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Clinical profile of lean NIDDM in South India

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Abstract

The majority (> 80%) of patients with non insulin dependent diabetes mellitus (NIDDM) present in Europe and America are obese. In developing countries like India, most NIDDM (> 60%) are non-obese and many are actually lean with a body mass index (BMI) of < 18.5 and are referred to as 'lean NIDDM'. This paper compares the clinical profile of a cohort of 347 lean NIDDM, with a group of 6274 NIDDM of ideal body weight (IBW) and 3252 obese NIDDM attending a diabetes centre at Madras in South India. The lean NIDDM who constituted 3.5% of all NIDDM patients seen at our centre, had more severe diabetes and an increased prevalence of retinopathy (both background and proliferative), nephropathy and neuropathy. Although a larger percentage of the lean NIDDM patients were treated with insulin, 47% of the males and 53% of the females were still on oral hypoglycaemic agents even after a mean duration of diabetes of 9.2 ± 8.1 years. Studies of GAD antibodies, islet cell antibodies (ICA) and fasting and stimulated C-peptide estimations done in a small subgroup of the lean NIDDM showed that they were distinct from IDDM patients. More studies are needed on metabolic, hormonal and immunological profile of lean NIDDM seen in developing countries like India. © 1997 Elsevier Science Ireland Ltd.

Keywords: Lean NIDDM; Non-obese; NIDDM; Complications; Retinopathy; Nephropathy; Neuropathy; C-peptide; Gad-antibodies; Islet cell antibodies; South India

1. Introduction

The WHO study group report classifies patients with non insulin dependent diabetes mellitus (NIDDM) into 'obese' and 'non-obese' groups, using a body mass index (BMI) criteria of > 25

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for women and >27 for men to define obesity [1]. Among Europeans, the majority of NIDDM patients are over the age of 50 years and obese [2]. In the Indian subcontinent, the onset of NIDDM is at a younger age and the majority of patients with NIDDM are non-obese [3–5], with a proportion of patients actually being lean with low body mass indices. Although there are a number of studies on the clinical profile of lean NIDDM from different parts of India [6–10], most of them have been based on small numbers of patients and have used varying definitions of 'leanness' with BMI varying from 18 to 20 in different studies [6–10]. At a recent Consensus meeting on Diabetes Peculiar to the Tropics held at Cuttack in India, the subject of 'lean NIDDM' was discussed at length and the need for more studies on the subject based on larger numbers of patients was stressed [11]. At this meeting, a consensus was reached on the definition of leanness, i.e. a body mass index of less than 18.5 [11]. In this paper we present the clinical profile of a large cohort of lean NIDDM seen in South India using the new body mass index criteria.

2. Patients and methods

The mode of selection of patients for the study was as follows: Of consecutively registered patients seen at the M.V. Diabetes Specialities Centre at Madras during a period of 18 months, only patients with NIDDM were included in the study. NIDDM was diagnosed according to the WHO study group report classification of diabetes [1]. All patients who had an abrupt onset of diabetes, acute weight loss, ketosis or ketoacidosis at any time or required insulin from the time of diagnosis were classified as insulin dependent diabetes mellitus (IDDM) and excluded from the study. Patients with fibrocalculous pancreatic diabetes (FCPD), Gestational Diabetes, known cases of secondary diabetes, e.g. endocrine diseases, and those with impaired glucose tolerance (IGT) or lesser degrees of glucose intolerance were also excluded. A total of 392 patients were thus excluded. The process of registration of patients was continued until 10 000 patients diagnosed as NIDDM on clinical grounds were recruited for the study (approximately 96% patients at our centre have NIDDM type diabetes. Out of this number, 127 NIDDM patients had to be excluded from the analysis due to incomplete case notes or inability to undergo certain tests. The study cohort thus consisted of 9873 NIDDM patients.

All patients were studied at the time of first registration at our centre before any changes in treatment was made. A complete clinical workup was done on all patients. Blood pressure was recorded in the right upper limb in the sitting posture using a mercury sphyghmomanometer. Height and weight was recorded and the BMI was calculated using the formula: weight (kg) divided by the height (m^2). Based on BMI, the patients were divided into the following groups: lean NIDDM when the BMI was less than 18.5; ideal body weight (IBW) NIDDM, if the BMI was between 18.5 and 25 in women and 18.5 and 27 in men; and obese NIDDM when BMI was >25 in women and >27 in men.

