CASE REPORT

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Death Following Crude Oil Aspiration

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ABSTRACT: This is a report on three deaths following oil aspiration by workers in petrol tankers. Lung aspiration was demonstrated by the presence of a yellowish-brown material in the alveolar spaces, which was difficult to identify by optic microscopy. Volatile hydrocarbons from petroleum were identified in lung samples by gas chromatography/mass spectrometry.

KEYWORDS: pathology and biology, petroleum products, aspiration, oil, lung aspiration

Crude oil is a complex mixture of aliphatic, alicyclic, and aromatic hydrocarbons, which includes a range from the lightest liquid components, with 4 or 5 carbon atoms, to the heaviest, with 40 atoms. Its composition varies greatly according to its geological source, and it is classified into paraffinic, naphthenic, and aromatic petroleum, depending on the predominant fraction of hydrocarbon present.

Gas oil is the fraction of hydrocarbon obtained by fractionated distillation from crude oil at a boiling point over 275°C [*1*].

The principal acute effect of crude oil on humans is narcosis [*2*], which is a consequence of the depression of the central nervous system (CNS), typical of all hydrocarbons; this effect is preceded by stimulation. From a chemical point of view, highly volatile aromatic hydrocarbons are associated with an elevated risk of toxicity to the CNS due to their high liposolubility.

The inhalation of high concentrations of the vapors given off by petroleum can produce a chemical pneumonitis due to irritation of the pulmonary mucous membranes [*3*], which

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gives rise to pulmonary edema and hemorrhage. Pulmonary toxicity is primarily related to aspiration of the poison [4], which occurs during ingestion or vomiting.

Cases 1 and 2

These two victims were brothers working in a petrol tanker. One of them went down into a tank to repair it, and when he did not return, his brother went to the rescue without taking any protective equipment with him. The second brother did not return either. Wearing artificial breathing apparatus, their companions brought them out, but they showed no sign of life.

At autopsy, the first one exhibited cuts and contusions in different parts of the body. There was a large hematoma at the cranial apex and slight cerebral edema. The lungs were large and congested, with a great number of subpleural petechiae scattered through both lungs. There were subepicardial pointed petechiae on the left side of the heart. The following determination was made: the possible cause of death was asphyxia by inhalation of toxic gases, probably with loss of consciousness, given the traumatic lesions.

Blood, the gastric contents, and a fragment of brain were taken for toxicological analysis, and fragments of lung were preserved in formalin for histopathological study.

The second brother showed more evident signs of asphyxia. There were no contusions or injuries, but there was slight ecchymosis around the nostrils. Subarachnoid petechiae and congestive meninges were present, as well as cerebral edema. Submucosal ecchymosis was seen in the trachea and bronchi. The lungs were large, with rounded edges, congestive, and edematous, with a great quantity of pointed petechiae scattered through both lungs and more extensive ecchymosis in the left interlobular sulcus.

Blood and fragments of brain, lung, and liver were received for toxicological analysis, and fragments of lung were placed in formalin for histopathological study.

The viscera of both brothers smelt intensely of burning, probably because of the crude oil saturating them.

Case 3

The victim was a 57-year-old man, a dockyard worker who died cleaning a gas oil tanker in a ship. He lost consciousness and fell, producing unimportant bruises in the frontal and mandibular regions.

At autopsy, the most evident pulmonary findings were edema and congestion in the right lung lobule and in the lower lobule of the left lung.

Taking into account the fact that the inhalation of the poison or irritant produced loss of consciousness, as a hypothesis of the mechanism of death, we suggest that the gas oil caused this effect by a mixed mechanism of toxemia/anoxemia.

Blood from the lung was received for toxicological analysis, as well as a fragment of lung in formalin for histopathological study.

Histopathological Study

The samples of lung in formalin from the three cases were embedded in paraffin, cut at 6 μm, and stained with hematoxylin and eosin and van Gieson's stain. Microscopic examination showed alveolar edema, hemorrhage, and a strange material, difficult to identify by optic microscope, consisting of yellowish-brown-colored particles of different shapes occupying some alveoli. In the third case, areas of emphysema and abundant anthracosis were seen (Figs. 1, 2, and 3).

These histopathological findings were interpreted as pulmonary aspiration.

