Screening for arterial stiffness and its determinants in a population of black African women in Senegal

Dépistage de la rigidité artérielle et de ses déterminants dans une population de femmes noires africaines

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Summary

Background: arterial stiffness is an independent cardiovascular risk factor. It is a loss of arterial compliance in large-caliber arteries related to a modification of viscoelastic mechanical properties.

Objective: this work aims to detect arterial stiffness and its determinants in initially healthy Senegalese women.

Methodology: we conducted a cross-sectional and prospective study over a seven-month period from September 2024 to March 2025. The study was carried out at the Laboratory of Physiology of the Faculty of Medicine, Pharmacy and Odontostomatology of the Cheikh Anta Diop University of Dakar. Were included voluntary women, a priori healthy. An assessment of bio-anthropometric, cardiovascular and biological parameters was carried out. The arterial stiffness was determined by measuring the finger-toe pulse wave velocity (ft-PWV) using a device called pOpmètre®.

Results: we collected a cohort of 57 women whose mean and median age was 50.63 ± 8.10 years and 51.00 years (Q1 = 45.00 years; Q3 = 55.00 years). The mean and median ft-PWV of the study population was 7.56 ± 1.98 m/s and 7.37 m/s (Q1 = 5.90 m/s; Q3 = 8.95 m/s). Among the study subjects, only 7 women or 12.28% had an abnormally high ft-PWV (> 10 m/s). The group of subjects with abnormally high ft-PWV, compared to the group of subjects with normal ft-PWV, had higher waist circumference values (respectively, 103.14 ± 8.36 & 92.72 ± 15.32; p=0.018), visceral fat level (respectively, 11.79 ± 2.69 & 9.04 ± 3.41; p=0.042) and total body fat mass (respectively, 45.77 ± 4.16 & 40.30 ± 8.01; p=0.014). ft-PWV was significantly and positively correlated with age (rho=0.412 p=0.040), percentage of glycated hemoglobin (rho=0.269 p=0.043), serum triglyceride level (rho=0.320 p=0.015) and the ratio Apolipoprotein B/Apolipoprotein A atherogenic index (rho=0.330 p=0.012).

Conclusion: the prevalence of arterial stiffness appears to be relatively low in middle-aged adult women. Cardiovascular and metabolic risk factors such as obesity, chronic hyperglycemia, and dyslipidemia would contribute to early and increased stiffening of the artery wall in young adult black women.

Keywords: arterial stiffness, cardio-metabolic risks, Senegalese women.

Résumé

Contexte : la rigidité artérielle constitue un facteur de risque cardiovasculaire indépendant. Il s'agit d'une perte de la compliance artérielle au niveau des artères de gros calibre en rapport avec une modification des propriétés mécaniques viscoélastiques.

Objectif : ce travail a pour but de dépister la rigidité artérielle et ses déterminants chez des femmes sénégalaises à priori saines.

Méthodologie : nous avons mené une étude transversale et prospective sur une période de sept mois allant de Septembre 2024 à Mars 2025. L'étude a été réalisée au Laboratoire de Physiologie de la Faculté de Médecine, de Pharmacie et d'Odontostomatologie de l'Université Cheikh Anta Diop de Dakar. Ont été inclus des sujets volontaires, à priori saines et de sexe féminin. Une évaluation des paramètres bio-anthropométriques, cardiovasculaires et biologiques a été effectuée. La rigidité artérielle a été déterminée par mesure de la vitesse de progression de l'onde de pouls doigt-orteil (VOPdo) à l'aide d'un dispositif appelé pOpmètre ®.

Résultats : nous avons colligé une cohorte de 57 femmes dont l'âge moyen et médian était de 50,63 ± 8, 10 ans et de 51,00 ans (Q1 = 45,00 ans; Q3 = 55,00 ans). La VOPdo moyenne et médiane de la population d' étude était de 7,56 ± 1,98 m/s et de 7,37 m/s (Q1= 5,90 m/s ; Q3 = 8,95 m/s). Parmi les sujets de l'étude, juste 7 femmes soit 12 ,28 % avaient une VOPdo anormalement élevée (> 10 m/s). Le groupe des sujets avec VOPdo anormalement élevée, comparé au groupe des sujets avec VOPdo normale, avait des valeurs plus élevées du tour de taille (respectivement, 103,14 ± 8,36 & 92,72 ± 15,32 ; p=0,018), du niveau de la graisse viscérale (respectivement , 11,79 ± 2,69 & 9, 04 ± 3,41 ; p=0,042) et de la masse grasse totale (respectivement , 45,77 ± 4,16 & 40,30 ± 8,01 ; p=0,014). La VOPdo était significativement et positivement corrélée à l'âge (rho=0,412 p=0,040), au pourcentage d'hémoglobine glyquée (rho=0,269 p=0,043), au taux sérique de triglycérides (rho=0 ,320 p=0,015) et à l'indice athérogénique Apolipoprotéine B/Apolipoprotéine A (rho=0,330 p= 0,012).

