

Distribution of Estimated 5-Year Stroke Risk Using the Stroke Riskometer™ Among Adults in Kathmandu, Nepal: A Cross-Sectional Study

Rachana Nakarmi,¹ Sobin Pant,¹ Pramila Tamang,¹ Parbati Rana,¹ Salina Maharajan,¹ Anita Thapa,¹ Nooma Sharma,¹ Lekhjunga Thapa¹

¹National Neuro Center, Chakrapath, Kathmandu, Nepal.

ABSTRACT

Introduction: Stroke is a leading cause of death and disability globally, while Nepal is experiencing a growing burden of non-communicable disease. Community-level data on estimated individual stroke risk remain limited. This study characterized estimated 5-year stroke risk among adults in Kathmandu Metropolitan City using the Stroke Riskometer™.

Methods: A community-based cross-sectional study included 422 adults aged ≥20 years. Five wards were purposively selected, followed by random household sampling and Kish-grid respondent selection within wards. Five-year absolute stroke risk was estimated using the Stroke Riskometer™. Risk distributions were summarized using medians and interquartile ranges (IQR), with subgroup distributions presented descriptively.

Results: Median estimated 5-year stroke risk was 2.50% (IQR 1.40 –5.80%), ranging from 0.60% to 48.30%. Using study-defined descriptive thresholds, 261/422 (61.85%) participants had estimated risk ≥2.0%; these thresholds are not validated clinical categories. Median estimated risk increased from 1.40% among participants aged 20–39 years to 8.80% among those aged ≥60 years. Excluding 11 participants with previous TIA/stroke yielded a median risk of 2.40% (IQR 1.40 –5.60%).

Conclusions: Estimated 5-year stroke risk showed a wide, right-skewed distribution in this community sample. Prospective validation against incident stroke outcomes is required before clinical or public-health application in Nepal.



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Correspondence:

Name: Sobin Pant
Affiliation: National Neuro Center,
Chakrapath, Kathmandu, Nepal
Phone No: +977-9844001955



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INTRODUCTION

Stroke is the second leading cause of death and third leading cause of combined death and disability worldwide, with over 86% of stroke deaths occurring in low- and middle-income countries.^{1,2} In Nepal, stroke is the third leading cause of death, and rising hypertension, diabetes, and tobacco use are driving an epidemiological transition, particularly in urban areas.^{3–5} Most existing evidence is hospital-based and subject to referral bias, and community-level stroke risk data remain scarce.⁶

The Stroke Riskometer™, a validated mobile application developed by Auckland University of Technology, integrates 21 variables to estimate individualized 5-year and 10-year absolute stroke risk and has undergone validation and cross-cultural evaluation in multiple populations.^{7–9} Community-based data using the stroke Riskometer™ in Nepal remain limited.

This study aimed to estimate the distribution of 5-year absolute stroke risk among adults in Kathmandu, describe the distribution of key risk factors, and examine differences in risk across demographic and clinical subgroups.

METHODS

This was a community-based cross-sectional study. The study population comprised adults aged ≥20 years residing in Kathmandu Metropolitan City, Nepal. Data were collected between August and November 2025. Ethical approval was obtained from the Nepal Health Research Council (Proposal ID: 402-2025), and written informed consent was obtained from all participants. Adults aged ≥20 years who had resided in Kathmandu for at least 6 months were eligible for inclusion. Individuals were excluded if they declined consent, or were

unable to communicate due to severe illness or hearing/speech impairment.

The sample size was calculated using the single-proportion formula ($n = z^2 p[1-p]/e^2$). A conservative proportion of 50% was used in the absence of a reliable local estimate, with 95% confidence ($z=1.96$) and 5% absolute precision, yielding a minimum sample of approximately 384. This was increased by approximately 10% for anticipated non-response, giving a target of 422 participants, which was achieved. A two-stage sampling approach was used. In the first stage, five of Kathmandu Metropolitan City's 32 administrative wards (Wards 3, 12, 16, 26, and 32) were purposively selected by the research team. Approximately equal numbers of participants were recruited from each selected ward rather than using population-proportionate allocation. In the second stage, households were randomly selected from ward-level household sampling frames prepared with assistance from the respective ward offices. Where more than one eligible adult was present in a selected household, one respondent was selected using the Kish grid technique. Ward-specific population and recruitment details are provided in Supplementary Table 1. Because ward selection was purposive and allocation was not population-proportionate, the sample should not be interpreted as a probability sample representative of Kathmandu Metropolitan City as a whole.

