International Journal of Diabetes in Developing Countries (April–June 2025) 45(2):373–379
https://doi.org/10.1007/s13410-024-01365-x

ORIGINAL ARTICLE

Incidence and predictors of diabetes mellitus: A 7- year community cohort follow-up of urban, adult Sri Lankans

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Received: 4 April 2023 / Accepted: 29 May 2024 / Published online: 10 June 2024

Abstract

Background There is limited data on the prevalence and outcome of prediabetes and the incidence of type 2 diabetes in South Asia.

Objective We conducted a prospective, community-based study involving a cohort of urban adults in Sri Lanka, with a seven-year follow-up period.

Methods Participants were selected using age-stratified random sampling, initially screened in 2007, and reevaluated in 2014. Structured interviews, anthropometric measurements, liver ultrasound, and biochemical and serological tests were performed on both occasions.

Results A total of 2985 individuals were recruited in 2007 [54.8% women, median age (IQR) 53(47–59)]; 737 had diabetes [baseline prevalence 24.7% (95%CI:23.1–26.2)] and 525 had prediabetes [baseline prevalence 17.9% (95%CI:16.2–19.6)]. In 2014, 2148(71.6%) persons attended follow-up [57.5% women; median (IQR) 60(54–66) years], which included 1650 who did not have diabetes in 2007. By 2014, 436/1650(27.6%) persons had developed new diabetes [annual incidence 3.9% (95%CI:3.0–4.9)]. Poisson regression analysis showed that prediabetes, central obesity, low HDL, and nonalcoholic fatty liver disease at baseline were significantly associated with new-onset diabetes.

Of the 525 persons with prediabetes in 2007, 365(69.5%) were followed-up in 2014; 147/365(40.3%) remained in prediabetes, 201/365(55.1%) had progressed to diabetes, and 17/365(4.6%) had reverted to normoglycemia. The annual conversion rate of prediabetes to diabetes was 7.9%. An increase in waist circumference and low HDL levels from baseline predicted progression to diabetes.

Conclusions The presence of prediabetes, central obesity, low HDL, and nonalcoholic fatty liver disease at baseline predicted new-onset diabetes, whereas increase in waist circumference and low HDL predicted conversion of prediabetes to diabetes. Targeted lifestyle interventions are essential for individuals with metabolic risks to prevent future diabetes.

Keywords Diabetes · Prediabetes · Incidence · Predictors · NAFLD · Sri Lanka

Introduction

Diabetes ranks seventh in the global burden of disease [1]. The prevalence of type 2 diabetes (T2D) among adults aged 20–79 years is expected to increase from 463 million in 2019 to 578 million by 2030 and 700 million by 2045 [2]. The highest percentage increases in disease prevalence are likely to be in non-Western and developing nations, with significant increases in the Middle East, sub-Saharan Africa, India, Asia, and Latin America [3]. Asian countries currently contribute more than 60% of the world's diabetic population [4], and India is known as the world's diabetes capital [5].

Prediabetes is an at-risk state for the development of future T2D [6]. It is defined as an impaired fasting glucose

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(IFG) or impaired glucose tolerance (IGT) [2]. The American Diabetes Association defines prediabetes as HbA1c 5.7–6.4% [7]. Individuals with either IFG or IGT have an increased risk of developing T2D and a higher prevalence of cardiovascular disease than normoglycemic individuals [8]. Prediabetes has an annual global conversion rate to T2D of 5–10%[6]. However, prospective community-based studies and information on the outcomes of prediabetes in the South Asian region are scarce.

Obesity is a significant predictor of T2D [9]. Obesity and T2D are independent predictors of the development of cardiovascular disease [10], which is the leading cause of death worldwide among adults [11]. The early detection of modifiable risk factors (such as obesity), prediabetes, and T2D will help reduce the cardiovascular disease burden in any community. Detecting at-risk populations and implementing risk-reduction strategies is the mainstay for reducing the incidence of T2D worldwide [12].

Although there are many reports on the prevalence of T2D, there are not many studies on its incidence. Very few community-based cohort studies have reported incident T2D, especially the South Asian region [2].

