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La Revue Africaine de Médecine Interne (R.AF.M.I.) est une revue destinée aux médecins internistes et spécialistes d'organes. Les publications peuvent être présentées en Français et en Anglais. La revue offre diverses rubriques :

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Les articles originaux présentent le résultat d'études non publiées et comportent une introduction résumant les hypothèses de travail, la méthodologie utilisée, les résultats, une discussion avec revue appropriée de la littérature et des conclusions.

Le résumé structuré (français et anglais) doit comporter: 1) Propos (état actuel du problème et objectif(s) du travail),

2) Méthodes – (matériel clinique ou expérimental, et méthodes utilisées), 3) Résultats, 4) Conclusion.

Le résumé ne doit pas excéder 250 mots. Le texte ne doit pas excéder 4500 mots et comporter plus de 40 références.

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Les articles de synthèse ont pour but de présenter une mise à jour complète de la littérature médicale sur un sujet donné. Leur méthodologie doit être précisée ; Le résumé n'est pas structuré (français et anglais). Le résumé ne doit pas excéder 250 mots. Le texte ne doit pas excéder 4500 mots et 60 références.

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Le Journal ouvre son espace éditorial aux articles d'opinion sur des questions médicales, scientifiques et éthiques ; le texte pourra être accompagné d'un commentaire de la rédaction. Il ne devra pas dépasser 800 mots.

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La première page comportera exclusivement le titre (et sa traduction en anglais), les prénoms et noms des auteurs, l'institution et l'adresse de correspondance, avec numéros de téléphone, de téléfax et adresse e-mail.

La deuxième page contiendra le résumé en français (maximum 250 mots). Ainsi que 3 à 5 mots-clés en français.

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2. Bieleli E, Kandjigu K, Kasiam L. Pour une diététique du diabète sucré au Zaïre. Méd. Afr. Noire 1989 ; 36 : 509-512.

3. Drabo YJ, Kabore J, Lengani A, Ilboudo PD. Diabète sucré au CH de Ouagadougou (Burkina Faso). Bull Soc Path Ex 1996 ; 89 : 185-190.

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Les figures et illustrations seront soit des originaux, soit fournies sur support informatique en un fichier séparé du texte au format TIFF ou JPEG, avec une résolution de 300 DPI.
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Les auteurs s'engagent sur l'honneur, s'ils reproduisent des illustrations déjà publiées, à avoir obtenu l'autorisation écrite de l'auteur et de l'éditeur de l'ouvrage correspondant.
Pour les microphotographies, il y a lieu de préciser l'agrandissement et la technique histologique utilisés.
Les remerciements éventuels seront précisés en fin de texte et seront courts.
Les conflits d'intérêt potentiels et les considérations éthiques devront être déclarés dans le manuscrit.

III. Envoi
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Les articles sont soumis pour avis à un comité scientifique de lecture et d'autres experts extérieurs à ce Comité. Une fois l'article accepté, il sera publié après payement des frais d'un montant de 150 000 f CFA ; par Western Union ou Money Gram ou virement bancaire.

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Post-stroke central neuropathic pain in Sub-Saharan Africa: insights from a Cameroonian referral hospital

Douleur neuropathique centrale post-AVC en Afrique subsaharienne : expérience d'un hôpital de référence Camerounais

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Summary

Description: Post-stroke central neuropathic pain (PSCNP) is a disabling complication of stroke that may remain underrecognized in routine clinical practice. Data on its burden and associated factors remain limited in Sub-Saharan Africa.

Objective: To determine the prevalence of PSCNP and identify factors associated with PSCNP among stroke survivors, while comparing the frequency of neuropathic pain with that observed in a non-stroke population.

Methods: We conducted a hospital-based cross-sectional analytical study in the Neurology Department of the Douala General Hospital. Participants included patients with confirmed stroke occurring at least three months before inclusion (stroke+) and controls without a history of stroke (stroke−), frequency-matched for age, sex, and vascular risk factors. Neuropathic pain was assessed using the Douleur Neuropathique 4 (DN4) questionnaire. Associated factors were identified using logistic regression analysis.