Biochemical studies were done on Corning Express Plus Auto Analyser (Corning, USA). Fasting and postprandial plasma glucose (glucose oxidase method using Boehringher Mannhiem kit), serum cholesterol (CHOD-PAP method), serum triglycerides (GPO-PAP method), blood urea (modified GLDH kinetic method) and serum creatinine (modified kinetic method of Jaffe) were done on all patients. Glycosylated haemoglobin (HbA_{1c}) was estimated by HPLC method using the Variant machine (Bio Rad, USA). In a randomly selected subgroup of 31 lean NIDDM, 39 patients of IBW and 48 of obese NIDDM, antibodies to GAD and fasting and stimulated C-peptide estimations and in a smaller number, islet cell antibodies (ICA) were carried out using previously described methods [12]. A computerized resting 12 lead electro-cardiogram (ECG), chest radiograph and peripheral doppler studies (Kody Vaslab, Madras) were taken in all cases.

Ocular screening included a routine visual acuity examination using Snellen's test charts. Ocular pressure was recorded using a shiotz tonometer. Biomicroscopic examination of the anterior segment was done routinely in all the cases. One drop of both Phenylephrine 10% and Tropicamide

Table 1
Clinical profile of study groups

NIDDM group	Number (%)	Sex distribution		Duration of diabetes (years)	Mean age at diagnosis of diabetes (years)
		Males ($n = 6177$)	Females ($n = 3696$)		
Lean	347 (3.5%)	233 (67.1%)	114 (32.9%)	9.2 ± 8.1	45 ± 13
IBW	6274 (63.5%)	4744 (75.6%)	1530 (24.4%)	7.9 ± 7.9	45 ± 10
Obese	3252 (32.9%)	1200 (36.9%)	2052 (63.1%)	6.4 ± 6.5	45 ± 10

IBW, Ideal body weight.

1% was then instilled into both eyes and the best possible mydriasis was obtained. Retinal examination was done by both direct and indirect ophthalmoscopy by a trained retinal specialist (MR) who had previously participated in studies in the UK, involving grading of retinal lesions [13]. A study of the vitreous cavity and a detailed examination of the retina with the Goldmann three mirror lens or central fundus lens was done wherever necessary. The following definitions were used for diagnosis of various complications:

Diabetic retinopathy was classified as non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR).

Ischaemic Heart Disease (IHD) was considered to be present when either myocardial ischaemia or infarction was present.

Myocardial Ischaemia was diagnosed if there was a history of exertional chest pain (angina) with unequivocal T wave changes in the electrocardiogram (ECG), but no evidence of infarction.

Myocardial Infarction: A history of myocardial infarction documented by hospital records along with ST/T wave or Q wave changes on ECG suggestive of a recent or past myocardial infarction.

Neuropathy was defined as bilateral absence of ankle jerks and/or bilateral distal sensory neuropathy. The latter was documented by Biothesiometry (Bio Medical Instrument, Newbury, Ohio,) by previously described methods [14].

Nephropathy was defined as persistent proteinuria of more than 500 mg/day in the absence of urinary tract infection or other non-diabetic proteinuria, but in the presence of any degree of retinopathy. Renal insufficiency was defined as serum creatinine > 1.2 mg/dl.

Peripheral vascular disease (PVD) was diagnosed by doppler studies which included a recording of the pressure tracings were done using the KODY Vaslab Machine (Kody Labs, Madras). The Ankle/Brachial (A/B) index was calculated in all cases. An A/B index of 0.8 or less was defined as PVD.

3. Statistical analysis

All analysis was done separately for males and females. Values are expressed as mean $\pm$ S.D. Comparison of mean values was made using Student's t-tests. Log transformation of the data was done wherever necessary. The χ^2 test was used to compare frequencies between different groups. Statistical analysis was done using the SPSS program (Version 4.0.1) on an IBM PC compatible computer. $P < 0.05$ was considered to be statistically significant.

4. Results

The clinical details of the study groups are shown in Table 1. It can be seen that 347 patients (3.5%) of the total NIDDM patients were classified as lean NIDDM. 6274 (63.5%) were of ideal body weight and 3252 patients (32.9%) were obese.

