Fibrocalculous Pancreatic Diabetes in Pune, India

Clinical features and follow-up for 7 yr

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OBJECTIVE — To study clinical features of fibrocalculous pancreatic diabetes from this clinic, to compare these with the published criteria of malnutrition-related diabetes mellitus, and to conduct serial follow-up of these patients to study difficulties in their treatment.

RESEARCH DESIGN AND METHODS — Details of presenting symptoms, anthropometry, diabetic tissue damage, treatment, and follow-up of 55 patients with fibrocalculous pancreatic diabetes (pancreatic calculi demonstrated on X-ray and sonography) treated during the last 7 yr were studied.

RESULTS — Many patients did not fit the accepted criteria of malnutrition-related diabetes. Thus, 17 (31%) were diagnosed after 30 yr of age and 23 (42%) had a body mass index >18 kg/m^2, and the daily dose of insulin in these patients (mean 0.8 U/kg) was similar to that in the IDDM patients (mean 1.0 U/kg). The two pathognomonic complaints (pancreatic pain and steatorrhea) were not always present. Many patients took very irregular treatment, but none suffered diabetic ketoacidosis despite stopping insulin for long periods of time; 33% of patients had some diabetic tissue damage when first seen. Fourteen patients were lost to follow-up, and 11 died during the follow-up.

CONCLUSIONS — Clinical features of these fibrocalculous pancreatic diabetes patients were somewhat different than the classic descriptions. A need exists to reconsider classification of FCPD under malnutrition-related diabetes mellitus. Many patients receive irregular treatment, and a substantial proportion die within a few years of diagnosis, many as a result of preventable causes.

FCPD has attracted considerable attention (1–3) since it was proposed as a major class of diabetes in the tropics (4). FCPD is associated with tropical calcific pancreatitis, a chronic calcifying pancreatitis of unknown etiology in developing tropical countries. Zuidema (5) first reported FCPD from Indonesia, and other reports followed from Uganda (6) and India (7). A WHO study group (4) classified FCPD as a subvariety of MRDM, the diagnosis of which is usually by Ahuja's criteria (8). This paper discusses clinical features of patients with FCPD and analyzes the progress of these patients during the first 7 yr of follow-up.

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FCPD, FIBROCALCULOUS PANCREATIC DIABETES MELLITUS; MRDM, MALNUTRITION-RELATED DIABETES MELLITUS; WHO, WORLD HEALTH ORGANIZATION; IDDM, INSULIN-DEPENDENT DIABETES MELLITUS; NIDDM, NON-INSULIN-DEPENDENT DIABETES MELLITUS; BMI, BODY MASS INDEX; WHR, WAIST-TO-HIP RATIO; OGTT, ORAL GLUCOSE TOLERANCE TEST; FPG, FASTING PLASMA GLUCOSE

RESEARCH DESIGN AND METHODS — In this diabetes clinic, all patients are interviewed about exocrine pancreatic symptoms (e.g., pain and steatorrhea). If pancreatic disease is suspected, abdominal X ray and sonography are performed. These are also done in patients <35 yr of age, in those with excessive weight loss, and in those who do not show ketonuria in presence of severe hyperglycemia. Those patients who show pancreatic calculi on X rays (confirmed on sonography) are considered to have FCPD if a significant history of alcohol intake is ruled out. Because of our interest in this condition, patients diagnosed to have FCPD by outside clinics are referred to us for management.

A total of 55 FCPD patients (33 men) were treated between September 1984 and July 1991. Of these, 26 were referred to this clinic (10 for management of diabetes in whom the additional diagnosis of FCPD was made). Diagnosis of pancreatitis was suggested by a history of abdominal pain in 44 patients, but 11 patients did not recollect any episode of abdominal pain. The latter were suspected to have FCPD either because of steatorrhea (4) and/or severe malnutrition (6) and/or absence of ketonuria despite very high (>30 mM) plasma glucose levels (8). One of these patients received treatment for dysentery on and off for 3 yr before steatorrhea was diagnosed; 48 patients had never consumed alcohol, 4 drank occasionally, and 3 started drinking after the diagnosis. Sonography did not show obstructive hepatobiliary disease in any patients. The majority (50 patients) were born and brought up in Maharashtra state (Centra

