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Impact of Hypertension on Type 2 Diabetes in Mysore Population of South India*

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ABSTRACT

Objective: The study aims to explore the prevalence of hypertension and its impact on Type 2 diabetes in a Mysore population of the Indian subcontinent. **Methods:** 636 participants volunteered for the study. Anthropometric measurements and blood pressure were recorded while plasma was analyzed for biochemical markers. The IDF and JNC 7 diagnostic criteria were followed to define diabetes and hypertension. **Statistical Analyses:** One-way analysis of variance, χ^2-test and Logistic regression analysis were performed to assess differences of the mean, proportion and the independent effect of hypertension on the development of type 2 diabetes. **Results:** Hypertension was observed to be prevalent in 37.1% of the studied population with an insignificant gender difference. Rate of occurrence of hypertensives was found to be significantly higher in type 2 diabetes (51.9%), obese subjects (45.2%), long-term smokers (49%) and alcohol addicts (48%) than control groups. The risk of development of diabetes was significantly higher in hypertensives than normotensive. However, when creatinine and blood urea nitrogen were included in the model, the significance was nullified. **Conclusions:** The prevalence of type 2 diabetes and hypertension is increasing at an alarming rate. This study reveals that the significance of hypertension as a parameter in predicting the risk of type 2 diabetes was influenced by the renal function and lipid profile.

Keywords: Hypertension; Type 2 Diabetes; Prevalence; Kidney Dysfunction; Mortality; Mysore Population; India

1. Introduction

The incidence of Type 2 Diabetes (T2D) is increasing globally from 2.8% in 2000 to 4.4% in 2030 [1]. The prevalence of T2D in Asian Indians ranges from 2.7% in rural India to 14% in urban India. India has the highest number of diabetes in the world [2,3]. The National Urban Diabetes Survey reported 12.1% of diabetes and 14% of impaired glucose tolerance [4]. The prevalence of hypertension (HTN) among adults is expected to rise by 60% resulting in a total of 1.56 billion affected individuals by 2025. Approximately 70% of diabetics are hypertensives, as diabetics are prone to HTN twice more likely than normoglycemic individuals [5]. Similarly, the presence of HTN precedes the onset of diabetes mellitus (DM) [6,7]; and among diabetics, HTN develops into diabetes nephropathy and retinopathy. The co-occurrence of HTN and T2D affects up to 60% of patients leading to higher risk of developing cardiovascular morbidity and mortality [8]. Though cardiovascular risks are common in both, in conjunction they accelerate cardiac, cerebral and renal dysfunctions [9]. The United Kingdom Prospective Diabetes Study (UKPDS) revealed that blood pressure control helps to avoid cardiovascular complications in patients with T2D [10]. The decrease in mean systolic blood pressure by 10 mm/Hg reduces the risks of developing complications in diabetes by 12%, mortality by 15%, myocardial infraction by 11% and microvascular complications by 13% among diabetics respectively [11].

The prevalence varies across different ethnic and religious groups in Asia; the co-occurrence of diabetes with HTN shows an increasing trend and has become an epidemic of a great concern [12]. About 50% of diabetes cases in India show the co-occurrence of HTN [13,14].

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Diabetes in other Asian countries such as Saudi Arabia (53%) [15,16], Jordan (72.4%) [17], Oman (21.5%), Turkey (32%), Bahrain (38%) and Taiwan (39%) [18-21] also shows a similar trend. In addition, studies reported higher tendency of HTN among UK Afro-Caribbean (82%) [22], UK Caucasian (74%), Italian (74.4%) and Spanish diabetes (73%) [23-25].

Few epidemiological studies asserting incidence rates of T2D and HTN have been carried out in various sectors of Karnataka. In the rural population of Davanagere, 18.3% of HTN has been reported, where males recorded a higher prevalence rate (19.1%) than females (17.5%) [26]. Heritability of HTN in families of Tumkur population was reported, wherein the young normotensive with a positive family history of HTN had significantly higher blood pressure [27]. In Karnataka, the prevalence of T2D has been observed to be 3.77% of Suttur population [28], 10.0% in Kolar population [29], 16% of the Udupi population [30] and 17.3% in Dharwad urban population [31]. The incidence of obesity among T2D of Mysore population was reported [32], while the awareness of diabetes and their attitude to patients of Bijapur have been reported [33]. But there is no known record of the prevalence of HTN among T2D or vice versa, implying how frequent HTN exacerbates T2D in southern India.

