Fine Needle Biopsy of Tuberculomas

To the Editor:

Most tuberculomas present as noncharacteristic lesions indistinguishable from other granulomas,1 bronchogenic carcinoma,2 or solitary metastasis.3,4 If these lesions can be diagnosed preoperatively, thoracotomy may be avoided and antituberculosis chemotherapy may be applied. Operations, if necessary, may be limited to local resections instead of lobectomy or pneumonectomy.

Radiography and other modern imaging methods (CT) have, with few exceptions (certain patterns of calcification), not been shown to be reliable enough for an accurate diagnosis without cytologic or histologic diagnosis it may even be hazardous and lead to unnecessary operations, or where malignancy is overlooked, to nonresectability, inoperability and finally to incurability. Fine needle biopsy (FNB) has proved to be a simple, safe, fast, accurate, inexpensive and minor procedure, which often can be performed on an outpatient basis, if an appropriate technique is used.5 Other techniques (transbronchial biopsy, bronchial brushing) may also be used, but may be less effective, more complicated, time-consuming and have the disadvantage of possible contamination; they may therefore give misleading results on smears and cultures.

Our clinical material consists of 2,796 patients for whom 5,300 FNBs were performed, resulting in the diagnosis of 1,664 malignancies. In 613 cases, inflammatory lesions were found (229 nonspecific and 303 specific, eg, tuberculous, fungal, etc); in 46 cases abscesses or empyemas of different origin3,4 and in 30 cases other inflammatory lesions were diagnosed. There were 107 tuberculosis. Cases of tuberculosis coexistent with malignancy4 were classified as carcinoma, even if their radiologic appearance and cytologic findings initially inferred the presence of a tuberculosis. In approximately 10 percent of the cases with tuberculosis, tubercle bacilli were found on direct smears and in another 10 percent tubercle bacilli were diagnosed on cultures. In 15 cases (total 202 granulomas) mycetomas (aspergillomas, histoplasmosomas) were found.13 Other granulomas (17 out of 202) were: Wegener’s granuloma, three; histiocytoma, three; plasma cell, four; eosinophil two. False positives were discovered in 1-2 percent. False negatives in earlier series were as high as 15 percent; with strict requirements, such as three negative samples from representative material obtained from different sites of the lesion, they decreased to 5-10 percent and were under optimal conditions in some series 3-4 percent. Complications consisted in the occurrence of pneumothorax in 27 percent (for three puncture events at the same session). However, only 7.7 percent had to be treated (½ by chest tube and ½ by exsufflation of air with fine caliber needle). In 11 percent there was local bleeding at the puncture site of the lesion, and in 2-5 percent (peripheral, two; central, 5 percent) hemothysis occurred; none of the latter two had to be treated. There was no fatality in this series.

W. N. Sinner, M.D., Karolinska Hospital, Stockholm, Sweden

REFERENCES
1 Sinner WN. Needle biopsy of histoplasmosis Fortschr Röntgenstr 1980; 133:578
2 Sinner WN. Fine needle biopsy of tuberculosis coexistent with carcinoma of the lung. ROEFO 1983; 139:173
3 Sinner WN. Fine needle biopsy of solitary pulmonary metastasis. Europ J Radiol (in press)
4 Sinner WN. Fine needle biopsy of pulmonary infectious disease. (to be published)

Hypokalemic Myocytolysis and Rhythm Disturbances

To the Editor:

The arrhythmogenic effects of hypokalemia (Hpk) are well known. However, cardiac arrhythmias are attributed mostly to premyocytolytic levels of Hpk. A few reports exist about myocardial damage due to Hpk.14

I have observed three patients with rhabdomyolysis (tetraplegia) and cardiac myocytolysis (CM) associated with severe Hpk (<2 mEq/L) secondary to furosemide excess, chlordihalidine therapy and familial hypokalemic periodic paralysis, respectively. Cardiac myocytolysis was documented by the elevation of MB fraction of creatine-kinase (CKMB>9 percent), a positive pyrophosphate heart scan (diffuse captation) and a reduction of left ventricular ejection fraction (LVEF<50 percent) detected by 2D echo. In all three patients, 96 h Holter monitoring was obtained which disclosed sinus bradycardia (<50 beats/min), but failed to show any tachyarythmia. After five days of KCl infusion (80 mEq/die) and oral aldactone (200 mg/die) together with a normalization of serum K+, there was an increase in sinus rate (>60 beats/min), good recovery of skeletal muscle function, and normalization of LVEF (>65 percent).

My impression is that hypokalemic CM is a reversible (within certain limits) process, responsible for hypokinetic rather than hyperkinetic arrhythmias. However, these patients suffered no other heart diseases except an electrolyte imbalance.

Further studies are necessary to confirm this aspect in cardiac patients.

Andrea Frustaci, M.D., Research Fellow, Cardiology Department, Catholic University of the Sacred Heart, Rome, Italy

Reprint requests: Dr. Frustaci, Istituto Cardiologia, Universita Cattolica S. Cuore, 00168 Rome, Italy

REFERENCES