There were 6177 males and 3696 females. This male predominance which has been reported in

Table 2
Clinical and biochemical details in male NIDDM patients

Variable	Lean ($n = 233$) Mean ± S.D.	IBW ($n = 4744$) Mean ± S.D.	Obese ($n = 1200$) Mean ± S.D.
Systolic BP (mmHg)	127 ± 19	133 ± 17*	135 ± 15*
Diastolic BP (mmHg)	80 ± 7	84 ± 12*	86 ± 9*
Blood sugar			
Fasting plasma glucose (mg%)	208 ± 88	171 ± 62*	165 ± 54*
Postprandial plasma glucose (mg%)	310 ± 102	263 ± 78*	255 ± 72*
Serum cholesterol (mg%)	189 ± 42	202 ± 38*	206 ± 41*
Serum triglyceride (mg%)	136 ± 104	186 ± 128*	199 ± 196*
Serum creatinine (mg%)	0.90 ± 0.63	0.82 ± 0.38	0.80 ± 0.24***
Blood urea (mg%)	31 ± 16	27 ± 8*	26 ± 7*
Glycosylated haemoglobin (%)	10.6 ± 1.6	10.2 ± 1.5*	10.1 ± 1.4*

* $P < 0.001$, compared to lean NIDDM; *** $P < 0.05$, compared to lean NIDDM.

our earlier studies [1–3] was seen in the lean and IBW groups. In the obese group, however there was a female predominance.

There were no significant differences in the age at diagnosis or the duration of diabetes between the three study groups. There was no difference in the smoking pattern between the three study groups. Among males, 41 of the lean NIDDM (17.6%), 696 of the ideal body weight group (14.7%) and 205 (17.1%) of the obese group smoked. Only six of the entire female diabetic patients smoked as smoking is very uncommon among Indian women.

Table 2 shows the clinical features of the male NIDDM patients. Both systolic and diastolic blood pressure were significantly lower in the lean NIDDM compared to the IBW and obese groups ($P < 0.001$). Fasting and postprandial plasma glucose were higher in lean NIDDM compared to the obese and ideal body weight groups ($P < 0.001$). Glycosylated haemoglobin (HbA_{1c}) level was also significantly higher in the lean group compared to the other groups. Serum cholesterol and triglycerides levels were lowest in the lean group and were progressively higher in the ideal body weight and obese groups ($P < 0.001$).

Table 3 shows the results in the females which shows similar results as the males with respect to blood pressure, fasting and post prandial plasma glucose levels and glycosylated haemoglobin levels. The serum cholesterol levels were surprisingly higher in the lean group, although this was not statistically significant ($P > 0.2$). The serum triglyceride levels however showed similar results as in the males with higher levels in the ideal body weight and obese groups ($P < 0.001$).

Table 4 shows the results of the GAD antibodies and ICA which were estimated in a randomly selected subgroup of the patients. It can be seen that three out of 31 (9.6%) lean NIDDM compared to two out of 39 (5.1%) IBW NIDDM and two out of 48 (4.2%) obese NIDDM patients were positive for GAD antibodies. The differences between the three groups were not statistically significant. With regards to ICA, none of the lean NIDDM studied, i.e. 0/10 (0%) were ICA positive whereas four out of 30 (13.3%) IBW NIDDM and three out of 40 (7.5%) obese NIDDM showed ICA positivity.

There were no significant differences in the fasting or stimulated C-peptide levels between the three study groups.

Table 5 shows the vascular complications in the study groups. Among males, the prevalence of background diabetic retinopathy (BDR) was significantly higher in the lean group compared to both IBW ($P < 0.05$) and obese groups ($P < 0.001$). Among females, the prevalence of BDR was higher among the lean group but the differences were significant only in comparison to the obese group ($P < 0.05$). The prevalence of proliferative retinopathy was higher in the lean NIDDM group both among males and females.

Table 3
Clinical and biochemical profile of female NIDDM patients

Variables	Lean ($n = 114$) Mean ± S.D.	IBW ($n = 1530$) Mean ± S.D.	Obese ($n = 2052$) Mean ± S.D.
Systolic BP (mmHg)	133 ± 22	134 ± 19	137 ± 18*
Diastolic BP (mmHg)	82 ± 7	83 ± 20	85 ± 8*
Blood sugar			
Fasting plasma glucose (mg%)	211 ± 83	192 ± 68***	183 ± 63**
Postprandial plasma glucose (mg%)	316 ± 92	291 ± 87***	273 ± 79*
Serum cholesterol (mg%)	222 ± 67	216 ± 43	214 ± 41
Serum triglyceride (mg%)	155 ± 75	189 ± 129*	184 ± 113*
Serum creatinine (mg%)	80 ± 0.2	78 ± 0.2	76 ± 0.2
Blood urea (mg%)	27 ± 7	26 ± 7	25 ± 6***
Glycosylated haemoglobin (%)	10.9 ± 1.5	10.5 ± 1.5***	10.4 ± 1.5*

* $P < 0.001$, compared to lean NIDDM; ** $P < 0.01$), compared to lean NIDDM; *** $P < 0.05$, compared to lean NIDDM.

