



ACUTE PERITONITIS SECONDARY TO ACCIDENTAL INGESTION OF LIQUID BUTANE, A CASE REPORT

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SUMMARY

Butane poisoning is relatively rare (2.3% of domestic poisoning cases). It usually occurs after butane inhalation and only one case of ingestion poisoning has been reported in the literature in the last twenty years. We propose to describe the diagnostic and therapeutic modalities of acute generalised peritonitis secondary to accidental ingestion of liquid butane in a 39-year-old patient. On admission, our patient presented with severe asthenia, respiratory distress syndrome and peritoneal irritation syndrome. We suspected inhalation pneumonitis associated with perforating peritonitis. Emergency laparotomy revealed an intraperitoneal gas effusion, frostbite lesions on the gastric wall and no gastric perforation. Postoperative management was uneventful. This study questions the association between pneumoperitoneum and gastric perforation in butane ingestion.

Key words : *acute generalized peritonitis, butane ingestion, case report*

1-Introduction

The abuse of volatile substances is a global problem. Among these, butane gas is implicated in 2.3% of domestic poisonings [1,2].

Butane (C₄H₁₀), also known as butyl hydride, is a colourless, odourless, highly flammable and volatile saturated hydrocarbon. Naturally gaseous at atmospheric pressure (1 bar) and ambient temperature (15°C), it becomes liquid at pressures of 1.5 to 7 bar and temperatures below 0°C. This liquid state allows it to be handled, transported and stored in small volumes ; for example, 1 litre of liquid butane releases 239 litres of gas (at 15°C under 1 bar pressure). [3]

Usually intended for domestic and industrial use, butane is increasingly being diverted for recreational or even autolysis purposes. From 2000 to date, 42 articles have reported the use of butane in poisoning. While the main route of intoxication is inhalation, there is only one rare case of butane ingestion in the literature, which occurred during a suicide attempt. [2]

The aim of this study is to describe the diagnosis and management of acute generalised peritonitis following accidental ingestion of liquid butane.

2-case report

A 39-year-old patient, a bricklayer by trade with no relevant medical history, was admitted to the Yaounde Emergency Centre (CURY) with severe physical asthenia, dyspnoea and generalised abdominal pain (VAS 8/10) secondary to accidental ingestion of a large quantity of liquid butane gas in an unlabelled water bottle at his place of work (construction site of a domestic gas bottling plant).

He was admitted one hour after ingestion of the toxic substance, and on primary assessment his airway was clear; oxygen saturation was 83% on room air, with tachypnoea at 36 cycles per minute, prompting high concentration mask oxygen therapy which increased saturation to 96%. His blood pressure was 132/90

mmHg with a regular pulse of 92 beats per minute. The patient was conscious and well oriented in time and space, with a Glasgow score of 15/15 and isochoric and reactive pupils. His temperature was 37.2°C. Secondary assessment revealed NYHA 4 dyspnoea with a normal cardiac and pulmonary examination. The patient's abdomen was distended and symmetrical. Generalised guarding and umbilical rebound tenderness were noted, as well as resolution of prehepatic dullness. The neurological examination was completely normal with no sensory or motor deficits and no signs of meningeal irritation.

The findings of peritoneal irritation and acute respiratory distress syndrome led us to suspect acute generalised peritonitis, probably associated with inhalation pneumonitis. A standard preoperative work-up (Table 1), a frontal chest x-ray and a plain abdominal x-ray (upright and frontal) were performed (Figure 1). The main findings were large bilateral pneumoperitoneum, thrombocytopenia and hypokalemia. Surgery was indicated and the patient underwent surgery 14 hours after admission.

A median supraumbilical laparotomy was performed which revealed a strong odour of domestic gas emanating from the peritoneal cavity, multiple whitish crusts scattered over the body of the stomach (frostbite), no visible perforation and no peritoneal effusion. Exploration of the abdominal cavity and lavage of the peritoneum were the only procedures performed.