Currently, there are limited epidemiological studies edifying the relationship between T2D and HTN in Indian context. There is an ongoing debate regarding the consideration of high blood pressure over other metabolic components (conjointly involved in T2D and HTN), as a predictor of T2D in Indians. Further, social and cultural diversity in India necessitates the exploration of the mentioned relationship in various sections of this country. Therefore we hypothesize that the risk of incidence of T2D is higher in the subjects with HTN. The present study aims to assess the prevalence of HTN among T2D subjects and its contribution in the occurrence of T2D in Mysore population of Karnataka in South India.

2. Materials and Methods

2.1. Study Population

This case-control study was conducted among participants in the diabetes health check-up programs organized by Amrita Kripa Polyclinic and Lion's Club of Mysore (R) in Mysore district of Karnataka State, India, during 2010 to 2011 including both non-diabetes and diabetes patients, without any mental impairment.

2.2. Sample Size

A total of 654 subjects volunteered and gave consent to participate in the study, out of which 636 were included in the study. The subjects including 343 males and 293 females, aged between 30 - 80 years were enrolled for the study. Subjects with abnormal renal or chronic liver dysfunction were excluded from the study.

2.3. Sampling Procedure

The study protocol was reviewed and approved by the Institutional Ethics Committee, Kolkata and also the Ethical Committee of University of Mysore. Informed consents were obtained from each participant in the study. The study was conducted according to the ethical guidelines for biomedical research on human populations (http://icmr.nic.in/ethical_guidelines, ICMR 7). Each participant of the study was about 12 hours of fasting period before the collection of blood. 5 ml blood sample was collected in 10 ml BD vacutainer by a phlebotomist, stored at 4˚C and transported to the laboratory immediately for further processing. Postprandial plasma glucose was measured after 2 hours of administering 75-grams of glucose to the subjects (OGTT, WHO, 1999).

2.4. Data Collection

Questionnaire: Data was collected on standardized questionnaire that included personal information, lifestyle, habitual behaviors (smoking and alcohol intake), clinical history of associated complications and blood pressure was recorded under the supervision of a physician.

Anthropometry: Height, weight, waist and hip circumference were measured by physical anthropologists using anthropometer (Holtaine, UK) and digital weighing machine (Tanita Corporation, Tokyo, Japan) as per WHO international manual [34]. Waist circumference (WC) was measured at the midpoint at the bottom of the rib cage and the top of the lateral border of the iliac crest during minimal respiration.

Laboratory Examination: Fasting plasma glucose (FPG), Glycated Haemoglobin (HbA1c), High-density lipoprotein (HDL), Low-density lipoprotein (LDL), Total Cholesterol (CHO), Triglycerides (TRIG), Creatinine (CRE), Blood urea nitrogen (BUN) and Postprandial glucose (PPG) were measured on Auto analyzer EM 360 (Transasia, ERBA Mannheim, Germany).

Operational Definitions: Body mass index (BMI) was calculated as weight in kilograms divided by the squared value of height in meters (kg/m^2). BMI was categorized as normal (<25 kg/m^2), overweight (>25 and < 30 kg/m^2), and obese (>30 kg/m^2) [35]. Blood pressure (Systolic and Diastolic) of each subject was measured using a standardized sphygmomanometer (Elko, India), in supine position. An average of two readings of both systolic (SBP) and diastolic blood pressure (DBP) was taken. HTN was defined following the criteria of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7

criteria) [36]. Participants were divided (as per their baseline BP) into, normotensive (SBP < 120 mmHg and DBP < 80 mmHg), Pre-hypertensive (SBP 120 - 140 mmHg and DBP 80 - 90 mmHg) and Hypertensive (SBP > 140 mmHg and/ or DBP > 90 mmHg), or presently taking anti-hypertensive medication. Further, hypertensives (presently taking anti-hypertensive medication or/ and with a history of HTN diagnosed by a medical physician) including the pre hypertensive were combined in one group opposed to normotensive controls. Self reported cases and individuals with FPG > 126 mg/dl and PGLU > 200 mg/dl were defined as diabetes [37]. Diabetics under treatment and long term management of blood glucose were defined as controlled diabetes with HbA1c values 6% - 8%. On the contrary above 8% were considered uncontrolled diabetes [38]. All self-reported cases were further validated by medical record review and supplementary questionnaires.

