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Prevalence and associated factors with cognitive disorders in patients with type 2 diabetes at the Laquintinie Hospital in Douala

Prévalence et facteurs associés aux troubles cognitifs chez les patients atteints de diabète de type 2 à l'Hôpital Laquintinie de Douala

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Article Original

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Key words: Prevalence, cognitive impairment, associated factors, type 2 diabetes.

Mots-clés: Prévalence, troubles cognitifs, facteurs associés, diabète de type 2.

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ABSTRACT

Background: Cognitive disorders can lead to poor therapeutic adherence, inadequate glycemic control and progressive loss of autonomy. In the long term, these disorders increase the risk of dementia, particularly of the Alzheimer's or vascular type, thereby worsening morbidity, and dependency. The objective of our study was to investigate the epidemiology of cognitive disorders in patients with type 2 diabetes at Laquintinie Hospital in Douala (HLD).

Methods: We conducted a case-control study over a 4-month period, including 234 diabetic patients. Non-diabetic subjects served as controls. Cognitive function was assessed using the Folstein Mini Mental State Examination. Statistical analyses were performed using SPSS version 20.0.

Results: The mean age of participants was 58.59 ± 11.4 years. Cognitive disorders were more prevalent among diabetic patients (40.6%, 95/234) compared to non-diabetic controls (16.8%, 41/243) ($p < 0.01$). Risk factors significantly associated with cognitive impairment included advanced age (OR = 8.81; $p = 0.006$), systolic blood pressure (OR = 9.54; $p < 0.001$), retinopathy (OR = 6.5; $p < 0.001$), nephropathy (OR = 5.66; $p < 0.001$), cardiovascular disease (OR = 8.57; $p < 0.001$), history of hypoglycemia (OR = 3.15; $p < 0.001$), sedentary lifestyle (OR = 5.16; $p < 0.001$), and abnormal glycated hemoglobin levels (OR = 4.32; $p = 0.006$).

Conclusion: Cognitive disorders were significantly more common in patients with type 2 diabetes than in non-diabetics, and were associated with aging, vascular complications, sedentary lifestyle, and poor glycemic control.

RESUME

Introduction : Les troubles cognitifs augmentent le risque de démence, aggravant la morbidité et la dépendance. L'objectif de notre étude était d'étudier l'épidémiologie des troubles cognitifs chez les patients atteints de diabète de type 2 à l'Hôpital Laquintinie de Douala (HLD).

Méthodologie : Nous avons mené une étude cas-témoins sur une période de 4 mois au cours de laquelle nous avons inclus 234 patients diabétiques. Les témoins étaient des sujets non diabétiques. Les fonctions cognitives étaient évaluées à l'aide du Mini Mental State de Folstein. Les analyses statistiques étaient réalisées avec le logiciel SPSS version 20.0.

Résultats : L'âge moyen des patients était de 58,59 ± 11,4 ans. Les troubles cognitifs étaient plus fréquents chez les diabétiques comparés aux non-diabétiques ($p < 0,01$). Les facteurs de risque de survenue de troubles cognitifs étaient : l'âge (OR = 8,81 ; $p = 0,006$), la pression artérielle systolique (OR = 9,54 ; $p < 0,001$), la rétinopathie (OR = 6,5 ; $p < 0,001$), la néphropathie (OR = 5,66 ; $p < 0,001$), les maladies cardiovasculaires (OR = 8,57 ; $p < 0,001$), les antécédents d'hypoglycémie (OR = 3,15 ; $p < 0,001$), la sédentarité (OR = 5,16 ; $p < 0,001$), l'hémoglobine glyquée anormale (OR = 4,32 ; $p = 0,006$).

Conclusion : Les troubles cognitifs étaient plus fréquents chez les patients diabétiques de type 2. Leur survenue était associée à l'âge, aux complications vasculaires, à la sédentarité et au mauvais contrôle glycémique.