Conclusion : la prévalence de la rigidité artérielle serait relativement faible chez la femme adulte d'âge moyen. Des facteurs de risques cardiovasculaires et métaboliques tels que l'obésité, l'hyperglycémie chronique et la dyslipidémie contribueraient à un raidissement précoce et accru de la paroi des artères chez la femme noire adulte jeune.

Mots clés : rigidité artérielle, risques cardio-métaboliques, femme sénégalaise.

Introduction

Arterial stiffness represents a loss of elasticity of the arteries. It is a loss of arterial compliance, a consequence of biological aging, that is to say, arteriosclerosis **[1]**. It is a witness to the parietal alteration which modifies the physical properties of the artery **[2]**. Increased arterial stiffness is associated with an increased risk of cardiovascular events such as myocardial infarction, high blood pressure, heart failure, and stroke **[3-5]** which are currently one of the leading causes of death in the world. It is now considered an important predictor of cardiovascular diseases and also constitutes a cardiovascular risk factor independent of other classical risk factors **[3]**.

The pulse wave velocity (PWV) is currently recognized as the gold standard for arterial stiffness. It is measured between two arterial sites and assesses the speed at which the pulse wave propagates along the arteries. The stiffer the arteries, the faster the speed of the wave's progression **[6]**.

The pOpmeter® (Axelife SAS, France) is a device that measures the velocity of progression of the finger-toe pulse wave velocity (ft-PWV). It is the speed at which the pulse wave of blood, emitted by the heart during ventricular systole, propagates through the arteries at the level of the finger and toe **[7]**. It is a device that allows easy, fast, and above all reproducible measurement. It has been validated by comparison to the carotid-femoral pulse wave velocity (cf-PWV) **[8]** which is a validated evaluative measure of aortic arterial stiffness.

The arterial stiffness is therefore an important cardiovascular risk indicator whose early detection and identification of specific determinants would contribute to the management, especially the preventive, of cardiovascular disease.

Screening for subclinical arterial stiffness in apparently healthy black African women has not yet been carried out in our region to our knowledge. It is in this context that this study was conducted, the objective of which was to screen for arterial stiffness and its determinants in a population of Senegalese women.

Methodology

Type and period of study

This was a prospective and cross-sectional study. It took place over the period from September 2024 to March 2025 at the Laboratory of Physiology and Physiological Functional Explorations of the Faculty of the Faculty of Medicine, Pharmacy, and Odontostomatology at the University of Cheikh Anta Diop in Dakar (UCAD).

The study involved a sample of 57 women. They were recruited and then informed of the full labor process and they provided their free and informed consent.

We included women who were presumably healthy and aged at least 35 years old, recruited at home and among those accompanying patients to the hospital. We had not include pregnant or breastfeeding women and/or non-consenting women.

Clinical investigation method

Each subject had a general clinical examination, including an assessment of the anthropometric parameters, body composition and cardiovascular constants necessary for the study.

The measurement of height, waist circumference and hip circumference was carried out using a tape meter, to the nearest cm. Then the waist-to-hip ratio was calculated. During the measurements, the participants were barefoot in a standing position, and with both arms hanging freely on the corresponding side, palms facing the thighs.

The waist circumference (cm) was measured at the midpoint between the inferior costal margin and the iliac crest in a perpendicular plane, with the patient standing on both feet approximately 20 cm apart, and with both arms hanging freely. According to the WHO, for women, a waist measurement of less than 80 cm is the normal reference.

The hip circumference was always measured with the same tape to the nearest 0.1 cm at the widest circumference on the buttocks by a research assistant kneeling on the side of the participant, so that the maximum extension level can be normally seen. Thus, for the waist-to-hip ratio, a value less than 0.88 is considered normal for them.

