

# Petrol ingestion poisoning in a pregnant woman

M. Fracas, J. Wabersich

*Department of Gynecology and Obstetrics, Padua University*

## Summary

A 38-week pregnant patient, weighing 70 Kgs, with poisoning by petrol ingestion, showing mixed acidosis and with a fetus with cardiotocographic alterations and oligohydramnios, underwent an urgent caesarean section under general anaesthesia. After 15 days from ingestion no complications were observed, the mother's vital parameters were normal and no severe complications were seen in the newborn (2900 g; 49 cm).

There are no similar cases of intense hydrocarbon ingestion in pregnant women as far as we know. Thus, in order to evaluate the toxicity of such chemical agents on pregnant patients and fetuses, we must consider the quantity of ingested hydrocarbons, gestation time and metabolism modifications which physiologically occur during pregnancy.

## Introduction

Hydrocarbons are organic compounds present in commonly used products like kerosene, petrol, gas oil, oil for lamps and various cleaning products [11].

Usually acute intoxication is due to absorption by inhalation from professional causes.

Absorption can also occur through the skin while ingestion always has a self-damaging intent with adults and is accidental with children [12].

The main targeted organs are: lungs, nervous central system, myocardium, kidney, liver and adipose tissue [12].

Cases of acute ingestion of hydrocarbons by a pregnant patient have been never described. It is therefore not possible to know what toxic effects could occur in the fetus.

## Case Report

A 28-year-old pregnant woman in the 38th week of gestation with a weight increase of 13 Kgs was found in a clearly soporific state two hours after ingestion of a non-specified quantity of petrol (the patient referred to having ingested 7-8 swallows probably equal to 500 cc).

After giving oxygen at 100% with a mask, the patient was transferred to the Obstetrics Clinic where she arrived alert and oriented. Monitoring of the patient's vital parameters never showed significant alterations in the circulatory system even if mixed acidosis and unbalanced electrolytes were present due to intense hypoxia.

Arterial blood gas values after ventilation showed a pH of 7.4, pCO<sub>2</sub> 28.4, pO<sub>2</sub> 104, HCO<sub>3</sub><sup>-</sup> 18, BE 6.9, and pSO<sub>2</sub> 98%.

The Anti-Poison Center of Milan, Niguarda Ca' Grande Hospital, was contacted and confirmed that no information regarding similar experiences was available and there was a lack of updated literature.

It was therefore not possible to know the toxic effects in the fetus.

During cardiotocography monitoring a reduction in cardiac frequency and variability was found. The basal frequency found was 105 b/min for a period of about 10 minutes; tracing

afterwards showed characteristics of a saltatory tracing. Considering the gestation time and the oligohydramnios, we decided to a caesarean section under general anaesthesia avoiding peripheric anaesthesia as to not risk an "ab ingestis" pneumonia. During caesarean section no particular alteration in the patient's vital parameters were seen and the operation went smoothly.

A live female fetus, weighing 2900 g and 49 cm long, was extracted with three rounds of umbilical cord around the neck.

The APGAR score at the 1st. min. was 4/10 and at the 5th. min. was 8/10. The newborn was intubated but due to her quick and good adaptive reaction she was promptly extubated.

The patient, after awakening, was followed inside the Intensive Care Unit for four days as respiratory depression and late arterial hypotension were expected as possible complications of the hydrocarbon ingestion.

After having excluded any possible complication, the patient was transferred to Psychiatric Services where acute paranoid reaction was diagnosed.

After 10 days from ingestion and caesarean section the patient's circulatory system was stable arterial blood gas values and electrolytes were normal and no respiratory problems were found.

The chest x-ray taken after ingestion and repeated after 10 days showed good diffusion transparency and no basilar infiltrates.

As far as the newborn is concerned, she remained under observation for two days and, considering the good general condition, she was transferred to the nursery.

At the 5th day of life the newborn was discharged as no evident problems were observed.

## Discussion

Acute ingestion of hydrocarbons is one of the most common causes of accidental poisoning in children [23, 6]. There are several studies describing intoxication due to ingestion of petroleum by-products.

Bysoni and Rubben, observed that this is the cause of 5-25% of all under 5-year-old poisoning causes [10].

Inhalation of hydrocarbons due to chronic exposure is very common [12], while hydrocarbon intoxication by acute ingestion in a pregnant woman has not previously been described in the literature [17].

Received July 15, 1997

revised manuscript accepted for publication October 10, 1997

Notwithstanding that the effect of these poisons on living organisms is known, nowadays we do not know how toxicity affects the fetus and how hydrocarbons influence pregnancy [17, 20].

This brings about two problems:

- the first is connected to the toxic effects on the organism and specifically on a pregnant woman with modified basal metabolism.
- the second is connected to the consequences on the fetus.