Data were collected primarily through household visits. Where household visits were not feasible because of participant unavailability or scheduling constraints, interviews were conducted at the local ward office or a community centre by prior arrangement. The proportion of interviews conducted at each location was not separately recorded. Data collection was undertaken by trained data collectors. Systolic blood pressure was measured using an aneroid sphygmomanometer, height using a portable stadiometer, and body weight using a portable digital weighing scale. Demographic, lifestyle, and medical-history information was obtained from participants. Interviewer-assisted assessments were conducted where self-administration was not feasible.

Stroke risk was assessed using the Stroke Riskometer™ mobile application, version 20.0.2.21, developed at Auckland University of Technology. The Stroke Riskometer™ estimates individualised 5-year and 10-year stroke risk using demographic, lifestyle, and clinical information. The original validation included 9,501 participants from New Zealand, Russia, and the Netherlands and compared its performance with established stroke-risk prediction algorithms.⁷ Subsequent cross-cultural evaluation has also been reported.⁹

The study questionnaire reproduced the questions and response options presented in the application. Original participant responses were entered into the application, and the estimated 5-year stroke-risk score generated by the application was recorded. No study-specific recoding was used to calculate the Stroke Riskometer™ score. The application assessment included age, sex, ethnicity, height, weight, smoking, alcohol

intake, fruit/vegetable intake, physical activity, emotional stress/depression, parental history of stroke or myocardial infarction before 65 years, systolic blood pressure, blood-pressure-lowering medication use, diabetes, heart disease, enlarged heart, atrial fibrillation, cognitive impairment/dementia, poor memory, traumatic brain injury, and previous transient ischemic attack (TIA)/stroke.

For descriptive analysis and presentation, selected variables were subsequently regrouped. Exact age was categorized as 20–39, 40–59, and ≥ 60 years. Smoking responses were grouped as never smoking, former smoking, and current smoking. Alcohol responses were grouped as lifetime non-drinker or ever drinker. For heart disease, enlarged heart, atrial fibrillation, cognitive impairment/dementia, traumatic brain injury, and previous TIA/stroke, responses indicating diagnosis either more than or less than 12 months previously were combined as “yes” for descriptive reporting. Fruit/vegetable intake and physical activity were retained using the original application response categories. Blood-pressure-lowering medication use, diabetes, emotional stress/depression, and poor memory were retained according to their original yes/no responses.

The present analysis focused on estimated 5-year absolute stroke risk. The continuous percentage score was considered the primary outcome. For secondary descriptive reporting, scores were grouped as $<2.0\%$, $2.0\text{--}4.9\%$, and $\geq 5.0\%$. These thresholds were defined a priori by the study team solely for descriptive stratification and are not externally validated diagnostic, prognostic, treatment, or clinical decision thresholds. Because the Stroke Riskometer™ was developed primarily in the context of first-ever stroke prevention, the score was not interpreted as a separately validated recurrence-specific prediction model among participants with previous TIA/stroke. A sensitivity analysis excluding these participants was therefore performed.

Data were cleaned and analysed using IBM SPSS Statistics version 26.0. Continuous variables are presented as median (IQR), while categorical variables are presented as frequency and percentage. The estimated 5-year stroke-risk distribution was markedly right-skewed and was therefore summarized using median and IQR. Risk distributions across demographic, lifestyle, and clinical subgroups were presented descriptively without inferential hypothesis testing or p-values. Several subgroup characteristics are themselves inputs to the Stroke Riskometer™ algorithm. Differences between groups defined by these variables therefore partly reflect construction of the calculated score and should not be interpreted as independent epidemiological associations. Multivariable regression of the calculated score on its component variables was not performed because this would introduce model circularity.

RESULTS

A total of 570 households were approached across five wards. 56 households could not be contacted and 53 were excluded on eligibility screening. Among 461 eligible households, 39

declined, yielding a final sample of 422 participants (response rate 91.54%). Ward-specific recruitment details are provided in Supplementary Table 1.

A total of 422 adults were included, with no missing data. The sample was slightly male-predominant (53.79%), with a median age of 46.00 years (IQR 34.00–60.00). Most participants were never-smokers (80.57%) and lifetime non-drinkers (63.98%) (Table 1).