Abdominal obesity is defined based on the parameters of the central fat distribution, which are the waist circumference (WC) or waist size and the waist-to-hip ratio (WHR). According to the NCEPIII, in women, abdominal obesity is retained in front of a WHR $\geq$ 80 cm and/or a WHR > 0.85 **[9]**.

Body weight, body mass index (BMI), body fat percentage, and visceral fat level were assessed using a bio-impedancemeter (Tanita® BC-601 brand 8 touch electrode global and segmental body impedance analyzer). The participants were barefoot, wearing light minimal clothing and standing in the center of the bio-impedancemeter.

Blood pressures, namely systolic and diastolic blood pressure, and heart rate were assessed using a validated Omron® electronic sphygmometer (Series 7, blood pressure monitor with ComFit arm Model BP760CAN) with a properly sized arm

circumference brace, according to proper guidelines. The mean arterial pressure (MAP) was calculated using the formula of Messaï E. Ed. Arnette Blackwell (Paris) 1995: MAP = (SBP + 2 DBP) / 3 the normal value should be between 70 mm Hg and 105 mm Hg **[10]**.

The blood pressures, namely systolic and diastolic blood pressure, and heart rate were assessed using a validated Omron® electronic sphygmometer (Series 7, ComFit® Cuff Blood Pressure Monitor Model BP760CAN) with a cuff appropriate for the arm circumference, according to the appropriate guidelines.

The pulse pressure (PP), in other words the differential pressure, was calculated by taking the difference between systolic and diastolic blood pressure: SBP - DBP. It is considered abnormal when the difference was ≥ 60 mm Hg **[11]**.

Evaluation of biochemical parameters

All study subjects were summoned at 8 a.m. after a 12-hour overnight fast to perform the necessary blood samples for the measurement of the following biological parameters:

- Assessment of glycemic balance: the fasting blood glucose; the glycated hemc
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  Model Assessment of Insulin Resistance: HOMA-IR). The normal value for fasting blood glucose must be < 1.10 g/L and the percentage of glycated hemoglobin must be less than 5.7% **[12]**. The normal value of the HOMA-IR index is < 2.5.
- Evaluation of lipid parameters: Total cholesterol, HDL cholesterol, LDL cholesterol, Triglycerides, Apolipoprotein A and Apolipoprotein B**.**

For lipid parameters, we considered that the normal LDL cholesterol level should be < 1.15 g/l **[13, 14]**. The normal total cholesterol level should be less than 2 g/l, the normal triglyceride level should be < 1.50 g/l, and HDL cholesterol level should be greater than 0.46 g/l **[9, 14, 15]**. The apolipoprotein B/apolipoprotein A ratio (Apolipoprotein B/A) should be ≤ 0.8 **[9]**.

Screening for arterial stiffness

The arterial stiffness parameter we chose is the finger-toe pulse wave propagation velocity (ft-PWV), measured by the pOpmeter® (Axelife SAS, France) according to the recommendations of Hallab et al. **[16]**.

The pOpmeter® is a non-invasive medical device. It hass two photodiode sensors, like the oximetry sensors (the sensor is composed of an emitting diode and a receiving diode), and a cardiac activity sensor to calibrate itself with respect to the R wave. It is also equipped with a signal acquisition and processing system and data visualization and recording software.

The system acquires the useful signals delivered by the sensors. It transmits to a PC, via a USB port, the image of these formatted signals, as well as all the useful data needed to display and processing of information.

In summary, a pOpmeter device includes the following:

- **2 photoplethysmographic sensors**
- **1 power supply (220V)**
- **1 pOpmeter® electronic box**
- **1 data processing software: PopScope®**
- **1 USB-RS232 cable**
- **1 5m USB extension cable.**

The pOpmeter® measures transit times between the finger and toe based on the ECG R wave. It calculates the ft-PWVdo based on the height of patient divided by the difference in transit times.

In the lying position and after ten minutes of rest, the sensors were positioned on the finger of patient (right index finger) and toe (right second toe) following the recommendations for use of the device. Then the difference between the finger wave and that of the toe was measured for 20 seconds. The travel distance was estimated based on the height of patient.

