patients with similar coronary arterial disease without aneurysm.11

In our case, rupture had occurred, with formation of a false aneurysm. Surgical intervention was believed to be imperative to prevent rupture into the pericardium. To our knowledge, this is the first reported case of a spontaneous rupture of a coronary artery, with formation of a false aneurysm and with successful surgical repair. The etiology is believed to be atherosclerosis. Whether there was a limited dissection prior to rupture cannot be ascertained. The aneurysm was not excised, and the exact mechanism of rupture is not known. With the advent of coronary angiography, antemortem recognition of this problem is possible and will lead to further clarification of the natural history and prognosis.

References
1 Morgagni JB: De Sedibus et Causis Morborum (tome I, liber 11, epistle 27, article 28). Venetus, 1761
2 Bougon: Bibli Med 37:183, 1812
3 Griffith TW: Remarks on aneurysm of the coronary arteries of the heart. Br Med J 00:268-269, 1901

Respiratory Tract Burns after Aspiration of Hot Coffee

Ralph C. Jung, M.D., F.C.C.P.,* and Leon S. Gottlieb, M.D., F.C.C.P.**

We present the findings in a patient with acute thermal burn to the upper and lower airway who developed mucosal edema followed by patchy areas of granuloma-like lesions in the trachea and bronchi. A four-month follow-up showed resolution of the gross lesions, but functional alterations remained. This patient illustrates the necessity for repeated direct observation and functional evaluation of the lower airway following thermal injury, which can be a life-threatening disorder.

Injury to the respiratory tract due to inhalation has been extensively documented.1-3 Commonly, the insulting agent is either smoke and its noxious products or chemical gases. The pulmonary complications following the aspiration of gastric contents and liquid chemical agents have also been extensively reported.4 Direct injury to the airway and lung following aspiration of thermal liquid agents and, particularly, the consequences thereof are not well recorded. We are reporting the case of a patient who received a direct burn to the upper and lower airway from the aspiration of hot coffee. Serial direct visualization of the airway via bronchoscopic examination and follow-up studies of pulmonary function to detect residual effects on the respiratory tract are presented to emphasize that direct thermal burn to the lower airway can occur in appropriate circumstances.

Case Report

The patient is a 28-year-old black woman who entered the medical center on Feb 7, 1975, after having ingested an indeterminate amount of meprobamate, barbiturates, and other medications following a spell of mental depression. She was found in a semicomatose state by a relative early on the day of admission. An attempt was made to induce vomiting by offering vinegar mixed with mustard followed by milk. The patient refused the “cocktail.” Next, hot coffee was poured into the patient’s mouth. When all of these attempts failed to revive the patient, she was rushed to a local hospital.

On initial examination, the patient’s mouth, lips, and tongue were markedly edematous and erythematous, requiring

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Respiratory Tract Burns after Aspiration of Hot Coffee

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Chest, 72:1, July, 1977
Table 1—Data on Arterial Blood Gas Levels and Ventilation

<table>
<thead>
<tr>
<th>Date and Time</th>
<th>pH</th>
<th>PaCO₂</th>
<th>Bicarbonate, mm Hg</th>
<th>Base</th>
<th>PaO₂</th>
<th>Oxygen Saturation</th>
<th>Arterial</th>
<th>Percent Oxygen Content, ml/100 ml</th>
<th>Systemic Compliance, cm H₂O</th>
<th>PEEP, cm H₂O</th>
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<tr>
<td>2/7, 5:30 PM</td>
<td>7.42</td>
<td>28</td>
<td>18</td>
<td>-6</td>
<td>0.2</td>
<td>58</td>
<td>91</td>
<td>15.9</td>
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<td>...</td>
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<td>26</td>
<td>17</td>
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<td>98</td>
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<td>16</td>
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<td>494</td>
<td>100</td>
<td>17.5</td>
<td>22</td>
<td>10</td>
</tr>
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<td>24</td>
<td>1</td>
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<td>98</td>
<td>12.7</td>
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<td>10</td>
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<td>12.6</td>
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<td>97</td>
<td>12.6</td>
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<td>70</td>
<td>95</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

*Base excess of extracellular fluid. **FrO₂, Fraction of oxygen in inspired air. †Compliance of ventilation system. ‡Off ventilator.

an oropharyngeal airway. After instituting appropriate measures to support her respiration, the patient was then transferred to the Los Angeles County/University of Southern California Medical Center. Prior to transfer, her arterial blood gas levels showed an arterial oxygen tension (PaO₂) of 62 mm Hg, an arterial carbon dioxide (PaCO₂) of 50 mm Hg, pH of 7.25, and a bicarbonate level of 21 mEq/L. On arrival, the patient was in grade 2 coma, with a pulse rate of 112 beats per minute, temperature of 37.0°C (98.6°F), respiratory rate of 38/min, and blood pressure of 130/80 mm Hg. There were full-thickness burns on the oropharynx, tongue, and labial mucosa. Some areas were markedly white and beginning to slough. Serosanguinous fluid was present in the mouth. Rhonchi and wheezes were heard throughout both pulmonary fields, with accentuated respiratory excursions. Because of the marked swelling of the oropharynx, hypopharynx, and uvula, an immediate tracheostomy was performed. The initial chest x-ray film showed essentially clear pulmonary fields.

