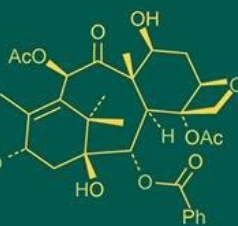
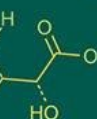


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
 ISSN Online: 2617-4707
 NAAS Rating (2025): 5.29
 IJABR 2025; 9(12): 882-888
www.biochemjournal.com
 Received: 17-09-2025
 Accepted: 22-10-2025

All authors' names and affiliations are given below, after the references.

Assessment of plasma magnesium levels in subjects who have suffered cryptogenic ischemic strokes

Moustapha Djite, Mame Ndoumbé Mbacke, Néné Oumou Kesso Barry, Niokhor Ndane Diouf, Ndiaga Matar Gaye, Pape Matar Kandji, Ndèye Marème Thioune, Siny Ndiaye, Najah Fatou Coly-Gueye, El Hadji Malick Ndour, Fatou Gueye-Tall, Dominique Doupa, Amadou Gallo Diop and Papa Madieye Guey

DOI: <https://www.doi.org/10.33545/26174693.2025.v9.i12k.6678>

Abstract

Introduction: Cryptogenic ischemic stroke (CIS) is a major public health problem, especially in our region where young people are increasingly affected. Several cardiovascular risk factors are being evaluated, including blood magnesium levels. The objective of this study was to evaluate serum magnesium levels in subjects who had suffered cryptogenic IS.

Methodology: This was a case-control study. Subjects were recruited from the neurology department of the Fann University Hospital between November 2021 and May 2022. The samples were analyzed in the biochemistry laboratory of the same facility using enzymatic methods and according to the supplier's recommendations. Statistical analysis was performed using XLSTAT 2022 software, and a p-value <0.05 was considered statistically significant.

Results: The study included 40 subjects with cryptogenic IS, 40 subjects with IS of known cause, and 40 healthy control subjects. In cryptogenic AVCI, the average age was 47 ± 11 years with a predominance of males (57.5%). Magnesium levels were 18.16 ± 2.51 mg/L, and hypomagnesemia was found in 40% of subjects. Furthermore, it was significantly different in smokers and non-smokers ($p=0.024$). A significant correlation was also found between magnesium and triglycerides ($p=0.012$).

Conclusion: Magnesium levels appear to be a relevant parameter to include in the initial assessment of cryptogenic IS due to the high frequency of hypomagnesemia. It could represent a potential biomarker in subjects who have suffered a cryptogenic IS.

Keywords: IS, cryptogenic, magnesium, cardiovascular risk factor

Introduction

The World Health Organization (WHO) defines stroke as the rapid development of localized or global clinical signs of cerebral dysfunction with symptoms lasting more than 24 hours that can lead to death without any apparent cause other than vascular causes ^[1]. The severity of the sequelae depends on the region of the brain affected, the functions it controls, and the duration of the stroke. The larger the area deprived of oxygen, the more severe the sequelae are likely to be.

Strokes are the second leading cause of death worldwide, especially in developing countries. They are the leading cause of acquired disability in adults and the second leading cause of disability worldwide ^[2]. In Senegal, they are estimated to account for 45.78% of neurological conditions requiring hospitalization in Dakar and are responsible for nearly two-thirds of deaths at the Neurological Clinic of the Fann University Hospital Center in Dakar, Senegal ^[3].

The term stroke encompasses: cerebral infarction (CI), also known as ischemic stroke, accounting for 80 to 90% of strokes; hemorrhagic stroke, with intracerebral hemorrhage (ICH) in 10 to 20% of cases; and subarachnoid hemorrhage, which accounts for less than 2% of cases ^[1]. Depending on their etiology, the TOAST classification designates five subtypes of IS (large artery atherosclerosis, cardio embolism, small vessel occlusion, rare cause stroke, and cryptogenic stroke) ^[4]. It has been found that 25% of IS can be classified as cryptogenic ^[5]. Several risk factors have been implicated, the most important of which are advanced age,

Corresponding Author:
Moustapha Djite
 1) Pharmaceutical
 Biochemistry Laboratory,
 Faculty of Medicine,
 Pharmacy, Cheikh Anta Diop
 University, Dakar, Senegal
 2) Biochemistry Laboratory,
 Fann National University
 Hospital, Dakar, Senegal

high blood pressure, diabetes, dyslipidemia, alcoholism, and obesity, among others [6]. As a result, stroke is considered a multifactorial disease involving inflammation and the disruption of several metabolic parameters. Several risk factors are currently being evaluated, including hypomagnesemia [7, 8]. Magnesium has been reported to play a role in atherogenesis, vascular calcification, and the proliferation and migration of endothelial cells and smooth muscle cells that make up the vascular wall [8]. It also plays a key role in vascular balance, neuromuscular transmission, and regulation of arterial tone [9, 10]. However, few studies have been conducted on magnesium and cardiovascular risk, or more specifically on cryptogenic IS. Therefore, the overall objective of this study was to evaluate plasma magnesium levels in subjects with cryptogenic or unknown cause IS.