Results: A total of 301 participants were included, comprising 100 stroke survivors and 201 non-stroke participants. Neuropathic pain identified by the DN4 was observed in 27.0% of stroke survivors and 8.95% of non-stroke participants (OR = 3.76; 95% CI: 1.95–7.24; p < 0.001). Among stroke survivors, 27 participants fulfilled the predefined clinical and neuroanatomical criteria for PSCNP. All PSCNP cases had a thalamic lesion on neuroimaging. In multivariable analysis, alcohol use (OR = 9.70; 95% CI: 2.63–35.81; p = 0.001), hemorrhagic stroke (OR = 8.53; 95% CI: 2.07–35.21; p = 0.003), and obesity (OR = 9.32; 95% CI: 2.49–34.96; p = 0.001) were independently associated with PSCNP.

Conclusion: PSCNP was observed in 27% of stroke survivors in this hospital-based study. Neuropathic pain identified by the DN4 was more frequent among stroke survivors than among the non-stroke comparison group. Alcohol use, hemorrhagic stroke, and obesity were independently associated with PSCNP. Given the cross-sectional design, single-center setting, and limited number of PSCNP cases, these associations should be interpreted as exploratory and do not establish causality or predictive value. Prospective multicenter studies are needed to confirm these findings.

Keywords: Associated factors - Neuropathic pain - Prevalence - Rehabilitation - Stroke - Sub-Saharan Africa.

Résumé

Description : La douleur neuropathique centrale post-AVC (DNCP-AVC) est une complication fréquente mais sous-diagnostiquée de l'accident vasculaire cérébral (AVC), en particulier en Afrique subsaharienne où les données restent limitées. Cette étude visait à déterminer la prévalence de la DNCP-AVC et les facteurs qui lui sont associés à l'Hôpital Général de Douala.

Objectif : Déterminer la prévalence de la douleur neuropathique centrale post-AVC et identifier les facteurs associés chez les patients victimes d'AVC, tout en comparant la fréquence de la douleur neuropathique à celle observée dans une population témoin sans AVC.

Méthodes : Nous avons mené une étude analytique transversale hospitalière dans le service de neurologie. Deux groupes ont été inclus : (1) des patients ayant présenté un AVC confirmé depuis au moins trois mois (groupe AVC+) sans antécédent de douleur neuropathique, et (2) des témoins sans antécédent d'AVC (groupe AVC−), appariés en fréquence selon l'âge, le sexe et les facteurs de risque vasculaire. La DNCP-AVC a été évaluée à l'aide du questionnaire Douleur Neuropathique 4 (DN4). Une régression logistique a été utilisée pour identifier les facteurs indépendamment associés à la DNCP-AVC au sein du groupe AVC+.

Résultats : Au total, 301 participants ont été inclus, dont 100 patients ayant présenté un AVC et 201 participants sans antécédent d'AVC. Une douleur neuropathique identifiée par le questionnaire DN4 était observée chez 27,0% des patients ayant présenté un AVC et chez 8,95% des participants sans AVC (OR = 3,76 ; IC95% : 1,95–7,24 ; p < 0,001). Parmi les patients ayant présenté un AVC, 27 répondaient aux critères cliniques et neuroanatomiques prédéfinis de douleur neuropathique centrale post-AVC. Tous les cas de DNCP-AVC présentaient une lésion thalamique à l'imagerie cérébrale. En analyse multivariée, la consommation d'alcool, l'AVC hémorragique et l'obésité étaient indépendamment associés à la DNCP-AVC.

Conclusion : Une DNCP-AVC était observée chez 27 % des patients ayant présenté un AVC dans cette étude hospitalière. La douleur neuropathique identifiée par le DN4 était plus fréquente chez les patients ayant présenté un AVC que chez les participants sans AVC. La consommation d'alcool, l'AVC hémorragique et l'obésité étaient indépendamment associés à la DNCP-AVC. Compte tenu du caractère transversal, monocentrique et du nombre limité de cas, ces associations doivent être considérées comme exploratoires et confirmées par des études prospectives.