The Ragama Health Study (RHS) is an ongoing, prospective study of non-communicable diseases in an extensive community cohort. The RHS is a collaboration between the Faculty of Medicine, University of Kelaniya, Ragama, Sri Lanka, and the National Center for Global Health and Medicine, Tokyo, Japan [13]. The original cohort was recruited in 2007, and the study was conducted in the Ragama Medical Officer of Health (MOH) area in the District of Gampaha, Sri Lanka. The objective of the current study was to investigate the incidence and predictors of T2D and the outcome of prediabetes in this cohort after seven years of follow-up (Fig. 1).

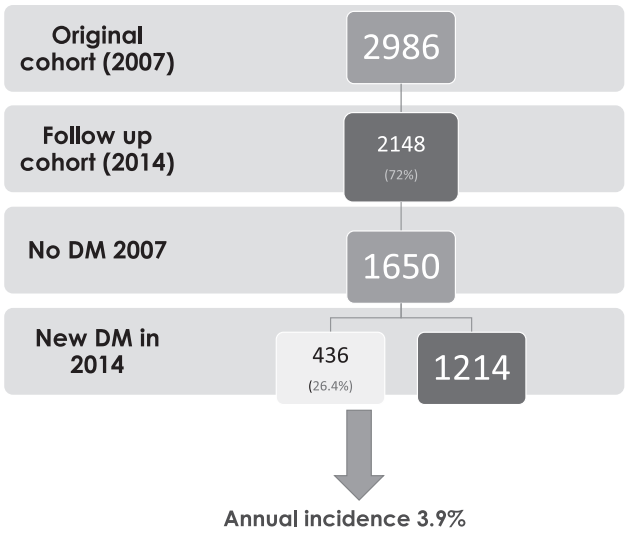

Fig. 1 Follow up population. DM = Type 2 diabetes

Methods and Materials

The Ragama MOH administrative area is situated approximately 18 km north of Colombo, the commercial capital of Sri Lanka. Ragama is a bustling urban township with a multi-ethnic population. Participants in the RHS were resident adults initially selected from electoral lists using age-stratified random sampling. The cohort was initially screened in 2007 when participants were aged 35–64 years and was invited back for re-evaluation after seven years in 2014 when participants were aged 42–71 years.

In 2007 and 2014, structured interviews were used to assess participants who also underwent clinical assessment, anthropometric measurements, ultrasound scanning of the liver, and biochemical/serological tests. Further details regarding screening of the inception cohort are described elsewhere [13].

On both occasions, trained personnel interviewed the cohort to obtain relevant information, including sociodemographic variables and lifestyle habits (diet, alcohol consumption, and physical activity). The medical records of the subjects, whenever available, were analysed to obtain more details. Blood pressure (BP) and anthropometric parameters including weight, height, and waist circumference (WC) were measured using standard methods. Changes in WC were classified as a reduction of $>5\%$, a reduction of $\leq 5\%$, an increase of $<5\%$ (no change), and an increase of $\geq 5\%$. Changes in weight were classified as loss $>5\%$, loss $\leq 5\%$,gain $<5\%$ (no change), gain $\geq 5\%$, and gain $\geq 10\%$ [14]. Total body fat (TBF) and visceral fat percentage (VFP) were measured with a body composition monitor using the bioelectrical impedance method (Omron HBF-362 body composition monitor, Omron Healthcare, Lake Forrest, Illinois, United States). Abnormal TBF was defined as TBF $>32\%$ in females and TBF $>25\%$ in males. Abnormal VFP was defined as VFP $>10\%$ in both sexes [14].

A10-mL sample of venous blood drawn from each participant was used to determine fasting serum triglycerides (TG), high-density lipoprotein (HDL), glycosylated hemoglobinA1c (HbA1c), hepatitis B surface antigen (HBsAg), and anti-hepatitis C virus antibodies (anti-HCV). Diabetes was defined as HbA1c $\geq 6.5\%$ (48 mmol/mol) or currently on treatment for diabetes and pre-diabetes was defined as HbA1c 5.7–6.4% [7]. HbA1c was tested using Ion Exchange HPLC method. Hepatitis serological tests were performed using CTK Biotech ELISA kits.

