Thus, the role of electrical management may be more desirable in these situations. Pacemaker usage offers a mode of therapy which does not depress fetal heart rate or contractility.

Pacemaker therapy during pregnancy was first reported by Shouse and Acker in 1964. Since that time, approximately nine additional cases have been reported, all, significantly, without maternal or fetal mortality. The indication for pacing has been complete heart block, congenital or acquired, occasionally associated with ventricular tachycardia. Prior to pacemaker availability, heart block in pregnancy was accompanied by a 6 percent incidence of heart failure, 9 percent incidence of toxemia, a 13 percent maternal death rate and 15 percent fetal loss. These statistics are not necessarily the result of Stokes-Adams episodes, but may represent the potential role of brady-arrhythmias for accentuating heart failure and anoxic injury to the fetus.

The only morbidity associated with pacemaker use has been with permanent units installed prior to pregnancy. These have included local irritation with ulceration from a unit located in the breast region, transient pacemaker competition, and temporary loss of capture which required an increase in milliamperage. No episodes of thromboembolism occurred.

In summary, pacemaker therapy offered a favorable alternative to drug therapy in the treatment of a refractory arrhythmia during pregnancy. There appears to be no associated fetal morbidity or mortality.

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REFERENCES


Esophageal Ulceration and Oral Potassium Chloride Ingestion*

T. Rosenthal, M.D.; R. Adar, M.D.; J. Militianu, M.D.; and V. Deutsch, M.D.

In a patient with rheumatic heart disease and a large left atrium compressing the esophagus, potassium chloride produced severe esophageal ulceration, possibly contributing to the patient's demise. This is the second report of esophageal ulceration caused by oral potassium therapy.

Ulceration of the bowel due to oral potassium therapy has been previously reported. Most lesions described were of the ulcerating-stenosing type. Experimentally, similar lesions were produced both in small and large bowel, however, there is only one report of an esophageal ulceration due to oral potassium therapy. The potassium ulcers described in the small bowel were mostly treated surgically, and the esophageal ulcer described by Pemberton apparently healed spontaneously on withdrawal of the drug.

The present case is the second reported in the literature of an esophageal ulcer. It is somewhat unique because the potassium ulcer may have contributed to the death of the patient.

CASE REPORT

A 65-year-old woman was admitted with pain in the chest growing progressively worse over the preceding two months. At the age of ten, she had rheumatic fever and at 44, following an episode of hemoptysis and palpitations, a diagnosis of rheumatic heart disease with mitral stenosis and aortic regurgitation was made. A mitral commissurotomy was performed.

Four years after the operation, she again developed signs of cardiac failure. Chest x-ray examination showed cardiomegaly with marked enlargement of the left atrium compressing the middle third of the esophagus against the vertebral column, and causing a slight delay in the passage of barium through the mid-esophagus. The patient received diuretic treatment with oral potassium supplement in the form of 0.5 gm tablets of noncoated potassium chloride, taken with water at the time of meals.

Careful history taken during the present admission revealed that for two months prior to admission, swallowing dry food resulted in a dull pain which began beneath the xiphoid and spread upwards along the sternum, radiating towards the neck and the left shoulder. A sensation of strangulation accompanied the dysphagia.

During the two weeks prior to her admission, these symptoms became more prominent culminating in severe acute precordial pain on the day of admission. The physical examination revealed the known cardiac signs, as well as evidence of left heart failure. The abdomen was normal except for a palpable liver. Laboratory tests were noncontributory.

Barium examination of the esophagus and stomach revealed dilatation of the mid-esophagus caused by extrinsic compression. In the middle of this segment a large ulceration

*From the Departments of Internal Medicine, General and Vascular Surgery and Diagnostic Radiology, the Chaim Sheba Medical Center, Tel-Hashomer, and the Tel-Aviv University Medical School, Tel-Aviv, Israel.

Reprint requests: Dr. Deutsch, Chaim Sheba Medical Center, Tel-Hashomer, Tel-Aviv, Israel.
swollen
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seen
ROSENTHAL

A bland liquid diet was given and the symptoms improved slightly, but several days later a second sudden episode of severe pain, this time unrelated to the intake of food, was followed by hypotension and oliguria. The shock proved resistant to therapy and the patient died within 14 hours.

Postmortem examination revealed rheumatic valvulitis of the mitral aortic and tricuspid valves. The mitral stenosis was the most prominent lesion, and the left atrium was extremely dilated. The right atrium and ventricle were also enlarged. Stenosis of the origin of the left descending coronary artery was present, but there was no evidence of acute myocardial infarction.

There was a large ulcer in the mid-esophagus (Fig 2) with a necrotic base. A severe acute inflammatory reaction was seen in the base of the ulcer with peri-esophagitis, and clumps of cocci were identified in the inflammatory base of the ulcer. A localized mediastinitis was present around the involved esophagus.

There was no hiatus hernia nor any inflammatory change in the lower third of the esophagus. There was no aberrant gastric mucosa or any neoplastic process in the esophagus, nor was there any evidence of a specific bacterial or fungal infection in the esophagus or in the mediastinal lymph nodes.

