CASE REPORT

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Fatal Hydrocarbon Lipoid Pneumonia and Pneumonitis Secondary to Automatic Transmission Fluid Ingestion

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ABSTRACT: This is the first reported case of an unusual exogeneous lipoid pneumonia with marked interstitial pneumonitis and fibrosis along with generalized diffuse omental oozing developing in a 14-month-old child following ingestion of automatic transmission fluid.

KEYWORDS: toxicology, pathology and biology, hydrocarbons, pneumonia

J. D. was a 14-month-old white boy who was received in transfer from Wesley Medical Center on June 17, 1991 where he had been initially evaluated and treated for ingestion of approximately 5 to 10 cc of automatic transmission fluid (ATF). Reportedly, on June 4, 1991, the boy's father was changing the transmission fluid and had placed it in a "ninja turtle cup" from which the child drank. Shortly after ingestion, the parents stated that the child had a "coughing and choking spell." They "pounded" on his back to try and help him to cough up the fluid but were minimally successful. Shortly thereafter, he was noted to have "some increase in respiratory rate and developed sleepiness with grunting." He was taken to a Hutchinson (Kansas) Hospital Emergency Room where he was admitted by his family physician.

He remained stable in the Hutchinson Hospital while on 2L of O_2 per mask until the morning of June 5, 1991 when he required 6L of O_2 per mask to keep his oxygen saturation up to 92%. His respiratory rate during the initial phase of hospitalization was in the 60s, but increased to 100. Initially he was afebrile, but developed temperatures ranging from 100 F to 104°F. He was treated with one dose of decadron while in Hutchinson and two doses of rocephin. At Wesley Medical Center a chest X-ray showed evidence of aspiration. He continued to do poorly while there and was eventually transferred to Arkansas Children's Hospital (ACH) for extracorporeal membraneous oxygenation (ECMO) therapy.

The following day at ACH, he developed two pneumothoraces that were treated by

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chest-tube placement. On June 29 he was noted to have a tense and distended abdomen. An abdominal X-ray showed persistent barium in the colon, but no evidence of free air. The following day, an exploratory laparotomy revealed generalized diffuse oozing with a large number of clots in the greater omentum and in the lesser sac. No obvious bleeding points were identified.

On July 2, 1991 lung and omental biopsies were performed, revealing an organized hematoma in the omentum and an exogenous lipoid pneumonia with marked interstitial pneumonitis and fibrosis in both upper and lower lobe wedge biopsies (Fig. 1). Electron microscopy confirmed the presence of an interstitial pneumonitis with fibrosis along with the presence of lipid/oil droplets in the increased numbers of alveolar macrophages. There was also an abnormal lysosomal accumulation of material present in alveolar and interstitial macrophages as well as in Type II pneumocytes, which was believed to represent the aspirated hydrocarbon material (Fig. 2). He continued to do poorly and was pronounced dead on July 8, 1991 at 3:08 P.M. following removal of life support.

Postmortem

The gross examination on the unembalmed body of the 14-month-old white boy was remarkable for a markedly distended abdomen; the abdominal circumference was 53 cm as contrasted with a head circumference of 49 cm, and a chest circumference of 48 cm. On opening the body, the thoracic fat pad measured 0.6 cm in thickness and the abdominal fat pad measured 0.8 cm.

Examination of the left chest cavity revealed multiple fibrinous adhesions and adherent clots along with approximately 10 cc of free blood within the left pleural space. Within the abdominal cavity there was approximately 50 cc of dark red blood with a 50 cc organized clot. The latter was adherent between the omentum and the peritoneum.

The heart and lungs were removed en bloc. The lungs were bilaterally heavy and firm. The combined lung weight was 300 gm. (Average combined weight 140 to 160 g.) They