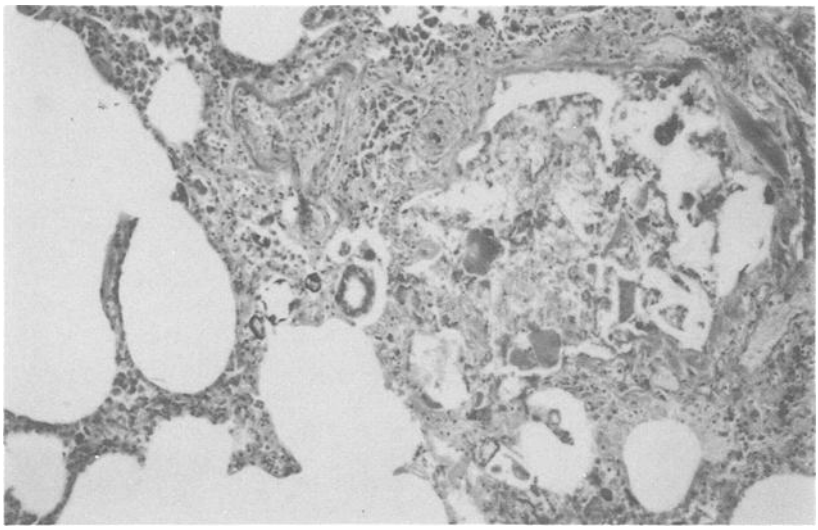

FIG. 1—*Yellowish-brown-colored particles occupying alveolar spaces in Case 1 (hematoxylin and eosin stain; original magnification, ×100).*

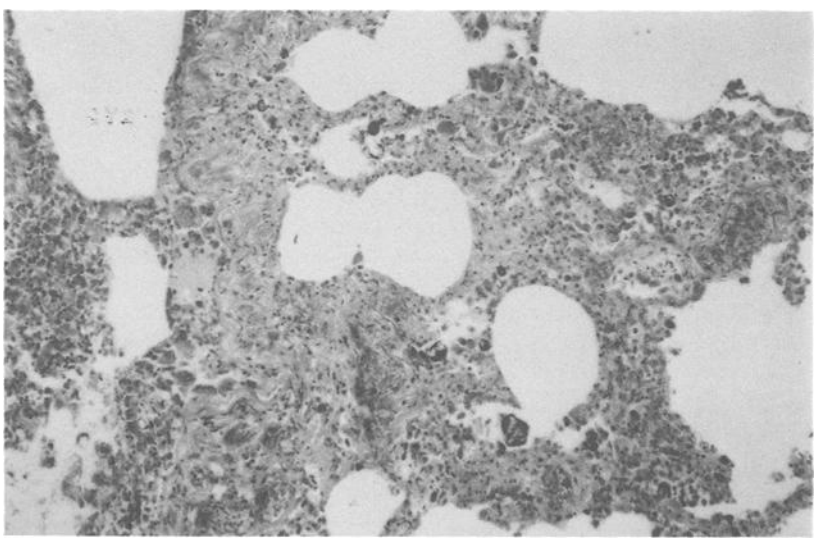

FIG. 2—*Alveolar edema, hemorrhage, and yellowish particles in Case 2 (hematoxylin and eosin stain; original magnification, ×100).*

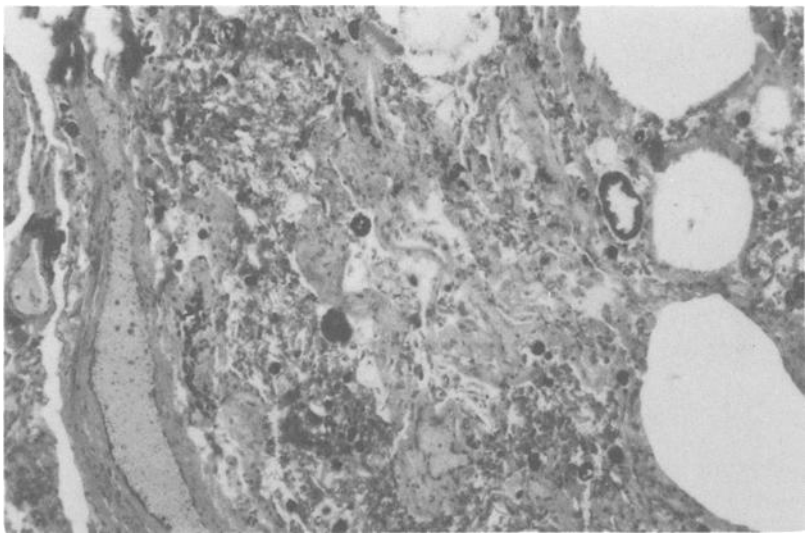

FIG. 3—*Alveolar edema, hemorrhage, anthracosis particles, and unknown material in Case 3 (hematoxylin and eosin stain; magnification, ×100).*

Toxicological Analysis

Gas Chromatography

The analysis of solvents and other volatile substances was carried out by gas chromatography using the headspace technique with a Hewlett-Packard Model 5710 A gas chromatograph attached to a Hewlett-Packard Model 3388 A integrator and equipped with a flame ionization detector (FID). Separation was attained with a 10-m by 0.530-mm inside-diameter fused silica column with 20 M Carbowax coating. The column temperature was held for 2 min at 85°C, then raised to 200°C at a rate of 5°C/min, and held 8 min. The flow rate was 5 mL/min. The helium injector and detector was operated at a temperature of 250°C.