Among the most toxic common effects on the organism there is the well known respiratory depression syndrome due to acute pneumonitis-adult respiratory distress syndrome (ARDS)-subordinate above all due to “ab ingestis” more than to gastrointestinal absorption [2, 5, 16].

Hydrocarbons, in fact, are low viscosity chemical compounds which could provoke lung alterations of gas exchanges with consequence of liquid interstitial inhibition and intensive ipoxia [9, 12].

Several cases of subordinate ARDS have been described from ingestion and inhalation of hydrocarbons by children [6, 15, 23]. Following inhalation “ab ingestis” in favor of ARDS, Giammona demonstrated that only endovenous infusion or endotracheal inhalation are able to provoke lung damage in animals, and not hydrocarbons ingestion [9].

Toxic effects on the central nervous system such as respiratory problems, soper, coma and death are quite common and are probably due to ipoxia and acidosis [24].

The literature reports various other effects due to intoxication by acute ingestion of hydrocarbons which were not evident in our case. Precisely, acute nephric damage with hypouresis [3, 13], hepatocellular damage [22], intracellular haemolysis [1], gastric ulcers [10], and finally cardiac toxicity with inotropism depression are described [19].

As far as our patient is concerned, the last echography showed the presence of normal amniotic liquid while 8 hours after ingestion of the poison we found oligohydramnios prompting us to proceed with an urgent caesarean section to avoid fetus damage.

Concerning metabolism modifications in pregnant women, it is known that during pregnancy there is an increase of basal metabolism with increased oxygen consumption of about 15%.

There is also liquid retention inside the extracellular compartment and increased adipose tissue deposits. Inside the lungs the current volume and breathing capability progressively increases,  $pCO_2$  decreases considerably, decreased plasmatic bicarbonates are observed and arterial pH stays at 7.40. Following the increase of cardiac flow we can observe an increase of nephric plasmatic flow and glomerular filtrate. Secretion, tone and gastric motility decrease, average time of gastric leakage increases and there is a reduction of the speed of gallbladder emptying [21].

It is well known that after absorption, hydrocarbons accumulate in adipose tissue where they are slowly released;

12% is eliminated without alterations through breathing, 0.1% goes to the urine while the main part is metabolized by the liver [21].

Concerning the toxic effects of hydrocarbons on the fetus, we could not find similar experiences described in the literature.

The only information comes from studies on professional cronic exposure by inhalation or skin contact [4] and from research on lab animals [8].

An epidemiological study on pregnant women working in an oil company and therefore exposed to cronic inhalation has shown a higher incidence of fetal malformations, such as palatoschisis and diaphragmatic hernia, growing defects and thymus regression with respect to the general population [4]. These types of malformations observed are influenced by the pregnancy time in which exposure occurs.

The only data about the effects on the fetus after hydrocarbons acute ingestion by the mother, come from animals while for human beings we can only hypothesize. The studies on lab animals have permitted us to analyze these effects using a wide case series which on the contrary, cannot be completely superimposed on human physiology [7, 8].

## Conclusions

To conclude we noticed small reversible alterations of the central nervous system (sopor) and mixed acidosis corrected by giving oxygen and an infusion of buffer solutions. ARDS signs were not observed nor was there kidney, liver or cardiac damage. The fetus after initial symptoms (cardiotocography alterations, oligohydramnios, APGAR 4/10 at the 1st minute) survived and today does not show any negative effects.

Based on our experience, we believe that in order to evaluate toxicity in cases of poisoning by hydrocarbons acute ingestion by pregnant women, it is important to consider, together with the quantity of chemical agent ingested, the pregnancy term.

In fact, the lack of toxic effects noticed in our patient (ARDS, hepatonephro-cardiotoxicity) could be a consequence of the fact that she ingested a small quantity of toxic agent, that metabolic alterations were promptly adjusted and also that the increase of circulating liquid volume, along with an increase of adipose tissue in this case, further diluted the concentration of the toxic agent.

Therefore, the absence of any toxic effect on the fetus could derive from the quantity of poison ingested, from its availability and, above all, from pregnancy time when ingested.

In our case, we had a fetus at the 38th week and it was possible to proceed with an urgent caesarean section as the fetus maturity allowed this.

It remains that the problem of what the consequences on the newborn and its future life could be are still unpredictable.