Methodology

Ethical approval

This study was approved by the Research Ethics Committee (CER) of Cheikh Anta Diop University (UCAD) in accordance with the rules laid down by the National Ethics Committee for Health Research (CNERS) of Senegal under number: 0412/2019/CER/UCAD.

Study setting and type

This is a case-control study of subjects who have suffered cryptogenic strokes. Patients were recruited from the

neurology department of the FANN National University Hospital Center in Dakar. Biochemical parameters were measured at the CHNU/FANN Biochemistry Laboratory.

Study population

Our study involved 40 subjects who had suffered cryptogenic cerebral infarcts, according to the TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification, referred to as Embolic Stroke of Undetermined Source (ESUS) [11, 12] (Figure 1). IS was diagnosed on clinical grounds and confirmed by CT scan data in all patients, and the cryptogenic origin was confirmed by the absence of high-risk embolic heart disease or stenotic (>50%) extracranial or intracranial atherosclerosis. Subjects who had a stroke related to PFO-ASIA (patent foramen ovale-atrial septal aneurysm), non-stenotic (<50%) potentially embolic atheroma, dissection, vasculitis, or small cerebral artery disease (≤ 2.0 cm) were not included in the study.

Two groups of control subjects were recruited, and blood samples were taken under the same conditions as for subjects who had suffered cryptogenic IS. The first group of control subjects consisted of 40 subjects who had suffered IS of known cause and were being monitored in the same neurology department. The second group of control subjects consisted of 40 healthy subjects with no clinically apparent pathologies recruited from blood donors.

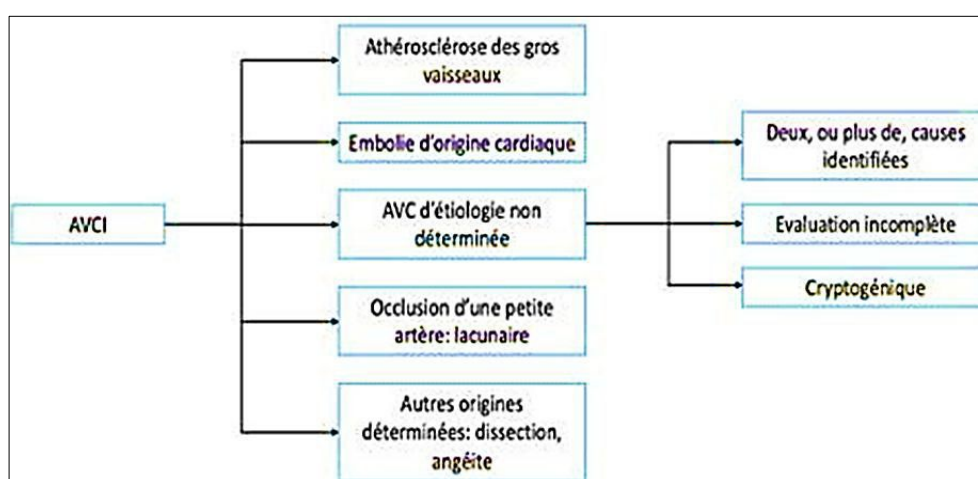


Fig 1: TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification [13]

Parameters studied

For our entire population, we studied epidemiological and clinical parameters, including diabetes, which was determined according to the 2006 WHO criteria (<https://www.who.int/fr/news-room/fact-sheets/detail/diabetes>), as well as physical inactivity (<https://www.who.int/fr/news-room/fact-sheets/detail/physical-activity>). Smoking and alcoholism were determined on the basis of a questionnaire and according to the patient's response. Chronic alcoholism was defined as men consuming 5 drinks per day, or 50 g of ethanol per day, and women consuming 3 drinks per day, or 30 g of ethanol per day. For smoking, a threshold of 5 cigarettes per day was defined, or 50 ug of nicotine absorbed per day. Basic biochemical parameters were

measured in serum using the Architect ci4100 automated analyzer (Abbott, Illinois, USA) using enzymatic techniques in accordance with the manufacturer's recommendations.