Table 1—*Clinical and biochemical characteristics of study subjects*

	FCPD patients	IDDM patients	NIDDM patients	Control subjects
n	55	81	72	73
Sex (M/F)	33/22	52/29	49/23	41/32
Age at diagnosis (yr)	22 (9–59)	15 (4–34)*	35 (16–39)†‡	33 (21–40)‡§
BMI (kg/m^2)	17.0 (10.7–24.1)	15.7 (12.0–25.7)	23.8 (14.7–40.2)*‡	23.2 (16.8–30.0)*‡
WHR				
Men	0.88 (0.82–1.00)	0.83 (0.77–0.91)†	0.92 (0.80–1.07)†‡	0.87 (0.79–0.94)‖¶
Women	0.80 (0.67–0.93)	0.81 (0.73–0.98)	0.80 (0.71–0.93)	0.77 (0.68–0.87)‖#
Triceps skin fold (mm)	8 (3–16)	6 (3–11)	13 (6–34)*‡	13 (5–28)*‡
Subscapular skin fold (mm)	10 (3–30)	7 (3–13)†	29 (8–50)*‡	24 (8–45)*‡#
HbA_1 (%)	10.6 (5.3–18.9)	12.1 (4.9–20)	9.3 (5.7–18.3)§*	6.3 (5.4–7.1)*‡[b]

Data are medians with ranges in parentheses. Both NIDDM patients and control subjects were < 40 yr of age.
*Different from FCPD, $P < 0.001$.
†Different from FCPD, $P < 0.01$.
‡Different from IDDM, $P < 0.001$.
§Different from FCPD, $P < 0.05$.
‖Different from IDDM, $P < 0.05$.
¶Different from NIDDM, $P < 0.01$.
#Different from NIDDM, $P < 0.05$.
*Different from IDDM, $P < 0.01$.
[b]Different from NIDDM, $P < 0.001$.

India); 3 were from South and 2 from North India. Twenty-four patients were very poor (monthly family income <1000 rupees), 20 admitted income ≤3000 rupees/mo, and 11 were more affluent (>3000 rupees/mo). Eight patients were illiterate, 29 attended secondary school, and 18 received higher education. Twenty patients were rural and 30 urban.

First examination included anthropometry (height, weight, triceps, and subscapular skinfold thickness, WHR) and an OGTT (75 g of anhydrous glucose, WHO 1985 criteria) as part of a prospective study of β-cell function in FCPD. Twenty-nine patients were diagnosed as diabetic for the first time; others were known already to have diabetes (duration 1 mo to 30 yr, median 18 mo).

Demographic data from 81 IDDM (ketosis prone) and 72 young NIDDM patients (<40 yr) in this clinic is shown for comparison (Table 1). Clinical classification was made according to WHO guidelines (4). Abdominal X-ray and sonography did not show pancreatic calculi in these two groups of patients. The control subjects (<40 yr of age, no family history of diabetes, and no pancreatic symptoms) had been outpatients for minor illnesses.

Plasma glucose (glucose oxidase) was measured on an Abbott VP Super autoanalyzer (Irving, TX) and HbA_1 by a colorimetric method (9). The significance of the differences between groups was calculated by Mann-Whitney test.

RESULTS

Age at diagnosis

The IDDM patients were the youngest, NIDDM the oldest, and FCPD intermediate (Fig. 1). Three (6%) FCPD patients were <10 yr of age, and 17 (31%) were >30 yr, including 3 >50 yr.

Signs of malnutrition and measures of obesity

Seven FCPD patients were cachectic; 6 patients showed skin and hair changes of malnutrition (Table 1). Eleven patients (including the 7 above) showed enlargement of parotid glands. Ten IDDM and 2 NIDDM (both with pulmonary tuberculosis) patients were cachectic; none showed skin and hair changes or parotid enlargement like those with FCPD. Only 32 (58%) of FCPD patients had a BMI

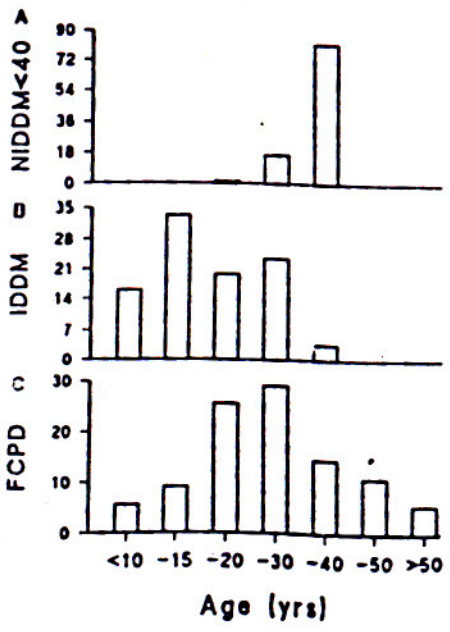

Figure 1—*Distribution of age at diagnosis in young (<40 yr) NIDDM patients (A), IDDM patients (B), and FCPD patients (C).*