Table 1. Sociodemographic and lifestyle characteristics (n = 422)

Characteristic	n(%)
Sex	
Male	227(53.79)
Female	195(46.21)
Age group (years)	
20–39	155(36.73)
40–59	160(37.91)
≥60	107(25.36)
Smoking status	
Never smoked	340(80.57)
Stopped smoking	16(3.79)
Current smoker	66(15.64)
Alcohol use	
Lifetime non-drinker	270(63.98)
Ever drinker	152(36.02)
Fruit/vegetable servings per day	
≤1 serving	25(5.92)
2–3 servings	334(79.15)
4–5 servings	0(0.00)
≥6 servings	63(14.93)
Physical activity (hours/week)	
≤1 hour	91(21.56)
1–2 hours	209(49.53)
≥2.5 hours	122(28.91)
Emotional stress/depression (past year)	
No	379(89.81)
Yes	43(10.19)
Parental stroke or MI before age 65	
No	419(99.29)
Yes	3(0.71)

Blood-pressure-lowering medication use was reported by 105 (24.88%) participants, and diabetes mellitus by 68 (16.11%). Cardiac comorbidities and prior TIA/stroke were each reported by fewer than 7% of participants. No participant reported cognitive impairment, poor memory, or traumatic brain injury (Table 2).

Median estimated 5-year stroke risk was 2.50% (IQR 1.40–5.80%), ranging from 0.60% to 48.30%, with a right-skewed distribution (Figure 1).

Using the study-defined descriptive thresholds, 161 (38.15%) participants had estimated risk <2.0%, 138 (32.70%) had estimated risk of 2.0–4.9%, and 123 (29.15%) had estimated risk ≥5.0%. Overall, 261 (61.85%) participants had estimated

risk ≥2.0%. These thresholds are descriptive only and do not represent validated clinical categories.

Table 2. Clinical comorbidity profile (n = 422)

Comorbidity	n(%)
Blood-pressure-lowering medication use	
No	317(75.12)
Yes	105(24.88)
Diabetes mellitus	
No	354(83.89)
Yes	68(16.11)
Heart disease (any)	
No	402(95.26)
Yes	20(4.74)
Enlarged heart	
No	398(94.31)
Yes	24(5.69)
Atrial fibrillation	
No	395(93.60)
Yes	27(6.40)
Prior TIA or stroke	
No	411(97.39)
Yes	11(2.61)
Cognitive impairment/dementia	
No	422(100.00)
Poor memory	
No	422(100.00)
Traumatic brain injury	
No	422(100.00)

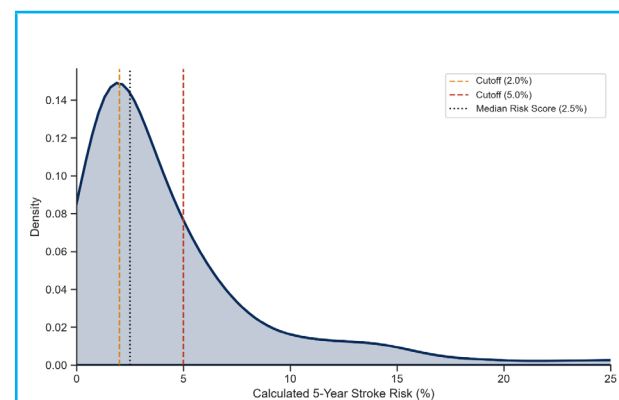


Figure 1. Right-skewed density distribution of estimated 5-year stroke risk (n = 422).

Median estimated risk increased across age categories, from 1.40% (IQR 0.90–2.00%) among participants aged 20–39 years to 2.80% (IQR 1.80–4.48%) among those aged 40–59 years and 8.80% (IQR 6.10–13.60%) among those aged ≥60 years (Figure 2).

Median estimated risk was 3.30% among males and 1.70% among females. Higher median estimated scores were also observed among participants reporting blood-pressure-lowering medication use, diabetes mellitus, heart disease, enlarged heart, or atrial fibrillation compared with those not reporting these characteristics. These comparisons are descriptive because

several subgroup-defining characteristics contribute directly to calculation of the Stroke Riskometer™ score.

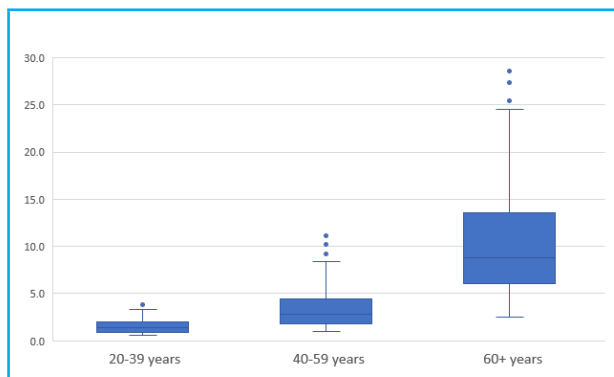


Figure 2. Box-and-whisker plots of estimated 5-year stroke risk by age group (n=422). Horizontal lines represent medians, box boundaries represent the IQR, and whiskers show the 5th–95th percentiles, and points beyond the whiskers represent outliers.

