Journal of Advances in Medicine and Medical Research

34(23): 334-342, 2022; Article no.JAMMR.92687
ISSN: 2456-8899
(Past name: British Journal of Medicine and Medical Research, Past ISSN: 2231-0614, NLM ID: 101570965)

Obesity and Dyslipidemia as Risk Factors of Vascular Cognitive Impairment in Adult Hypertensive Nigerian

Oluyinka Bamidele Aborisade [a*], Mabel Ayebatonyo Charles–Davies [a], Mayowa Ojo Owolabi [b] and Emmanuel Oluyemi Agbedana [a]

[a] Department of Chemical Pathology, College of Medicine, University of Ibadan, Ibadan, Nigeria.
[b] Department of Medicine, College of Medicine, University of Ibadan, Ibadan, Nigeria.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JAMMR/2022/v34i234869

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/92687

Original Research Article

Received 20 August 2022
Accepted 23 October 2022
Published 25 November 2022

ABSTRACT

Hypertension impairs the cerebral vasculature resulting in vascular cognitive impairment [VCI]. The role of obesity and plasma lipids shown as risk factors in development of VCI in adults with hypertension is unclear and is investigated in this study.
Normoglycemic individuals [n=216] aged 40-75 years were enrolled into this study between January and December 2019. They included Newly Diagnosed Hypertensives with [NDHCI, n-69] and without Cognitive Impairment [NDH, n-81], age-matched with 66 apparently healthy individuals [Controls]. Anthropometric measures [height, weight, waist circumference [WC], Hip circumference [HC], waist hip rratio [WHR] and body mass index [BMI] and socio-demographic indices, lifestyle and blood pressure were obtained using standard methods. Plasma lipids [Total cholesterol [TC], high density lipoprotein cholesterol [HDL-C], Triglyceride [TG] were determined spectrophotometrically, while low density lipoprotein cholesterol [LDL-C] was calculated using Friedewald formula. Neuropsychological assessment based on cognitive score [CS] was done using community screening instrument for dementia [CSID]. Data were analyzed using statistical package for social sciences [SPSS] software 17.0 version. Analysis of variance [ANOVA] and Post Hoc test were used for comparison of variables while Chi square test was used to find associations between variables. These were considered significant at $p<0.05$.
The obesity indicators [WHR, WC and BMI] and dyslipidemia indicators [TC, LDL-C and triglyceride]

**Corresponding author: E-mail: oluyinkaaborisade@gmail.com;*

levels were significantly higher in NDHCI [WHR=76.97±0.89, WC=37.67±0.61, BMI=28.09±0.49; TC=206.89±3.91, LDL-C=135.76±3.81, triglyceride=135.38±1.63] and NDH [WHR=70.71 ± 0.10] WC=34.74 ± 0.04,BMI= 28.30 ± 0.49 and TC=192.83 ± 4.28,LDLC= 120.35 ± 3.22,Triglyceride=127.00 ± 2.69] relative to control [WHR=51.99 ± 0.83, WC=32.94 ± 0.22, BMI=23.89 ± 0.30 and TC=104.70 ± 7.19,LDLC= 94.80 ± 1.90,Triglyceride=72.77 ± 3.70], p<0.01. Significantly low cognitive scores were found in NDHCI [3.48±0.38] compared with NDH [18.77±0.50] and control [28.76±0.16], p<0.001. WHR and LDL-C had inverse relationships with cognitive score in NDH [β= -14.627, p= 0.306] and NDHCI [β= -0.031, p=0.005] respectively. Moreover, WC [β=12.315, p=0.049], HC [β=12.241, p=0.036] and WHR [β=496.374, p=0.043] in NDH group, had a significantly positive relationship with DBP.
Obesity and atherogenic dyslipidaemia may be associated with the progression of hypertension to cognitive impairment in Nigerian hypertensive adults.

Keywords: Obesity; atherogenic dyslipidemia; cognitive impairment; adult hypertensive.

1. INTRODUCTION

"Dementia, one of the major causes of disability in older people is a complex syndrome characterized by global and irreversible cognitive decline that is severe enough to undermine daily functioning. About a decade ago, the American Psychiatric Association reported that 57.7% of people living with dementia lived in developing countries; a proportionate increase to 70.5% by 2050 is anticipated" [1]. "Dementia can result from impairment of cognition, which is one of the foremost health complications in normal aged life if left uncontrolled" [2].