The postoperative period was characterised by normalisation of respiratory rate and oxygen saturation in ambient air from D0 postoperatively and progressive normalisation of platelet levels. The patient was fasted for four days. Once the patient was allowed to eat again, his course remained uncomplicated and he was discharged on the sixth postoperative day.

3-discussion

Our patient was an adult male who accidentally ingested liquid butane. According to Vahabzadeh et al, the main route of butane poisoning is inhalation (53 of 54 cases). Most of these patients were men (81%), usually prisoners, who used butane as a substitute for narcotics to which they no longer had access. Apart from this recreational use, three cases of butane poisoning were associated with attempted autolysis, two by inhalation and only one by ingestion of butane.

In our study, the clinical manifestations were respiratory and gastrointestinal, with pneumoperitoneum and thrombocytopenia on further investigation. In the literature, cases of butane inhalation are generally associated with respiratory complications (asphyxia due to laryngospasm), cardiac complications (severe coronary spasm and ventricular fibrillation), neurological complications (euphoria, narcosis and somnolence) [4] and, secondarily, renal complications (rhabdomyolysis) [5]. The only reported case of butane ingestion did not present with dyspnoea, but with lethargy, epigastric pain and diffuse tympany on physical examination. He had sinus tachycardia, predominantly neutrophilic hyperleukocytosis and pneumoperitoneum on paraclinical examination.

The presence of signs listed in both butane inhalation and butane ingestion cases leads us to believe that it is likely that our patient ingested and inhaled butane. However, neurological signs were absent in our patient, whereas they were constant in the cases reported in the literature. In the absence of an electrocardiogram, the possibility of cardiac damage in our patient could not be ruled out.

Our therapeutic approach is similar to that of Cheng et al in their case of butane ingestion. However, our operative findings were very different. One was a gaseous effusion without visible perforation and the other was a gaseous effusion with perforation of the small curvature of the stomach. In a study similar to ours, which looked at lesions associated with nitrogen ingestion [6], the following lesions were reported: 3 cases of pneumoperitoneum associated with gastric perforation (anterior wall of the lesser curvature of the stomach), 1 case of pneumoperitoneum without gastric perforation, 1 case of abdominal distension, 1 case of combined abdominal distension and subcutaneous emphysema, and 1 case of combined bilateral pneumothorax with subcutaneous emphysema and gastric perforation. Thus, the presence of pneumoperitoneum after ingestion of these gases does not always correlate with macroscopically visible gastric perforation. These differences could be related to: the context of ingestion (accidental in our case and voluntary in all the others) and therefore the amount of toxic ingested, and probably the delay in treatment. In his study, Yuemei Zheng et al. attribute the rarity of lesions in the mouth, oropharynx, upper respiratory tract and oesophagus to the Leidenfrost effect, in which a liquid encountering a temperature well above its boiling point forms an insulating vapour layer that slows thermal transfer. We believe that the absence of gastric lesions in our patient may be due to this effect.

The favourable outcome of our patient after treatment is in line with the literature, which gives a good prognosis for cases of butane ingestion and a disastrous functional and vital prognosis (80% death) for cases of butane inhalation. However, in the absence of a visible perforating lesion, we question the real therapeutic impact of our patient's surgical treatment on his clinical course.

CONCLUSION

Poisoning by ingestion of butane is very rare. The clinical picture is one of peritoneal irritation syndrome with or without perforation, but with persistent pneumoperitoneum on the plain abdomen in the upright position. Surgical treatment seems to be the norm for this lesion and the postoperative course is generally uncomplicated.

TABLES AND FIGURES

table 1 : pre-operative laboratory tests

		Values
Hémogramme	White blood cells	8.85 10⁶/ml
	Red blood cells	4.71 10⁹/ml
	Haemoglobin	13.8 g/dl
	Platelets	132 10⁶/ml
Urea		0.34 g/l
Creatinine		11,97 mg/l
Sodium		141.8 mEq/ l
Potassium		3.32 mEq/ l
Chlorine		102.5 mEq/ l
Prothrombin rate		100%
Activated partial thromboplastin time		21.0 s
Blood group		O positive



figure 1 : thorax radiograph, upright, front view

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