All participants were subjected to an ultrasound scan of the liver using a 5-MHz, 50 mm, convex probe (MindrayDP-10 Ultrasound Diagnostic Systems, Mindray Medical International Limited, Shenzhen, China). Ultrasound

examination was performed by medical personnel specially trained in liver ultrasonography. Nonalcoholic fatty liver disease (NAFLD) was diagnosed using ultrasound criteria for fatty liver (2/3 or more of the following three criteria: increased liver echogenicity compared to the kidney and spleen, blunting of the vascular architecture of the liver, attenuation of the ultrasonic signal in deep view), safe alcohol consumption (Asian standards of < 14 units/week for men and < 7 units/week for females), and absence of hepatitis B and C markers [15].

Data were entered into the Epi Info 7 (Center for Disease Control and Prevention, Atlanta, GA, USA). Logical and random checks were performed. Statistical analysis was performed using SPSS ver. 22.0 (SPSS, Chicago, IL, USA). Prevalence and incidence rates with 95% confidence intervals (95% CI) were calculated using a standard formula for proportion estimates. Continuous and categorical data were described using the median with interquartile range (IQR) and frequency with percentages, respectively. Group comparisons were made using the Mann–Whitney U test and Pearson's chi-square test for continuous and categorical variables, respectively.

Poisson regressions with robust error variance models was used to investigate variables associated with new-onset T2D. The following exposure variables in 2007 were analyzed for association with incident T2DM: sex, age, educational level (i.e., less than General Certificate of Education, Ordinary Level), income (i.e., less than the median income in the cohort), unsafe alcohol use, smoking pack-years, prediabetes, central obesity (waist circumference ≥ 90 cm for males and ≥ 80 cm for females), raised blood pressure (systolic BP ≥ 140 or diastolic BP ≥ 90 mmHg, or treatment of previously diagnosed hypertension), low HDL [< 40 mg/dL (1.03 mmol/dL) in males and < 50 mg/dL (1.29 mmol/dL) in females, or on specific treatment for this lipid abnormality], raised TG [≥ 150 mg/dL (1.7 mmol/L) or on specific treatment for this lipid abnormality], dyslipidemia (total cholesterol > 140 mg/dL, raised TG, reduced HDL or on lipid-lowering therapy), NAFLD and percentage gain in body mass index (BMI), weight, and waist circumference. A stepwise variable selection method was used to develop the final model. Multi-collinearity of independent variables was assessed using Variance Inflation Factor (VIF). All the independent variables showed a VIF of less than 3 indicating a low correlation among variables. Statistical significance was set at $P < 0.05$.

Written informed consent was obtained from all participants. Ethical approval was obtained from the Ethical Review Committee of the Faculty of Medicine, University of Kelaniya, Ragama, Sri Lanka.

Results

A total of 2985/3012 (99.1%) [1636 women (54.8%); median age (IQR) 53 (47–59) years] participants initially recruited in 2007 had complete data for analysis. A total of 2148/2985 (71.6%) of the original cohort attended follow-up in 2014 [1237 (57.5%) women; median (IQR), 60 (54–66) years]. Participant profiles were similar among the inception and follow-up cohorts, except for more females and those with central obesity in the follow-up cohort (Table 1) [14].

Of the 2985 subjects in the inception cohort, 737 had established T2D, with a prevalence of 24.7% (95%CI:23.2% – 26.3%). Those with established T2D were more likely to be older, have a lower income, have higher body weight, body mass index (BMI) and waist circumference, hypertension, coronary artery disease, low HDL, dyslipidemia, and NAFLD at baseline, compared to those without T2D (Table 2).

From the inception cohort of 2007, 1650/2246 participants without T2D attended follow-up in 2014. Of these, 436/1650 (27.6%) had developed new-onset T2D, with an annual incidence of 3.9% (95% CI:3.0%—4.9%) (Fig. 1). Those with incident T2D were more likely to be female and have a higher body weight, BMI, and waist circumference,

Table 1 Profile of the participants in 2007 who attended and did not attend follow-up in 2014 [14]

	Attended follow-up in 2014 (N = 2148)	Did not attend follow-up 2014 (N = 837)	*p* value
Males (%)	910 (42.4)	439 (52.4)	< 0.001
Median age (IQR)	53.0 (47.0 – 59.0)	53.0 (46.0 – 59.7)	0.630
Central obesity	1213 (56.5%)	405 (48.4%)	< 0.001
Median FBG (IQR)	104.0 (96.0 – 117.0)	104.5 (97.0 – 124.0)	0.035
Median SBP (IQR)	132.0 (119.0 – 147.0)	132.0 (120.0 – 147.0)	0.461
Median DBP (IQR)	78.0 (71.0 – 87.0)	79.0 (72.0 – 87.9)	0.340
Median TG (IQR)	115.0 (85.0 – 162.0)	121.0 (86.0—161.0)	0.226
Median HDL (IQR)	50.0 (48.0 – 52.0)	50.0 (48.0 – 52.0)	0.759