Let us note as follows the transit time to the finger (ttd), the transit time to the toe (tto), the difference in arrival time of the toe-finger wave (DOD). We deduce according to the size the velocity of the upper limb pulse wave (PWVs), velocity of the lower limb pulse wave (PWVi), and the pulse wave velocity index (PWVI) which is equal to the ratio of the PWVi to the PWVs. We also deduce the finger-toe pulse wave velocity (ft-PWV) which is equal to the DOD ratio by the size coefficient that approximates the measurement of the carotid-femoral pulse wave velocity (cf-PWV).

Pulse wave propagation velocity (PWV) is the ratio of the travel distance between two sensors to the travel time. It is in the order of 5 to 15 meters per second. If the two sensors are located on the finger and toe, respectively, then this is referred to as ft-PWV.

The ft-PWVdo values ≥ 10 m/s will be considered indicators of abnormal arterial stiffness, in accordance with the recommendations of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC) **[17]**.

We took three measurements with the pOpmeter® and then we took the mean of the the three measurements.

Statistical analysis

All variables have been recorded in an Excel table. Quantitative variables were described using the mean ± standard deviation, and qualitative variables were described using absolute values and percentages. The Student t-test was used to compare the mean of quantitative variables that followed Gaussian distribution. The Kruskal-Wallis test was used to compare the median of quantitative variables that did not follow Gaussian distribution. Spearman correlation tests were performed to determine associations between the pulse wave velocity and other clinical and biological parameters studied.

The results were considered significant for a value of p-value ≤ 5%. Data analysis was performed using SPSS software version 23.0.

Results

Descriptive results

The mean age of the study population was 50.63 ± 8.10 years with extremes of 36 years and 69 years and the median age was 51.00 years (Q1 = 45.00 years ; Q3 = 55.00 years). In the study population, the age group 35 years to 50 years old concerned 49.12% of the subjects (28 women), that of 51 years to 60 years old oncerned 40.35% of subjects (23 women) and that of 61 years old or more concerned 10.53% of subjects (6 women).

Based on lifestyle, health conditions, and medical history, 15 subjects (26.32%) of the study population engaged in physical activity. It was a regular walk or then gymnastics. There were no smokers or alcohol consumers in the study population. The mean and median fasting blood glucose levels were 0.83 ± 0.66 g/l and 0.82 g/l, respectively (Q1 = 0.75 g/l; Q3 = 0.89 g/l).

Table I: the bio-anthropometric, the cardiovascular and the characteristics of the carbohydrate-lipidic of the study subjects

Variables	Mean ± SD	Median (Q1 ; Q2)
Body mass index (kg/m²)	29.04 ± 6.61	30.60 (23.50 ; 33.65)
Waist circonference (cm)	94.05 ± 14.85	93.00 (81.00 ; 105.50)
Waist / hip ratio	0.87 ± 0.08	0.87 (0.82 ; 0.91)
Visceral fat level	9.37 ± 3.43	9.00 (7.00 ; 12.00)
Body fat mass (%)	40.98 ± 7.83	40.00 (36.65 ; 47.70)
Systolic blood pressure (mm Hg)	134.53 ± 21.70	135.00 (116.00 ; 149.00)
Diastolic blood pressure (mm Hg)	89.07 ± 15.33	89.00 (75.50 ; 101.50)
Pulse pressure (mm Hg)	45.46 ± 11.29	43.00 (36.50 ; 54.00)
Heart rate (bpm)	76.45 ± 10.70	75.00 (69.00 ; 83.00)
Pulse wave velocity (m/s)	7.56 ± 2.00	7.37 (5.90 ; 8.95)
Glycated hemoglobin (%)	5.33 ± 0.66	5.00 (5.00 ; 6.00)
HOMA-IR	1.28 ± 1.38	1.00 (0.00 ; 1.92)
Total cholesterol (g/l)	2.12 ± 0.45	2.13 (1.87 ; 2.39)
HDL cholesterol (g/l)	0.60 ± 0.15	0.59 (0.47 ; 0.71)
LDL cholesterol (g/l)	1.41 ± 0.37	1.41 (1.22 ; 1.60)
Triglycerides (g/l)	0.73 ± 0.29	0.73 (0.50 ; 0.91)
Apolipoprotein B/A	0.67 ± 0.22	0.64 (0.51 ; 0.84)

HOMA-IR : Homeostatic Model Assessment of Insulin Resistance ; SD : standard deviation ; Q : quartile

Analytical results

Evaluation of the ft-PWV characteristics

In the global population, 7 subjects or 12.28% had a ft-PVW higher than normal.