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Introduction

Type 2 diabetes (T2D) is a chronic metabolic condition characterized by high plasma glucose levels related to insulin resistance and/or insufficient insulin production [1] . It is the most common form of diabetes with nearly 90% of cases [2,3] . Patients with this pathology develop long-term complications such as retinopathy, nephropathy, neuropathy, stroke and coronary artery disease that make the disease serious [2,4,5] . Cognitive functions are an umbrella term that includes memory, the ability to learn new things, mental flexibility, attention and executive functions [4,6,7] . The alteration of these latter has a negative impact on the quality of life of patients and constitutes a major public health problem, especially in geriatrics [4,8,9] According to the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM-V 2014), a neurocognitive disorder refers to a decline in one or more cognitive functions; significant, progressive and not explained by a confessional state or by a mental illness [10] . Brain damage from diabetes is increasingly studied and most of these studies focus on people over 65 years of age [5,8,11] . Screening uses cognitive tests; the most widespread being the Mini Mental State Examination (MMSE) of Folstein [12] . Microvascular and macrovascular complications of diabetes causing cerebral ischemia and infarction, chronic hyperglycemia leading to oxidative stress through mitochondrial dysfunction, and cerebral insulin resistance would explain the link between cognitive impairment and type 2 diabetes [13] The clinical picture includes impairment without alteration of quality of life (Mild cognitive impairment) which represent between 25 and 36% of cases and impairment with alteration of quality of life or dementia. According to data from the International Diabetes Federation (IDF) published in 2019, diabetes affects nearly 463 million people worldwide, or 1 in 10 adults, and this prevalence is projected to reach 690 million people in 2030. The prevalence of type 2 diabetes is projected to reach 439 million people in 2030, or 7.7% of the world population [14]. The association between diabetes and impaired cognitive function has been the subject of numerous studies [3,14,15] . A study conducted in France concluded that in 2020 and 2040, the forecasts were respectively 42.3 million Alzheimer's disease (AD), of which 8.1 million would be attributable to diabetes, and 81.1 million AD, of which 24.3 million would be associated with diabetes [16]. A systematic review and meta-analysis published in 2021 concluded that the global prevalence of mild to moderate cognitive impairment in type 2 diabetics was estimated at 45%, i.e. 36.6% in Europe and 46.4% in Asia [3] . In the Middle East, Naguib et al on 269 patients in Saudi Arabia found a frequency of 80.3% patients with impaired cognitive function, including 33.8% cases of severe cognitive impairment [16] . In Africa, the issue of glycemic imbalance and impaired cognitive function is poorly documented; Ossou-Nguiet et al. in Congo reported a prevalence of 57% [9] . In Cameroon, Abba et al found a prevalence of 14.8% at the Douala General Hospital [17] . A better understanding of cognitive disorders in diabetic subjects would allow us to better understand the extent of the problem and to propose optimal management of diabetic subjects, taking into account the impact of these disorders on compliance with antidiabetic therapies, the risk of hypoglycemia and their potential progression to dementia [18] . The present work therefore aimed to study cognitive disorders in type 2 diabetic subjects at Laquintinie Hospital in Douala and to make our contribution to the lack of information on the subject.

Materials and method

The study was conducted over a period of 4 months, from January to May 2021, in the consultation units of the Centre for Hypertension, Diabetes and Obesity (CENHADOB) as well as in the hospitalization unit of Laquintinie Hospital in Douala. The study included two groups of patients: cases, who were patients with type 2 diabetes aged 21 years and over whose diagnosis had been confirmed by a specialist and who were being followed at the Centre for Hypertension, Diabetes and Obesity (CENHADOB) and at the diabetology inpatient unit of the Laquintinie Hospital in Douala and who had consented to participate in the study. Controls were non-diabetic subjects included after a fasting blood glucose level of 0.7-1g/L, including nursing staff and those accompanying patients, after verification that the fasting capillary blood glucose level was normal. To achieve our objectives, we conducted a cross-sectional study over a period of 4 months in the consultation and hospitalisation units of the Laquintinie Hospital in Douala. The sampling technique was non-probabilistic with non-exhaustive collection of diabetic patients seen at the CENHADOB and in the hospitalization unit. Controls were represented by nursing staff and patients' companions after verification that fasting capillary glycaemia was normal. A structured questionnaire and patient records were used to collect sociodemographic, biological and therapeutic data. The questionnaire was completed after explaining the purpose of the study to the participants during a 10- to 15-minute interview. The first part of the questionnaire consisted of sociodemographic data, the second part of biological data, and the third part of therapeutic data, including data on the diagnosis of cognitive disorders. Diagnosis of cognitive disorders using patient records and questioning; screening for cognitive disorders was carried out using the Folstein MMSE. The MMSE was interpreted in accordance with the metanalysis by Ciesielska et al in Poland, comparing the MMSE with the MOCA as follows: 27-30: normal, 20-26: mild cognitive impairment, 15-19: moderate cognitive impairment, 3-15: severe

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impairment, <3: severe impairment.