2.5. Statistical Analysis

The analysis has been carried out after segregating the cases and controls further into hypertensives and normotensives, resulting into four groups in total. The results have been reported as mean ± standard deviation. One-way analysis of variance (ANOVA) was used to analyze the statistical differences in the mean of various parameters between the groups. χ^2-test was used to compare the proportion of the subjects between different dichotomized variables. Logistic regression analysis was performed to assess the independent effect of hypertensive status on the odds of occurrence of diabetes, after adjusting for confounders. Statistical analyses were performed using SPSS version 12.0 software (SPSS, Chicago, IL, USA). All the reported P-values were two-tailed, and those less than 0.05 were considered statistically significant.

3. Results

Table 1 shows the distribution of HTN across sex, age groups, diabetes status and addiction habits. The overall prevalence rate of HTN was found to be 37.1%, 33.8% of male and 41% in females. However, the intergroup difference is statistically similar (P > 0.05). Among different age groups, the rate of HTN increased with age from

Table 1. Distribution of hypertension across age, gender, BMI, habits and clinical history.

Variables	Category	Normotensive		Hypertensive		Total		P value
		N	%	N	%	N	%	
Diabetes status	Type 2 Diabetes	142	48.1	153	51.9	295	100	<0.001
	Non Diabetes	258	75.7	83	24.3	341	100	
Sex	Female	173	59	120	41	293	100	0.63
	Male	227	66.2	116	33.8	343	100	
Age groups	30 - 39	79	86.8	12	13.2	91	100	<0.001
	40 - 49	141	76.2	44	23.8	185	100	
	50 - 59	67	50.8	65	49.2	132	100	
	60 - 69	70	47.6	77	52.4	147	100	
	70 - 79	34	50	34	50	68	100	
	80+	9	69.2	4	30.8	13	100	
BMI groups	Normal (<25)	179	69.6	78	30.4	257	100	0.009
	Overweight (25 - 30)	158	59.8	106	40.2	264	100	
	Obese (>30)	65	54.8	53	45.2	118	100	
Smoking	Yes	76	71.7	30	28.3	106	100	0.03
	No	296	62.3	179	37.7	475	100	
	Quit	28	50.9	27	49.1	55	100	
Alcohol intake	Yes	100	67.6	48	32.4	148	100	NS
	No	287	62	176	38	463	100	
	Quit	13	52	12	48	25	100	
Glycated haemoglobin A1c	<6%	270	71.8	106	28.2	376	100	<0.001
	6% - 8%	87	50.9	84	49.1	171	100	
	>8%	43	48.3	46	51.7	89	100	
	Total	400	62.9	236	37.1	636	100	

30 - 69 years and then gradually decreased in subjects of 70 years and above, and the trend was found to be significant ($P < 0.001$). Unconventionally, HTN was significantly less prevalent among smokers than non-smokers whereas long term ex-smokers showed the highest prevalence ($P < 0.05$). A similar trend was observed among alcoholics but was found to be statistically insignificant. Obese subjects were observed to have significantly ($P < 0.01$) higher prevalence rate of HTN (45.2%) than overweight (40.2%) and normal (30.2%) subjects. HTN was found to be more prevalent ($P < 0.001$) among diabetics (51.9%) than non-diabetics (24.3%). This was further substantiated by the significantly ($P < 0.001$) higher prevalence rates among uncontrolled diabetes (51.7%) and controlled diabetes (49.1%) subjects than non-diabetic subjects (28.2%) as categorized by the HbA1c levels.

The variables are described in **Table 2** after classifying the subjects into four groups based on clinical history of diabetes and/or HTN. HTN was more prevalent in subjects of the older age group, irrespective of the presence or absence of T2D. Both diabetes normotensive and hypertensive subjects were observed to have a significantly higher waist circumference than non-diabetic normotensive subjects. Individuals affected by both of the mentioned disorders had higher BMI and CRE levels than normal controls. As anticipated, both systolic and diastolic blood pressure was higher among hypertensive groups than normotensive. Fasting glucose, postprandial glucose, glycated haemoglobin and triglyceride levels were significantly higher in the diabetes groups, irrespective of HTN. On the contrary, HDL levels were found to be low in the diabetes subjects. Levels of BUN were found to be significantly higher in diabetes hypertensives when compared to diabetes normotensive. CHO and LDL levels showed no significant difference between the groups.