## References

- [1] Algren J. T., Rodgers G. C.: "Intravascular hemolysis associated with hydrocarbon poisoning". *Pediat. Emerg. Care*, 1992, 8, 34.
- [2] Arena J. M.: "Hydrocarbon poisoning, current management". *Pediat. Ann.*, 1987, 16, 879.
- [3] Banner W., Walson P. D.: "Systemic toxicity following gasoline aspiration". *Am. J. Emer. Med.*, 1983, 3, 292.
- [4] Chen Z., Chen C., Dong S.: "Epidemiological studies on risk for adverse pregnancy outcomes in women neighboring a petrochemical works". *Chunh. Hua. Yu. Fang. I. Hsueh. Tsa. Chih.*, 1995, 29 (4), 209.
- [5] Dice W. H., Ward G., Kelley J., Kilpatrick W. R.: "Pulmonary toxicity following gastrointestinal ingestion of kerosene". *Ann. Emerg. Med.*, 1982, 11, 138.
- [6] Ervin M. E., Manske M. G.: "Petroleum distillates and turpentine". Chap. 76 in: "Haddad L., Winchester J.: "Clinical Management of Poisoning and Drug Overdose". 2nd ed. Philadelphia, WB Saunders, 1990, 1177.
- [7] Feuston M. H., Mackerer C. R.: "Development toxicity of clarified slurry oil, syntower bottoms, and distillate aromatic extract administered as a single oral dose to pregnant rats". *J. Toxicol. Environ. Health.*, 1996, 49 (1), 45.
- [8] Feuston M. H., Mackerer C. R.: "Development toxicity study in rats exposed dermally to clarified slurry oil for a limited period of gestation". *J. Toxicol. Environ. Health.*, 1996, 49 (2), 207.
- [9] Giammoma S. T.: "Effects of furniture polish on pulmonary surfactant". *Am. J. Dis. Child.*, 1967, 113, 658.
- [10] Bysani G. K., Rucoba R. J., Noah Z. L., F.C.C.P.: "Treatment of hydrocarbon pneumonitis". *Chest*, 1994, 106, 300.
- [11] Haddad L., Winchester J.: "Clinical Management of Poisoning and Drug Overdose". Philadelphia, WB Saunders, 1990, 1177.
- [12] Harrison T. R.: "Principi di medicina interna". Wilson J. D. *et al.*, 1ª ed. italiana sulla 12ª originale. McGraw-Hill Italia, 1992, Milano.
- [13] Janssen S., Van der Geest S., Meijer S., *et al.*: "Impairment of organ function after oral ingestion of refined petrol". *Intensive Care Med.*, 1988, 14, 238.
- [14] Klein B. L., Simon J. E.: "Hydrocarbon poisonings". *Pediat. Clin. North Am.*, 1986, 33, 411.
- [15] Luis J. P., Simao C., Rodrigues G., Carvallho A., Almeida H., Correia M., Sequeira J. S.: "Adult type respiratory distress syndrome (ARDS) induced by hydrocarbons". *Acta Medica Portuguesa*, 1996, 9 (1), 45.
- [16] Machado B., Cross K., Snodgrass W. R.: "Accidental hydrocarbon ingestion cases telephoned to a regional poison center". *Ann. Emerg. Med.*, 1988, 17, 804.
- [17] Marchetti M.: "Azione dei veleni sul prodotto del concepimento". *Minerva Ginecologia, Atti delle Società Regionali di Ostetrica e Ginecologia*, 1973, 25 (9), 539.
- [18] Marrubini Bozza M. L., Laurenzi Ghezzi R., Uccelli P.: "Intossicazioni acute. Meccanismi, diagnosi e terapia". 2° ed. Organizzazione Editoriale Medico Farmaceutica, Milano, 1987, 760.
- [19] Okechukwu A., Castello F. V.: "Myocardial dysfunction after hydrocarbon ingestion". *Critical. Care Medicine*, 1994, 528.
- [20] Onnis A., Grella P., Marchesoni D.: "I farmaci in gravidanza". 2ª ed. Piccin Editore, Padova, 1983.
- [21] Pescetto G., De Cecco L., Pecorari D., Ragni N.: "Manuale di ginecologia e ostetricia". 2nd ed. Società Editrice Universo, 1989, Roma.
- [22] Plummer J. L., Hall P., Ilsley A. H., *et al.*: "Influence of enzyme induction and exposure profile on liver injury due to chlorinated hydrocarbon inhalation". *Pharmacol. Toxicol.*, 1990, 67, 329.
- [23] Truemper E., Rejes de la Rocha S. R., Atkinson S. D.: "Clinical characteristic, pathophysiology, and management of hydrocarbon ingestion: case report and review of the literature". *Pediatr. Emerg. Care*, 1987, 3, 187.
- [24] Wolfsdorf J.: "Kerosene intoxication: an experimental approach to the etiology of the CNS manifestations in primates". *J. Pediatr.*, 1976, 88, 1037.

Address reprint requests to:

J. WABERSICH  
Institute of Gynecology and Obstetrics  
University of Padova  
Via Giustiniani, 3  
35128 Padova (Italy)