(IQR—inter quantile range; FBG—fasting blood glucose; SBP- systolic blood pressure; DBP, diastolic blood pressure; TG—triglyceride; HDL—high-density lipoprotein)

Table 2 Characteristics of those with established diabetes compared with those without diabetes in 2007

Parameter in 2007	No Diabetes Number (%) or Median (IQR) N = 2246	Diabetes Number (%) or Median (IQR) N = 737	*p*-value
Age (years)	52.0 (46.0 – 58.0)	55.0 (50.0 – 60.0)	< 0.001
Male gender	1033 (46.0)	315 (42.7)	0.134
Low education level	1043 (46.4)	328 (44.5)	0.383
Household income < median	620 (38.5)	224 (45.7)	0.005
Smoking > 1 pack year	333 (14.8)	85 (11.5)	0.030
Unsafe alcohol	433 (19.3)	121 (16.4)	0.093
Weight (kg)	58.4 (51.0 – 66.5)	60.8 (53.8 – 68.3)	< 0.001
Waist (cm)	84.6 (77.5 – 91.9)	89.0 (83.3 – 95.1)	< 0.001
BMI (kg/m^2)	23.7 (20.8 – 26.4)	24.5 (22.3 – 27.4)	< 0.001
BMI > 23 kg/m^2	1271 (56.8)	503 (68.5)	< 0.001
Central obesity	1129 (50.3)	488 (66.3)	< 0.001
Hypertension	908 (40.0)	434 (58.8)	< 0.001
Elevated TG or on treatment	707 (98.5)	417 (97.9)	0.623
Low HDL or on treatment	594 (26.8)	270 (37.5)	< 0.001
Dyslipidemia	1368 (61.1)	540 (74.1)	< 0.001
NAFLD	614 (37.8)	325 (74.5)	< 0.001
Coronary artery disease	71 (3.2)	63 (8.5)	< 0.001

(IQR-inter quantile range; BMI-body mass index; TG-triglyceride; HDL-high-density lipoprotein; NAFLD-nonalcoholic fatty liver disease)

hypertension, low HDL, dyslipidaemia, NAFLD, coronary artery disease, and prediabetes in 2007, than those who remained nondiabetic (Table 3).

Poisson regressions with robust error variance models showed that prediabetes, central obesity, low HDL, and NAFLD were significantly associated with incident T2D (Table 4).

Abnormal VFP was observed in 234 (54.3%) participants with new-onset T2D and 426 (37.6%) non-diabetic participants in 2014 ($p < 0.001$). Abnormal TBF was observed in 324 (75.2%) new-onset T2D participants and 801 (70.6%) non-diabetic participants in 2014 ($p = 0.085$).

Of the 525 participants with prediabetes in 2007, 365 (69.5%) attended the follow-up in 2014. 201/365 (55.1%) of them had developed new-onset T2D, giving an annual conversion rate of prediabetes to T2D of 7.9%; 147/365 (40.3%) remained in prediabetes and 17/365 (4.6%) had reverted to normoglycemia. In those with prediabetes in 2007, increase in WC [for each 1 cm increase RR = 1.02 (95% CI: 1.01—1.03), $p < 0.001$] and low HDL [RR = 1.25 (95% CI: 1.05—1.49), P = 0.014] predicted progression to T2D.

Discussion

In this community cohort follow-up study of an urban aging adult population in Sri Lanka, the annual incidence of T2D was 3.9%. Prediabetes, central obesity, low HDL, and NAFLD were associated with new-onset T2D. Central adiposity, as evidenced by abnormal VFP, was also significantly more prevalent in persons with new-onset T2D. The annual conversion rate of prediabetes to T2D was 7.9%. The presence of features of metabolic syndrome at baseline predicted the conversion of prediabetes to T2D, and only a small proportion of prediabetes reverted to normoglycemia after seven years.