The influence of HTN on risk for development of T2D was analyzed based on HTN status after adjusting the probable cofounder and has been depicted in **Table 3**. The crude odds of occurrence of T2D due to HTN was 3.349 ($P < 0.0001$). Although the odds ratio was reduced after adjusting for the confounders; age, sex, smoking, alcoholism, BMI yet the level of significance was constant. The significance levels were reduced to $P < 0.05$ after the inclusion of FPG, PPG and HbA1c to the previously mentioned models. Though the inclusion of CRE and BUN in the model distorted the significance, but the significance completely disappeared when adjusted for TRIG, CHO, LDL and HDL. When possible confounders were controlled, the odds ratio was found to be 1.45 and was observed to be insignificant. The confounding variables that were found to be significant on inclusion of all variables in the model were Age (OR = 1.053, P =

Table 2. Characteristics of the subjects grouped based on presence or absence of diabetes and hypertension.

Biomarker	Diabetic hypertensive	Diabetic normotensive	Nondiabetic hypertensive	Nondiabetic normotensive
	Mean ± S.D	Mean ± S.D	Mean ± S.D	Mean ± S.D
	N = 153	N = 142	N = 83	N = 258
Age (years)	59.04 ± 10.50$^{*\#}$	54.26 ± 12.49*	56.34 ± 11.65*	48.33 ± 12.81
Waist (cm)	92.83 ± 11.45$^{*\wedge}$	89.98 ± 12.51*	88.44 ± 11.18	86.21 ± 11.05
Body mass index (kg/m)^2	27.37 ± 4.51*	26.04 ± 4.25	26.71 ± 5.18	25.48 ± 4.55
Systolic blood pressure (mmHg)	152.51 ± 15.68$^{*\#}$	120.86 ± 12.97$\wedge$	153.88 ± 19.25*	110.87 ± 12.11
Diastolic blood pressure (mmHg)	92.48 ± 12.13$^{*\#}$	82.56 ± 10.79$\wedge$	96.07 ± 11.71*	80.08 ± 9.93
Fasting glucose (mg/dl)	142.56 ± 59.19$^{*\wedge}$	145.44 ± 58.93$^{*\wedge}$	90.20 ± 11.17	88.45 ± 9.97
Postprandial glucose (mg/dl)	228.16 ± 97.34$^{*\wedge}$	228.57 ± 93.62$^{*\wedge}$	115.71 ± 24.45	109.39 ± 22.71
Triglyceride (mg/dl)	184.13 ±111.94$^{*\wedge}$	186.65 ±105.48$^{*\wedge}$	149.05 ± 85.89	143.86 ± 91.58
Cholesterol (mg/dl)	175.18 ± 45.78	174.06 ± 51.09	171.78 ± 49.75	168.58 ± 47.08
High density lipoprotein (mg/dl)	44.74 ± 9.95$^{*\wedge}$	47.09 ± 10.72$^{*\wedge}$	50.49 ± 7.95	50.08 ± 8.31
Low density lipoprotein (mg/dl)	101.79 ± 23.63	102.70 ± 22.15	100 ± 21.76	98.05 ± 20.42
Creatinine (mg/dl)	1.05 ± 0.49*	0.99 ± 0.32	0.98 ± 0.36	0.95 ± 0.32
Blood urea nitrogen (mg/dl)	10.30 ± 4.1$^{\#}$	9.12 ± 3	9.78 ± 3.24	9.43 ± 3.32
Glycated haemoglobin A1c (%)	7.45 ± 2.09$^{*\wedge}$	7.25 ± 2.13$^{*\wedge}$	5.11 ± 1.36	4.95 ± 1.06

$^{*}P < 0.05$ as compared to nondiabetes normotensive. $\wedge^{\#}P < 0.05$ as compared to diabetes normotensive. $P < 0.05$ as compared to nondiabetes hypertensive.

Table 3. Logistic regression analysis for assessing risk of diabetes according to hypertension status.