Statistical analysis was performed using XLSTAT 2019 software. Data were presented as frequencies and percentages for categorical variables and as mean $\pm$ standard deviation for continuous variables. All continuous variables were tested for normal distribution using the Shapiro-Wilk test, and the significance of differences between groups was tested using the Wilcoxon-Mann-Whitney test and the Kruskal-Wallis test. Categorical variables were compared using the chi-square test and the association between variables was assessed using Spearman's and Pearson's correlation tests. A p-value less than 0.05 was considered significant.

Results

Our study population consisted of 40 patients with cryptogenic IS and 40 patients with IS of known cause followed at the Fann Neurological Clinic. The mean age of patients with cryptogenic IS was 47 ± 11 years, with extremes of 33 and 70 years. The distribution by gender showed a male predominance with a sex ratio of 1.35. Several cardiovascular risk factors were studied, and among them, the most frequently found parameters were hypertension (45%), dyslipidemia (22.5%), and diabetes (15%). Regarding lifestyle parameters, sedentary lifestyle,

smoking, alcoholism, and drug use were found at frequencies of 25%, 25%, 15%, and 5%, respectively.

The assessment of serum magnesium levels showed an average value of 18.16 g/L in cryptogenic IS and 18.08 g/L in IS with known causes.

Analysis of lipid profile parameters revealed a cholesterol level of 1.96 g/L in subjects with cryptogenic IS compared to 1.92 g/L in subjects with IS of known cause. Statistically significant differences were found for blood glucose, CRP, urea, and PAL between cryptogenic IS and IS with known causes (Table 1).

Table 1: Epidemiological, clinical, and biological characteristics of the study population

	Cryptogenic stroke	Determined stroke	Controls	p
Epidemiological parameters				
Mean age (years)	47 ± 11	57.02	47 ± 12	<0.001
Sex ratio	1.35	1.35	1.35	-
Biological parameters				
Magnesium (mg/l)	18.16	18.08	-	0.896
Total cholesterol e (g/l)	1.97	1.93	1.96	0.904
HDL-c (g/l)	0.46	0.44	0.59	0.353
LDL-c (g/l)	1.05	1.04	1.29	0.762
Triglycerides (g/l)	0.87	0.91	0.78	0.725
Blood glucose (g/l)	0.96	1.18	-	<0.001
Urea (g/l)	0.22	0.29	0.16	0.007
Creatinine (mg/L)	7.43	7.48	7.98	0.254
Uric acid (g/L)	55.95	56.74	48.07	0.912
CRP (g/l)	3.41	43.05	1.92	<0.001
AST (IU/L)	34.25	58.28	23.54	0.012
ALT (IU/L)	25.64	34.07	14.61	0.123
GGT (IU/L)	60.72	102.53	32.72	0.080
ALP (IU/L)	60	90	-	0.026
Clinical and lifestyle parameters				
HTA (%)	45	84.61	-	0.002
Diabetes (%)	15	61.53	-	0.040
Dyslipidemia (%)	22.50	64.10	-	0.012
Sedentary lifestyle (%)	25	51.28	-	<0.001
Smoking (%)	25	17.94	-	<0.001
Alcoholism (%)	15	2.56	-	<0.001
Drugs (%)	5	-	-	-

Analysis of our data showed that 40% of subjects who suffered cryptogenic IS had hypomagnesemia. A higher

frequency was found in subjects who suffered definite IS, with a rate of 46.15% (Figure 2).

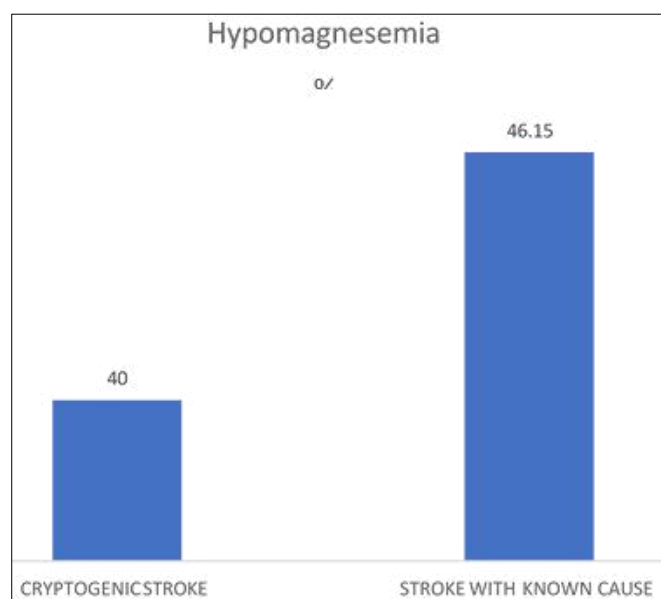


Fig 2: Frequency of hypomagnesemia s in the study population

Analysis of clinical risk factors in our study population following magnesium showed that hypomagnesemia was associated with higher frequencies in subjects with