Former smokers had a median estimated risk of 8.55%, compared with 2.30% among never-smokers and 2.60% among current smokers. Former smokers were substantially older, with a median age of 72.50 years (IQR 57.50–78.00), compared with 35.50 years (IQR 29.00–50.25) among current smokers. Median age among never-smokers was 46.50 years (IQR 35.00–60.00). (Table 3).

Because participants reporting previous TIA/stroke represent a secondary-prevention subgroup, a sensitivity analysis was performed excluding these 11 participants.

Among the remaining 411 participants, median estimated 5-year stroke risk was 2.40% (IQR 1.40–5.60%), compared with 2.50% (IQR 1.40–5.80%) in the full cohort. Using the same study-defined descriptive thresholds, 252/411 (61.31%) participants had estimated risk $\geq 2.0\%$, compared with 261/422 (61.85%) in the full cohort.

Exclusion of participants with previous TIA/stroke therefore produced little change in either the continuous estimated risk distribution or the secondary descriptive categorization.

DISCUSSION

This study describes the distribution of estimated 5-year stroke risk using the Stroke Riskometer™ among adults recruited from selected wards of Kathmandu Metropolitan City. The principal finding was a wide and markedly right-skewed distribution, with a median estimated risk of 2.50% (IQR 1.40–5.80%) and values ranging from 0.60% to 48.30%. The continuous estimated risk distribution represents the primary outcome of this study.

Using study-defined descriptive thresholds, 61.85% of participants had estimated risk $\geq 2.0\%$. This finding requires cautious interpretation because the thresholds used in this study have not been externally validated as clinical, diagnostic,

Table 3. Median estimated 5-year stroke risk by subgroup (n = 422)

Variable/Group	n	Median (%)	IQR (%)
Age group (years)			
20–39	155	1.40	0.90–2.00
40–59	160	2.80	1.80–4.48
≥ 60	107	8.80	6.10–13.60
Sex			
Male	227	3.30	2.00–7.40
Female	195	1.70	1.00–3.50
Blood-pressure-lowering medication use			
No	317	2.00	1.20–3.60
Yes	105	6.60	4.00–12.30
Diabetes mellitus			
No	354	2.20	1.20–4.33
Yes	68	7.15	4.30–12.60
Heart disease (any)			
No	402	2.40	1.40–5.63
Yes	20	5.35	3.08–9.68
Enlarged heart			
No	398	2.40	1.40–5.60
Yes	24	5.55	2.98–9.78
Atrial fibrillation			
No	395	2.40	1.40–5.40
Yes	27	5.60	3.20–10.30
Prior TIA or stroke			
No	411	2.40	1.40–5.60
Yes	11	8.40	2.20–19.90
Smoking status			
Never smoked	340	2.30	1.20–5.60
Current smoker	66	2.60	2.00–4.88
Former smoker	16	8.55	5.40–22.85
Physical activity (hours/week)			
≤ 1 hour	91	2.60	1.50–5.60
1–2 hours	209	3.00	1.80–6.40
≥ 2.5 hours	122	1.80	1.10–4.35
Alcohol use			
Lifetime non-drinker	270	2.25	1.18–6.03
Ever drinker	152	2.65	1.83–5.13
Emotional stress/depression			
No	379	2.40	1.40–5.70
Yes	43	2.80	2.00–6.90
Fruit/vegetable servings/day			
≤ 1 serving	25	2.40	0.90–7.25
2–3 servings	334	2.60	1.50–5.60
4–5 servings	0	-	-
≥ 6 servings	63	1.90	1.20–6.40

prognostic, or treatment categories. The finding therefore should not be interpreted to mean that 61.85% of adults in Kathmandu require clinical intervention. Rather, these thresholds provide an additional descriptive representation of the continuous risk-score distribution.

Eleven participants reported previous TIA/stroke and therefore represented a secondary-prevention subgroup. Sensitivity analysis excluding these participants produced little change in

the overall distribution: median estimated risk changed from 2.50% to 2.40%, while the proportion with study-defined estimated risk $\geq 2.0\%$ changed from 61.85% to 61.31%. The principal description of the risk distribution was therefore not materially dependent on inclusion of participants with previous cerebrovascular events. Nevertheless, because the complete cohort contains participants with and without previous TIA/stroke, it should not be described as exclusively a primary-prevention population, and the estimated score among participants with previous events should not be interpreted as a separately validated recurrence-risk estimate.