Models	Odds ratio (Hypertensive vs. normotensive)	CI interval	P value
1	3.349	2.392 - 4.690	<0.0001
2	2.940	2.057 - 4.203	<0.0001
3	2.953	2.058 - 4.236	<0.0001
4	2.589	1.789 - 3.746	<0.0001
5	2.220	1.225 - 4.024	0.009
6	1.862	1.007 - 3.444	0.048
7	1.830	0.984 - 3.403	0.056
8	1.431	0.723 - 2.831	0.303
9	1.452	0.732 - 2.881	0.286

Model 1: Unadjusted. Model 2: Adjusted for Age and gender. Model 3: Adjusted for model 2 + smoking + Alcoholism. Model 4: Adjusted for model 3 + BMI. Model 5: Adjusted for model 3 + FPG + PPG + HbA1c. Model 6: Adjusted for model 5 + BMI. Model 7: Adjusted for model 6 + CRE + BUN. Model 8: Adjusted for model 6 + TRIG + HDLC + LDLC + CHO. Model9: Adjusted for model 8 + CRE + BUN.

0.001), FPG (OR = 1.06, P < 0.0001) PGLU (OR = 1.043, P < 0.0001), HbA1c (OR = 1.06, P < 0.0001), BMI (OR = 1.099, P < 0.01), HDLC (OR = 0.863, P < 0.0001) and LDLC (OR = 1.033, P < 0.004).

4. Discussion

Several studies have reported the prevalence of HTN and T2D in rural as well as urban India. However, studies linking HTN to T2D in India are minimal. According to Screening India's Twin Epidemic (SITE) study, the prevalence rate of the co-occurrence of HTN and T2D in individual of eight states was 20.6%, with 34.7% of T2D and 46% of HTN. In Karnataka, the frequency of co-occurrence of HTN and T2D is 17.4%, whereas HTN and T2D occur alone at 32.1% and 34.5% respectively [39]. In the present study, cooccurrence of HTN and T2D was observed to be 24.1% of the Mysore population with a higher prevalence rate of HTN (37.1%) and T2D (46.4%) than reported by SITE study for the entire Karnataka population. Our study shows a higher rate of incidence of these disorders within Karnataka, more specifically in populations of Mysore district. The incidence of HTN among T2D patients in the present study population (51.9%) is comparable to T2D incidence in Kashmir (42%) and varies from other populations of India [13-25]. The differences observed in the incidences of HTN in T2D among different populations can possibly be attributed to ethnicity, population dispersion, physical characteristics and the multiple definitions adopted for T2D/ HTN and surveillance procedures in the previous studies.

Studies on the glycemic control stages marked by HbA1c levels are limited in various populations. Studies have shown that intensive blood sugar control is effective in reducing the risk of HTN by approximately 25%. Our findings are in conformity with earlier studies, where the incidence of HTN among T2D subjects having glycemic control (HbA1c < 8%) is lower than in uncontrolled group (HbA1c > 8%) [40,41]. Customarily, hypertensivity in diabetes advances with age as reported in other studies [42-45]. Contrastingly, we observed a lag in the occurrence of HTN in subjects beyond the age of 70 years, which can be attributed to the lower levels of BMI. In accordance with earlier findings higher BMI group showed a high prevalence rate for HTN [44-46]. Further, high prevalence of HTN among ex-smokers projects the probable association of the disorder with the duration of addictive behaviors. An unusual trend has been reported in prior studies of higher prevalence of hypertensives amongst former smokers and non smokers than smokers which harmonize with our results [47,48]. The possible answer could be the unwillingness of individuals to disclose their addictive behavior and also occurrence of obese/overweight subjects under the smokers' category.