Mots-clés : Accident vasculaire cérébral - Afrique subsaharienne - Douleur neuropathique - Facteurs associés - Prévalence - Réadaptation.

Introduction

Stroke is a major global health burden, particularly in low- and middle-income countries, and may lead to neuropathic pain due to somatosensory system lesions **[1]**. The increasing burden of non-communicable diseases contributes to this trend **[2]**. Several post-stroke complications remain insufficiently investigated in Cameroon. Recent studies have documented a substantial burden of post-stroke cognitive impairment among stroke survivors, highlighting the need to better characterize other disabling neurological sequelae such as central neuropathic pain **[3]**.

Post-stroke central neuropathic pain (PSCNP) remains underdiagnosed and poorly characterized in routine clinical practice, especially in resource-limited settings **[4]**. While several studies have reported PSCNP prevalence ranging from 5% to 35% in high- and middle-income countries, data from Sub-Saharan Africa remain scarce, heterogeneous, and often limited by small sample sizes or restricted stroke subtypes **[5-8]**.

In Cameroon specifically, available data on PSCNP are either outdated or do not comprehensively evaluate both its prevalence and associated clinical factors using standardized diagnostic tools. Moreover, few studies have incorporated a comparative non-stroke population to contextualize the burden of neuropathic pain.

This study aimed to determine the prevalence of PSCNP and identify associated factors in a tertiary hospital setting in Cameroon, while providing a comparative estimate of neuropathic pain frequency in a matched non-stroke population.

Methods

Ethics

This study was approved by the Ethics Committee of the University of Douala (N° 1696 CEI-UDo/06/2019/T). All participants provided informed consent to the anonymous use of their clinical and paraclinical data.

Study design

This was a hospital-based, cross-sectional analytical study conducted over five months, from January to May 2019, in the Neurology Department of the Douala General Hospital (DGH), a tertiary university hospital in Cameroon. The study protocol, eligibility criteria, data collection procedures, and operational definitions were established before and during the data collection period in 2019. Patient recruitment was completed during the study period in 2019. The subsequent interval before data verification, database cleaning, statistical analysis, and manuscript preparation was partly related to disruptions in hospital activities and research operations during the COVID-19 pandemic and its aftermath. These activities were subsequently completed in 2023-2024. No changes were made to participant eligibility criteria or outcome definitions based on the observed results.

The primary objective was to estimate the prevalence of post-stroke central neuropathic pain (PSCNP) and identify associated factors among stroke patients. A secondary objective was to compare the frequency of neuropathic pain between stroke patients and a frequency-matched non-stroke population.

Inclusion criteria

The study population consisted of two groups:

1. Stroke+ Group: Patients with age ≥ 18 years, history of a clinically confirmed stroke (ischemic or hemorrhagic), supported by neuroimaging (CT or MRI). Stroke onset at least 3 months prior to inclusion (to ensure chronic phase). Ability to provide informed consent and participate in interviews. Regular follow-up in the Neurology departments of the study hospital.
2. Stroke- Group: Participants without a history of stroke were included for comparison of neuropathic pain prevalence only and not for etiological analysis, frequency-matched with the stroke+ group in terms of age, sex, and cerebrovascular risk factors (CVRFs). **Non-stroke comparison group:** Participants in the stroke− group were recruited from patients attending other clinical departments of the Douala General Hospital during the same study period. They had no previous history of stroke and were frequency-matched with the stroke+ group according to age, sex, and cerebrovascular risk factors. This group was included solely to provide a comparative estimate of the frequency of neuropathic pain and was not considered a population-based control group.

Exclusion Criteria

Patients were excluded if they have known peripheral neuropathic pain (e.g., diabetic neuropathy, post-herpetic neuralgia) or any other chronic pain condition not attributable to a central lesion. Patients with severe aphasia or cognitive impairment that impaired reliable pain reporting were excluded. Current severe psychiatric illness (e.g., major depression with psychotic features, schizophrenia) that could interfere with accurate symptom reporting. Severe comorbid medical conditions (e.g., terminal illness, advanced cancer)

that could confound pain symptoms or compromise participation. Use of centrally acting pain medications (e.g., anticonvulsants, antidepressants) within 2 weeks prior to assessment, which could alter pain perception.