hypomagnesemia, with the exception of dyslipidemia. However, no significant differences were found except for smoking ($p=0.024$) (Table 2).

Table 2: Evaluation of magnesium concentrations based on clinical parameters and lifestyle Cryptogenic ISA

		mean (sd) (g/l)	median [Q25-75]	min	max	p
Alcohol	NO	17.9 (2.50)	18.3 [17.5 - 19.2]	8.84	22.1	0.066
	YES	19.8 (2.04)	19.9 [19.3 - 20.3]	16.6	22.9	-
Diabetes	NO	18.0 (2.61)	18.5 [17.5 - 19.6]	8.84	22.9	0.75
	YES	18.8 (1.85)	18.6 [17.9 - 19.2]	16.6	22.1	-
Sickle cell disease	NO	18.1 (2.54)	18.4 [17.5 - 19.6]	8.84	22.9	0.66
	YES	18.9 (-)	18.9 [18.9 - 18.9]	18.9	18.9	-
Drug	NO	18.1 (2.55)	18.4 [17.5 - 19.4]	8.84	22.9	0.25
	YES	19.6 (0.636)	19.6 [19.3 - 19.8]	19.1	20.0	-
Dyslipidemia	NO	17.9 (2.69)	18.4 [17.1 - 19.2]	8.84	22.9	0.24
	YES	19.0 (1.55)	19.4 [17.8 - 20.4]	16.7	20.9	-
HTA	NO	17.5 (2.76)	18.2 [17.5 - 19.0]	8.84	20.9	0.14
	YES	19.0 (1.95)	18.8 [17.8 - 20.3]	15.7	22.9	-
Migraine	YES	18.4 (2.17)	18.6 [16.7 - 19.7]	12.4	22.9	0.71
	NO	17.7 (3.01)	18.4 [17.6 - 19.1]	8.84	20.5	-
Sedentary lifestyle	NO	18.4 (2.21)	18.5 [17.5 - 19.6]	12.4	22.9	0.78
	YES	17.6 (3.33)	18.5 [17.0 - 19.3]	8.84	20.4	-
Smoking	NO	17.7 (2.61)	18.2 [16.7 - 19.2]	8.84	22.1	0.024
	YES	19.6 (1.53)	19.4 [18.7 - 20.3]	17.7	22.9	-

Analysis of blood test parameters based on serum magnesium levels showed lower values in cryptogenic IS patients with hypomagnesemia. A statistically significant

difference was found for triglycerides, with a p-value of <0.005 (Table 3).

Table 3: Evaluation of biological factors based on plasma magnesium levels in patients with cryptogenic IS

	Hypomagnesemia NO (n = 24)	Hypomagnesemia YES (n = 16)	p
Age (mean $\pm$ SD)	49.6 (11.9)	43.1 (11.3)	0.089
ALT (mean $\pm$ SD) (IU/L)	24.8 (23.6)	26.9 (25.6)	0.93
ASAT (mean $\pm$ SD) (IU/L)	33.1 (15.8)	36.0 (16.3)	0.42
Total cholesterol (mean $\pm$ SD) (g/L)	2.08 (0.840)	1.81 (0.388)	0.44
Creatinine (mean $\pm$ SD) (mg/L)	7.59 (1.70)	7.20 (1.35)	0.45
GGT (mean $\pm$ SD) (IU/L)	64.1 (62.8)	55.7 (54.5)	0.93
HDL-c (mean $\pm$ SD) (g/L)	0.45 (0.115)	0.47 (0.156)	0.4
LDL-c (mean $\pm$ SD) (g/L)	1.12 (0.610)	0.95 (0.226)	0.76
Magnesium (mean $\pm$ SD) (mg/L)	19.6 (1.20)	16.1 (2.51)	<0.001
PAL (mean $\pm$ SD) (IU/L)	59.2 (21.5)	61.1 (21.7)	0.73
Triglycerides (mean $\pm$ SD) (g/L)	0.96 (0.461)	0.73 (0.228)	<0.005
Urea (mean $\pm$ SD) (g/L)	0.24 (0.161)	0.20 (0.0918)	0.56

Pearson correlation tests were used for variables following a normal distribution, while Spearman's test was applied for triglycerides. The results showed that no significant correlation was found with LDL cholesterol ($p = 0.91$),

HDL cholesterol ($p = 0.63$), or total cholesterol ($p = 0.59$). However, a significant correlation was observed between triglycerides and serum magnesium ($p = 0.012$) (Table 4) (Figure 3).