By the fourth hour after admission, diffuse moist rales and rhonchi were noted throughout both pulmonary fields. Initial and subsequent determinations of arterial blood gas levels are shown in Table 1. The tracheal aspirate contained many white blood cells but no organisms. The patient was given therapy with supplemental oxygen, methylprednisolone (30 mg/kg intravenously every eight hours), and intravenous administration of penicillin. Her oral temperature rose to 37.8°C (100°F). Later the same evening, the chest x-ray film showed diffuse bilateral interstitial infiltrates (Fig 1).

Because of deteriorating levels of arterial blood gases (Table 1), the patient was placed on a volume ventilator with 60 percent oxygen, and positive end-expiratory pressure (PEEP) of 10 cm H₂O, and the pulmonary artery was cannulated. The pulmonary arterial pressure was 15/9 mm Hg, with a mean of 11 mm Hg and a wedge pressure of 8 mm Hg. The inspired oxygen concentration was eventually decreased to 30 percent. The other settings on the ventilator remained unchanged. Because of persistent wheezes, the patient continued to receive a continuous intravenous infusion of aminophylline. She showed slow, progressive improvement in ventilation, despite the development of staphylococcal pneumonia requiring intravenous therapy with methicillin.

On the fourth day of hospitalization, a flexible fiberoptic bronchoscope passed through the tracheostomy tube showed that the mucosa of the trachea and both bronchi were hemorrhagic and denuded (Fig 2). The right bronchus was edematous and showed multiple superficial mucosal ulcerations. The carinal spur was sharp, but the base appeared to be thickened. Scattered superficial mucosal ulcerations were seen bilaterally to the level of the segmental bronchial spurs. In all lobar orifices of the right lung, mucosal edema was also prominent. There were minimal secretions.

Bronchoscopic examination on the 12th day of hospitalization showed considerable healing of all areas; however, small granuloma-like lesions were now visible on the bronchial wall of both main bronchi (Fig 3). By this time, there was significant improvement in ventilation. The patient left the hospital on the 17th day after the burn.

Repeat bronchoscopic examination performed one month later failed to demonstrate any abnormality beyond slight blunting of the carina. Studies of pulmonary function performed four months after the injury showed a generalized depression of the forced expiratory volume in one second (FEV₁) of 80 percent. The patient was continued on a tracheostomy, with feeds given by means of a nasogastric tube. She was discharged home on the 70th day after the injury.

Figure 1. Chest x-ray film approximately eight hours after admission, showing extensive bilateral interstitial-alveolar radiodensities.

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Figure 2. Bronchosopic view of right main bronchus four days after injury, showing extensive areas of erythema and edema with isolated ulcerated areas.

decrease in the expiratory flow rates and lung volumes (Table 2). The arterial blood gas levels were normal. The patient had no symptomatic evidence of any residual damage. Her lungs were clear, and the oropharynx was essentially healed. The wound from the tracheostomy had healed. The patient failed to return for further follow-up care.

Discussion

Observations on this patient's clinical course present, we believe, the first direct evidence of not only the morphologic characteristics of an acute contact burn with hot liquid but also are indicative of the natural history of such lesions occurring below the vocal cords, namely, that residual long-term effects, such as mucosal scarring and formation of granulomas in the tracheobronchial tree, as well as parenchymal damage, are mild, though likely, consequences of this type of injury.

Injuries due to inhalation have usually been reported to produce damage to the respiratory tract from either particulate matter or the chemical components of smoke.

Table 2—Pulmonary Function Studies Four Months after Injury

<table>
<thead>
<tr>
<th>Factor*</th>
<th>Determined</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, L</td>
<td>2.7</td>
<td>3.7-4.2</td>
</tr>
<tr>
<td>Maximal expiratory flow, L/min</td>
<td>259</td>
<td>278-348</td>
</tr>
<tr>
<td>FEF25-75%, L/min</td>
<td>150</td>
<td>176-224</td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>2.2</td>
<td>2.9-3.3</td>
</tr>
<tr>
<td>FEV₁/FVC%</td>
<td>82</td>
<td>73-80</td>
</tr>
<tr>
<td>Maximal inspiratory flow, L/min</td>
<td>317</td>
<td>174-244</td>
</tr>
<tr>
<td>Maximal breathing capacity, L/min</td>
<td>122</td>
<td>104-127</td>
</tr>
<tr>
<td>TLC, L</td>
<td>4.3</td>
<td>5.0-5.7</td>
</tr>
<tr>
<td>FRC, L</td>
<td>2.4</td>
<td>2.7-3.1</td>
</tr>
<tr>
<td>RV, L</td>
<td>1.5</td>
<td>1.4-1.8</td>
</tr>
<tr>
<td>RV/TLC%</td>
<td>33</td>
<td>28-31</td>
</tr>
</tbody>
</table>

*FVC, Forced vital capacity; FEF25-75%, mean forced expiratory flow during middle half of FVC; FEV₁, forced expiratory volume in one second; TLC, total lung capacity; FRC, functional residual capacity; and RV, residual volume.