Data collection

For each patient, the following data were collected:
Socio-demographic information: Age, sex, and other relevant details.
Cerebrovascular Risk Factors: Information on hypertension, diabetes, dyslipidaemia, alcoholism, smoking, uric acid levels, coronary heart disease, and atrial fibrillation.
Clinical and neurological evaluation: Blood pressure, pulse, weight, height, and body mass index (BMI) were measured. Stroke severity was assessed using the National Institute of Health Stroke Scale (NIHSS), and functional outcomes were evaluated with the modified Rankin Scale (mRS).
Pain Characteristics: Data on pain type, onset delay post-stroke, and sensory system findings from neurological examinations were documented.
PSCNP was defined as neuropathic pain occurring after a confirmed stroke and considered attributable to the central nervous system lesion. Diagnosis required: (1) a documented history of stroke; (2) onset of pain after the stroke; (3) a pain distribution anatomically compatible with the affected central somatosensory region; (4) corresponding positive or negative sensory abnormalities on neurological examination; (5) confirmation of the stroke lesion by brain CT or MRI; and (6) absence of a more plausible peripheral, musculoskeletal, or other cause of pain. Neuropathic characteristics were assessed using the Douleur Neuropathique 4 (DN4) questionnaire, with a score ≥4 indicating a neuropathic component. The DN4 was therefore used as part of the diagnostic assessment and not as the sole criterion for PSCNP. **Diagnostic approach for PSCNP:** In stroke survivors reporting pain, the diagnosis of PSCNP required concordance between the temporal relationship to stroke, the anatomical distribution of pain, the location of the central lesion on neuroimaging, and sensory abnormalities in the painful territory. Alternative causes of pain, including peripheral neuropathic, musculoskeletal, and other nociceptive conditions, were considered and excluded when clinically identifiable.
Pain intensity was measured on a visual analog scale (VAS), categorized as mild (1-3), moderate (4-6), or severe (7-10).
Therapeutic Medications: Information on medications used to treat PSCNP in stroke+ patients was collected.
Biological Data: Laboratory results from within three months of the study were reviewed, including creatinine (normal range: 62-120 µmol/l), lipid profile (LDL cholesterol < 70 mg/dL; HDL cholesterol > 40 mg/dL for men, >50 mg/dL for women; total cholesterol < 200 mg/dL; triglyceridemia: 30-150 mg/dL), and fasting blood glucose (normal < 126 mg/dL).

Operational definitions

Post-Stroke Central Neuropathic Pain (PSCNP): Defined as pain arising as a direct consequence of a lesion or disease affecting the somatosensory system after stroke. Diagnosed based on clinical criteria from the DN4 questionnaire, with a score ≥ 4 indicating neuropathic pain.
Stroke: Defined according to the World Health Organization (WHO) criteria as rapidly developing clinical signs of focal or global disturbance of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin. Diagnosis was confirmed by brain imaging (CT or MRI).
Obesity: Defined as a body mass index (BMI) ≥ 30 kg/m² based on measured height and weight at inclusion.
Alcoholism: Defined as consumption of alcohol exceeding 14 drinks/week in men or 7 drinks/week in women or based on a positive CAGE screening test (≥ 2 positive responses).
Hypertension: Defined as a history of hypertension diagnosed by a physician, current antihypertensive treatment, or a blood pressure ≥ 140/90 mmHg at two separate measurements.
Diabetes Mellitus: Defined by a history of diabetes diagnosed by a physician, current use of antidiabetic medication, or fasting blood glucose ≥ 126 mg/dL.
Dyslipidemia: Defined by a total cholesterol ≥ 200 mg/dL, LDL ≥ 130 mg/dL, HDL < 40 mg/dL in men and < 50 mg/dL in women, triglycerides ≥ 150 mg/dL, or ongoing lipid-lowering treatment.
Thalamic lesion: Presence of infarct or hemorrhage in the thalamic region, confirmed by neuroimaging.
Time since stroke onset: Chronic stroke was defined as more than 3 months after stroke onset.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as means and standard deviations (SD), while categorical variables are presented as frequencies and percentages. Associations between categorical

