period of months. Diffusing capacity was not measured by these investigators. Although our patient did not have extensive surface burns, he did have a severe reduction in carbon monoxide diffusing capacity. It is possible that he did have subclinical noncardiogenic pulmonary edema which resolved over a period of months. His initial chest roentgenograms are consistent with this hypothesis. In this regard, abnormalities of gas exchange have been noted to persist for months following episodes of clinical adult respiratory distress.\textsuperscript{9} It is possible that with the application of more sensitive tests such as DCO, abnormalities of gas exchange would be noted with greater frequency following smoke inhalation.

The etiology of the endobronchial polyps is probably the impaction of superheated particles at mucosal sites. It is possible that their development is similar to that of the bronchiolitis obliterans reported following exposure to fumes\textsuperscript{29,30} where granulation tissue plugs form within the lumen of small airways often extending into the alveolar ducts. The prevalence of endobronchial polyps following smoke inhalation is unknown. Since these polyps appear to be a late development and since patients' airways are generally only studied acutely after smoke inhalation, we suspect that tracheobronchial polyps may be a relatively common sequela of smoke inhalation. Their potential hazards would be hemorrhage, or obstruction of a major bronchus by larger polyps. However, in a limited experience, they would appear to be a benign, delayed complication of smoke inhalation which spontaneously resolve without specific treatment presumably by compromise of their vascular supply with sloughing of the polyp.

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Small Cell Lung Carcinoma and Bowen's Disease 40 Years after Arsenic Ingestion\textsuperscript{*}

R. Heddle, M.B.B.S.; and G. D. Bryant, M.B.\textsuperscript{†}

A non-smoking woman presented with ectopic ACTH syndrome associated with disseminated small cell carcinoma of lung. The patient had Bowen's disease and had taken oral arsenic for psoriasis 40 years ago. It is postulated that the previous therapeutic arsenic ingestion caused both her arsenical dermatosis and her small cell carcinoma of lung.

Carcinoma of the lung is most frequently attributed to cigarette smoking, but many other etiologic agents, including arsenic, have been implicated. We describe a patient who presented with ectopic ACTH syndrome associated with disseminated small cell carcinoma of the lung.

CASE REPORT

A 72-year-old woman presented with abdominal distension, constipation and recent onset of peripheral edema. Physical examination revealed hepatomegaly, ascites, peripheral edema and multiple scaly red plaques on the back and legs. Biopsy of these plaques showed Bowen's disease and basal cell carcinoma.

Chest x-ray examination showed a left lower lobe mass and hilar enlargement. At bronchoscopy, there was abnormal mucosa in the left main bronchus and brushings gave results positive for small cell carcinoma (Fig 1). Serum electrolytes were Na\textsuperscript{+}, 144 mmol/L; K\textsuperscript{+}, 4.4 mmol/L; Cl, 91 mmol/L; and HCO\textsubscript{3} greater than 40 mmol/L. The plasma cortisol level was greater than 1,600 nmol/L (reference range 760-950 nmol/L) 12 hours after 1 mg oral dexamethasone. A radionucleotide liver-spleen scan showed multiple hepatic defects suggestive of metastatic deposits. Fine needle aspiration of these yielded small cell carcinoma (Fig 2). Bone marrow biopsy also showed carcinoma. The patient's edema worsened and she developed severe hyperglycemia requiring therapy with insulin. Hyperkalemia persisted despite 300 mg per day of spironolactone and aldosterone.

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Figure 1. Bronchial brush smear. Upper half-normal bronchial cells; lower half-small cell undifferentiated carcinoma cells. Alcohol fixed Papanicolaou stain, original magnification \times 300.
Lymphomatoid Granulomatosis with Impaired Cellular Immunity*
Eight Year Survival without Treatment

Edith Firstater, M.D.; Israel Yust, M.D.; Marcel Topilsky, M.D.; Boris Tartakovsky, M.Sc.; Sriaga Segal, Ph.D.; and Ari Abramov, M.D.