"Vascular cognitive iimpairment [VCI] is a board spectrum of cognitive and behavioral changes associated with cerebral vascular pathology, characterized by impairment of attention and executive function [including planning, task flexibility, problem solving] ranging from early cognitive decline to dementia. It is a chronic illness that arises from the interplay of genetic, environment and behavioral factors that severely affect social / physical activities and quality of llife" [2,3]. "Goldstein reported an association of hypertension with a wide variety of cognitive deficits including reduced abstract reasoning, executive dysfunction, impaired memory, attention deficits and slowing of mental processing speed" [4]. "Also, hypertensives have shown signs of small vessel disease on conventional MRI which are related to VCI, these include: recent subcortical infarcts [clinically symptomatic], white matter magnetic resonance hyperintensities, lacunes [clinically silent], prominent perivascular spaces, cerebral microbleeds, and atrophy" [5]. "The presence and progression of cerebral atropy is another potentially relevant manifestation [although still poorly characterized] of the small vessel disease in hypertensive patient with VCI" [6].

Hypertension [HTN] is a public health problem in both developed and developing countries, with increasing importance as the major cause of cardiovascular diseases; the leading cause of death worldwide [7] It is estimated to affect about 1.56billon people globally by 2025 [6]. "An estimate of 60 to 70% of HTN in adults is attributable to adiposity [7]. Abdominal obesity, elevated waist circumference and increased body mass index [BMI≥30kg/m2] are independent risk factors for the development and progression of hypertension" [6].

"Hypertension is the leading risk factor for intracranial and extracranial atherosclerosis. Extra cranial lesion is characterized by lipid accumulation in carotid and vertebral arteries, often associated with ulceration and atheroma. This is caused by accumulated fatty deposit and scar tissues which leads to restriction of the circulation and a risk of thrombosis, instability and protrusion which are linked to artery –to artery embolism. Intracranial lesions affect the circle of willis and its major branches which could result in vascular occlusion and final ischemic stroke" [8].

Hypertension in Nigeria affects over 70% of the population [9,10]. It is therefore necessary to prevent the onset of dementia via vascular cognitive impairment by identifying important risk factors for the progression of hypertension [11,12]. This study is aimed at evaluating obesity and dyslipidemia as probable independent risk factors for the progression of hypertension to vascular cognitive impairment.

2. MATERIALS AND METHODS

2.1 Study Design

This study is a case control. Participants were enrolled at Medical Outpatient Unit of the University College Hospital between January and December, 2019 on their Clinic days.

2.2 Study Area

The study area was the University College Hospital Ibadan and its environs.

2.3 Study Population

Two hundred and sixteen participants, aged 40-75 years were enrolled into this study. They consisted of newly diagnosed hypertensives without cognitive impairment, NDH [n=81], newly diagnosed hypertensives with cognitive impairment, NDHCI [n=6] and non hypertensives, non-diabetics with intact cognition, Controls (n=66).

2.3.1 Participants with hypertension

Newly diagnosed hypertensives with systolic blood pressure [SBP] of ≥140 and diastolic blood pressure [DBP] of ≥90mmHg were enrolled into this study. They were non-diabetic, without stroke, without lipid lowering drugs or antihypertensive drugs and had no family history of vascular cognitive impairment. The diagnosis of hypertension was based on the guidelines of the Joint National Committee on hypertension [JNC 7, 2012]. Stage 1 hypertension was SBP =140 – 159mmHg and DBP 90-99mmHg while Stage 2 hypertension was SBP: ≥160mmHg and DBP≥100 mmHg. Diagnosis was made by a consultant Nephrologists at the Medical outpatients Department of the University College Hospital Ibadan.

2.3.2 Participants with hypertension and cognitive impairment

Newly diagnosed hypertensives with cognitive impairment participants whose SBP were ≥140 and DBP of ≥90mmHg and cognitive score were between 0 and 20 [normal reference Range being 0-30score] were enrolled into this study. They were non-diabetic, without stroke, without lipid lowering drugs or antihypertensive drugs, and had no family history of vascular cognitive impairment. Diagnosis of hypertension with cognitive impairment was based on the guidelines of JNC 7 and CSI-D. The CSI-D was validated by the systematic mini mental state examination [SMMSE] [2]. Diagnosis was made by a Consultant Neurologist at the Medical Outpatient Department of the University College Hospital Ibadan.