There was no significant difference in the occurence of nephropathy between the three study groups. Renal insufficiency was however more common in the lean NIDDM group but only among males ($P < 0.05$ compared to ideal body weight and obese groups).

Among males, peripheral neuropathy was more common in lean NIDDM group compared to IBW and obese groups ($P < 0.05$), but no significant difference was noted among the three female study groups.

There was no significant difference in the prevalence of myocardial ischaemia or myocardial infarction between the three study groups either in men or women.

The prevalence of peripheral vascular disease (PVD) was surprisingly higher among the lean NIDDM males compared to the obese group ($P < 0.001$) but the number of patients with PVD was low.

Table 6 shows the pattern of treatment given to the patients in the three study groups after the initial studies at our centre were completed. All patients were initially given a trial of oral hypoglycemic agents and if this failed to achieve euglycemia in maximal doses, insulin was given. It can be seen that both among males and females, a higher percentage of the lean NIDDM group received insulin.

5. Discussion

This paper reports on the clinical profile of a large cohort of NIDDM patients attending a diabetes centre in South India. Studies on NIDDM in India are of great interest because of several differences from Europeans [3–5]. The prevalence of NIDDM is very high [4], the prevalence of obesity is low [4,5], the onset of diabetes is at a younger age [3,5], the prevalence of ischaemic heart disease is high [15,16] and that of peripheral vascular disease is low [17]. There has been considerable interest in the entity called lean NIDDM because such a subgroup of NIDDM is rarely seen among Europeans and clinical characterization of this subgroup is obviously of great importance.

In this paper, we report that lean NIDDM comprises about 3.5% of all patients seen at our centre. The prevalence of lean NIDDM varies at different centres depending on the socio-economic status of the people and the type of clinic, e.g. government hospital clinics would be expected to have a larger percentage of lean NIDDM than private diabetic centres. In Orissa State, where poverty is more rampant, up to 22% of patients with NIDDM have been reported to be lean (using BMI < 19 criteria) [7]. Another report [9] found that 17.9% of NIDDM patients seen at Hyderabad were lean, using BMI < 20 as the criteria.

Table 4
ICA and GAD antibody in study groups

	C-peptide (pmol/ml)		Anti-GAD positive	ICA positive
	Fasting	Stimulated		
Lean NIDDM ($n = 31$)	0.74 ± 0.52	1.51 ± 0.89	3/31 9.6%	0/10 0%
IBW NIDDM ($n = 39$)	0.90 ± 0.48	1.74 ± 0.73	2/39 5.1%	4/30 13.3%
Obese NIDDM ($n = 48$)	0.88 ± 0.51	1.88 ± 0.72	2/48 4.2%	3/40 7.5%

None of the differences between the study groups were statistically significant.

The lower serum cholesterol levels seen in our lean male NIDDM patients has also been observed by Das [7]. Comparison of vascular complications in lean NIDDM from different centres is difficult because of small numbers of patients studied and lack of standardized testing procedures. One study [8] suggested that neuropathy was more common in lean NIDDM. Another study [9] suggested that ischaemic heart disease was less common among lean NIDDM, but the differences were not statistically significant.

In this study we report that retinopathy (both background and proliferative) is more frequent among lean NIDDM, both in males and females. Additionally, renal insufficiency and neuropathy are more common among lean male NIDDM patients. The higher prevalence of these complications is probably related to the higher plasma glucose and glycosylated haemoglobin levels. The higher prevalence of peripheral vascular disease is intriguing but this may be an artefact due to the small numbers of patients with PVD. We have reported previously on the lower prevalence rates of PVD in South Indian NIDDM patients [17].

It is not surprising that a much higher percentage of patients were treated with insulin among the lean group. This could be due to several reasons: the diabetes is more severe [6,8,18,19] and the leanness itself could have influenced the use of insulin. It is of interest however, that nearly 47% of the lean male NIDDM and 53% of the lean female NIDDM patients were successfully treated with diet and oral hypoglycaemic agents, even after a mean duration of 9.2 ± 8.1 years of diabetes.

The grossly elevated glycosylated haemoglobin and plasma glucose values probably reflect the poor level of primary care treatment of diabetes in our country. All patients were studied at the time of first registration at our centre before optimal regulation of their diabetes had been done. Obviously, after treatment at our centre, in all patients, better control of diabetes was achieved. The results of the follow-up studies after initiation of therapy are still not available. However, preliminary results show that with control of diabetes there is a modest weight gain with an increase of BMI, approximately by a factor of one, in all three groups which did not change the classification of the patients in the majority of cases. This also has been shown by other groups [6–9].

