Diabetes mellitus, pulmonary tuberculosis and chronic calcific pancreatitis revisited

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Summary

The prevalence of chronic calcific pancreatitis (CCP) was determined in 25 successive patients with both diabetes mellitus and newly diagnosed pulmonary tuberculosis. Twenty patients (80%) were alcoholics and all were black. Of these, 9 (45%) had CCP. In only 3 of these 9 patients was the history compatible with the condition diagnosed. Clinical steatorrhoea was absent in the patients with CCP. Pulmonary tuberculosis was extensive with major involvement of three or more lung zones in 36% of patients. Mainly basal involvement of the lungs was present in 8% of patients.

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The risk of developing pulmonary tuberculosis is increased by chronic disabling diseases, such as diabetes and alcoholism.[1] Defective chemotaxis of polymorphonuclear leucocytes from patients with diabetes mellitus has been reported.[2] Impaired bactericidal function of granulocytes in patients with poorly controlled diabetes has also been demonstrated.[3] Ethyl alcohol also depresses leucocyte migration to inflamed areas, which may contribute to the poor resistance of alcoholics to infection.[4]

The incidence of pulmonary tuberculosis in a series of 3 106 diabetics was 8,4%.[5] In a previous study the prevalence of diabetes among patients with pulmonary tuberculosis admitted to an isolation department was found to be 2,1%.[6] Thirty per cent of patients with chronic calcific pancreatitis (CCP) have or have had tuberculosis at some stage of their disease or during follow-up.[7] The reported incidence of overt diabetes in patients with non-calcific pancreatitis was 30% as compared to 70% in patients with calcific pancreatitis.[8]

A study was undertaken in order to establish the prevalence of CCP in patients with both diabetes and newly diagnosed pulmonary tuberculosis admitted to our local teaching hospitals over a 22-month period.

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Patients and methods

Pulmonary tuberculosis was diagnosed on clinical, radiographic and sputum findings. Diabetes was diagnosed in non-established cases according to WHO criteria.[9] A diagnosis of CCP was made after straight radiography of the abdomen. A detailed history of alcohol consumption, smoking, abdominal pain, steatorrhoea and a family history of diabetes was obtained.

Straight radiography of the abdomen was also performed in a control group of 25 patients. These were patients with recently diagnosed pulmonary tuberculosis but without diabetes and ill enough to be admitted to an isolation ward. The mean age of this group was 41,2 years and 56% were men.

Results

During the study period 25 patients with diabetes and pulmonary tuberculosis were found. Table I summarises some of the data from this group of patients. Twenty of the 25 patients were heavy alcohol consumers and all 20 were black. They had used alcohol daily or binged over weekends for periods of 5 -20 years. Nine of these 20 patients had radiographic evidence of CCP. A history consistent with acute intermittent pancreatitis was obtained in only 4 patients and of these only 3 had radiographic evidence of CCP. No history compatible with malabsorption or clinical steatorrhoea could be obtained from any of the patients. No patients in the control group had radiographic evidence of CCP. Nine patients (36%) from the control group admitted to past heavy alcohol consumption.

TABLE I. CLINICAL CHARACTERISTICS OF PATIENTS AT ENTRY INTO STUDY

Characteristic	No.	%
Mean age (yrs)	**46,4**	—
Men	**16**	**64**
Therapy		
Insulin	**13**	**52**
Oral hypoglycaemic agent	**10**	**40**
Diet only	**2**	**8**
History of smoking	**19**	**76**
Heavy alcohol consumption*	**20**	**80**
History consistent with AIP	**4**	**16**
CCP	**9**	**36**
Family history of diabetes	**3**	**12**

*Defined as daily or weekend binge drinking.
AIP = acute intermittent pancreatitis.

The mean body mass index (BMI) was 18,4 kg/m^2 (range 12,2 - 22,3 kg/m^2) and 21,6 kg/m^2 (range 14,7 - 25,6 kg/m^2) for men and women respectively. In 6 of the 25 patients the diagnoses of pulmonary tuberculosis and diabetes were made simultaneously. Four of the 25 patients had a previous history of pulmonary tuberculosis. The pattern of lung involvement is shown in Table II.

