Communications to the Editor

Pulmonary Lipid Peroxidation in Cigarette Smokers and Lung Cancer Patients

To the Editor:

The article by Petruzelli and colleagues, which appeared in the October 1990 issue of Chest, was of particular interest to us. While working with various tissue homogenates in sucrose solution (0.25 mol/L) for the assay of malondialdehyde (MDA), we noted a yellow-orange color development upon heating with thiobarbituric acid (TBA) in boiling water. A literature search revealed that sucrose homogenates are unsuitable for estimation of TBA-reactive material. Subsequently, other common carbohydrates in strong solution (0.25 mol/L) were found to interfere with the assay; of these, sucrose showed maximal interference. The yellow-orange color formation is proportional to the concentration of the sucrose in the assay medium. The absorbance in the presence of sucrose was four to five times higher than that in its absence. The great simplicity of the method has encouraged its wide use in investigations of lipid peroxidation, and in our experience interference by sucrose cannot be ignored.

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To the Editor:

In response to the communication from Anand and colleagues, we offer the following comments: When we used sucrose for lung tissue preparation, a blank with sucrose was used for reading the absorbance at λ = 535 nm, so that the MDA levels reported in our article are true values. It can be assumed that the real differences would have been much larger if sucrose had interfered. However, since 1987, we have measured MDA concentration in lung tissue cytosol (not in S-12 preparations) by using 0.15 mol/L of KCl (pH 7.5) instead of sucrose. From the available results of this ongoing study, we have confirmed a twofold higher level of MDA in lung tissue of recent smokers as compared to ex-smokers. This difference was apparent in patients with squamous cell carcinoma and was less pronounced in patients with adenocarcinoma. Therefore, although the criticism that sucrose may interfere with lipid peroxidation is valid, a second round of studies has shown that the effect reported is correct (ie, that recent smokers have a higher level of MDA in lung tissue cytosol that was prepared in the absence of sucrose).

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Chylothorax in Familial Lymphedema of the Meige Type

To the Editor:

Hereditary lymphedema (HL), or Nonne-Milroy-Meige disease, is a primary form of lymphatic edema, usually localized in the lower limbs bilaterally. The disorder is caused by inadequate lymphatic drainage due to insufficient development of the lymphatic vessels. It is a hereditary condition with autosomal dominant transmission. Hereditary lymphedema can be classified on the basis of the patient's age at appearance of the disease into three types: (1) congenital or infantile HL, which appears at birth or immediately afterward (also known as Nonne-Milroy disease); (2) lymphedema praecox, or juvenile HL, which appears at puberty (also called Meige disease) or, more commonly, before the age of 35; and (3) HL tarda, the rarest form, which appears after the third decade of life. Its association with chylothorax has been rarely reported in the literature. We present a familial case (brother and sister) of HL, localized in the left lower extremity, which was associated with chylothorax.

A 24-year-old man was hospitalized in January 1988 because of a shooting pain at the left base of the thorax, fever, dry cough, and severe dyspnea. Clinical examination revealed the presence of painless opacification of the left lower extremity up to the fold of the buttock. The skin was white and tough. Edema had set in at the age of 8 years (Fig 1). Chest x-ray examination revealed homogeneous opacification of the left hemithorax except for the apical zone.

A specimen obtained by pleural puncture was a yellowish, nonodorous liquid, which, when centrifuged, did not become clear (empyema was excluded). Laboratory studies disclosed the following values: cholesterol, 110 mg/dl; triglycerides, 430 mg/dl; lymphocytes, 100 percent (pseudochylothorax was excluded).
right costodiaphragmatic sinus and opacification of the right horizontal fissure. A specimen obtained by means of thoracentesis was a moderately lactescent liquid; the supernatant did not clarify when centrifuged. Laboratory examination revealed the following values: lymphocytes, 100 percent; cholesterol, 93 mg/dl; triglycerides, 412 mg/dl. After evacuation, the liquid reappeared. Clinical examination did not show changes in the nervous system. The patient had normal height and secondary sexual characteristics, as well as positive sex chromatin. Like her brother, the patient has no other congenital anomalies (eg, craniofacial or visceral abnormalities or yellow nails), although they were assiduously sought.

Family anamnesis showed that the parents (aged 43 and 41) are not kindred, are in good health, have no lymphedema and no pulmonary complaints in their antecedents, and have no other children. No cases similar to those of the patients described were reported in the family among more distant relatives, nor were there any other congenital anomalies or genetic diseases. Paternity tests and confidential anamnesis allowed rejection of the hypothesis that the father was not in fact the biologic father of the patients.

The peculiarity of these cases resides in the unilaterality of the lymphedema, the peripubertal spontaneous occurrence of the condition, the association with chylothorax, and the familial character of the disease, which, however, did not manifest itself in the parents, a fact that contravenes the dominant transmission observed in other studies.8 Two hypotheses are possible: (1) The parents may have crude, oligosymptomatic forms of the disease or may have undiscovered congenital anomalies not associated with lymphedema. (2) The transmission of the disease is autosomal recessive, which could be a novelty in the literature.

![Figure 1. Tense swelling of the left lower limb, with trophic ulcerations and local globular swellings.](image1)

![Figure 2. Circumferential edema of the left shank and foot, which was irreducible by compression or elevation.](image2)

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Myxedematous Pleural Effusion

To the Editor:

We would like to comment on myxedematous pleural effusion because our recent experience was somewhat different from that described by Gottehrer et al in the November 1990 issue of Chest. They stated that “patients with hypothyroid pleural effusions are asymptomatic” and that the “small, noninflammatory effusions” are “of minor clinical importance.” On the contrary, however, our patient had bilateral, massive, refractory pleural effusions and several episodes of apnea in spite of various kinds of treatment. Definitive diagnosis was delayed until six months after admission. All of his clinical problems, such as the refractory pleural effusion, respiratory embarrassment, and even the hearing impairment, disappeared after thyroid hormone replacement. In addition, the predominant cells in the pleural fluid were neutrophils in the beginning but subsequently became lymphocytes (Table 1). Furthermore, the positive periodic acid-Schiff stain of the pleural effusion cytologic specimen and the high protein content of the pleural fluid in this patient support the thesis of extravasation of the