Table 4: Correlation between magnesium and biochemical parameter

	Correlation coefficient	p	Test	Correlation coefficient (95% CI)
Age	0.228	0.16	Spearman	-
ALAT	-	0.71	Pearson	0.0599 (-0.256; 0.365)
ASAT	0.00546	0.97	Spearman	-
Total cholesterol	-	0.59	Pearson	0.0884 (-0.229; 0.389)
Creatinine	-	0.082	Pearson	0.279 (-0.0361; 0.543)
GGT	-	0.62	Pearson	0.0817 (-0.236; 0.383)
HDL-c	-	0.63	Pearson	0.0774 (-0.240; 0.380)
LDL-c	-	0.91	Pearson	0.0188 (-0.294; 0.328)
PAL	-	0.78	Pearson	0.0462 (-0.269; 0.353)
Triglycerides	0.393	0.012	Spearman	-
Urea	0.177	0.27	Spearman	-

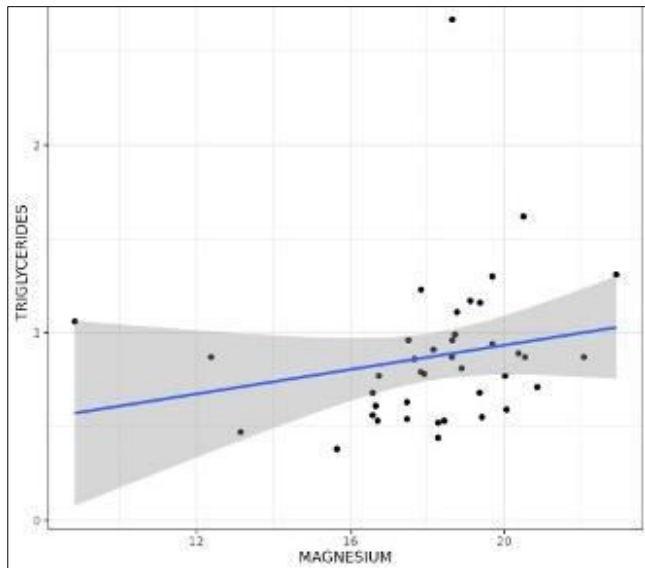


Fig 3: Correlation line between magnesium and triglycerides

Discussion

Stroke is now a major health problem in developing countries. This study is part of research into biological markers of predisposition to stroke. The overall objective was to evaluate plasma magnesium levels in subjects who had suffered cryptogenic ischemic stroke.

Our results showed that cryptogenic ISUs occur at an earlier age compared to other subtypes of ISUs, with a mean age of 47 years. This result is similar to that found by Hajar *et al.*, who reported a mean age of 46 years. It is also close to that found in studies by Mathieu Zuber *et al.* and Putala J *et al.*, which confirmed that more than 40% of cryptogenic AF occur in subjects under the age of 50 [14-16]. However, a study conducted in France found a slightly higher average age of 57 ± 7.13 years [17]. In developed countries, IS occurs 10 to 20 years later, according to studies conducted in Japan and Norway [18, 19]. This disparity seems to be linked to higher life expectancy in industrialized countries and to etiological factors that contribute to the occurrence of stroke. Furthermore, the relatively young age of our population could reflect an under-identification of embolic causes (such as paroxysmal atrial fibrillation) or rare causes (patent foramen ovale, arterial dissection), which are often overlooked due to a lack of advanced diagnostic tools. Several authors emphasize the need for a thorough etiological assessment in young subjects, particularly through prolonged cardiac monitoring or transesophageal echocardiography [20].