variables were assessed using the chi-square test or Fisher's exact test, as appropriate. The frequency of neuropathic pain identified by the DN4 was compared between the stroke+ and stroke− groups. Within the stroke+ group, univariable logistic regression was used to assess associations between demographic, vascular, clinical, and imaging variables and PSCNP. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Variables with $p < 0.05$ in univariable analyses were entered into the multivariable logistic regression model. Statistical significance was defined as a two-sided $p < 0.05$. Model fit was assessed using the Hosmer–Lemeshow test.

The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines **[9]**.

Results

Sociodemographic data:

A total of 301 patients were included in the study, including 100 patients with stroke (stroke+ group) and 201 patients without stroke (stroke- group). The mean age was 57.7 ± 11.9 years in the stroke+ group and 58.6 ± 11.2 years in the stroke- group ($p > 0.05$). Hypertension was the most common CVRF in both groups, $p = 0.584$ (Table I and II).

Table I: Sociodemographic and Clinical Characteristics of the Stroke-Positive (stroke+) and Stroke-Negative (stroke−) populations

Variables	Stroke − (n=201)	Stroke + (n=100)	Total (n=301)	p-value
Sex				
Female	**86 (42.8)**	**51 (51.0)**	**137 (45.5)**	**0.178**
Male	**115 (57.2)**	**49 (49.0)**	**164 (54.5)**	
Age group				
< 45 years	**30 (14.9)**	**15 (15.0)**	**45 (15.0)**	
45–60 years	**85 (42.3)**	**44 (44.0)**	**129 (42.9)**	**0.950**
> 60 years	**85 (42.3)**	**39 (39.0)**	**127 (42.2)**	
Mean age (years)	**58.66 ± 11.22**	**57.72 ± 11.94**	**58.3 ± 11.4**	**0.503**
Marital status				
Single	**21 (10.4)**	**11 (11.0)**	**32 (10.6)**	
Married	**145 (72.1)**	**71 (71.0)**	**216 (71.8)**	**0.637**
Divorced	**3 (1.5)**	**0 (0.0)**	**3 (1.0)**	
Widowed	**32 (15.9)**	**18 (18.0)**	**50 (16.6)**	
Occupational sector				
Private sector	**19 (9.5)**	**17 (17.0)**	**36 (12.0)**	
Student	**1 (0.5)**	**0 (0.0)**	**1 (0.3)**	
Public sector	**33 (16.4)**	**18 (18.0)**	**51 (16.9)**	**0.036**
Informal sector	**44 (21.9)**	**9 (9.0)**	**53 (17.6)**	
Unemployed	**104 (51.7)**	**56 (56.0)**	**160 (53.2)**	
Health coverage				
Insurance	**16 (8.0)**	**18 (18.0)**	**34 (11.3)**	
Self-financed	**93 (46.3)**	**9 (9.0)**	**102 (33.9)**	**<0.001**
Self + Family	**39 (19.4)**	**32 (32.0)**	**71 (23.6)**	
Family	**53 (26.4)**	**41 (41.0)**	**94 (31.2)**	

Table II: Vascular risk factors in stroke and non-stroke groups

Risk factors	Stroke− (n=201)	Stroke+ (n=100)	Total	p-value
Hypertension	**166 (82.6)**	**80 (80.0)**	**246 (81.7)**	**0.584**
Dyslipidemia	**37 (18.4)**	**21 (21.0)**	**58 (19.3)**	**0.591**
Smoking	**12 (6.0)**	**1 (1.0)**	**13 (4.3)**	**0.067**
Alcohol use	**89 (44.3)**	**45 (45.0)**	**134 (44.5)**	**0.906**
Atrial fibrillation	**18 (9.0)**	**4 (4.0)**	**22 (7.3)**	**0.159**
Cardiopathy	**15 (7.5)**	**4 (4.0)**	**19 (6.3)**	**0.318**
Epileptic seizure	**5 (2.5)**	**0 (0.0)**	**5 (1.7)**	**0.174**
Angina/MI	**2 (1.0)**	**0 (0.0)**	**2 (0.7)**	**1.000**
Obesity	**46 (22.9)**	**25 (25.0)**	**71 (23.6)**	**0.470**

Neuropathic pain identified by the DN4 was observed in 27.0% of stroke survivors and 8.95% of participants in the non-stroke comparison group. This difference was statistically significant (OR = 3.76, 95% CI: 1.95–7.24; p < 0.001). Among stroke survivors, those fulfilling the predefined clinical criteria for PSCNP were classified as having PSCNP (Figure 1).