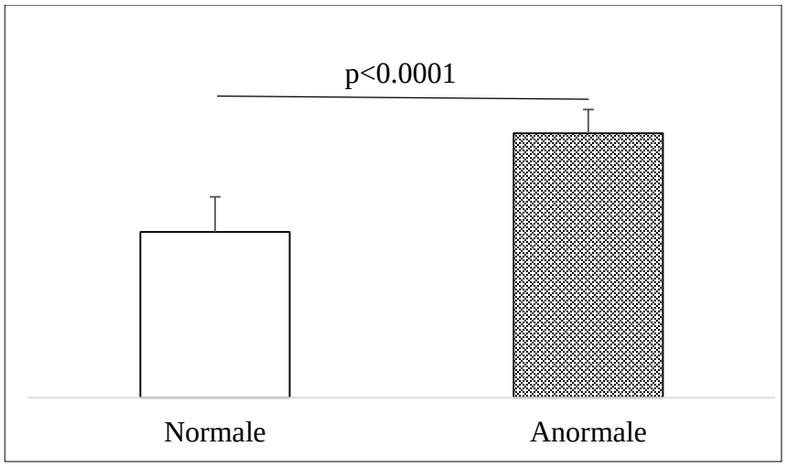

Figure 1: comparison of the mean ft-PWV value between women without arterial stiffness and those with arterial stiffness

Comparison of pulse wave velocity according to anthropometric and body composition parameters, measured and calculated arterial pressures, carbohydrate-lipid parameters

Table II shows that ft-PWV was significantly higher in subjects with abnormally high glycated hemoglobin percentage than in subjects with a normal glycated hemoglobin percentage (respectively, 8.13 ± 2.07 m/s & 7.43 ± 1.98 m/s; p=0.034). The ft-PWV was also significantly higher in subjects with abnormally high total cholesterol levels than in subjects with normal total cholesterol levels (respectively, 7.88 ± 2.07 m/s & 6.74 ± 1.57 m/s; p=0.028).

The ft-PVW was also significantly higher in subjects with abnormally high total cholesterol than in subjects with normal total cholesterol (respectively, 7.88 ± 2.07 m/s & 6.74 ± 1.57 m/s; p=0.028).

Table II: variation in the mean values of anthropometric, cardiovascular and carbohydrate-lipid parameters and ft-PWV

Variables		Numbers N (%)	Mean ± SD	p-value	ft-PWV Mean ± SD	p-value
BMI (kg/m²)	Normal	20 (35.10)	21.89 ± 2.26	<0.0001	7.44 ± 1.75	0.668
	Elevated	37 (64.90)	32.91 ± 4.63		7.67 ± 2.22	
Waist size (cm)	Normal	12 (21.10)	75.25 ± 4.31	<0.0001	7.03 ± 1.45	0.181
	Elevated	45 (78.90)	99.07 ± 12.40		7.74 ± 2.14	
Waist-Hip Ratio (cm)	Normal	34 (59.60)	0.82 ± 0.06	<0.0001	7.32 ± 2.16	0.286
	Elevated	32 (56.10)	0.94 ± 0.06		7.67 ± 2.22	
Visceral fat level	Normal	40 (70.20)	7.60 ± 2.30	<0.0001	7.40 ± 1.80	0.401
	Elevated	17 (29.80)	13.53 ± 1.42		7.95 ± 2.39	
Body fat mass (%)	Lean	11 (19.30)	28.97 ± 5.27	<0.0001	8.00 ± 1.12	0.594
	Normal	19 (33.30)	38.23 ± 1.60		7.53 ± 2.06	
	Elevated	27 (47.40)	47.80 ± 2.19		7.84 ± 1.44	
SBP (mm Hg)	Normal	35 (61.40)	121.23 ± 13.15	<0.0001	7.34 ± 2.02	0.290
	Elevated	22 (38.60)	155.68 ± 14.54		7.92 ± 1.97	
DBP (mm Hg)	Normal	30 (52.60)	77.30 ± 8.78	<0.0001	7.32 ± 2.21	0.359
	Elevated	27 (47.40)	102.15 ± 9.08		7.81 ± 1.76	
MBP (mm Hg)	Normal	21 (36.86)	86.41 ± 5.89	<0.0001	7.32 ± 2.16	0.499
	Elevated	36 (63.14)	114.61 ± 11.62		7.71 ± 1.92	
Pulse pressure (mm Hg)	Normal	51 (89.50)	43.02 ± 8.99	<0.0001	7.53 ± 2.06	0.644
	Elevated	6 (10.50)	66.17 ± 6.52		7.84 ± 1.44	
HbA_1c (%)	Normal	39 (68.40)	4.97 ± 0.30	<0.0001	7.14 ± 1.72	**0.034**
	Elevated	18 (31.60)	6.02 ± 0.14		8.49 ± 2.29	
HOMA-IR	Normal	46 (80.70)	0.78 ± 0.79	<0.0001	7.43 ± 1.98	0.321
	Elevated	11 (19.30)	3.55 ± 1.09		8.13 ± 2.07	
Total cholesterol (g/l)	Normal	19 (33.33)	1.63 ± 0.27	<0.0001	6.74 ± 1.57	**0.028**
	Elevated	38 (66.67)	2.36 ± 0.30		7.88 ± 2.07	
HDL cholesterol (g/l)	Normal	53 (93.00)	0.62 ± 0.13	<0.0001	7.45 ± 2.00	0.353
	Elevated	4 (7.00)	0.33 ± 0.06		8.41 ± 1.72	