In 2007, we reported a prevalence of 24.7% for T2D in this adult population [13]. The prevalence increased to 52% after seven years. The International Diabetes Federation estimates that the age-adjusted comparative diabetes prevalence among adults (aged 20–79 years) in Sri Lanka is 10.7% [2]. The much higher prevalence in the present cohort highlights the increase in the prevalence of T2D with age, which is a well-described phenomenon [16].

In China, the age-standardized incidence of T2D was 9.5 per 1000 person-years for men and 9.2 for women from 2006 to 2014 [17]. A retrospective nationwide longitudinal study in Taiwan from 1999 to 2004 showed that the age-standardized prevalence of T2D increased from 4.7 to 6.5% in men and from 5.3 to 6.6% in women [18]. The annual incidence of T2D in the current study was lower than that reported in these studies. Despite many studies reporting on prevalence, to our knowledge no community-based studies have previously investigated the incidence and predictors of new-onset T2D among South Asian populations.

Table 3 Comparison of characteristics between those with new-onset diabetes and those who remained nondiabetic in 2014

Parameter in 2007	New-onset Diabetes in 2014 Number (%) or Median (IQR) (N = 436)	Non-diabetic in 2014 Number (%) or Median (IQR) (N = 1143)	*p*-value
Male gender	157 (36.0)	526 (46.0)	< 0.001
Age (years)	53 (47 – 58)	52 (46 – 58)	0.050
Low education level	242 (55.5)	632 (55.3)	0.985
Household income < median	169 (40.1)	504 (44.7)	0.122
Unsafe Alcohol	62 (19.0)	218 (19.1)	0.029
Smoking > 1 pack year	49 (11.2)	157 (13.7)	0.217
Weight (kg)	62.2 (54.8 – 68.5)	57.4 (50.3 – 65.7)	< 0.001
Waist (cm)	88.9 (82.5 – 95.2)	83.7 (76.9 – 90.5)	< 0.001
BMI (kg/m^2)	25.4 (23.1 – 27.9)	23.3 (20.5 – 26.0)	< 0.001
BMI > 23 kg/m^2	328 (75.6)	602 (52.8)	< 0.001
Central obesity	304 (69.7)	537 (47.0)	< 0.001
Prediabetes	201 (46.1)	164 (14.3)	< 0.01
Hypertension	191 (43.8)	456 (39.9)	0.175
Elevated TG or on treatment	181 (98.9)	317 (98.1)	0.770
Low HDL or on treatment	153 (35.6)	288 (25.5)	< 0.001
Dyslipidemia	315 (72.6)	668 (58.6)	< 0.001
NAFLD	175 (55.6)	287 (35.1)	< 0.001
Coronary artery disease	21 (4.8)	22 (1.9)	0.003

(IQR-inter quantile range; BMI-body mass index; TG-triglyceride; HDL-high density lipoprotein; NAFLD-nonalcoholic fatty liver disease)

Table 4 Independent predictors of diabetes in those with no diabetes in 2007

Variable – in 2007	Coefficient	SE	P-value	RR	95% CI for RR
Central obesity	0.3786	0.1210	0.002	1.460	1.152 1.851
Prediabetes	0.9276	0.0908	< 0.001	2.528	2.116 3.021
Low HDL	0.2746	0.0921	0.0003	1.316	1.099 1.577
NAFLD	0.2795	0.1079	0.010	1.323	1.070 1.634

(NAFLD-nonalcoholic fatty liver disease, SE-standard error, RR-risk ratio, CI-confidence interval)

NAFLD, markers of obesity, and impaired glucose tolerance are positively associated with future T2D [19]. Several previous studies, including one from the present cohort, have shown that moderate to severe NAFLD is predictive of T2D as it occurs in association with features of metabolic syndrome, including insulin resistance [20–22]. A meta-analysis of 117020 persons from 20 prospective studies found that the presence of NAFLD predicted the development of incident T2D over a median follow-up of 5 years [23].

The development of metabolic abnormalities and T2D correlates with the abdominal and visceral accumulation of adipose tissue because visceral fat reduces insulin activity by increasing fatty acids. Visceral fat cells are known to release proteins that contribute to inflammation, atherosclerosis, dyslipidemia, and hypertension. It is likely that visceral adipose tissue is more closely associated with T2D than other obesity indices [24]. Several parameters, such as body mass index, waist circumference, waist-to-hip ratio, and waist-to-height ratio, are used to determine general and central obesity. Waist-to-height ratio and waist circumference are better parameters for predicting T2D, as they directly correlate with central obesity [25]. VFP, measured by bioelectrical impedance, is an indirect measure of visceral fat deposition that correlates closely with the metabolic risk posed by obesity [26]. In our cohort, central obesity (as measured by WC), low HDL, and NAFLD at inception showed a significant association with new-onset T2D seven years later. Abnormal VFP was significantly more prevalent in persons with new-onset T2D.

