240 µmol). The patient was **transferred to the nursing department on the 16th day** after ingestion with satisfactory status, with need for assistance in everyday life due to previous comorbidities.

Conclusion

We report the case of an elderly man who accidentally ingested chloroform and recovered after supportive treatment with NAC.

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Acknowledgements

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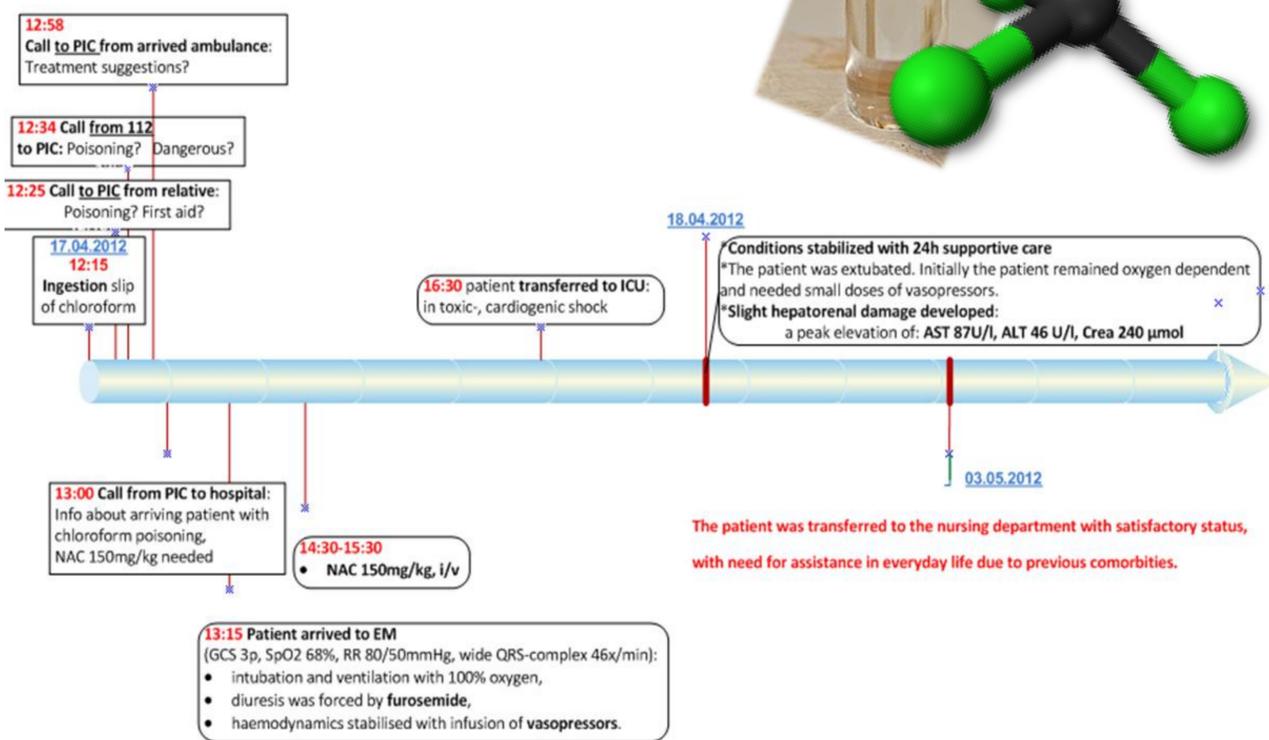


Fig.3 Flow chart :Poisoning with chloroform, treatment and outcome 17.04.- 03.05.2012

References

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2. Choi, S., H et al. (2006) Diagnostic radiopacity and hepatotoxicity following chloroform ingestion: a case report. Emerg. Med. J. 2006 May; 23(5): 394–395
3. Dell, A et al. (2010) Acute Chloroform Ingestion Successfully Treated with Intravenously Administered N-acetylcysteine. Journal of Medical Toxicology, June 2010, Vol 6 (2):143-146