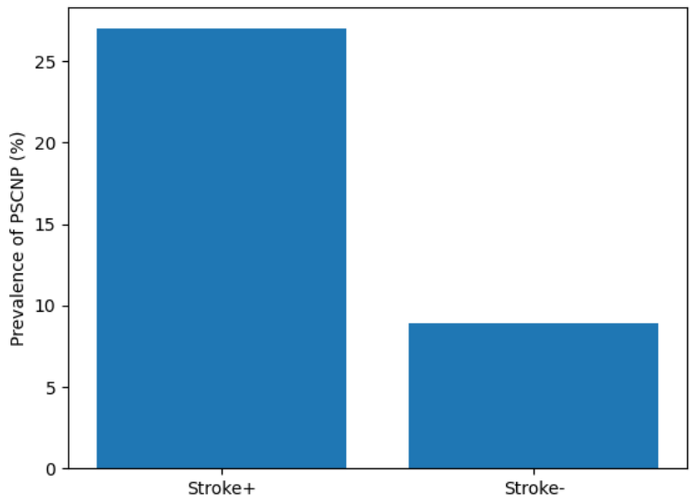

Figure 1. Frequency of neuropathic pain identified by the DN4 among stroke and non-stroke participants.

Neuropathic pain identified by the DN4 was more frequent among stroke participants (27.0%) than among non-stroke participants (8.95%; p < 0.001). PSCNP was diagnosed only among stroke participants based on the predefined clinical and neuroanatomical criteria.

Factors associated with post-stroke central neuropathic pain

Significant cerebrovascular risk factors for PSCNP included high blood pressure (p = 0.013), alcohol use (p < 0.001), obesity (p < 0.001), and dyslipidemia (p = 0.017) (Table III). Within the clinical and paraclinical characteristics of stroke patients, factors significantly associated with PSCNP were obesity (p = 0.001), hemorrhagic stroke (p = 0.001), and thalamic involvement (p = 0.002) (Table IV). No significant association was found between biological parameters and PSCNP.

Table III : Factors associated with neuropathic pain in stroke patients

Variables	NP - (73)	NP+ (27)	Total (%)	OR (95% CI)	p-value
Hypertension	54 (74.0)	26 (96.3)	80 (80.0)	**9.15 (1.16 – 72.11)**	**0.013**
Alcohol use	23 (31.5)	22 (81.5)	45 (45.0)	**9.57 (3.22 – 28.43)**	**< 0.001**
Obesity	10 (13.7%)	15 (55.6)	25 (25.0)	**7.88 (2.87 – 21.64)**	**<0.001**
Dyslipidemia	11 (15.1)	10 (37.0)	21 (21.0)	**3.32 (1.21 -9.11)**	**0.017**

NP : Neuropathic pain ; **OR :** Odds Ratio ; **CI :** confidence interval

Table IV : Clinical and neuroimaging characteristics according to the presence of PSCNP among stroke survivors

Variable	Stroke NP− (%)	StrokeNP+ (%)	OR (95% CI)	p-value
BMI >30 kg/m²	12.3	37.0	7.22 (2.08–25.08)	0.001
Hemorrhagic stroke	13.7	44.4	5.04 (1.83–13.85)	0.001
Thalamic lesion	0.0	100.0	—	0.002*

BMI: Body mass index

* Thalamic lesion: an odds ratio could not be estimated because all participants with PSCNP had a thalamic lesion, whereas no thalamic lesion was observed among participants without PSCNP.