2.3.3 Controls

These were apparently healthy, normotensive and non-diabetics participants with intact cognitive function as certified by the Neurologist. They were neither on lipid lowering nor antihypertensive medications.

2.4 Demographic and Anthropometric Characteristics

Demographic Indices: Semi structured questionnaire was completed by each participant in order to obtain demographic data; gender, age, smoking history, alcohol consumption, family history of hypertension, cognitive impairment, drug and dietary history, presence of undiagnosed diabetes, chronic kidney disease, educational status, marital status, occupation and lifestyle.

Anthropometric Indices: The anthropometric indices measured were; height, weight, body mass index [BMI], waist circumference [WC], hip circumference [HC] and waist hip ratio(WHR), using standard methods.

2.4.1 Body weight

These were taken using a balance beam scale. The participants dressed in light clothing, stood bare footed on the scale placed on a flat surface. The weight was moved until the beam balances [the arrow aligned] and the weight was recorded to the nearest 0.1 kg.

2.4.2 Height

The height of each participant was measured in meters. The participants stood bare footed as upright as possible on a hard level ground against a vertical wall without raising their heels from the ground. Their feet were kept together while the back and heel were aligned with a ruled bar against the vertical surface. Measurement was made by moving a sliding headpiece to the vertex of the participants head and the reading at that point was recorded to the nearest 0.1 m.

2.4.3 Body mass index [BMI]

This was calculated from the body weight and height by dividing the body weight by the height square as indicated below:

BMI=Body weight [kg]/ Height square [m2].

2.4.4 Waist circumference [WC]

The waist circumference [cm] of each participants was measured using a measuring tape placed at the umbilical level. The subjects stood upright, undressed from waist up to the chest and the waist circumference was measured from the tip of the iliac Crest up the boarder of the 12th rib using standard tape. The measurements were recorded accordingly to the nearest 0.1 cm.

2.4.5 Hip circumference [HC]

The hip circumference of each subject was measured in centimetres [cm]. The subjects stood as upright as possible and the hip circumference was measured as the widest circumference of the hip over light clothing using a non- stretchable tape measure, without any pressure to the body surface and was recorded to the nearest 0.1 cm.

2.4.6 Waist hip ratio [WHR]

This was calculated by dividing the waist circumference by the hip circumference.

> WHR= Waist circumference [cm] / Hip circumference [cm].

2.5 Blood Pressure

"BP measurements were performed using a mercury sphygmomanometer. Adequately sized cuffs [standard cuff of 23×12cm] according to arm circumference were placed on the non-dominant arm. The first and the fifth Korotkoff sounds were taken as the systolic and diastolic measurements respectfully. The measurements were taken after the patient had rested for 10minutes in the sitting position, rested their back, legs resting on the ground [not crossed], empty bladder, with arm comfortably placed at the heart level. Two measurements were taken at 2minutes intervals. The mean of the set of two measurements was calculated to give SBP and DBP respectively. Clinical hypertension was defined as a BP≥140\90mmHg" [AHA, 2019].

2.6 Measurements of Cognitive Function

"The Community screening instrument for dementia [CSID] was used to assess cognitive function and results in a score of 30 [normal] to 0 [impaired]. It provides a global score of cognitive ability that correlates with function in activities of daily living. The CSID measures various domains of cognitive function including orientation to time and place, registration, concentration, short-term recall, naming familiar items, repeating a common expression construct a diagram, and follow a three-step verbal command. It provides opportunity for those that cannot read and write, provides a baseline score of cognitive function and pinpoints specific deficits that can aid in forming a diagnosis. The CSID was validated using SMMSE a reliable instrument that allows practitioners to accurately measure cognitive deficits and deterioration over time" [13].

2.7 Sample Collection

Venous blood [10 mL] was collected aseptically from the participants after an overnight fasting by venipuncture. This was done by applying tourniquet 4-6inches [10-15 cm] above the intended puncture site to obstruct the return of blood to the heart and to distend the vein. The site of the puncture, the media cubital vein in the antecubital fossa was first cleansed with alcohol, the blood was collected with new disposable pyrogen free needle and syringe after the skin had dried.

