before.” Of course, Dr. Pecora’s work could have been additionally referenced at that stage of our paper and we regret this oversight. However, the previous publications were concerned with lung resection in patients who had pulmonary tuberculosis. These patients usually have healthy contralateral lungs. Our paper presents data on regional pulmonary function in lung cancer where the incidence of concomitant obstructive lung disease in the non-tumor-bearing lung parenchyma is a significant complicating factor.

We are fully aware of the relative inaccuracy of our predictions for the outcome of smaller resections compared to pneumonectomy. This has been clearly stated in both the abstract and the body of our paper. Finally, we support and agree with Dr. Boysen’s editorial in Chest and we feel that the great majority of thoracic surgeons, when given the choice, would prefer to augment their clinical impressions with physiologic data.

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Self-terminating Torsades de Pointes or Holter Conversion?

To the Editor:

Undoubtedly, the case of “Holter auto-thumping” for termination of presumed quinidine-induced ventricular tachycardia was read with some interest and great amusement (Chest 1980; 78:874). There is, however, a more plausible explanation for the patient’s cardioversion based upon an analysis of the case history and ECG rhythm strips. It is clear from such analysis that the patient has a prolonged QT interval at the time of his normal sinus rhythm, undoubtedly related to quinidine therapy, followed by the degeneration to an arrhythmia characterized by an alternating pattern of electrical polarity where the QRS complexes appear to spiral around the isoelectric line. These features assure that the patient, in fact, experienced a classic episode of torsades de pointes, (TDP) as described by Dessertenne et al1 in 1966 and which was recently reviewed by Smith and Gallagher.2

The importance of this distinction is that TDP is usually self-terminating after a couple seconds to minutes, and this is the probable reason for apparent cardioversion without the aid of standard resuscitation in this patient. In addition, since TDP is rarely converted successfully by standard methods of cardioversion,3 it is hardly plausible that the “Holter auto-thump” was responsible.

Also, we must await further investigations and case reports to assess the role of the Holter monitor as a therapeutic modality.

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References

Hepatoma Presenting as a Single Cavitary Lung Mass and Leukemoid Reaction

To the Editor:

I read with interest the article by Cooper and Hayes published in Chest, February, 1980.1 I recently had the opportunity to see a similar case referred in consultation because of a leukemoid reaction white blood cell count of 40,000 with a platelet count of 1,200,000 and normal hemoglobin of 14 gm.

The patient complained of evening fever up to 39°C associated with night sweats and pleuritic chest pains. On examination, decreased breath sounds were heard in the right lower hemithorax. A chest x-ray film showed a 2.5 × 3 cm lobulated cavitary lesion in the right lung base. Her alkaline phosphatase level was greater than 500 (normal up to 250), LDH, 557; SCOT, 88; and bilirubin 2.2. Isotope liver scan and CAT scans showed two large defects in the right and left lobe of the liver.

A needle biopsy of the liver demonstrated multiple foci of active granulomatous inflammation compatible with mycobacterial infection or fungal infection. The patient received a course of antituberculosis therapy with INH, ethambutol and rifampin. The patient’s condition continued to deteriorate and she became semicomatose. Her white blood cell count increased to 70,000 and the platelet count to 1,500,000. Her leukocyte alkaline phosphatase was very high (215) compatible with a leukemoid reaction.

At this time repeat sections of the original liver biopsy were made which revealed a moderately differentiated hepatocellular carcinoma (hepatoma). The patient expired two weeks after her admission and, unfortunately, an autopsy was not obtained. This case is interesting since it represents the second reported case of a hepatoma presenting as a single cavitary lung mass. Also, the very severe leukemoid reaction and the granulomatous lesions in the liver misled us originally to the presumptive diagnosis of tuberculosis.

Hepatomas, as do many other neoplasms, often produce a leukemoid reaction2 and hepatocellular carcinomas have produced secondary polycythemia in a number of reported cases,3,4 due in some instances to ectopic erythropoietin production.5

As in the case reported by Cooper and Hayes,1 our patient had a very high alkaline phosphatase level which probably is the most commonly elevated laboratory test in hepatoma.

We wish to emphasize that these interesting tumors could masquerade as polycythemia or even, as in our case, as cavitary pulmonary tuberculosis with liver involvement and severe leukemoid reaction.

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