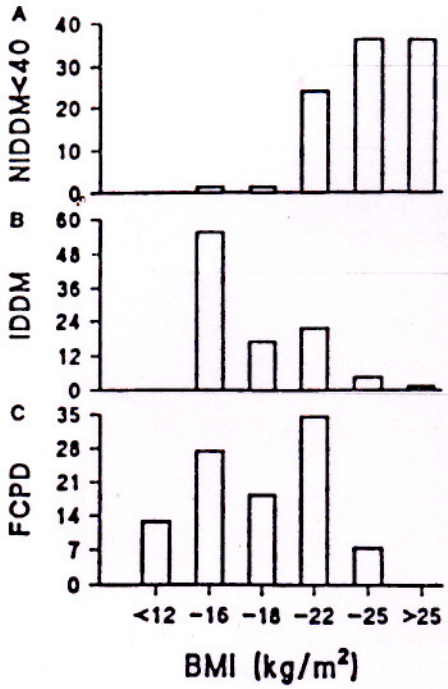

Figure 2—*Distribution of BMI (kg/m²) in young (<40 yr) NIDDM patients (A), IDDM patients (B), and FCPD patients (C).*

<18 kg/m², the commonly suggested cutoff point for MRDM, 58 (72%) of IDDM patients also had a low BMI (Fig. 2). Overweight (BMI >25 kg/m²) subjects were mostly in the NIDDM group (36%). Skinfold thickness measurements showed similar results to those of BMI. Despite their low BMI, WHR in FCPD patients was similar to that in nondiabetic controls.

Dietary habits

None of these FCPD patients from Maharashtra state had consumed cassava at any time; one patient from Kerala had eaten cassava off and on. Jawar (*Sorghum vulgare*) was a common staple (main cereal at least 4 days/wk) for many of the FCPD diabetic patients (60%). FCPD patients consumed 1560 kcal (median, range 850-2100) and 46 g protein/day (range 28–78) amounting to 1.1 g protein/kg body weight (0.6–2.3).

Glycemic control and antidiabetic treatment

HbA_1 concentration at first visit was similar in FCPD and IDDM patients. NIDDM patients showed a lower concentration. Of 26 referred FCPD patients, 22 received insulin. Most patients received insulin once per day and not very regularly, and 4 patients took sulphonylureas. Of 29 newly diagnosed patients, 6 had mild hyperglycemia that was controlled with diet alone (1 received insulin for a short period at the time of pancreatic surgery). These patients have maintained good glycemic control during follow-up (6 mo to 4 yr from diagnosis). Two patients refused insulin treatment and persisted with oral hypoglycemic agents despite poor control (FPG >11 mM). Other patients were treated with insulin. The insulin dose during the initial weeks of treatment, especially in those admitted to the hospital was quite high (≤ 8 U · kg^{-1} · day^{-1}); those treated as outpatients received much smaller doses. After stabilization (~3 mo later), the insulin dose was 0.85 U/kg (median, range 0.2–2.4), similar to that in IDDM patients of 1.0 U/kg (0.5–2.3). No significant relation existed between body weight and insulin dose. Most patients received conventional (porcine-bovine mixture) insulins, except 3 who could afford purified insulins. Despite a free supply of insulin and syringes many took insulin irregularly and stopped insulin use from time to time, sometimes for months. None of those patients had diabetic ketoacidosis during follow-up (except possibly 1 who died in a peripheral hospital).

Associated conditions

Two FCPD patients had adult polycystic disease of the kidneys. Repeated urinary infections were common in these patients. Three subjects developed pulmonary tuberculosis, and 1 of those died after long neglect of diabetes as well as tuberculosis; the other 2 improved after antitubercular treatment. Of the IDDM patients, 3 (but none of the NIDDM patients) suffered pulmonary tuberculosis during follow-up and were successfully treated.

Diabetes-related problems

At the first visit the following problems were noted in these FCPD patients.

Peripheral neuropathy. Many patients complained of tingling and numbness. Objective evidence of peripheral neuropathy (diminished sensations and diminished or absent ankle jerks) was found in 16 patients. Six of these were newly diagnosed, and 10 were known diabetic patients (median duration of diabetes 9 yr, range 1.5–30 yr).