This study was conducted according to the guidelines for clinical research on experimental models for clinical research on humans, as indicated by the ministry of public health of Cameroon. Administrative authorizations were issued by the institutional human health research ethics committee of the University of Douala (N°2050 CEI-Udo/05/2021/T) and Laquintinie hospital (N°358 AR/MINSANTE/HLD/DM/04/20). The aim of objective of study were explained to each participant in the language they understood best (french or english). Only participant who signed and informed consent were admitted to the study. Participation in the study was voluntary and patients were free to refuse to answer all relevant questions and to whithdraw from the study if the wished at the any time. Also there was not difference in management between patients who agreed to take part in the study and those who did not.

The data were entered into an Excel spreadsheet (Microsoft Office, USA) and analysed using Statistical Package for Social Sciences (SPSS) version 20.0 for Windows (SPSS, IBM, Chicago,IL ,USA). Qualitative variables were presented as frequencies (N,n) and percentages (%), and quantitative variables as mean ± standard deviations(sd) and medians with their interquartile ranges. The Fisher or Pearson chi2 tests of independence were used to determine the dependence of the qualitative variables between the two groups. The Shapiro Wilk normality test was used to study the distribution of the quantitative variables. Parametric comparison tests were performed for variables following a Gaussian distribution, and non-parametric comparison tests were performed for variables following a non-Gaussian distribution. The parametric Mann-Whitney test was used to compare the mean age between the two groups. Binomial logistic regression was used to calculate odds ratios, confidence intervals and pvalues to identify factors associated with cognitive impairment. For all these tests, the confidence interval for the null hypothesis was set at 95%, and the margin of error at 5% (Ho rejected when p-value <0.05).

Results

A total of 477 patients, including 243 cases and 234 controls, were included in our study. Table 1 describes the sociodemographic characteristics of the participants. The mean age of the diabetic patients was 58.6 ± 11.4 years, ranging from 31 to 85 years, and that of the controls was 60.4 ± 10.2 years, ranging from 31 to 84 years. No statistically significant difference was found between these two ages (p-value=0.3). The most represented sex was female with a sex ratio of 0.6. The majority of participants were married (53.1%), had insufficient income (92.9%) and had studied for more than 7 years (65.7%).

Table 1. Socio-demographic characteristics of participants

	Case (N = 243)		Controls (N = 234)		
Parameters	**n**	**%**	**n**	**%**	**p-value**
Age (Years)	58.6 ± 11.4 [31 - 85]		60.4 ± 10.2 [31 - 84]		0,3
					0.09
< 40	16	6	14	5	
[40 - 59[	108	44	99	42	
≥ 60	119	50	121	51	
Sexe					0.01*
Male	100	41	93	39	
Female	143	59	141	61	
Marital status					0.02*
Married	122	50.2	131	55.9	
Unmarried	121	49.8	103	44.1	
Income					0.02*
Sufficient	15	6.1	19	8.1	
Not sufficient	228	93.9	215	91.8	
Level of Study					0.02*
Less than 7 years	79	32.6	85	36.3	
more than 7 years	164	67.4	149	63.7	

Continuous data were presented in the form of mean and standard deviation (Mean ± SD). Categorical data were presented like frequency (N,n) and percentage (%). P-value: continuous data (Mann- Whitney rank sum test); categorial data (Fisher's exact test). * :means that the p-value is significant

Table 2 describes the biological and clinical profile of diabetic patients. This table shows that 60.1% of diabetic patients were overweight or obese, 33.62% were sedentary, the average duration of diabetes was 9.59±8.49 years in our study population, 4.27% had diabetes for less than 10 years and 95.72% for more than 10 years, 80.31% presented with diabetic imbalance, 73.6% had fasting blood glucose above 1.1 g/L, 24.1% were hypertensive. Complications such as hypoglycaemia, retinopathy, neuropathy and nephropathy were present in significant proportions, associated with cardiovascular disease (16.3%) and a disturbed lipid profile.