Blood [2 mL] was dispensed into fluoride oxalate bottle, this was used to analyse fasting plasma glucose immediately after sample collection [Randox Laboratory Ltd, Ireland]. The remaining blood [3mL] was dispensed into ethylene diamine tetra acetic acid [EDTA] bottle for the analysis of lipids [total cholesterol, high density lipoprotein and triglyceride] [Randox Laboratory Ltd, Ireland] while LDL-C was calculated using frieldewald's formulae. All bottles were labeled appropriately and centrifuged at 500g for five minutes after which plasma was extracted and stored in the fridge in small aliquots at -20°C until analysis.

2.8 Statistical Analysis

Data from the study population were collected and analyzed using the Statistical Package for Social Sciences [SPSS] software 17.0 vversion [SPP Inc., Richmond, CA]. Data analysed were considered significant at $p<0.05$.

2.8.1 For quantitative variables

Analysis of variance was used to test significance of variations and Post Hoc test was used for comparison of multiple variables. Linear regression analysis was employed to determine relationship between variables.

2.8.2 For non quantitative variables

Chi square analysis was used for determination of associations between variables.

3. RESULTS

Table 1. Age, anthropometry and blood pressure in hypertensives, hypertensives with cognitive impairment and apparently healthy normotensive individuals with intact cognition

Index	Control [n=66]	NDH [n=81]	NDHCI [n=69]	P1	P2	P3	P4
Age (years)	61.71±0.98	63.88±1.01	62.15±0.97	0.249	0.121	0.216	0.763
Anthropometry							
Weight [kg]	58.42 ± 0.82	69.14±1.09	72.36± 0.88	<0.001∗	<0.001∗	<0.001∗	0.018∗
Height [m]	1.57±0.01	1.57±0.01	1.61±0.01	0.001∗	0.934	0.001∗	0.001∗
BMI [kg/m2]	23.89 ±0.30	28.30±0.49	28.09±0.36	<0.001∗	<0.001∗	0.000∗	0.696
WC [cm]	32.94±0.22	34.74±0.57	37.67±0.61	<0.001∗	0.001∗	<0.001∗	<0.001∗
HC [cm]	38.79±0.16	39.32±0.24	40.36±0.22	<0.001∗	0.080	0.001∗	0.001∗
WHR	51.99±0.83	70.71±0.10	76.97±0.89	<0.001∗	0.031∗	<0.001∗	0.001∗
Blood pressure							
SBP [mmHg]	102.12±1.28	167.54±2.83	161.1±2.39	<0.001∗	<0.001∗	<0.001∗	0.051
DBP[mmHg]	77.36±1.10	98.64±1.61	100.76±1.27	<0.001∗	<0.001∗	<0.001∗	0.279

n=number of subjects, ∗=significant at p<0.05, P1=values obtained from ANOVA, P2= values compared between hypertensives and controls, P3= values compared between hypertensives with cognitive impairment and controls, P4=values compared hypertensives and hypertensives with cognitive impairment. BMI=Body mass index, WC=Waist circumference, HC=Hip circumference, WHR= Waist hip ratio, SBP=Systolic blood pressure, DBP=Diastolic blood pressure. NDH=newly diagnosed hypertensives, NDHCI= newly diagnosed hypertensives with cognitive impairment. Values are in mean±SD

Table 2. Lipids and cognitive score in hypertensives, hypertensives with cognitive impairment and apparently healthy normotensive individuals with intact cognition

Index	Control [n=66]	NDH [n=81]	NDHCI [n=69]	P1	P2	P3	P4
Lipids							
TC[mg/dL]	104.70±7.19	192.83±4.28	206.89±3.91	0.001∗	0.001∗	<0.001∗	0.055
TG [mg/dL]	72.77±3.70	127.00±2.69	135.38±1.69	0.001∗	0.001∗	0.001∗	0.007∗
HDL[mg/dL]	52.29±2.97	44.65±0.29	40.30±2.97	0.001∗	0.001∗	0.001∗	0.877
LDL[mg/dL]	94.80±1.90	120.35±3.22	135.76±3.81	0.001∗	0.001∗	0.001∗	0.001∗
Cognitive score							
0-30	28.67±0.16	18.77± 0.50	3.48±0.38	0.001∗	0.001∗	0.001∗	0.001∗

N=number of participants, ∗=significant at p<0.05, P1=values obtained from ANOVA, P2= values compared between hypertensives and controls, P3= values compared between hypertensives with cognitive impairment and controls, P4=values compared hypertensives and hypertensives with cognitive impairment, TC=Total cholesterol, TG=Triglyceride, HDL=High density lipoprotein, LDL=Low density, Values are in mean ± SD