Retinopathy. Background diabetic retinopathy (including microaneurysms, hemorrhages, and hard and soft exudates) was seen in 6 patients, and 1 showed maculopathy. All of these were known diabetic individuals (median duration 5 yr, range 1–15 yr). One patient was blind in one eye as a result of central retinal artery occlusion.

Proteinuria. Albustix (Ames, Elkhart, IN) were used to test for proteinuria, which was present in 2 patients. These individuals were known to have diabetes for 2 and 10 yr.

Macrovascular disease. None of the patients showed significant peripheral vascular disease (claudication or diminished foot pulses on palpation or both); resting electrocardiogram was normal in all. None of the IDDM or NIDDM patients showed any significant neuropathy, retinopathy, or proteinuria when first seen, but 5 NIDDM patients showed electrocardiographic ST-T wave changes (coronary possible, Minnesota code).

Follow-up and outcome

Thirty FCPD patients came for regular follow-up; 14 have been lost to follow-up (for >1 yr), and 11 of these FCPD patients died within the follow-up period. Common reasons for loss to follow-up include low socioeconomic status and education and distance from the hospital; 4 patients moved. Many could not be convinced of the necessity of reg-

Table 2—*Details of FCPD patients who died during follow-up*

Patient no.	Age at diagnosis (yr)	Sex	Age at death (yr)	Causes contributing to death
1	10	F	11	Chronic diarrhea, septicemia
2	15	F	15	Possible hypoglycemia
3	14	M	16	Road accident
4	14	F	16	Possible diabetic ketoacidosis
5	19	F	25	Perinephric abscess, septicemia
6	25	F	27	Chronic malnutrition, neglect
7	35	M	38	Pulmonary tuberculosis
8	28	F	39	Chronic renal failure, polycystic kidneys
9	45	M	46	Possible hypoglycemia
10	30	F	46	Postoperative portal vein thrombosis, septicemia
11	54	M	57	Postoperative portal vein thrombosis, septicemia

ular treatment because they did not become acutely ill after stopping insulin use for long periods of time. A change to alternative medicine played a role in some patients. Many died quite young and within a short time from diagnosis. Details of these patients are shown in Table 2.

CONCLUSIONS— This study highlights the heterogeneity in socioeconomic background, symptoms, age, and nutritional status (including BMI) of FCPD patients. Many of these patients differed from the classic description (7) and did not fulfill the criteria for MRDM (4,8). Thus, many were diagnosed after 30 yr of age, had a BMI >18 kg/m^2, and belonged to higher socioeconomic groups. Severe malnutrition was seen in only 13% and in only 58% was the BMI <18 kg/m^2. A low BMI was also seen in 72% of these IDDM patients implying that diabetes-related malnutrition is a significant factor, although in FCPD exocrine pancreatic deficiency would add to the problem. Reports from Madras (1,10) and Indonesia (11) also failed to find gross malnutrition in many of their FCPD patients. Thus, classification of FCPD under MRDM is not without problems. Moreover, low BMI in many newly diagnosed IDDM patients argues against using malnutrition at diagnosis as a major criterion to diagnose MRDM. This is not to say that malnutrition does not lead to β-cell dysfunction and glucose intolerance. Indeed, malnutrition in early life (12–14), even in utero (15,16), is associated with β-cell dysfunction in later life. This important aspect needs to be addressed in carefully planned prospective studies in humans.

No information exists in FCPD patients on body fat distribution and central obesity, a well-known risk factor for diabetes (17), especially in Asian Indians (18,19). Lack of excess central obesity in our FCPD patients suggests that insulin resistance and other metabolic characteristics related to central obesity might not be important in its pathogenesis.

The most useful indication of FCPD was pancreatic pain, steatorrhea was less common. Ketosis resistance helped diagnosis in some patients. Pancreatic pain needs to be inquired about carefully because of the varied presentation (7) and because pancreatic pain may not be remembered if suffered in early childhood. In 1 patient the telltale branding marks (skin burns caused by a hot iron) in the epigastric region suggested the diagnosis. Steatorrhea is infrequent, possibly because of low dietary fat, and might be misdiagnosed as dysentery. Ketosis resistance of these patients helps distinguish them from the usually ketosis-prone IDDM patients; possibly this may be multifactorial in origin (20), but some patients may become ketotic (1). A high index of suspicion will help identify pancreatic etiology in some patients tentatively diagnosed as having primary diabetes.