It has been established that co-existence of T2D and HTN accelerates the progression of metabolic abnormalities more than their independent outcomes. Hence, there is always a chance of significant variability in metabolic characteristics between individuals suffering independently with both disorders or with the coexistence [49]. In our study the population is categorized into four groups (Non diabetes normotensive, Non diabetes hypertensive, diabetes normotensive and diabetes hypertensive) referring to four conditions, with significant observed differences among the groups. Insulin resistance in T2D causes inhibition of lipolysis leading to hyperinsulinemia and elevated triglyceride [50]. Our findings are in agreement with the aforesaid inference, wherein elevated levels of fasting glucose, postprandial glucose, glycated

haemoglobin, triglyceride levels of FPG, PPG, HbA1c, TRIG and low levels of HDL levels were observed in diabetes hypertensives and normotensive groups. Our finding is in concordance with earlier studies, suggesting the association of T2D with dyslipidemia, particularly with high triglycerides accompanied with a simultaneous decrease in HDL cholesterol [51]. Positive association between HTN and abnormal lipid profiles in rural population of Bagalkot in Karnataka [52] and among hospital patients have been reported in previous studies on Indian population [53,54].

Elevated BUN has been reported to be a marker for activating the sympathetic nervous system and an unregulated rennin angiotensin system. Thus increased levels of BUN in T2D with HTN distinctly reveals the risk of renal and atherosclerotic complications in diabetes hypertensives than diabetes normotensive [55-59]. In the present study, the co-incidence of T2D and HTN as defined by higher BMI and CRE levels strengthens the notion that obesity and renal dysfunction are predictors of T2D associated with HTN.

Our study indicates that HTN plays a major role in the development of T2D, after the confounding effects of age, sex, BMI, glycemic index (FPG, PPG and HbA1c), lipid profile, CRE and other relevant factors had been adjusted. Our result concurs with the recent findings that aging, obesity, dysglyceamia and dyslipidemia co-exist with HTN and T2D [17,60,61]. Studies reported a precise and prominent role of HTN in the prevalence of T2D with crude relative risks of 2.34 (CI of 2.16 - 2.73) [62] and 2.65 (CI of 1.88 - 3.73) [63], relatively lower than the ratio obtained in our study. This evidently suggests that Mysore population is at a higher risk of T2D due to HTN. Previous studies on Asian populations proposed that baseline hyperglycemia and BMI are potential covariates determining the association of HTN with T2D. Thus, baseline HTN may be a potential predictor for incident diabetes, if the onset of diabetes is defined using parameters like FPG and PPG levels [61]. However, it has been concluded that obesity and metabolic syndrome does not explain the entire association between BP and incidence of T2D [63]. Hence, besides BMI, lipid profile and Glycemic index, we included CRE and BUN into these models. Accumulation of BUN and CRE is a direct indicator of renal dysfunction. Thus higher levels of these are observed in hypertensives, more precisely in untreated hypertensives and diabetes nephropathy cases [64]. Furthermore, it has been reported that renal failure rate is two to three times higher in patients of diabetes hypertensives than in non diabetes hypertensives [65]. Therefore, inclusion of these predictors can ascertain the association between T2D and HTN. Lack of correction for the predictor variables related to renal dysfunction can be one of the reasons for discrepancies in the earlier studies [61,62].

5. Limitations and Strengths

This study is the first of its kind in South Indian population. Glycemic index is defined more explicitly in this study by performing OGTT and HbA1c estimations compared to earlier studies, which failed to include glucose tolerance [62,66]. Some of the major limitations of this study are complete exclusion of the effects of residual confounding due to measurement errors, *i.e.* in the assessment of confounding factors or unmeasured dietary, social and economic factors. Data could not be stratified gender wise because of small sample size. The sample population was heterogeneous consisting of both urban and rural inhabitants, thus findings cannot be generalized to other populations of India. Although clinical data are available, duration and history of diabetes and HTN was not taken into consideration for the study. Some of the salient markers like micro albumin, urea and insulin are beyond the scope of this paper. Non availability of information regarding the treatment for HTN in subjects of the hypertensive group, to see the effect of treatment of diabetes, was one of the lacunae.

6. Conclusion

The high prevalence rate of T2D and HTN is major concerns in Mysore population. HTN plays a key role in the progression of T2D and is associated with vascular complications. Among hypertensives, BMI, Glycemic index, lipid profile and kidney dysfunction, markers are potential predictors of T2D. The assessment of nephropathic markers besides analyzing metabolic components and blood pressure management is better approaches to prevent the risk of development of T2D in hypertensives.

7. Authors' Contributions

MDS conceived and executed the study. SDG helped in sample collection, laboratory work and statistical analysis. CDS supervised the research, proof read the draft. DDX , BVR, JSR and LGL contributed in formulation of data collection. All authors read and approved the final manuscript.

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