Table 3. Multiple regression of anthropometric, lipids and cognitive score in hypertensives, hypertensives with cognitive impairment and apparently healthy normotensive individuals with intact cognition

Groups	Dependent	Predictors	B	T	P
NDH	DBP	WC	12.315	2.008	0.049*
		HC	12.241	2.143	0.036*
		WHR	496.374	2.070	0.043*
	Cognitive Score	WHR	-14.627	-2.932	0.004*
		Height	13.365	2.325	0.023*
	HC	HC	0.493	2.115	0.038*
NDHCI	SBP	WC	1.291	2.778	0.007*
	Cognitive Score	LDL	-0.031	2.934	0.005*
Control	Height	WHR	0.044	2.143	0.054

β=Standard coefficient, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, WC=Waist circumference, HpC= Hip circumference, WHR=Waist hip ratio, TG=triglyceride, LDL= Low density lipoprotein, NDH=Newly Diagnosed Hypertensives, NDHCI=Newly Diagonised Hypertensives with cognitive impairment

Table 1 shows comparison of age, anthropometry and blood pressure in NDH and NDHCI and control. The ages of all the 3 groups were similar [p>0.05]. Weight, WC and WHR measures in NDHCI progressed in NDH to NDHCI relative to control. The SBP, DBP and BMI were higher in NDH and NDHCI than control while, height and HC were higher in NDHCI relative to NDH and control [p<0.02].Table 2 shows comparisons of lipids and cognitive score in NDH, NDHCI and control. Plasma TC, TG, LDL-C progressed from NDH to NDHCI relative to control. HDL-C was lower in NDH and NDHCI than control [p<0.01].

Table 3 shows the relationship of Anthropometrics, Lipids and Cognitive score in NDH, NDHCI and control. WC, HC and WHR in NDH, had a significantly positive relationship with DBP [β=12.315, p=0.049; β=12.241, p=0.036; β=496.374,p=0.043], respectively. LDL-C had inverse relationships with cognitive score in NDH [β= -14.627, p= 0.306] and NDHCI [β= -0.031, p=0.005] respectively. There is progressive increase in the anthropometrics and lipids from control to NDH and NDHCI with a significant and corresponding decrease in the cognitive scores. This outcome may propose the involvement of lipid peroxidation in hypertension and the possibility of direct association of LDL-C in the progression of hypertension to cognitive impairment.

4. DISCUSSION

Hypertension is a modifiable risk factor that is associated with cognitive impairment, which has been attributed to underlying pathologies of dyslipidaemia. Thus, prevention of hypertension has been reported to delay cognitive loss [14]. In this present study, the ages of the NDH, NDHCI and control were similar [p>0.05], so the findings may not be attributed to age [15].

4.1 Dyslipidaemia in Hypertension and Cognitive Impairment

Hypertension advances development of atherosclerotic plaques in cerebral supply routes and arterioles, which may prompt blood vessel occlusion**.** Results with respect to relationship between dyslipideamia and cognitive impairment are conflicting [16]. However, in this research, there were progressive increases in total cholesterol, triglyceride, and low density lipoprotein cholesterol [LDL-c] from control to NDH to NDHCI with corresponding decrease in cognitive score. Only LDL cholesterol had an inverse relationship with cognitive score. Dyslipidaemia in hypertensives with and without cognitive impairment has been reported previously [16]. Increasing levels of these lipids particularly LDL-c may be associated with the progression from hypertension to cognitive impairment and may be early biomarkers of cognitive impairment in hypertensives. In cross sectional studies of men with vascular cognitive impairment disease, plasma triglyceride as well as LDL-c levels were increased [17]. Plasma triglyceride had previously been shown as a free risk factor for the development of VCI [18]. High level of Low density lipoprotein-cholesterol has been observed to be related to hippocampal volume and dementia [19,20]. Low HDL-c level is a risk factor for atherosclerosis and stroke and has been embroiled in dementia [21]. In this investigation, reduced level of HDL-C level was observed in NDH and NDHCI compared with control [p<0.01].