TABLE II. DISTRIBUTION OF LUNG LESIONS IN DIABETIC PATIENTS

	Upper zone	Mid zone	Lower zone
Right lung	**21**	**11**	**6**
Left lung	**7**	**8**	—

Discussion

The mean age of the patients was 46,4 years (range 14 - 60 years) and included 1 child, the latter being the only case of primary pulmonary tuberculosis in the present series. First-World patients with pulmonary tuberculosis and diabetes tend to be older and the relative incidence is highest in those > 65 years.[5] All our patients were black with the exception of a white woman.

Four patients had had a full course of chemotherapy for past pulmonary tuberculosis. In this group pulmonary tuberculosis was reactivated from 1 month to 14 years after the diagnosis of diabetes. One patient became sputum positive again 4 months after completion of a 12-month 'short' course of chemotherapy. This patient also suffered from CCP. Diabetics previously treated for pulmonary tuberculosis thus remain at high risk for reactivation and such patients must be carefully observed.

CCP was present in 45% of alcoholics with diabetes and recently diagnosed pulmonary tuberculosis. The prevalence might have been even higher had diagnostic tools such as computed axial tomography and endoscopic retrograde cholangiography been employed. In these patients CCP is most probably caused by alcohol, since they all consumed alcohol on a daily basis and/or were weekend binge drinkers. It is alarming that a history compatible with acute episodes of pancreatitis was present in only 3 of the 9 patients with CCP. A high index of suspicion is thus necessary to make the diagnosis of CCP in this population with both diabetes and pulmonary tuberculosis. The absence of clinical steatorrhoea in our patients emphasises the difference in clinical presentation of CCP in South Africa from that in England and USA.[7] No case of pulmonary tuberculosis was encountered in a series of 80 patients with both chronic pancreatitis and diabetes studied in Italy (A. Tiengo — personal communication). This is also in stark contrast to the South African experience.[7]

Pulmonary tuberculosis is often extensive at the time of diagnosis in diabetics.[1] Nine of our patients had major involvement of three or more lung zones. Only 2 patients (8%) exhibited the 'classic', mainly basal, involvement expected in diabetics.[1]

Although clinical steatorrhoea was absent in patients with CCP they were malnourished, as was evident from the mean BMI of 17,1 kg/m^2 for men in this group. This is consistent with the view that most patients with CCP have lost 30 - 50% of their body weight by the time they are seen or on follow-up.[7]

In conclusion, CCP featured prominently in a group of patients with both pulmonary tuberculosis and diabetes mellitus. The CCP was most probably of alcoholic aetiology and tended to be asymptomatic. A high index of suspicion was necessary in order to make the diagnosis of CCP, which greatly influences the prognosis of these patients. It is recommended that all patients with both diabetes and newly diagnosed pulmonary tuberculosis be thoroughly screened for CCP.

REFERENCES

1. Fraser RG, Pare JA. *Diagnosis of Diseases of the Chest.* 2nd ed. Philadelphia: WB Saunders, 1978: 756.
2. Mowat AG, Baum J. Chemotaxis of polymorphonuclear leucocytes from patients with diabetes mellitus. *N Engl J Med* 1971; **284:** 621-627.
3. Nolan CM, Beaty HN, Bagdale JD. Further characterization of the impaired bactericidal function of granulocytes in patients with poorly controlled diabetes. *Diabetes* 1978; **27:** 889-894.
4. Gilman AG, Goodman LS, Rall TW, Murad F, eds. *The Pharmacological Basis of Therapeutics.* 7th ed. New York: Macmillan, 1985: 377.
5. Marble A, Krall LP, Bradley RF *et al.*, eds. *Joslin's Diabetes Mellitus.* 12th ed. Philadelphia: Lea & Febiger, 1985: 741-742.
6. Marais RM. Diabetes mellitus in black and coloured tuberculosis patients. *S Afr Med J* 1980; **57:** 483-484.
7. Bank S. Chronic pancreatitis: clinical features and medical management. *Am J Gastroenterol* 1986; **81:** 153-167.
8. Bank S, Marks IN, Vinik AI. Clinical and hormonal aspects of pancreatic diabetes. *Am J Gastroenterol* 1975; **64:** 13-22.
9. WHO Study Group on diabetes mellitus. *WHO Tech Rep Ser* 1985; No. 727.