Prediabetes is a condition in which blood glucose is above normal but does not meet the criteria for the diagnosis of T2D. IGT and IFG are collectively referred to as prediabetes. When patients have both IGT and IFG, the cumulative incidence of T2D over six years is 65% higher than that of individuals with normal blood glucose levels [27, 28]. Identifying individuals with prediabetes together with targeted lifestyle interventions is an essential and cost-effective method for preventing the development of T2D and its complications in a given population [29, 30].

In our cohort, the annual conversion rate of prediabetes to T2D was 7.9%, which is comparable to the previously reported range of 5–10% for most other populations [6]. A fifth of those with prediabetes in the study cohort remained in prediabetes after seven years, while less than 5% reverted to normoglycemia in the absence of a formal, targeted, specific lifestyle or pharmacological intervention. The Diabetes Prevention Program Outcomes Study (DPPOS) in 2014 demonstrated a 56% reduction in the incidence of new-onset T2D among individuals with prediabetes who reverted to normoglycemia with intensive lifestyle interventions [31]. Since an increase in WC and decrease in HDL predicted the progression of prediabetes to T2D in our cohort, interventions that target weight reduction are likely to be ideal for achieving regression of prediabetes to normoglycemia [32].

This study had several strengths. This was a relatively large prospective community-based cohort follow-up study. The data collected prospectively at baseline in 2007 and follow-up in 2014 were robust and complete. More than 70% of the study participants attended follow-up after seven years. However, there were several limitations: TBF and VPF were not available at baseline for risk assessment, the follow-up sample consisted mainly of older adults, and although NAFLD was diagnosed based on accepted ultrasound criteria, the interobserver variability of the operators was not formally assessed. Longitudinal follow up was not carried out.

Poorly controlled T2D causes early mortality and morbidity through cardiovascular events, stroke, blindness, kidney failure, and lower limb amputation, which significantly affect the quality of life. T2D and its complications also impose a considerable financial burden on patients, their families, health systems, and national economies through direct medical costs, loss of productivity, and premature mortality [33]. Lifestyle interventions can potentially and effectively deter the progression to T2D in individuals with prediabetes [34].

Conclusions

This study demonstrates a higher prevalence of T2D in older, urban-dwelling Sri Lankan adults, compared to rates reported from other parts of Asia. The presence of components of metabolic syndrome predicted new-onset T2D and conversion of prediabetes to T2D, and only a small proportion of those with prediabetes reverted to normoglycemia. Our findings reiterate the need for targeted lifestyle interventions for individuals with metabolic risk factors, especially prediabetes and obesity, to prevent future T2D.

Acknowledgements We thank all those who continuously supported the Ragama Health Study. We thank the participants of this study for their continued cooperation.

Author Contribution SdeS and HJdeS conceptualized and designed the study. AK, SdeS, MAN, ARW, NK, and HJdeS were involved in establishing the Ragama Health Study cohort. SdeS, MAN, and AK collected data. DE analyzed the data assisted by TB, SdeS, AP, MAN, and HJdeS. SdeS and DE prepared and revised the manuscript. All authors read and agreed to the final version of the manuscript.

Funding We gratefully acknowledge grants from the Ministry of Higher Education of Sri Lanka and the National Center for Global Health and Medicine, Tokyo, Japan for conducting this study. The funding bodies played no role in the design of the study, collection, analysis, and interpretation of data, and in writing the manuscript.

The authors have no relevant financial or non-financial interests to disclose.

The authors have no financial or proprietary interests in any material discussed in this article.

Data Availability The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate Ethical approval was obtained from the Ethics Review Committee of the Faculty of Medicine, University of Kelaniya. Informed written consent was obtained from all participants in 2007 and 2014.

(Approval Nos – P/38/09/2006 and P/169/08/2014).

Competing Interests All authors have no conflicts of interest to declare.

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

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