4.2 Anthropometry, Hypertension and Cognitive Impairment

Abdominal obesity indicators [WC, WHR, and BMI] increased progressively from control to NDH and NDHCI in this study, linking altered athropometrics with cognitive impairment. These indicators of obesity may be early markers of cognitive impairment [22,23]. Moreover, WHR had an indirect relationship with cognitive score in hypertensives [p<0.004]. Increased HC [an indicator of subcutaneous obesity] was higher in NDHCI than NDH and control. The direct relationship of elevated HC with obesity may be linked with cognitive decline in hypertensives. Body mass index was associated with hypertension and not cognitive impairment in this study, as it was higher in NDH than NDHCI and control [p<0.02] Whitmer [2008] and Shenc, [2019], reported that “abdominal obesity could be more dangerous risk factor for dementia, cardiovascular disease and diabetes than total body obesity. Central obesity in midlife increased the risk of dementia independent of diabetes and cardiovascular comorbidities in their study” [23,24].

4.3 Hypertension and Cognitive Impairment

The SBP and DBP were higher in NDH and NDHCI than control, which suggests that hypertension is not the only contributor to cognitive decline. No significant relationship was observed between blood pressure and cognitive impairment in this study in all groups. Cognitive

impairment may be present irrespective of blood pressure readings in hypertensives contrary to report by Mc Donald *et al*. [2017]. Future research would focus on the assessment of cerebral atrophy and it's potential relationship with obesity and atherogenic dslipidaemia in hypertensive patients with VCI.

5. LIMITATION OF THE STUDY

This research did not capture the point at which dyslipidemia and obesity in hypertension begin to progress to vascular cognitive impairment. Future research should look into this.

6. CONCLUSION

Obesity and atherogenic dyslipidaemia may be associated with the progression of hypertension to VCI in adult hypertensive. Regular assessment of weight, waist circumference, serum lipids, waist hip ratio (altered anthropometrics), improved lifestyle including diet modification and physical activity aimed at weight reduction are recommended.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

Ethical approval was gotten from the UI/UCH joint ethical committee.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Jonathan Graff-Radford. Review of vascular cognitive impairment, continuum management study group of chinese society of cardiology of Chinese Medical association; 2019.
2. Dichgans M, Leys D. Cognitive functioning and JNC. Guidelines for hypertension in older adults J.Gerontol. A Biol.Sci. Med Sci. 2017;72(8):121-126.
3. Tadic M, Cuspidi C, Hering D Hypertension and cognitive dysfunction in elderly: Blood pressure management for this global burden. BMC Cardiovascular Disorders. 2016;16:208.
4. Goldstein FC, Hajjar TM, Dunn CB, Levey AI, Wharton W. The relationship between Cognitive functioning and JNC 8, guidelines for Hypertension in older adults J. Gerontol. A Biol.Sci. Med Sci. 2017; 72:121-126.
5. Teresa Gasull and Adria Arboix. Molecula mechanisms and pathophysiology of acute stroke. emphasis on Biomarkers in the different strokel0/ subtypes. Int. J. Mol.Sci. 2022;23:1497.
6. Muzaimi Mustache, Che Mohammed Nasril, Mazina Mohammed Ghazali. Cerebral small vessel disease (CSVD)-Lessons from animal model: Cerebrovasc Dia. 2010;30(2):157-166; Neurology. 2012; 79(20):2016-7.
7. Masaki Mogul. Could management of blood pressure prevent dementia in the elderly? Clinical Hypertension. 201925:27.
8. Shen C, Zhou Z, Laid S, TaoX,Zhao D, Dong W: Urban- rural- specific trend in prevalence of general and central obesity and association with hypertension in Chinese adults, age18 to 65years. BMC Public Health. 2019;30:19(1):66:1.
9. ChenX, Liu Y, Sun X,Yin Z, Li H, Deng K. Comparison of Body mass index, waist circumference, conicity indexed waist hip ratio for predicting incidence of hypertension. The Rural Chinese Cohort Study. J Hum Hypertension. 2018;32(3): 228-235.
10. Simpson JE, Fernando MS, Clark L, Ince PG, Matthews F, Forster G, O'Brien JT, Barber R, Kalaria RN, Brayne C, Shaw PJ, Lewis CE. Wharton: Cognitive function and ageing neuropathology study group white matter lesions in an unselected cohort of the elderly: astrocytic, microglial and oligodendrocyte precursor cell responses. Neuropathol Appl Neurobiol. 2007;33: 410–419.
11. Shalom Nwodo C.,Olubanke O. Ogunlana,Dominic E, Azuh, Emeka E. J, Iweala C, Uchuegbu, Mercy E, Idachaba, Victor Osanor. Correllation between BMI and WC in Nigeria adult; Implication as indicators of health status. J . Public Health Res. 2013;(2):e16.
12. ChenX, Liu Y, Sun X, Yin Z, Li H, Deng K. Comparison of Body mass index, waist circumference on the incidence of HTN in a community based Chinese population. circumference, conicity indexed waist

hip ratio for predicting incidence of hypertension in the rural Chinese Cohort study. J hum hypertension. 2018:32(3): 228-235.