Figure 1 describes the complications of diabetic patients in our study population. From this figure we find that 46.2% had cardiovascular disease, 30.8% had liver disease, 20.5% had kidney disease and 2.6% had undergone lower limb amputations.

Figure 2 shows the prevalence of cognitive disorders in diabetic and non-diabetic patients. From this analysis, we found that 40.6% of diabetic patients had cognitive disorders and 16.8% of non-diabetic patients had cognitive disorders. Pearson's test of independence showed that these disorders were significantly associated with diabetes (p = 0.01).

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Table 2: biological and clinical profile of diabetic patients

Variables	n(%) / median (IQR)
BMI (Kg/m2)	
] 18,5-24,9]	86 (36.90)
≥ 25	147 (60.10)
Sedentary lifestyle	
Yes	78 (33.62)
No	154 (66.38)
Duration of diabetes (years)	
Average duration and extremes	9.59 ± 8.49 (0.08-38)
<10	10 (4.27)
≥10	224 (95.72)
HbA1C%	
≤7%	25 (19.69)
>7%	102 (80.31)
Fasting blood glucose (g/L), median(IQQ)	1.59 (1.13-2.80)
High fasting blood glucose (≥1.1g/L)	153 (73.6)
Blood pressure	
Systolic arterial hypertension ≥140mmHg	92 (39.8)
Diastolic arterial hypertension ≥ 90 mmHg	78 (36.0)
Combined hypertension ≥ 140/90mmHg	56 (24.1)
Complications of diabetes	
Hypoglycaemia	
≤ 2 episodes	51 (62.2)
> 2 episodes	31 (37.8)
Retinopathy	106 (45.5)
Nephropathy	40 (21.5%)
Neuropathy	
Yes	142 (64.5)
No	79 (35.7)
Cerebrovascular accidents	
Yes	37 (16.3)
Lipid profile (g/L)	
LDL-C (g/L), median (IQR)	1.40 (1.11-1.65)
High LDL-C ≥ 1g/L	21 (29.2)
Total cholesterol (g/L), median(IQR)	2.09 (1.9-2.5)
High total cholesterol (≥2g/L)	48(64.0)
Triglycerides, median (IQQ)	1.52 (1.2-1.72)
Triglycerides, high ≥ 1.5g/L	39 (53.4)
HDL-c (g/L) median, IQQ	0.54 (0.35-0.62)
Low HDL-c <0.4g/L	24(32.9)

BMI: Body Masse Index; HbA1C%:Glycated haemoglobin; LDL-C : Low Density Lipoproteins-Cholesterol ;HDL-C: High Density Lipoproteins-Cholesterol; IQR: Inter-Quartile Range. Data are presented like frequency (n), persentage (%), median

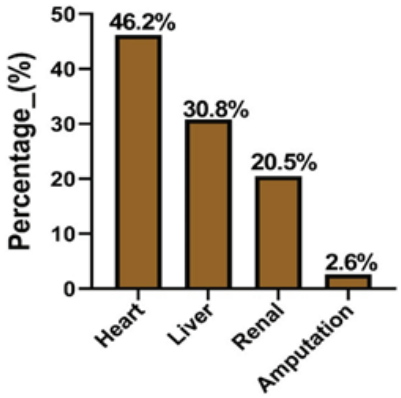

Figure 1: distribution of co-morbidities among cases

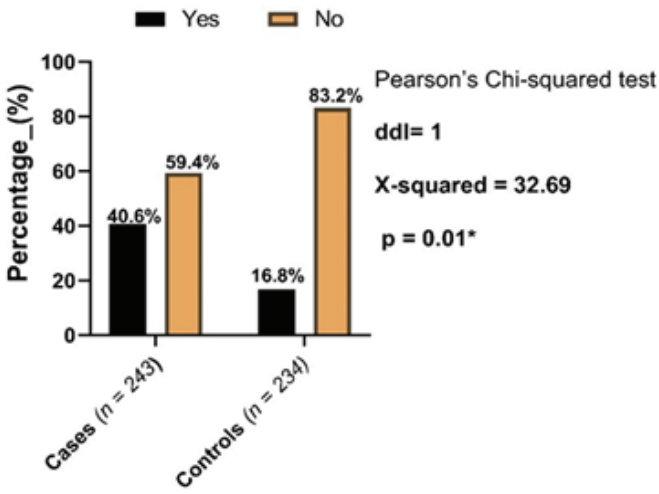

Figure 2: distribution of cognitive disorders in cases (diabetics) and controls (non-diabetics)

Table 3 is the result of binomial logistic regression looking for factors associated with cognitive impairment in diabetic patients.