Aside from supraglottic or hypopharyngeal lesions, such as edema, as the result of heat and chemical smoke, there have been no observations reported concerning the consequences of direct contact of the mucosal surfaces of the upper and lower airways with hot liquids. We had the opportunity to observe and follow the course of this patient, who is the second of two patients in whom direct damage to the upper and lower airways has resulted from contact burns from a lethal liquid.

The fact that, in this patient, aspiration of hot coffee also led to damage to the airway distal to the trachea indicates that one cannot assume that aspiration of hot liquid substances will produce severe damage only above the vocal cords. Not only is repeated examination of the upper airway mandatory when this occurs, but it is also necessary to evaluate the extent of damage more distally. Some of the aspirated material undoubtedly was deposited beyond the segmental bronchi, since direct mucosal injury was observed at the segmental orifices through the bronchoscope. These observations would tend to contradict previous observations on the effect of thermal injury due to inhalation. It has been shown experimentally that inhalation of hot steam or gas is most unlikely to produce injury below the trachea due to rapid cooling of the material; however, as this case illustrates, the laryngeal and tracheal cooling process can be overwhelmed by hot liquid.

This case also served to reemphasize the importance of repeated arterial blood gas analyses during the early stages of this disorder, not only to determine the progression of damage to the respiratory tract, but also to guide therapy. With the analysis of arterial blood gas levels, it is our practice, in these patients, to also calculate the alveolar-arterial oxygen pressure difference which we use as a sensitive indicator of worsening airway constriction or parenchymal congestion (or both), which this patient developed, as illustrated by the chest x-ray film. Guided by this calculation, early in the course of our patient's illness, we applied PEEP, which aided the rapid reversal of her shunt, as can be seen in Table 2.

Since therapy with corticosteroids has been considered useful in aspiration pneumonia, we administered...
high doses of this agent to the patient. Whether or not this contributed as much to her rapid progressive recovery as did the other measures instituted is uncertain. The anti-inflammatory action of steroids is common knowledge; therefore, it is conceivable that they were useful in assisting the recovery of this patient.

Of ultimate concern to patients who have suffered injury due to inhalation is the long-term effect on the tracheobronchial tree. By bronchoscopic observation one month after the injury, this patient's respiratory tract was remarkably clear; however, somewhat more significant were the results of the pulmonary function studies at four months, which showed loss of lung volumes and reduced expiratory flow rates, particularly the rather large reduction in the maximal mid-expiratory flow rate, a possible indicator of disease of the small airways. Therefore, although no gross abnormalities were visible, the patient still demonstrated impairment of pulmonary function. The patient gave no history of prior pulmonary disease and was not a smoker; therefore, presumably she had no preexisting chronic pulmonary disease.

Unfortunately, since the patient failed to return for further care, it was not possible to determine how permanent these functional changes were. It would have been most desirable to determine the severity and reversibility of her upper-airway obstruction during her immediate recovery and convalescent phases by repeated determinations of the flow-volume loop. Since the patient developed the other functional pulmonary stigmata of the adult respiratory distress syndrome, it is conceivable that she might have developed some irreversible parenchymal damage, as has been reported with this disorder. It is important to stress that our patient is an example of the type of patient who should receive long-term observation of pulmonary function so that should residual damage remain, this would be documented.

REFERENCES


ANNOUNCEMENTS

15th International Annual Meeting, Society of Nuclear Medicine

The 1977 meeting of the Society of Nuclear Medicine will be held in Groningen, the Netherlands, September 13-16. For information, contact the Organizing Committee, PO Box 290, Groningen.

Annual Otolaryngologic Assembly

The Annual Otolaryngologic Assembly of 1977 will be held September 10-16 in the Eye and Ear Infirmary of the University of Illinois Hospital, Chicago. Co-chairs are Drs. Emanuel M. Skolnik and Burton J. Soboroff. For information, contact: Otolaryngology, 1855 West Taylor Street, Chicago 60612.

Workshop in Echocardiography

Dr. Louis E. Teichholz will direct the Workshop in Echocardiography, September 1-4 at the Pine Isle Resort Hotel at Lake Lanier Islands, Buford, Georgia (40 minutes north of Atlanta). For information, contact: Ms. Billie N. Chiles, Tampa Tracings, PO Box 1245, Tarpon Springs, Florida 33590.

Fourth Annual Seminar, Topics in Pulmonary Disease

Colby College/Mid-Maine Medical Center, Waterville, Maine will sponsor the Fourth Annual Seminar in Pulmonary Disease August 21-25 at the College.

Direct inquiries to Mr. R. H. Kany, Director, Special Programs, Colby College, Waterville, Maine 04901.