Gas Chromatography/Mass Spectrometry

Hydrocarbon identification was accomplished by gas chromatography/mass spectrometry (GC/MS), using a GC/EM/Data System HP-5995 B, operated in electron impact (EI) mode and using a capillary injection system in splitless modality.

Case 1

The blood sample arrived split, and since it was thus not usable, the decision was made to investigate the presence of volatile hydrocarbons from petroleum in the other samples.

Part of the sample of the gastric contents was analyzed by gas chromatography of the fraction of vapor in equilibrium with the sample (headspace), which identified only the presence of ethyl alcohol, in a concentration of 7.01 g/L.

Since we did not have any lung tissue without fixative, a piece of lung preserved in formalin was extracted with organic solvents, and the solution was analyzed by gas chromatography/mass spectrometry; however, the different types of hydrocarbons present in petroleum were not identified as being present.

Case 2

Analysis of an aliquot fraction of blood did not reveal the presence of volatile hydrocarbons, but ethyl alcohol was found at a concentration of 0.32 g/L.

In a piece of the fragment of lung received, linear, branched, and cyclic saturated and unsaturated paraffin hydrocarbons, constituents of petroleum, were identified.

Case 3

An aliquot fraction of the blood sample was analyzed by gas chromatography (headspace method) to investigate the presence of volatile substances, such as solvents. Besides the characteristic components of gas oil, the presence of dichloromethane was detected and these substances were confirmed by gas chromatography/mass spectrometry.

Discussion and Conclusions

Ingestion is the most frequent form of exposure found in accidental poisoning by hydrocarbons [5]. In ingestion, hydrocarbons produce their toxic effects on several organs and systems, including the lung, CNS, gastrointestinal tract, liver, and heart. The lung is the most affected organ, and pneumonitis due to aspiration is the greatest cause of morbidity and mortality [6,7]. The CNS is affected, and it is unclear whether this is a direct effect or whether it occurs as a result of cerebral hypoxia due to chemically induced pneumonitis.

The toxicity of hydrocarbons is usually directly proportional to their volatility and inversely proportional to their viscosity [8] so that products with high viscosity have limited toxicity. Products with very low viscosity present a high risk of producing aspiration.

Materials such as petrol or kerosene contain a high percentage of aromatic hydrocarbons. These highly volatile solutions have low viscosity and surface tension and can easily and quickly spread over mucous surfaces. These properties permit the hydrocarbon to climb the lining of the esophagus and penetrate the trachea. Consequently, the risk of aspiration is very great [9].

Hydrocarbons are also gastric irritants and provoke vomiting, which favors the mechanism explained.

There are various studies which suggest that the pulmonary damage is a result of aspiration of the hydrocarbons through the trachea and not of the circulation of the blood. For example, studies in animals [7] have demonstrated that the administration of sublethal doses of hydrocarbons directly into the stomach, with subsequent binding of the esophagus to avoid contact with the trachea, produces little or no pulmonary damage. Another factor is the time required to produce pathological changes in the lung; in these cases, the time was short, which suggests aspiration rather than absorption.

In the three cases reported here, we found the following data. In the first case, as the sample of blood arrived spilt and the fragment of lung used for toxicological investigation had previously been fixed in formaldehyde solution, the results were negative. In the second case, we found that the blood sample analyzed was negative for petroleum hydrocarbons, while the GC/MS analysis of lung tissue was positive. In the third case, in contrast, the blood analysis by GC, verified by GC/MS, was positive, with findings of dichloromethane and components of gas oil. We must emphasize a fact indicated by the pathologist, which is that the blood had been extracted from the lung.

In the three cases, pulmonary aspiration was demonstrated by optical microscope. This, together with the fact that positive identification of the hydrocarbons that constitute petroleum was made in lung tissue and in blood from the lung, which leads us to believe that we have three cases of petroleum hydrocarbon poisoning. Furthermore, the hydro-

carbon entered the lungs by aspiration, and not by absorption from the blood, giving rise to lipid pneumonitis by aspiration [*10*], with consequent anoxemia and death.

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