In the multivariate analysis, three independent associated factors were identified: Alcohol use was significantly associated with PSCNP (OR = 9.70; 95% CI: 2.63–35.80; p = 0.001). Hemorrhagic stroke was also an independent associated factor (OR = 8.53; 95% CI: 2.07–35.21; p = 0.003). Obesity was also identified as an independent associated factor (OR = 9.32; 95% CI: 2.48–34.96; p = 0.001) (Table V).

Table V: Multivariable logistic regression analysis of factors associated with PSCNP among stroke survivors

Variable	Adjusted OR(95% CI)	p-value
Alcohol use	9.70 (2.63–35.81)	0.001
Hemorrhagic stroke	8.53 (2.07–35.21)	0.003
Obesity	9.32 (2.49–34.96)	0.001

Outcome

PSCNP developed in most cases within the first three months after stroke, with the highest frequency occurring within the first month (40.7% of patients). Tricyclic antidepressants, particularly amitriptyline, were the main treatment option prescribed, used by 18 patients (66.7%), followed by antiseizure medications in 5 patients (18.5%).

Discussion

This study found that neuropathic pain identified by the DN4 was more frequent among participants with a history of stroke than among the non-stroke comparison group. Among stroke survivors, 27% fulfilled the study criteria for PSCNP. The cross-sectional design, however, does not allow causal inference or estimation of the risk of developing PSCNP.

Given the cross-sectional design, causal relationships cannot be established. The inclusion of a non-stroke control group allowed estimation of relative prevalence but does not support causal inference and should therefore be interpreted cautiously. The frequency of PSCNP observed among stroke survivors in our study was 27%. This estimate is higher than the 5% reported by Bashir et al. among ischemic stroke survivors in Nigeria, while other studies have reported estimates of 29.5% in Italy and 34.5% in Egypt. **[8]**. Differences in study populations, stroke subtypes, diagnostic approaches, timing of assessment, and clinical settings may contribute to this variability. Other studies show similar findings to ours, with Osama et al. reporting a prevalence of 34.5% in Egypt, and Paolucci et al. finding a prevalence of 29.5% in Italy (7,10). This variability in prevalence could be explained by differences in study designs, patient populations, stroke subtypes, diagnostic criteria, and follow-up durations. PSCNP is thought to result from central sensitisation and maladaptive plasticity following disruption of spinothalamic pathways, particularly involving thalamic structures.

Time since stroke and reported pain onset: Among participants with PSCNP, the reported onset of pain occurred most frequently during the first three months after stroke, with the highest proportion reported during the first month. Because pain onset was assessed retrospectively in this cross-sectional study, these findings should be interpreted as reported timing of symptom onset rather than prospective incidence estimates. Bashir et al. found neuropathic pain (NP) in the first three months in 50% of patients, and Klit et al. in Denmark found 60% within the first three months, with about 40% in the first month (6,8). Patients often lacked continuous follow-up after hospitalization and frequently delayed seeking care until symptom worsening. The development after stroke of unusual symptoms several months later may be a sign of a poorly adaptive plastic change in neural organization or connectivity **[11]**.

Characteristics of pain: PSCNP varies among individuals, though it commonly affects the right half-body in our study, corresponding to the side with greater deficits. Vestergaard et al. showed PSCNP could affect small areas (e.g., the hand or foot) or larger regions (e.g., half-body) **[12]**. Leijon et al. also observed variable pain locations based on site **[11]**. Pain intensity was generally moderate, similar to findings from Klit et al. and Bashir et al. **[6]**. The most common features in our study were electric discharge sensations similar to findings from Leijon et al, Klit et al, and Demasles et al, despite variability across studies **[6, 11, 13]**.

Factors associated with PSCNP: Univariate analysis identified several cerebrovascular characteristics associated with PSCNP.

Hypertension: Hypertension was associated with PSCNP in univariable analysis but was not retained as an independent factor in the multivariable model. Although chronic vascular injury may theoretically influence central sensory pathways, the present cross-sectional study cannot determine whether hypertension contributes directly to PSCNP **[14, 15]**.