The gender distribution in our study showed a male predominance (57.5% vs. 42.5%), with a sex ratio of 1.35. This result is comparable to that obtained in a study conducted by Boyer *et al.*, which reported a male predominance of 56% [21]. This male predominance is regularly found in the literature. According to the study by Yaghi *et al.*, men are more exposed to IS, which could be explained by vascular risk factors that manifest earlier in men, particularly smoking, dyslipidemia, and uncontrolled hypertension [22]. In addition, a systematic review by Ntaios *et al.* also confirmed this male trend in the majority of cohorts of patients with IS [23]. In our study, patients with cryptogenic stroke had classic cardiovascular risk factors, including hypertension (45%), dyslipidemia (22.5%), and diabetes (15%). These frequencies are significantly lower

than those observed in patients with stroke of known cause, suggesting that cryptogenic IS may occur in the absence of a typical cardiovascular risk profile. This finding is consistent with the literature, which shows that cryptogenic IS often affects younger individuals with no apparent major comorbidities [20].

Biologically, our analyses revealed a decrease in HDL cholesterol and an increase in triglycerides, with no marked differences in total cholesterol and LDL cholesterol. These trends suggest a pro-atherogenic metabolic environment, which is typical of the silent metabolic profile [24]. Urea appeared to be the only parameter that differed significantly between the groups ($p = 0.007$), with a higher value in strokes with a determined cause. This elevation could reflect impaired renal function or a more pronounced catabolic state in these patients [25].

Furthermore, this study showed that 40% of cryptogenic SCA subjects were hypomagnesemia, compared to 46% of SCA subjects with a known cause. This confirms a notable prevalence of hypomagnesemia in cryptogenic IS (24.65%). This result could be explained by the importance of magnesium in the body. It acts as a vasodilator, an inflammation regulator, an oxidative stress moderator, and a stabilizer of cardiac ion channel. A prospective meta-analysis of more than one million participants showed that a 100 mg/day increase in dietary magnesium was associated with a 7% reduction in the risk of stroke (RR 0.93; CI 0.89-0.97) [26]. Chronic magnesium deficiency could therefore increase the risk of stroke through several mechanisms (inflammation, endothelial dysfunction, oxidative stress, electrolyte imbalances, and thrombotic diathesis) [27].

During the evaluation of clinical and biological parameters based on serum magnesium levels in patients with cryptogenic ischemic stroke, we observed that hypomagnesemia subjects had a more pronounced tendency toward hypertension (37.5%) and diabetes (12.5%), although the differences did not reach statistical significance. However, these data should not be overlooked, as the simultaneous presence of these factors, even at low intensities, increases overall cardiovascular risk. Indeed, several studies have shown that hypomagnesemia can precede the onset of traditional risk factors, acting as a trigger or aggravating factor [28]. Song *et al.* reported that insufficient dietary magnesium intake is associated with an increased incidence of type 2 diabetes and metabolic disorders [29]. In addition, magnesium deficiency promotes arterial stiffness and abnormal vascular reactivity, two mechanisms involved in hypertension [30].

Patients with decreased magnesium levels had significantly more frequent LDL hypercholesterolemia (41.7% vs. 6.25%, $p = 0.0361$). This observation is consistent with Spanish studies by Barragán *et al.* showing an association between magnesium and LDL cholesterol (association $p = 0.002$), although no clear link was found with triglycerides or HDL cholesterol [31]. In addition, Liu *et al.* found a significant association between hypomagnesemia and dyslipidemia in general (TC, LDL, HDL, TG) [24]. This relationship is corroborated by the meta-analysis by Simental Mendía *et al.*, which shows that magnesium supplementation can improve the lipid profile, particularly by reducing LDL cholesterol and triglycerides [32]. In our population, the significant correlation between triglycerides and hypomagnesemia ($p = 0.012$) further supports this finding. Hypomagnesemia could contribute to silent metabolic

dysregulation, which, in the absence of classic factors (atherosclerosis, atrial fibrillation), would promote a stroke of thromboembolic or microangiopathic origin.

Magnesium is an essential cofactor for enzymes such as lecithin-cholesterol acyltransferase (LCAT), which is involved in HDL metabolism [33]. Magnesium deficiency also leads to increased oxidative stress, chronic inflammation, and insulin resistance, three key factors in dyslipidemia. Population studies confirm that dietary magnesium intake significantly reduces the risk of stroke, heart failure, diabetes, and thus mortality [26]. What makes the impact of hypomagnesemia particularly concerning in the context of cryptogenic stroke is its independence from traditional risk factors. In a multicenter study (Kieboom *et al.*, 2016), moderate hypomagnesemia was associated with an increased risk of cardiovascular events, including stroke, even after adjusting for conventional factors [34].