13. Mufunda J, Mebrahtu G, Usman A, Nyarango P, Kosia A, Ghebrat Y, Ogbamarim A, Masjuan M, Gebremicheal A The prevalence of hypertension and its relationship with obesity: results from a national blood pressure survey in Eritrea. J Hums Hypertens. 2006;20.
14. Charles-Davies MA, Arinola OG, Fasanmade AA, Olaniyi OE, Oyewole OE, Owolabi MO, Hassan O, Ajobo Akinlade KS, Ebesurum MO, Popoola OO, Fabian UA, Rahman SK, Ogunlakin MA, Agbedana EO. Metabolic alterations in different stages of hypertension in apparently healthy nigerian population. International Journal of Hypertension. 2013; 2:351357.
15. Simpson JE, Hosny O, Wharton SB, Heath PR, Holden H, Fernando MS, Matthews F, Forster G, O'Brien JT, Barber R, Kalaria RN, Brayne C, Shaw PJ, Lewis CE, Ince PG Medical Research Council Cognitive Function and Ageing Study Neuropathology Group Microarray RNA expression analysis of cerebral white matter lesions reveals changes in multiple functional pathways. Stroke. 2009;40: 369–375.
16. Dodge HH, Chang CC, Kamboh IM, Ganguli M. Risk of alzheimer's disease incidence attributable to vascular disease in the population. Alzheimers Dement. 2011;7:356–360.
17. Sheng B, Cheng LF, Law CB, Li HL, Yeung KM, Lau KK. Coexisting cerebral infarction in Alzheimer's disease is associated with fast dementia progression: Applying the national institute for neurological disorders and stroke/association internationale pour la recherche et l'enseignement en neurosciences neuroimaging criteria in alzheimer's disease with concomitant cerebral infarction. J Am Geriatr Soc. 2007;55:918–922.
18. Feng L, Ng XT, Yap P, Li J, LeeTS, Håkansson K, Kua EH, Ng TP. Marital Status and cognitive impairment among community-dwelling Chinese older adults: The role of gender and social engagement. Dement Geriatr Cogn Dis Extra. 2014;4(3): 375–384.
19. Rebled GAC, Serrano SJ, Antón LRL, Aznar TC, Aragüés MG. Occupation and risk of cognitive impairment and dementia in people in over 55 years. A systematic review, Spain. Rev Esp Salud Publica. 2016;90:e1-e15.
20. Zuliani G, Cavalieri M,1 Galvani M, Volpato S, Cherubini A, Bandinelli S, Corsi AM, Lauretani F, Guralnik JM, Fellin R, Ferrucci L. Relationship between low levels of high-density lipoprotein cholesterol and dementia in the elderly. The Intl. Chianti Study. J Gerontol A Biol Sci Med Sci. 2010;65A(5):559–564.
21. Cheng Y, Jin Y, Unverzagt FW, Su L, Yang L, Ma F, Hake AM, Kettler C, Chen C, Liu J, Bian J, Li P, Murrell JR, Hendrie HC, Gao S. (2014). The relationship between cholesterol and cognitive function is homocysteine-dependent. Clin Interv Aging. 2014;9:1823–1829.
22. White M. Fernando. Grey matter lesions in an unselected cohort of the elderly: Molecular pathology suggests origin from chronic hypoperfusion injury. Stroke. 2006;37:1391–1398.
23. Neuropathology group of the medical research council cognitive function and ageing study pathological correlates of late-onset dementia in a multicentre, community-based population in England and Wales. Neuropathology Group of the Medical Research Council Cognitive Function and Ageing Study (MRC CFAS). Lancet. 2001;357:169–175.
24. Ni Mhuruchi C, Rodgers A, Pan WH, Gu DF, Woodward M. Asia pacific cohort studies collaboration. Body mass index and cardiovascular disease in Asia–Pacific Region: An overview of 33 cohorts involving 310,000 participants. Int J Epidemiol. 2009;3:751–758.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/92687