for *Mycobacterium tuberculosis* at three weeks. The patient continued with corticosteroids and antituberculosis drugs, and at three months was discharged on a supervised outpatient programme.

Comment

Early bacteriological confirmation of miliary tuberculosis may be difficult to obtain. Compared with the relatively high incidence of a positive smear and culture in fibrocavitary tuberculosis, the sputum yield or organisms in miliary tuberculosis is relatively low.¹ Liver biopsy yields a fairly high percentage of granulomas but does not confirm the diagnosis unless the A.F.B. stain is positive. Bone marrow biopsies usually show granulomas when anaemia, leucopenia, and monocytosis are present; but the incidence of caseation and positive A.F.B. stains is low.² Many other diseases may cause miliary mottling in the chest x-ray films, and biopsy techniques have been developed to help in the differential diagnoses without the need for thoracotomy. Nevertheless, both percutaneous needle aspiration and cutting needle biopsy are associated with frequent pneumothoraces.³ Andersen described transbronchoscopic lung biopsy via the rigid bronchoscope. While only 16% of his cases had inconclusive biopsies, pneumothorax was frequent.⁴ In 24 of 33 patients who underwent transbronchial lung biopsy using a flexible fiberoptic bronchoscope a diagnosis was established without pneumothorax occurring.

We think that this is the first report of the diagnosis of miliary tuberculosis via transbronchial lung biopsy. As this patient had six negative sputum smears for A.F.B. before the procedure, the risk of infection to the bronchoscopist was thought to be minimal. This technique is safe and extremely efficacious in the rapid diagnosis of miliary lesions of the lung in acutely ill patients.

¹ Sahn, S. A., and Neff, T. A., *American Journal of Medicine*, 1974, 56, 495.

² Cucin, R. L., et al., *Journal of Chronic Diseases*, 1973, 26, 355.

³ Zavala, D. C., and Bedell, G. N., *American Review of Respiratory Disease*, 1972, 106, 186.

⁴ Andersen, H. A., and Fontana, R. S., *Chest*, 1972, 62, 125.

⁵ Levin, D. C., Wicks, A. B., and Ellis, J. H., jun., *American Review of Respiratory Disease*, 1974, 110, 4.

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Carcinoembryonic Antigen in Differential Diagnosis of Carcinoma of Pancreas from Chronic Pancreatitis

The differentiation of pancreatic carcinoma from chronic pancreatitis may prove impossible even at operation. Having heard that carcinoembryonic antigen (C.E.A.) levels were nearly always raised in pancreatic cancers^{1 2} we investigated both C.E.A. levels and Lundh test results as a means of differentiating these two conditions.

Methods, Patients, and Results

Plasma C.E.A. was measured by a modified double antibody technique.³ Pancreatic exocrine function was estimated by the Lundh test⁴ in all 25 patients with chronic pancreatitis and 10 of the 12 patients with carcinoma of the pancreas. Patients with chronic pancreatitis showed at least three of the following features: pancreatic calcification on abdominal x-ray films; a mean trypsin activity (M.T.A.) in duodenal juice after a Lundh test meal ≤ 6 IU/ml⁴; at least one well-documented attack of acute pancreatitis with serum amylase ≥ 1100 IU/l; diagnosis of chronic pancreatitis at laparotomy; history of chronic pancreatitis of three or more years. Pancreatic carcinoma was diagnosed at operation within two weeks of C.E.A. and duodenal M.T.A. determinations. In six patients histological confirmation was not obtained. The tumour was apparently confined to the pancreas in four, and either extended beyond the pancreas or had metastasized in eight.

Ten patients with pancreatitis but none with carcinoma had normal plasma C.E.A. levels (≤ 12.5 μ g/l; $\chi^2 = 6.95$; $P < 0.01$). No patients with pancreatitis had C.E.A. levels above 40 μ g/l compared with five of those with carcinoma ($\chi^2 = 12.27$; $P < 0.001$). The two diseases overlapped from 12.5 to 40 μ g/l and

only one of the four patients with localized carcinoma had a level greater than 40 μ g/l. C.E.A. values indicated malignancy in five patients (those with values greater than 40 μ g/l), but the Lundh test suggested a pancreatic carcinoma in seven patients (see table).

Diagnostic Value of Plasma C.E.A. and Lundh Test in 10 Patients with Carcinoma of Pancreas*

Case No.	C.E.A. (μ g/l)	M.T.A. (IU/ml)	Lundh Test (Bile in Duodenal Juice)	Test Indicative of Malignancy	
				C.E.A.	Lundh
1	24.0	2.9	—	—	+
2	54.0	1.1	—	+	+
3	43.9	2.2	—	+	+
4	38.5	4.9	—	—	+
5	25.5	3.1	—	—	+
6	230.0	0.5	—	+	+
7	27.0	2.9	—	—	+
8	48.5	Unsuccessful†	Unsuccessful†	+	—
9	500.0	33.9	+	+	—
10	15.0	8.4	+	—	—

*Patients in cases 1-8 were jaundiced.

†Tube failed to pass third part of duodenum.

Discussion

Though C.E.A. was once thought to be specific for adenocarcinoma of the gut abnormal values have been recorded in many benign conditions also.² Our results confirmed a high incidence of raised C.E.A. levels in patients with pancreatic carcinoma, but C.E.A.'s role in the differential diagnosis of pancreatic disease was disappointing. Up to a level of 40 μ g/l chronic pancreatitis and pancreatic carcinoma overlapped considerably, but we did not record levels greater than 40 μ g/l in chronic pancreatitis. In the five patients with carcinoma who had levels above 40 μ g/l the diagnosis was usually suspected on other evidence. Only one patient with a localized lesion had a C.E.A. level greater than 40 μ g/l.

The high incidence of positive C.E.A. assays (68%) in chronic pancreatitis was not unexpected because biological measurements are often abnormal in any disease. Others have reported high C.E.A. levels in up to 64% of patients with chronic pancreatitis, but the reason for this abnormality is not clear.⁵ We could not incriminate alcohol intake, smoking habits, length of history, pancreatic calcification, or other gastrointestinal diseases though others have suggested alcohol intake and associated liver disease as causes of raised C.E.A. levels. C.E.A. levels did not correlate with the extent of pancreatic exocrine insufficiency as measured by the Lundh test, but the test does not measure the amount of inflammation or developing fibrosis in the pancreas, and they may be important.

Most of our patients with pancreatic cancers were also jaundiced; in such cases a low M.T.A. and absence of bile in the duodenal juice strongly indicate malignancy. Therefore, it was not surprising that the additional information of the Lundh test increased the diagnostic accuracy from five to nine patients. Though in a larger series C.E.A. levels may indicate a malignant cause of pancreatic exocrine insufficiency the fetal pancreatic antigens may prove more specific. Thus, measuring C.E.A. alone is usually unhelpful in differentiating pancreatic carcinoma from chronic pancreatitis, but together with the Lundh test it may be helpful.

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¹ Delwiche, R., Zamcheck, N., and Marcon, N., *Cancer*, 1973, 31, 328.

² Ona, F. V., et al., *Cancer*, 1973, 31, 324.

³ Laurence, D. J. R., et al., *British Medical Journal*, 1972, 3, 605.

⁴ Mottaleb, A., et al., *Gut*, 1973, 14, 835.

⁵ Dilawari, J. B., et al., *Gut*, 1975, 16, 255.

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