Conclusion

In the specific context of cryptogenic IS, where conventional causes are absent or not very pronounced, hypomagnesemia emerges as a potential cardiovascular risk factor, contributing to a silent metabolic imbalance. It could be considered both a predictive marker and a therapeutic target in the secondary prevention of these events.

Authors' Declaration

The authors declare having no conflict of interest in relation to this article.

References

1. Béjot Y, Giroud M. Épidémiologie des accidents vasculaires cérébraux. *Med Sci (Paris)*. 2009;25(8-9):727-732.
2. Walker RW, McLarty DG, Kitange HM, Whiting D, Masuki G, Mtasiwa DM, *et al.* Stroke in sub-Saharan Africa: a review. *Trop Med Int Health*. 2000;5(10):786-793.
3. Diop DS. Évaluation des facteurs de risques cardiovasculaires et du risque cardiovasculaire global de l'hypertendu: étude transversale chez 1229 travailleurs sénégalais [thèse de médecine]. Dakar: Université Cheikh Anta Diop; 2009.
4. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, *et al.* Classification of subtype of acute ischemic stroke: definitions for use in a multicenter clinical trial. *Stroke*. 1993;24(1):35-41.
5. Coulibaly A. Dépression post accident vasculaire cérébral dans le service de médecine de l'hôpital du Mali de Bamako: aspects épidémiologique et clinique. Bamako; 2012.
6. Organisation mondiale de la Santé. Plan d'action de la lutte contre les accidents vasculaires cérébraux 2012-2016. Genève; 2012.
7. Odom MJ, Zuckerman SL, Mocco J. The role of magnesium in the management of cerebral vasospasm. *Neurol Res Int*. 2013;2013:1-8.
8. Tangvoraphonkchai K, Davenport A. Magnesium and cardiovascular disease. *Adv Chronic Kidney Dis*. 2018;25(3):251-260.
9. Fondation des maladies du cœur et de l'AVC du Canada. Qu'est-ce qu'un AVC ? Ottawa; 2025.
10. Hachet O, Giroud M, Lorgis L, Cottin Y. Accident vasculaire cérébral et syndrome coronarien aigu. *Cardiologie Cardinale*. 2015;9:4-9.
11. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, *et al.* Classification of subtype of acute ischemic stroke: definitions for use in a multicenter clinical trial. *Stroke*. 1993;24(1):35-41.
12. Hart GR, Diener HC, Coutts SB, Easton JD, Granger CB, O'Donnell MJ, *et al.* Accidents vasculaires cérébraux de source indéterminée: le cas d'une nouvelle construction clinique. *Lancet Neurol*. 2014;13(4):429-438.
13. Timsit S, Breuilly C. Infarctus cérébraux cryptogéniques: de la classification au concept. *Presse Med*. 2009;38:1832-1842.
14. Putaala J, Tatlisumak T. Le moment idéal pour disséquer l'entité de l'accident vasculaire cérébral cryptogénique. *Stroke*. 2014;45(8):2484-2490. DOI:10.1161/STROKEAHA.114.004676.
15. El Omari H, *et al.* Embolic stroke of undetermined source (ESUS): étude comparative du profil étiologique et évolutif selon l'âge. *Rev Neurol (Paris)*. 2025;181(1):173-178. DOI:10.1016/j.neurol.2025.01.173.
16. Zuber M. ESUS (Embolic Stroke of Undetermined Significance): un concept utile ? Paris; 2025.
17. Bourgain M. Évaluation échocardiographique des dimensions de la racine aortique dans le cadre d'un AVC cryptogénique avec un foramen ovale perméable [mémoire]. Amiens: Université de Picardie Jules Verne; 2019.
18. Haute Autorité de Santé. Prévention vasculaire après un infarctus cérébral ou un accident ischémique transitoire. Paris; 2014.
19. Sene-Diouf F, *et al.* Prise en charge des accidents vasculaires cérébraux au Sénégal. *Rev Neurol (Paris)*. 2007;163(9):901-906. DOI:10.1016/S0035-3787(07)91465-4.
20. Hart RG, Diener HC, Coutts SB, Easton JD, Granger CB, O'Donnell MJ, *et al.* Accidents vasculaires cérébraux emboliques de source indéterminée: le cas d'une nouvelle construction clinique. *Lancet Neurol*. 2014;13(4):429-438. DOI:10.1016/S1474-4422(13)70310-7.
21. Boyer A. Épidémiologie de l'accident vasculaire cérébral à l'île de la Réunion consultant en centre hospitalier [mémoire]. Saint-Denis: Université de La Réunion; 2022.
22. Yaghi S, Bernstein RA, Passman R, Okin PM, Furie KL. Accident vasculaire cérébral cryptogénique: recherche et pratique. *Circ Res*. 2017;120(3):527-540.
23. Ntaios G, Papavasileiou V, Milionis H, Makaritsis K, Vemmou A, Koroboki E, *et al.* Accidents vasculaires cérébraux emboliques de source indéterminée dans le registre des AVC d'Athènes. *Stroke*. 2015;46(7):1764-1771. doi:10.1161/STROKEAHA.115.009334.
24. Yaghi S, Elkind MSV. Lipids and cerebrovascular disease: research and practice. *Stroke*. 2015;46(11):3322-3328. DOI:10.1161/STROKEAHA.115.011164.
25. Nawaz S, Khalil M, Amin M, *et al.* Role of urea and creatinine in determining the severity of ischemic stroke. *Pak J Med Sci*. 2016;32(4):1017-1020.