The table shows that the factors associated with cognitive disorders are: age (OR = 8.81; p = 0.006), systolic blood pressure (OR = 9.54; p<0.001), retinopathy (OR = 6.5; p <0.001), nephropathy (OR = 5.66; p < 0.001), cardiovascular disease (OR = 8.57; p < 0.001), history of hypoglycaemia (OR = 3.15; p < 0.001), sedentary lifestyle (OR = 5.16, p < 0.001), abnormal glycated haemoglobin (OR = 4.32; p = 0.006).

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Table 3: factors associated with cognitive impairment in diabetic patients

	Cognitive disorders					
	Yes		No			
Variables	n	%	n	%	OR (95%CI)	P-value
Age (years)						
<under 40	2	14.3	12	85.7	Ref.	
40-59	21	21.2	78	78.8	1.62 [0.34-7.79]	0.55
≥ 60	72	59.5	49	40.5	8.81 [1.89-41.14]	0.006
Systolic blood pressure (mmHg)						<0,001
≥140	65	70.7	27	29.3	Ref.	
< 140	28	20.1	111	79.9	9.54 [5.18-17.58]	
Diabetic retinopathy						<0,001
No	27	21.3	100	78.7	Ref.	
Yes	68	64.2	38	35.8	6.5 [3.63-11.63]	
Diabetic nephropathy						<0,001
No	53	29.3	128	70.7	Ref.	
Yes	34	85	6	15	5.66 [2.43-11.52]	
History of stroke						<0,001
No	63	33.2	127	38.7	Ref.	
Yes	30	81.1	7	18.9	8.57 [3.57-20.59]	
History of hypoglycemia						<0,001
No	47	30.9	105	69.11	Ref.	
Yes	48	58.5	34	41.5	3.15 [1.81-5.51]	
Sedentary lifestyle						<0,001
No	43	27.9	111	72.1	Ref.	
Yes	52	66.7	26	33.3	5.16 [2.87-9.29]	
HbA1c(%)						0.006
≤7	5	20	20	80	Ref.	
>7	53	33.1	123	69.9	4.32 [1.51-12.42]	

OR: Odds.ratio; CI: Confidence Interval; Data are presented as frequency (n) and percentage (%). P-value/OR: binomial logistic regression was used to identify factors associated with cognitive impairment. Variables with a P-value < 0.05 and OR > 1 were significantly associated with cognitive impairment.

Discussion

Type 2 diabetes is the most common form of diabetes, accounting for over 90% of cases worldwide. Every year, around 93 million people worldwide are diagnosed with type 2 diabetes, and more than a third die from it. Patients with this condition develop long-term complications that adversely affect their quality of life. Cognitive problems are one of the most serious complications of diabetes. In Cameroon, few data are available on this subject; the general objective of this study, which was to investigate the prevalence of and factors associated with cognitive disorders in patients with type 2 diabetes at the Laquintinie Hospital in Douala, shows a major interest in preventing disorders not only for Cameroonian patients but also for improving the therapeutic index.

We found that the frequency of cognitive disorders in diabetic subjects in our study was 40.6% and 16.8% in non-diabetics. These results are lower than those of Ossou-Nguiet et al. in Congo, who reported a prevalence of 57% in cases and 34% in controls [9]. This difference is essentially methodological, as the latter used the Grober and Buschke test, which is a more sensitive local test than the MMSE. A lower prevalence was reported by Abdel-latif et al. in Egypt, i.e. 22% in cases and 9% in controls [20]. This difference is mainly due to the young age group included in their study, and the level of education, which was university. [21]. Abba et al. at Douala General Hospital reported a prevalence of 14.8%. [17]. The difference between their results and those of our series is methodological. Their cut-off point for cognitive disorders was less than 25, while ours was less than or equal to 26[21]. Yang Deng et al. in China found a prevalence of 15.4 [22]. They used two screening tools, the MOCA and the MMSE. A meta-analysis and systematic review by You et al. in China showed that the prevalence of cognitive disorders worldwide was estimated at 45% [3]. In our series, diabetics were 3 times more likely to develop cognitive disorders than non-diabetics. These results are similar to those of Ossou Nguiet et al. in the Congo, who found a risk multiplied by 2.57. Biessel et al. showed that diabetic patients were 1.5-2 times more likely to develop cognitive disorders than the population without diabetes; the effect was more marked in patients in their sixties and older [23]. Among the cases, the cognitive disorders were mild, moderate and severe, respectively 78.9%, 18.9% and 2.1%. This distribution is close to that of Abba et al., who found frequencies of 84.84 for mild disorders, 12% for moderate forms and 3.03% for severe forms. However, in Saudi Arabia, Naguib et al. found 80.3% of diabetics with cognitive dysfunction, 33.8% of which were severe cases due to the use of MOCA. You et al. in a systematic review and metanalysis recommend the use of MMSE for elderly diabetic patients [3].