Alcohol use: The observed association between alcohol use and PSCNP may have several possible explanations, including nutritional deficiencies,

direct neurotoxicity, and effects on neuronal repair **(16)**. However, these mechanisms were not assessed in the present study and therefore remain hypothetical. Moreover, alcohol consumption was self-reported and may have been affected by recall or social desirability bias. The observed association should consequently not be interpreted as evidence of a causal relationship.

Obesity: Obesity was independently associated with PSCNP in our study. Several mechanisms could theoretically contribute to this association, including chronic low-grade inflammation, metabolic dysfunction, and alterations in pain processing **[17]**. However, these mechanisms were not directly investigated in our study. Therefore, obesity should be considered an associated factor rather than an established causal or predictive factor for PSCNP.

In addition to obesity and alcohol use, dyslipidemia may also contribute through lipotoxicity, impairing nerve fibers and contributing to neuropathic pain **[15, 18]**. All participants classified as having PSCNP in our study had a thalamic lesion on neuroimaging, whereas no thalamic lesion was observed among stroke survivors without PSCNP. This finding is consistent with the established role of thalamic and thalamocortical pathways in central pain processing **[19]**. However, because of the small number of PSCNP cases and the complete separation observed between the two groups, an effect estimate for thalamic involvement could not be calculated. This finding should therefore be interpreted as an anatomoclinical observation rather than as evidence of a predictive effect, and it requires confirmation in larger prospective cohorts.

Clinical implications:

Screening for neuropathic pain should be considered as part of post-stroke follow-up, particularly among patients reporting sensory symptoms or persistent pain. The associations observed with alcohol use, obesity, and hemorrhagic stroke may help generate hypotheses for future prospective studies but should not currently be considered established predictors of PSCNP.

Strengths and limitations

This study has several limitations. First, its cross-sectional design precludes causal inference and prospective estimation of the incidence or temporal risk of PSCNP. Second, the study was conducted in a single tertiary hospital, and the hospital-based recruitment of both stroke and non-stroke participants may limit generalizability and introduce selection bias. Third, the non-stroke comparison group was not population-based and was included only to provide a comparative estimate of neuropathic pain frequency. Fourth, neuropathic pain assessment relied on the DN4 as part of the diagnostic assessment and did not include systematic neurophysiological confirmation. Fifth, the diagnosis of PSCNP depended on clinical and neuroanatomical correlation, which may be affected by the availability and quality of neuroimaging. Sixth, some variables, particularly alcohol consumption, were self-reported and may have been subject to recall or social desirability bias. Finally, the relatively small number of PSCNP cases limits the precision of multivariable estimates, as reflected by the wide confidence intervals, and the observed associations should therefore be considered exploratory.

Conclusion

Approximately one-third of stroke patients developed PSCNP, most within three months post-stroke. PSCNP was more frequently observed in stroke patients compared to non-stroke individuals. Alcohol use, hemorrhagic stroke, and obesity were independently associated with PSCNP. These findings highlight the need for systematic screening and targeted management strategies to reduce the burden of PSCNP in low-resource settings.

The declarations

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Ethics approval and consent to participate

Ethical clearance was obtained from the institutional ethical clearance committee of the University of Douala (N° 1696 CEI-UDo/06/2019/T) while administrative authorization from the DGH was obtained from the administrative authority. Patients were informed of the aim of the study and only those who provided formal consent were included.

Consent for publication

All participants signed a consent form that outlined the study's details, including potential risks and benefits, before participating. Privacy rights were strictly observed.

Availability of data and material

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare no competing interests.

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Authors' contributions

A.M. M: wrote the main manuscript text. A.N. V., D.G. M., K.C. and C. K. contributed to the analysis of the results and prepared tables. Y.N. M. and C.T. K. worked on the manuscript and supervised this work. All authors reviewed the manuscript.

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Artificial Intelligence Statement

Artificial intelligence-assisted tools were used exclusively for English language editing and translation of the manuscript. The authors reviewed and validated all the final content and take full responsibility for the accuracy and integrity of the manuscript.

Trial Registration

This study was observational and non-interventional; therefore, trial registration was not required.

The authors does'nt report any conflict of interest.

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