In the absence of symptoms of pancreatitis, abdominal X ray, sonography, and tests of exocrine pancreatic function help diagnose pancreatic involvement. This clinic has reported abnormalities of serum pancreatic enzymes in the earlier stages of FCPD (nondiabetic and impaired glucose tolerant [21]) and has also shown that the enzyme levels are higher in our population than in white Caucasians from the U.K. (22,23). This suggests the existence of a subclinical pancreatopathy in this population. FCPD diagnosed by the present criteria (pancreatic stones) appears at the extreme end of the spectrum. Diagnosis in early stages obviously would be desirable. The criteria of Mohan et al. (24) for diagnosis of FCPD are comprehensive but need to be tested in large population studies. Interpretation of exocrine pancreatic tests in some diabetic patients, however, might be complicated by the interaction between the exocrine and endocrine pancreas (25,26).

The occurrence of FCPD in Maharashtra where cassava is not grown suggests that cassava cannot be the sole or even the main etiological factor. Role of other dietary cyanogens (i.e., jawar) needs further investigation (27).

The majority of these FCPD patients are treated with insulin. However, this clinic has not studied whether oral hypoglycemic agents are effective. The response to oral agents is probably dictated by residual β-cell function (1). We found an inverse relationship between plasma C-peptide and HbA_1 concentrations in these patients (21) and also that β-cell function improved after treatment of diabetes (28). Possibly, some patients can control diabetes with the use of oral agents.

Insulin-resistance is claimed to be an important feature of MRDM (8). Some of our patients required high doses of insulin during the first few weeks of treatment when blood glucoses were high, food intake was substantial, and weight gain was rapid. However, the patients soon needed smaller doses, and none required unusually large doses subsequently. Subsequent insulin doses of FCPD patients were similar to those of IDDM patients. The insulin dose in patients from Madras was also not unusually high (1). FCPD patients do not appear to be unusually resistant to exogenous insulin.

Evidence of diabetic tissue damage (retinopathy, neuropathy, and nephropathy) was present in several FCPD patients; most were known to be diabetic, but some were diagnosed for the first time and had neglected their diabetic symptoms for years before seeking medical help. Absence of evident peripheral vascular or coronary artery disease in these patients was probably because of their young ages.

Regular follow-up of these patients is difficult, and treatment frequently is irregular, usually because of socioeconomic reasons and lack of education. A relative immunity from ketoacidosis even after stopping insulin injections for long periods means that these patients can afford to stop treatment, this immunity also makes it difficult for the physician to convince the patient of the necessity for regular antidiabetic treatment. Many patients are lured away by the claims of practitioners of alternative medicine. The high mortality at young ages reflects a combination of these factors along with poor medical facilities in remote places. The distance that the patient lives from the hospital therefore becomes an important factor in disease management. Metabolic problems contributed to death in 3 of our patients, although it was difficult to be sure of the cause of deaths that occurred in remote places.

In summary, this report presents 7 yr of experience in treating FCPD patients. The clinical features of these patients showed a much wider spectrum than the classic descriptions, and many patients did not fulfill the criteria of MRDM (4). Treatment and follow-up of these patients were difficult, and many died within a relatively short period from diagnosis. Future studies should use wider criteria for diagnosis of FCPD, including the assessment of pancreatic function.