The average age of diabetics in our study was 58.59 years. This result is similar to that obtained by several authors. In the Congo, Ossou et al. found an average age of 56 for diabetics. [9]. In Saudi Arabia, Naguib et al. found an average age of 56[16]. However, some authors have reported an older age, as in the case of Bauduceau in France, who reported an age of 77±5 years, and Umegaki in Japan, who reported an average age of 74 years. [24]. In Egypt, Abdel-latif et al. found 50.7 ±6.6 years of age. The age range found in our series has been reported by several authors [8, 22, 55, 61, 82]. age greater than or equal to 60 was a non-significant factor associated with the onset of

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cognitive disorders, which may be justified by the fact that in the natural history of type 2 diabetes there is an insidious phase marked by carbohydrate intolerance or moderate fasting hyperglycaemia lasting about ten years, and the patient is not symptomatic; the circumstances in which diabetes is discovered are complications after decades of progression, which gives primary prevention its rightful place [28].

Data on gender are inconsistent in the literature [13,78, 89]. The sex-ratio in our study was 0.66 for cases and 0.67 for non-diabetics. Our results are similar to those of Ossou-nguiet et al. who reported a predominance of women with a sex ratio of 0.82 in diabetics. The distribution of cognitive disorders according to sex in our study showed a sex ratio of 0.53; the predominance of female cognitive disorders has been reported in several studies.[8,9,16]. However, some authors have reported a male predominance, as in the case of Rama et al. [30]. Our results can be explained by the fact that most of the patients seen for consultation were women, and the average age of the study population is a non-modifiable cardiovascular risk factor in women. The socio-cultural data in our series showed a significant association between low income, low level of education, not having a profession and the occurrence of cognitive disorders. These data can be explained by the average age of the study population. In addition, the Folstein Mini Mental State is a test that is sensitive to the level of education [21,31]. Patients with a primary level had poor performance in calculations and long-term memory. These data are consistent with those in the literature. Naguib et al. in Saudi Arabia reported that patients with a low level of education were 4.67 times more likely to suffer cognitive decline than those with a higher level of education.[16]. Ossou et al. found a significant association between cognitive impairment and low social status. These results are close to ours, respectively level of study, low income < 50000f cfa (OR : 4,28 p = 0,002). However, the confounding factor of depression must be taken into account. With regard to cardiovascular risk factors, systolic arterial hypertension increased the risk of cognitive decline by around 7 times. Several studies point in the same direction. Ossou et al. in the Congo found a significant association between arterial hypertension and cognitive decline [9]. Mark et al. suggested in a review that arterial hypertension was a risk factor for cognitive decline only when it was systolic with values above 180mmHg [32]. Cognitive disorders in hypertensive diabetics are thought to be related to the mechanical hyperpressure exerted on the vessels, leading to hypoxia and ischaemia of the cerebral cells [7, 8, 33, 61]. A sedentary lifestyle was found to be a factor associated with cognitive decline in our study, with a 5-fold risk compared with the active population. There are two possible explanations for this. Sport improves cerebral activity by increasing the volume of maximal cerebral oxygen, which would contribute to better cognition. In addition, sustained activity at a frequency of 3 times a week for 45 minutes would reduce the effects of metabolic syndrome, the action of sympathetic tone and provide psychological well-being [34].