One of the objections to this study might be that the so-called 'lean NIDDM' patients may in reality be (or may have included) patients with IDDM. This study being based on large numbers, we could not perform C-peptide estimations or look at immunological markers in the whole group. However as shown in Table 4, in a small subgroup ($n = 31$) of newly diagnosed 'lean NIDDM' we performed fasting and stimulated C-peptide estimations as well as looking at antibodies to glutamic acid decarboxylase (GAD) and in a smaller subgroup, antibodies to islet cell antibodies (ICA). The results show that while the C-peptide values were marginally lower in the lean NIDDM, they were not significantly different from the other two groups of NIDDM studied. Moreover the values were significantly higher than that seen in our IDDM patients who have a mean fasting value of 0.09 ± 0.10 pmol/ml and a mean stimulated value of 0.14 ± 0.08 pmol/ml [12]. The results of the GAD and ICA positivity in this group of lean NIDDM were also not significantly different from the IBW and obese

Table 5
Vascular complications in the study groups

Complication	Males			Females		
	Lean ($n = 233$)	IBW ($n = 1200$)	Obese ($n = 114$)	Lean ($n = 114$)	IBW ($n = 1530$)	Obese ($n = 2052$)
Retinopathy BDR	87 (37.3%)	1461 (30.8%)***	253 (21.1%)*	38 (33.3%)	468 (30.6%)	476 (23.2%)***
PDR	16 (6.9%)	199 (4.2%)	25 (2.1%)*	12 (10.5%)	61 (4.0%)**	47 (2.3%)*
Nephropathy	11 (4.7%)	218 (4.6%)	74 (6.2%)	5 (4.4%)	64 (4.2%)	74 (3.6%)
Renal insufficiency	22 (9.4%)	195 (4.1%)*	34 (2.8%)*	3 (2.6%)	57 (3.7%)	41 (2.0%)
Peripheral neuropathy	104 (44.6%)	1480 (31.2%)*	322 (26.8%)*	44 (38.6%)	505 (33.0%)	649 (31.6%)
IHD:ischaemia	31 (13.3%)	522 (11.0%)	114 (9.5%)	16 (14.0%)	282 (18.4%)	328 (16.0%)
Infarction	13 (5.6%)	289 (6.1%)	62 (5.2%)	8 (7.0%)	83 (5.4%)	107 (5.2%)
PVD	12 (5.2%)	161 (3.4%)	17 (1.4%)*	8 (7.0%)	57 (3.7%)	105 (5.1%)

BDR, background diabetic retinopathy; PDR, proliferative diabetic retinopathy; IHD, ischaemic heart disease; PVD, peripheral vascular disease.
* $P < 0.001$ compared to lean NIDDM; ** $P < 0.01$ compared to lean NIDDM; *** $P < 0.05$ compared to lean NIDDM.

NIDDM groups but different from our IDDM patients [12]. Hence, it appears likely that the vast majority of lean NIDDM group do indeed have NIDDM and not IDDM or latent autoimmune diabetes of adults (LADA) [20] type diabetes masquerading as NIDDM.

Moreover the mean age of the lean NIDDM group (45 ± 13 years) is about two decades higher than that of the IDDM patients seen at our centre. Finally, the fact that none of the patients were ketotic and that nearly 48% of the lean NIDDM patients still responded to diet or oral hypoglycemic agents after a mean duration of 9.2 ± 8.1 years, clearly distinguishes the lean NIDDM from IDDM patients.

In summary, we present clinical data on a large cohort of lean NIDDM seen in Southern India, which suggests that it probably represents a more severe form of diabetes with a higher risk of vascular complications. This underscores the need for aggressive treatment of diabetes. Obviously more studies are needed on the metabolic, hormonal and immunological characterization of lean NIDDM patients seen in India.

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Table 6
Treatment pattern in the study groups

Variable	Male			Female		
	Lean ($n = 2333$)	IBW ($n = 4744$)	Obese ($n = 1200$)	Lean ($n = 114$)	IBW ($n = 1530$)	Obese ($n = 2052$)
Treatment						
Diet alone	10 (4.3%)	62 (1.3%)*	12 (1.0%)*	2 (1.8%)	11 (0.7%)	21 (1.0%)
Oral drugs	100 (42.9%)	3449 (72.7%)*	976 (81.3%)*	58 (50.9%)	1018 (66.5%)*	1473 (71.8%)*
Insulin	123 (52.8%)	1233 (26.0%)*	212 (17.7%)*	54 (47.4%)	501 (32.7%)**	558 (27.2%)*

* $P < 0.001$ compared to lean NIDDM; ** $P < 0.01$ compared to lean NIDDM.

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