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References

1. Mohan V, Mohan R, Susheela L, Snehlata C, Bharani G, Mahajan VK, Ramachandran A, Viswanathan M, Kohner EM: Tropical pancreatic diabetes in South India: heterogeneity in clinical and biochemical profile. *Diabetologia* 28:229–32, 1985
2. Yajnik CS: Diabetes in tropical developing countries. In *Diabetes Annual/5*. Alberti KGMM, Krall LP, Eds. Amsterdam, Elsevier, 1990, p. 72–87
3. Yajnik CS: Diabetes in tropical developing countries. In *Diabetes Annual/6*. Alberti KGMM, Krall LP, Eds. Amsterdam, Elsevier, 1991, p. 62–81
4. WHO Study Group: *Diabetes Mellitus: Report of a WHO Study Group*. Geneva, World Health Org., 1985 (WHO Tech. Rep. Ser. no. 727)
5. Zuidema PJ: Cirrhosis and disseminated calcification of pancreas in patients with malnutrition. *Trop Geog Med* 11:70–74, 1959
6. Shaper AG: Etiology of chronic pancreatic fibrosis with calcification seen in Uganda. *Br Med J* II:1607–609, 1964
7. Geevarghese PJ: *Pancreatic Diabetes*. Bombay, India, Popular Prakashan, 1968
8. Ahuja MMS: Heterogeneity in tropical pancreatic diabetes (Letter). *Diabetologia* 28:708, 1985
9. Parker KM, England JD, Da Costa J, Hess RL, Goldstein DE: Improved colorimetric assay for glycosylated hemoglobin. *Clin Chem* 27:669–72, 1981
10. Mohan V, Chari ST, Ramachandran A, Jayanthi V, Malathi S, Madangopalan N, Viswanathan M: Fibro-calculous pancreatic diabetes and obesity. *Diabetes Res Clin Pract* 8:161–66, 1990
11. Wiyono P: Exocrine and endocrine pancreatic function in malnutrition-related diabetes mellitus (MRDM) in Yogyakarta, Indonesia. *Kobe J Med Sci* 34:215–26, 1988
12. Cook CG: Glucose tolerance after kwashiorkor. *Nature* (Lond) 215:1295–96, 1967
13. Swenne I, Crace CJ, Milner RDG: Persistent impairment of insulin secretory response to glucose in adult rats after limited period of protein calorie malnutrition early in life. *Diabetes* 36:454–58, 1987
14. Swenne I, Crace CJ, Jansson L: Intermittent protein calorie malnutrition in the young rat causes long-term impairment of the insulin secretory response to glucose in vitro. *J Endocrinol* 118:295–302, 1988
15. Shoeck A, Remacle C, Reusens B, Hoet JJ: Effect of a low protein diet during pregnancy on the fetal rat endocrine pancreas. *Biol Neonate* 57:107–18, 1990
16. Hales CN, Barker DJP, Clark PMS, Cox LJ, Fall C, Osmond C, Winter PD: Fetal and infant growth and impaired glucose tolerance at age 64. *Br Med J* 303:1019–22, 1991
17. Bjorntorp P: Obesity and diabetes. In *Diabetes Annual/5*. Alberti KGMM, Krall LP, Eds. Amsterdam, Elsevier, 1990, p. 373–395
18. Shelgikar KM, Hockaday TDR, Yajnik CS: Central rather than generalized obesity is associated with hyperglycemia in Asian Indian subjects. *Diabetic Med* 8:712–17, 1991
19. McKeigue PM, Shah B, Marmot MG: Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 337:382–86, 1991

20. Yajnik CS, Shelgikar KM, Naik SS, Kanitkar SV, Orskov H, Alberti KGMM, Hockaday TDR: The ketosis resistance in fibro-calculous pancreatic diabetes. 1. Clinical observations and endocrine-metabolic measurements during oral glucose tolerance test. *Diabetes Res Clin Pract* 15:149–56, 1992
21. Yajnik CS, Shelgikar KM, Sahasrabudhe RA, Naik SS, Pai VR, Alberti KGMM, Hockaday TDR, Katrak A, Dandona P: The spectrum of pancreatic exocrine and endocrine (β-cell) function in tropical calcific pancreatitis. *Diabetologia* 33:417–21, 1990
22. Yajnik CS, Kanitkar SV, Katrak A, Naik SS, D'Souza A, Dandona P: Serum immunoreactive trypsin in tropical pancreatic diabetes syndrome. *Ann Clin Biochem* 26:69–73, 1989
23. Yajnik CS, Sahasrabudhe RA, Naik SS, Katrak A, Shelgikar KM, Kanitkar SV, Narayanan VA, Dandona P: Exocrine pancreatic function in Indian diabetics. *Pancreas* 5:631–38, 1990
24. Mohan V, Ramachandran A, Viswanathan M: Diabetes in the tropics. In *Diabetes Annual/4*. Alberti KGMM, Krall LP, Eds. Amsterdam, Elsevier, 1988, p. 46–55
25. Henderson JR, Daniel PM, Fraser PA: The pancreas as a single organ: the influence of the endocrine upon the exocrine part of the gland. *Gut* 22:158–67, 1981
26. British Medical Journal: Pancreatic islet—acinar interactions (Editorial). *Br Med J* 283:570–71, 1981
27. Patole A, Lawate US, Raut KN, Shelgikar KM, Yajnik CS: Nutritional intake of young diabetics. *Diabetes Bull Res Soc Study Diabetes India* 8:43–48, 1988
28. Yajnik CS, Kanitkar SV, Shelgikar KM, Naik SS, Alberti KGMM, Hockaday TDR: Pancreatic C-peptide response to oral glucose in fibrocalculous pancreatic diabetes. *Diabetes Care* 13:525–27, 1990