Hypoglycaemia showed a significant association with cognitive decline. Mattishen et al. reported that hypoglycaemia increased the risk of dementia by 1.68 and that dementia increased the risk of hypoglycaemia by 1.61. They therefore concluded that there was a vicious circle between hypoglycaemia and dementia. They therefore concluded that there was a vicious circle[35]. Rozalina et al. showed that intensive treatment in patients with several comorbidities increased the risk of hypoglycaemia by 2 [36]. Yuan et al. reported in a review that repeated episodes of severe hypoglycaemia were responsible for cerebral metabolic stress at the origin of irreversible neuronal degeneration, responsible for atrophy of the hippocampus, a structure strongly involved in memory [37]. All these data underline the importance of individualizing blood glucose targets on a case-by-case basis, as recommended by learned societies [38]. Diabetic microangiopathy, in particular diabetic retinopathy and nephropathy, were significantly associated with cognitive decline. Several authors have reported an association between diabetic retinopathy and impairment of cognitive function, as in the case of Naguib who reported ($p < 0.05$) in patients with severe cognitive disorders [16]. Bauduceau et al., San Shan et al. who reported a strong association between retinopathy and the severity of cognitive disorders [8,39,40]. The link between diabetic retinopathy and cognitive disorders can be explained by the fact that retinal and cerebral vessels share the same embryological origin, and diabetes acts through oxidative stress, loss of pericytes, weakening of the capillary endothelium and its hyperpermeability. Visual problems begin at the stage of oedematous capillaropathy and impair vision, making it impossible to perform tasks such as reading, recognition and constructive praxis [41,42]. As for nephropathy, its association with cognitive decline in our study corroborates that of several authors; in fact, cognitive impairment in diabetic patients can be explained by a breakdown in the blood-meningeal barrier, which has characteristics similar to those of the glomerular filtration barrier. Sloten et al. have suggested that impairment of cognitive function can be anticipated by calculating the ratio of serum protein to serum albumin, which would reflect the severity of cognitive impairment [41]. Jinyu et al. reported a positive correlation between glomerular filtration rate and cognitive function (0.428) and a negative correlation between albuminuria and cognitive function (-0.327) [43]. Macroangiopathic complications in our study were stroke and coronary heart disease. Stroke

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was found to significantly increase the occurrence of cognitive impairment in diabetic patients. These results corroborate the data in the literature. Mapoure et al. evaluating cognitive disorders in stroke victims at Douala General Hospital reported a prevalence of 41.2%; in their series, they counted 16 diabetic patients, i.e. 14.8% of the population of subjects with stroke [44]. These results are close to those found in our series (15.8%). Mark et al. reported that cognitive disorders in stroke patients were related to multiple infarcts or infarcts located in strategic regions of the brain (frontal region, temporal region), and that oxidative stress leads to weakening of the vessels responsible for the deep microbleeds that are the precursors of haemorrhagic strokes [32]. Coronary artery disease was poorly represented in our series, with 2 patients (0.9%). This may be explained by diabetic neuropathy, which causes hypoesthesia and anaesthesia, thereby rendering angina and infarction silent. Cognitive decline in our series was also associated with a glycosylated haemoglobin of >7%. Similar results have been reported in the literature [3,37]. This link can be explained by the fact that the chronic accumulation of glucose will lead to oxidative stress by depletion of cofactors, which will be responsible for the production by the mitochondria of reactive oxygen species which, at the cerebral level, will be responsible for a breach of the blood-atomenic barrier at the hippocampal level, which would explain the cognitive disorders. We did not find any association between hyperglycaemia and the onset of cognitive disorders, and even in the literature we did not find such a link; rather, there is a link between fluctuations in blood glucose levels and cognitive disorders [37].

Conclusion

The prevalence of cognitive disorders was higher in cases compared to reference population. Factors associated with cognitive impairment in diabetic patients were age, systolic blood pressure, retinopathy, nephropathy, cardiovascular disease, history of hypoglycaemia, sedentary lifestyle, abnormal glycated haemoglobin.

Conflicts of interest: The authors declare no conflicts of interest.

Authors' contributions: MEND and OY conceived the idea and the study. MEND, OY and ML collected and entered the data in the field. MEND and NLF supervised data collection in the hospitals. Author DA, NMWS coordinated data entry, MEND, NMWS created figures, performed statistical analyses and interpreted the results with the help of OY drafted the first version of the manuscript with the help of NLF. Authors OY, ML and AMMM reviewed the paper for important intellectual content. Authors MEND and NLF supervised the work at all stages. All authors read and approved the final document before submission.

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