Median estimated risk increased substantially across age categories, from 1.40% among participants aged 20–39 years to 8.80% among those aged ≥ 60 years. Age is an established determinant of stroke incidence and cumulative vascular risk. [1,10] However, age is itself an input to the Stroke Riskometer™ algorithm. The observed gradient therefore describes the behaviour of the model-derived score within this sample rather than providing an independent estimate of the effect of age.

Participants reporting blood-pressure-lowering medication use, diabetes mellitus, heart disease, enlarged heart, or atrial fibrillation also had higher median estimated scores. These conditions are established contributors to stroke risk in external epidemiological research.^{2,11–13} However, they are also inputs to calculation of the Stroke Riskometer™ score. The differences observed in this study are therefore partly expected by construction and should not be interpreted as independent statistical associations or as validation of individual risk factors in the Nepalese population.

The proportion of participants reporting current blood-pressure-lowering medication use was 24.88%, while 16.11% reported diabetes mellitus. Blood-pressure-lowering medication use should not be interpreted as hypertension prevalence. Untreated or undiagnosed hypertension would not be captured through medication use, while some blood-pressure-lowering medications may be prescribed for indications other than hypertension.

Former smokers had higher median estimated scores than current or never-smokers. However, former smokers were substantially older than current smokers, with median ages of 72.50 and 35.50 years, respectively. Given the strong contribution of age to the calculated score, the higher median score among former smokers should not be interpreted as an adverse consequence of smoking cessation. Tobacco exposure remains an established causal risk factor for stroke based on external epidemiological evidence.²

Physical activity was recorded using the predefined response categories in Stroke Riskometer™ version 20.0.2.21 rather than as exact continuous hours per week. The apparent gaps or overlaps in the category labels therefore reflect the response options of the instrument used for data collection rather than study-defined categories. The results are presented using those original categories to preserve fidelity to the data-collection

instrument.

Several limitations merit acknowledgement. First, the cross-sectional design does not permit causal inference or assessment of actual future stroke incidence. The outcome was a model-derived estimated risk score rather than prospectively observed stroke events. The present study therefore does not establish calibration, discrimination, or predictive validity of the Stroke Riskometer™ in Nepal.

Second, several characteristics used to define the reported subgroups are themselves components of the Stroke Riskometer™ algorithm. Differences in estimated scores across these groups are therefore partly inherent to score construction and should not be regarded as independent epidemiological associations.

Third, five wards were purposively selected and approximately equal numbers were recruited from each rather than sampling proportionately to ward population. The sample therefore cannot be assumed to represent Kathmandu Metropolitan City as a whole, and generalisation to the wider metropolitan population or to rural and peri-urban Nepal should be cautious.

Fourth, although systolic blood pressure, height, and weight were obtained using study equipment, several clinical and lifestyle variables were self-reported and may be affected by recall, reporting, or classification error. Blood-pressure-lowering medication use does not identify untreated or undiagnosed hypertension. Alcohol exposure, dietary intake, physical activity, and emotional stress/depression were represented using predefined categorical responses and therefore do not permit finer-grained exposure assessment.

Finally, 11 participants reported previous TIA/stroke. Although sensitivity analysis demonstrated little change after their exclusion, their inclusion means that the complete cohort contains both primary- and secondary-prevention participants.

Despite these limitations, this study provides community-level descriptive information on model-estimated stroke risk in an urban Nepalese setting where such data remain limited. Prospective studies linking baseline Stroke Riskometer™ estimates with incident stroke outcomes are required to evaluate calibration and predictive performance in Nepalese populations before routine clinical or public-health implementation can be recommended.

CONCLUSIONS

This community-based cross-sectional study demonstrated a wide and markedly right-skewed distribution of estimated 5-year stroke risk among adults recruited from selected wards of Kathmandu Metropolitan City, with a median estimated risk of 2.50% (IQR 1.40–5.80%).

Median estimated scores differed across several demographic and clinical subgroups; because many of these characteristics

are themselves inputs to the Stroke Riskometer™ algorithm, these differences should be interpreted descriptively rather than as independent epidemiological effects.

Sensitivity analysis excluding participants with previous TIA/stroke produced little change in the overall estimated risk distribution. Prospective validation against incident stroke outcomes is required to establish the predictive performance and potential clinical or public-health utility of the Stroke Riskometer™ in Nepalese populations.

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Authors' Contribution: RN, NS, and LT conceived and

designed the study. RN, PT, PR, SM, and AT collected the data. RN and SP performed the statistical analysis. RN and SP drafted the manuscript. NS and LT supervised the study and critically

reviewed and revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Conflict of Interest: None.

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