The gallbladder showed mild chronic inflammation with some stones and there was no evidence of infection in the urinary tract. Death was thought to be due to the septic process in and around the esophageal ulcer in a patient with severe underlying cardiac disease.

**DISCUSSION**

Noncoated potassium chloride tablets are used quite frequently in our patients. In spite of the alleged erratic absorption of these tablets and the danger of small bowel ulceration, we have found this form of therapy effective and free of complications in the great majority of our patients. We have not encountered any case of small bowel ulceration within the last ten years.

Clinical and experimental evidence indicates that the local effect of the hypertonic potassium chloride combines with a relative vascular insufficiency to produce the acute ulceration.

In the present case, the stasis in the esophagus due to the marked compression by the large left atrium could well explain the ulcer formation by the potassium chloride. The patient was not receiving any other known irritant or ulcerogenic medication at the time. Although a full blown purulent mediastinitis or gross perforation were not found at autopsy, the localized mediastinitis may have contributed to the death in view of the severe underlying disease.

Despite the large number of patients with heart disease causing left atrial enlargement who receive potassium therapy, and the rarity of the complication reported here, the fact that this may result in a fatality dictates the need for more care in the use of oral potassium in these patients. Even the use of “Slow-K” as in the patient reported by Pemberton does not ensure safety; therefore, extreme caution is called for in the use of any form of potassium chloride tablets in patients in whom the left atrial enlargement is sufficient to cause a delay in the passage of barium in the upper mid-esophagus.

Furthermore, the appearance of dysphagia or any other complaint related to swallowing in these patients

**Figure 1.** Barium examination of the esophagus. The mid segment of the esophagus is dilated. In the middle of the dilated segment a large ulceration is seen (arrow), surrounded by edematous margins.

**Figure 2.** Macroscopic appearance of the mid-esophagus showing a large ulcer with a necrotic base and surrounded by swollen mucosa.
should be taken seriously as an indication for investigation of the esophagus, and for review of the forms of oral potassium therapy.

REFERENCES

Fibrous Histiocytoma of the Lung (Sclerosing Hemangioma Variant?)*

Sreedhar Nair, M.D., F.C.C.P., Kesavan Nair, M.D., and Irwin M. Weisbrot, M.D.

A case of fibrous histiocytoma (sclerosing hemangioma variant) of the lung occurring in an asymptomatic woman is presented. The clinical and histopathologic features are discussed and the literature briefly reviewed. Albeit rare, this entity enters into the differential diagnosis of any condition presenting as a "coin lesion" on the chest roentgenogram. We do not propose to reclassify all preceding reports of sclerosing hemangioma as fibrous histiocytoma because most are not. Based on a review of the literature, we believe the former term has been used to describe a variety of lesions, both vascular and inflammatory.

A name first coined by Liebow and Hubbel,4 "sclerosing hemangioma of the lung," is the descriptive term for a group of rare benign tumorlike lesions thought to have histologic features in common with certain dermal tumors, presently classified as fibrous histiocytoma by some authors. Fewer than 50 cases of sclerosing hemangioma of the lung have been reported since the publication of Liebow and Hubbel's original paper. Many of these, resembling such lesions as histiocytoma, xanthoma, reactive epithelial proliferation and vascular malformation, have been reported as sclerosing hemangioma. Some consider these to be the varied histologic appearances of a single entity. On the basis of available evidence, it is difficult to accept such a conclusion. In the present paper, a case of fibrous histiocytoma with the original diagnosis of sclerosing hemangioma of the lung is reported.

CASE REPORT

A 23-year-old white woman was admitted to the Norwalk Hospital in August, 1962 for evaluation of a density in the lung discovered a year before during a routine roentgen examination of the chest.

The year before she was admitted because of fibroadenoma in her right breast. No other abnormalities were detected on physical examination. A roentgenogram of the chest showed a solitary, well-circumscribed density 1.6 by 1.5 cm in the apical segment of the right upper lobe (Fig 1A, B). There was no history suggestive of tuberculosis or any other pulmonary disease. The patient smoked one package of cigarettes a day. A hemogram and other biochemical studies revealed normal results. The sputum and gastric washings contained no acid-fast bacilli or fungi on direct smear and culture. No tumor cells were detected in the sputum. A tuberculin test with intermediate PPD was negative. A few rales and expiratory rhonchi suggestive of bronchitis were present bilaterally. Physical examination otherwise revealed no abnormalities.

A thoracotomy was performed. The lesion was felt as a firm nodule in the upper lobe. A wedge resection of the right upper lobe was performed. The patient made an uneventful recovery and until now, ten years after operation, has had no respiratory symptoms.

PATHOLOGIC FINDINGS

Gross

Within a wedge of grossly normal lung was a 1.2-cm nodule composed of soft yellow tissue, adjacent to which

Figure 1A. Note soft round density measuring 1.5 cm in second anterior interspace on right side. Density is homogeneous and tomography did not show any calcifications. Margins are sharp, with no surrounding infiltrates.