Lymphomatoid granulomatosis (LYG), a non-neoplastic lymphoreticular disorder, was diagnosed in a 65-year-old woman. Chest radiographs demonstrated bilateral lower lobe nodular infiltrates. Percutaneous needle biopsy of the lung showed an infiltrate composed of plasma cells, lymphocytes and large histiocytic-like cells. Impairment of cellular immunity was found by in vivo as well as by in vitro tests. The clinical condition of the patient has remained stable for the last eight years without specific treatment.

In 1972 Liebow et al. described a number of patients with a condition they called lymphomatoid granulomatosis (LYG) of the lung. Since then, other authors have described patients with this entity which is characterized by vasculitis with lymphoreticular infiltrate. Patients with this disease usually seek medical attention because of cough, excess sputum production, fever, dyspnea and weight loss. The pulmonary lesions tend to be in the lower lung fields, may be cavitary, and are generally unassociated with hilar lymphadenopathy. While most of the cases described in the literature have been treated with steroids alone or in combination with cytotoxic drugs, the efficacy of this treatment has not been established.

We report a patient with LYG with impaired cell-mediated immunity. The patient has remained stable without specific treatment for the last eight years.

**CASE REPORT**

A 65-year-old woman was first admitted in 1975 with complaints of nausea, high fever, productive cough, breathlessness and chest pain. Her temperature was 40°C, the pulse rate was 120 beats per minute, and there were fine crepitant rales on both lung bases. Laboratory investigations at this time showed: Hb, 10.0 g/dl; WBC, 9,200 with a shift to the left; ESR, 80/110; albumin, 3.0 g; and globulin, 3.3 g; latex fixation test, 1:2,500-1:5,000; Rose Waaler, 1:64-1:128. Urine and blood cultures showed *E. coli* and sputum cultures grew *Klebsiella pneumoniae* and *Streptococcus viridans*.

Chest x-ray films taken on admission revealed the presence of multiple round-shaped lesions in the left lower lobe and in the right middle and lower lobes (Fig. 1). Radiograms of the sinuses, hand and spine were normal. Results of anti-DNA, ANF, LE cells, cryoglobulins, cold agglutinins, Weinberg-Casoni-Coombs tests, and bone marrow aspiration were all negative. She was treated with gentamicin (Garamycin) and cephalaxin (Reflax) and her condition improved gradually. A few weeks later she was readmitted with septic fever and this time sputum and blood cultures grew *Diplococcus pneumoniae*. She was again started on treatment with antibiotics and the fever gradually abated. The findings of gross nodular lesions in both lung fields remained unchanged throughout both hospital courses. At this time, immunologic evaluation was performed.

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**FIGURE 2.** Fine needle aspirate smear of liver. Upper right corner—normal hepatocytes; lower left—malignant cells. Air dried May-Grünwald-Giemsa stain, original magnification ×300.

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potassium supplements.

Following commencement of vincristine 1 mg/m², cyclophosphamide 1 g/m², and adriamycin 50 mg/m², six cycles at 21-day intervals, the edema and metabolic disturbances disappeared and the chest x-ray film showed clear. Twenty-one months later, the patient remains active and well on maintenance chemotherapy.

**DISCUSSION**

As the patient's husband smoked, we considered the possibility of passive smoking-induced lung carcinoma. However, the presence of Bowen's disease suggested the possibility of previous arsenic ingestion. After repeated questioning, the patient recalled taking (in the 1940s) arsenic solution daily for five years for psoriasis. We could not identify any other etiologic factor.

Robson and Jelliffe and Goldman reported eight patients with lung cancer and arsenic dermatosis (manifested by keratoses, basal cell carcinoma, Bowen's disease or pigmentation) together with a history of therapeutic arsenic ingestion decades before. In seven of these cases, arsenic had been given as a solution either as a tonic or for psoriasis. None of these cases was recognized as small cell carcinomas. Studies on smelter workers exposed to arsenic suggest a significant increase in the incidence of carcinoma of the lung, including small cell type.

We believe this is the first case report of small cell carcinoma of the lung associated with a past history of therapeutic arsenic ingestion and suggest that it may be helpful to enquire about previous arsenic treatment in nonsmokers presenting with carcinoma of the lung.

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