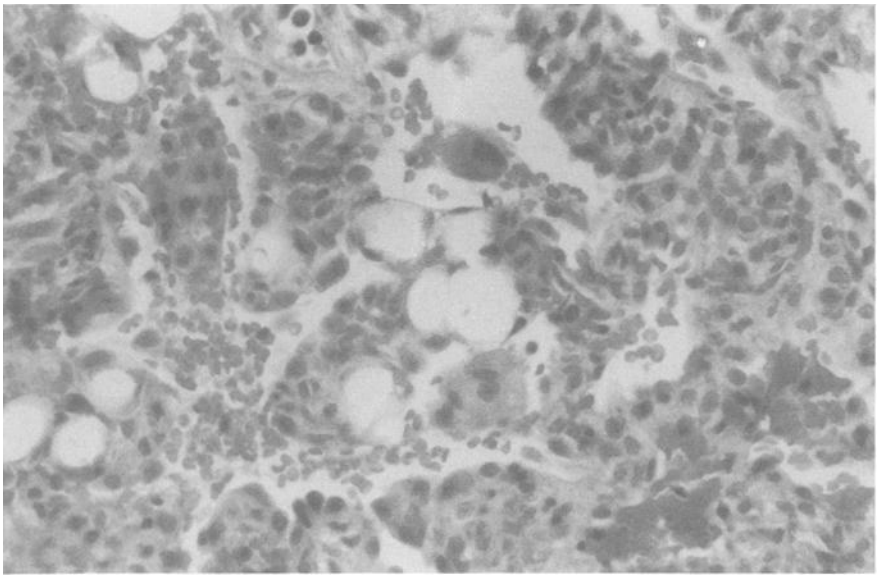

FIG. 1—*Microscopy of lung biopsy showing increased numbers of macrophages; some lipid-laden with giant cells in the alveolar spaces. The alveolar septums are thickened and contain predominately chronic inflammatory cells. (Plan 10 0.25)*

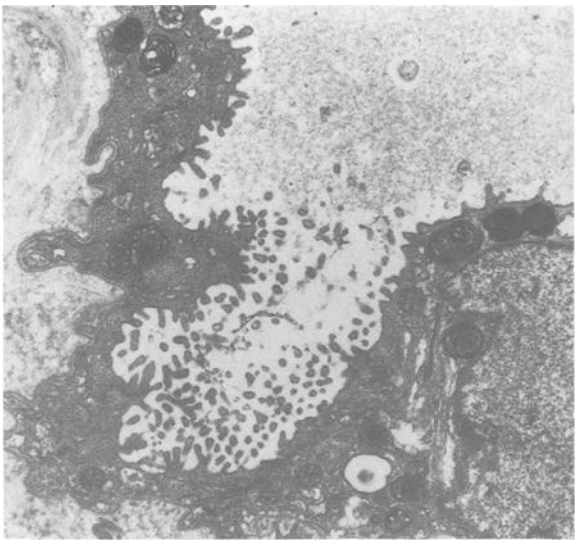

FIG. 2—*Electron microscopy on the lung biopsy confirmed the presence of lipoid droplets (cleared spaces) with the abnormal accumulation of material in Type II pneumocytes (dense whorled-appearing black material in lysomes), the latter is believed to be the aspirated hydrocarbon. (1.1 μm)*

were solid in appearance with absence of crepitance bilaterally. The parenchymal cut-surface was markedly congested and diffusely fibrotic.

Examination of the gastrointestinal tract showed scattered, various-sized area of subserosal hemorrhage involving both the small and large intestines and the omentum. Fresh hemorrhage was noted in the peripancreatic fat. These latter areas of recent hemorrhage were confirmed by microscopic examination. The sections from the lesser omentum revealed organizing fibrovascular tissue with adjacent layering fresh clot.

Microscopy of the lungs revealed patchy atelectasis with focal hyalin membrane formation, patchy focal areas of edema fluid or fresh hemorrhage, or both, in some alveolar spaces, marked septal thickening with acute and chronic inflammatory cells, the presence of lipoid/oil droplets with adjacent giant cells, hyperplastic type II pneumocytes, and an increase in the numbers of alveolar macrophages. A focal acute bronchitis and bronchiolitis with a developing adjacent early acute bronchopneumonia was also noted.

The remainder of the postmortem examination was essentially unremarkable.

Discussion

The ingestion of various hydrocarbons, in the form of petroleum distillates (including kerosene, gasoline, furniture polish, lighter fluid, mineral-seed oil, and turpentine), is a common pediatric emergency problem [*1*]. In the United States it accounts for about 5% of all calls reported to poison control centers.

Because these products are often attractively packaged and scented, it is not surprising that children under 3 years of age account for 50% of all patients admitted for hydrocarbon ingestion [*2*]. The medical literature concerning hydrocarbon ingestion is extensive; kerosene being the most frequent offender [*3–14*].

Petroleum distillates are crude oil by-products that range from benzene to lubricating oils. The exact content of each depends on the particular manufacturing process, which is specific for each product. In this case, we were unable to determine the brand of transmission fluid ingested.