26. Fang X, Wang K, Han D, He X, Wei J, Zhao L, *et al.* Dietary magnesium intake and the risk of cardiovascular disease, type 2 diabetes, and all-cause mortality. *BMC Med.* 2016;14:210.
27. Liu M, Dudley SC Jr. Magnesium, oxidative stress, inflammation, and cardiovascular disease. *Antioxidants (Basel).* 2020;9(10):907. DOI:10.3390/antiox9100907.
28. Fritzen R, Davies A, Veenhuizen M, Campbell M, Pitt SJ, Ajjan RA, *et al.* Magnesium deficiency and cardiometabolic disease. *Nutrients.* 2023;15(10):2355. DOI:10.3390/nu15102355.
29. Song Y, Manson JE, Cook NR, Albert CM, Buring JE, Liu S. Dietary magnesium intake and risk of cardiovascular disease among women. *Am J Cardiol.* 2005;96(8):1135-1141.
30. Rosanoff A, Weaver CM, Rude RK. Statut sous-optimal en magnésium aux États-Unis: les conséquences sur la santé sont-elles sous-estimées ? *Nutr Rev.* 2012;70(3):153-164. DOI:10.1111/j.1753-4887.2011.00465.
31. Găman MA, Dobrică EC, Cozma MA, Antonie NI, Stănescu AMA, Găman AM, *et al.* Crosstalk of magnesium and serum lipids in dyslipidemia and associated disorders. *Nutrients.* 2021;13(5):1411. DOI:10.3390/nu13051411.
32. Simental-Mendía LE, Simental-Mendía M, Sahebkar A, Rodríguez-Morán M, Guerrero-Romero F. Effect of magnesium supplementation on lipid profile. *Eur J Clin Invest.* 2016;46(5):386-397.
33. Nartea R, Mitoiu BI, Ghiorghiu I. The link between magnesium supplements and statin medication in dyslipidemic patients. *Curr Issues Mol Biol.* 2023;45(4):205. DOI:10.3390/cimb45040205.
34. Kieboom BCT, Niemeijer MN, Leening MJG, van den Berg ME, Franco OH, Deckers JW, *et al.* Le magnésium sérique et le risque de décès par maladie coronarienne et de mort cardiaque subite. *J Am Heart Assoc.* 2016;5(1):e002707. DOI:10.1161/JAHA.115.002707.

Authors name and details

Moustapha Djite

Moustapha Djite

- 1) Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal
- 2) Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Mame Ndoumbé Mbacke

Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Néné Oumou Kesso Barry

- 1) Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal
- 2) Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Niokhor Ndane Diouf

Department of Medical Biochemistry, Assane Seck University, Ziguinchor, Senegal

Ndiaga Matar Gaye

Neurological Clinic, Fann National University Hospital, Dakar, Senegal

Pape Matar Kandji

- 1) Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal
- 2) Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Ndèye Marème Thioune

Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Siny Ndiaye

Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal

Najah Fatou Coly-Gueye

Diamniadio Children's Hospital, Dakar, Senegal

El Hadji Malick Ndour

Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal

Fatou Gueye-Tall

Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal

Dominique Doupa

Department of Medical Biochemistry, Saint-Louis University, Saint-Louis, Senegal

Amadou Gallo Diop

Neurological Clinic, Fann National University Hospital, Dakar, Senegal

Papa Madieye Guey

- 1) Pharmaceutical Biochemistry Laboratory, Faculty of Medicine, Pharmacy, Cheikh Anta Diop University, Dakar, Senegal
- 2) Biochemistry Laboratory, Fann National University Hospital, Dakar, Senegal