The petroleum distillates physical properties, consisting of viscosity, surface tension, and volatility, determine the risk for aspiration and therefore, have the greatest effect on toxicity. Of these three, it is the viscosity, the tendency to resist flow or change form, which has been determined to provide the best estimate of the aspiration potential [*16*]. Distillates, such as kerosene, which have high volatility, decreased viscosity, and low surface tension are more likely to be aspirated and to subsequently result in severe respiratory injury such as a chemical pneumonitis. The lower viscosity appears to enhance penetration into the more distal airways; and the lower surface tension, which is the cohesiveness of the molecules on a liquid surface, facilitates spread over a large area of lung tissue [*11*].

In contrast, distillates, such as lubricating oils, which have low volatility, increased viscosity, and high surface tensions are less likely to produce severe injury [*15*] (for example, lipoid pneumonia is usually more localized and less inflammatory). But even though the process is more low-grade, the chronic granulomatous changes may produce a longer, more indulent course than the low-viscosity petroleum distillate pneumonias.

Differing incidences of various clinical manifestations have been reported by different authors, but the precise incidences are relatively unimportant. More important is the fact that respiratory symptoms and signs usually predominate.

Following petroleum-distillate ingestion most children who present to outpatient facilities are asymptomatic [*17*]. According to Anas, respiratory symptomatology if it develops, almost always begins within the first 6 h following ingestion, and children who are asymptomatic for 6 h are likely to remain so. Within 30 min of exposure symptoms of respiratory distress (choking and coughing) from aspiration usually but not always appear. Vomiting often precedes aspiration, however it is not a prerequisite [*15*]. Approximately 24 to 48 h postingestion is when the distress generally worsens, however, most patients recover by the third to eighth day [*18*].

Signs of distress, if any, can include varying degrees of cyanosis, nasal flaring, tachypnea, tachycardia, dyspnea, and retractions. Somnalence is the chief neurologic manifestation; coma and convulsions occurring infrequently. A moderate fever (38 to 39°C) is often observed on admission by three-fourths defervesce by 24 h postexposure [*17*]. The pathogenesis of the fever is unknown but is believed to be central in origin. If the fever persists 48 to 72 h postingestion, a bacterial superinfection should be suspected.

Radiographic abnormalities of the lungs usually appear within 30 min of exposure. The changes tend to reach a maximum of 72 h after exposure, but usually clear several days later. Occasionally, the changes persist for several months [*19*]. The chest X-ray abnormalities also tend to correlate poorly with the clinical symptoms [*2*].

Pathologically, again depending primarily upon the petroleum-distillate viscosity, microscopic changes may range from those seen in a chemical pneumonitis to those seen in a lipoid pneumonia. The pathology of the atelectasis is in part a consequence of the increase in surface tension resulting from the interaction of the hydrocarbon with pulmonary surfactant. Sometimes fibrinoid or hyalinlike membranes similar to those found in neonatal respiratory distress syndrome are observed [*19*]. Bacterial pneumonias, again, are infrequent, but can supervene in a protracted course.

Transmission fluid is a lubricating oil, composed primarily of mineral oil (75 to 80%, depending on grade); a polymer (~5%), which is used to increase the viscosity, and an antitoxidant.[3] Again, as with all distillates the manufacturing process is specific for each product. The exact brand of transmission fluid in our case is unknown; however the clinical history and the pathologic changes in the lungs support that J.D. aspirated a significant quantity.

[3]Personal communication, Chevron and Rocky Mountain Petroleum distillate research and testing departments.

Of interest is the fact that mineral or olive oil was previously administered in order to thicken the hydrocarbon and increase its viscosity. It was hoped that with substances like kerosene that this would decrease the risk of aspiration; however, the administration of such oils, according to Beamon et al. resulted in an increased incidence of pneumonitis instead [*20*]. Our findings consisted of a lipoid pneumonia and a marked interstitial pneumonitis with fibrosis.

Gastrointestinal symptoms are frequent but usually minor in nature, as are gastrointestinal findings, even though it is thought that some absorption from the gastrointestinal tract into the systemic circulation ensues. Because hydrocarbons are highly hydrophobic we hypothesize in our case that some of the ingested material could have been stored in vascular cell membranes or neutral body fat (omental fat), which eventually led to the disruption of the vascular lining, and thus resulted in the slow omental oozing that we found in the abdominal cavity.

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