LDL cholesterol (g/l)	Normal	12 (21.10)	0.91 ± 0.18	<0.0001	7.45 ± 1.86	0.723
	Elevated	45 (78.90)	1.55 ± 0.28		7.69 ± 2.36	
Triglycerides (g/l)	Normal	55 (96.50)	0.70 ± 0.24	0.012	7.41 ± 1.92	0.335
	Elevated	2 (3.50)	1.56 ± 0.08		10.23 ± 2.36	
Apolipoprotein B/A	Normal	42 (73.70)	0.57 ± 0.14	<0.0001	7.50 ± 2.08	0.920
	Elevated	15 (26.30)	0.97 ± 0.14		7.55 ± 1.86	

BMI : body mass index ; SBP : systolic blood pressure ; DBP : diastolic blood pressure ; MBP : mean blood pressure ; HbA_1c : glycated hemoglobin ; HOMA-IR : homeostatic model assessment of insulin resistance ; ft-PWV : finger-toes pulse wave velocity ; SD : standard deviation

Variations in anthropometric parameters, cardiovascular constants and carbohydrate-lipid parameters according to normal or high ft-PWV

The mean value of age (49.82 ± 7.95 years & 56.43 ± 7.25 years ; p=0.050), waist circumference (92.78 ± 15.32 cm & 103.14 ± 8.36 cm ; p=0.018), body fat mass (40.30 ± 8.01 % & 45.77 ± 4.16 % ; p=0.014), visceral fat level (9.04 ± 3.41 & 11.71 ± 2.69 ; p=0.042), glycated hemoglobin (5.23 ± 0.54 % & 5.74 ± 0.51% ; p=0.040) and triglyceride level (0.70 ± 0.27 g/l & 0.98 ± 0.31g/l ; p=0.050) were significantly elevated in the group of subjects with high ft-PWV when compared to the values of subjects with normal ft-PWV.

Table III: variations in the anthropometric parameters, the cardiovascular constants and carbohydrate-lipid parameters according to normal or high ft-PWV

Paramètres	Normal ft-PWV n = 50	High ft-PWV n = 7	p-value
Age (years)	49.82 ± 7.95	56.43 ± 7.25	**0.050**
Body mass index (kg/m²)	28.66 ± 6.74	31.80 ± 5.13	0.179
Waist circonference (cm)	92.78 ± 15.32	103.14 ± 8.36	**0.018**
Waist – Hip ratio	0.86 ± 0.08	0.93 ± 0.09	0.090
Body mass fat (%)	40.30 ± 8.01	45.77 ± 4.16	**0.014**
Visceral fat level	9.04 ± 3.41	11.71 ± 2.69	**0.042**
Systolic blood pressure (mm Hg)	134.48 ± 22.89	134.86 ± 10.61	0.943
Diastolic blood pressure (mm Hg)	88.92 ± 15.59	90.14 ± 14.31	0.839
Mean blood pressure (mm Hg)	104.11 ± 17.50	105.05 ± 12.72	0.865
Pulse pressure (mm Hg)	45.56 ± 11.72	44.71 ± 8.14	0.814
Heart rate (bpm)	76.11 ± 10.05	79.00 ± 15.71	0.677
Glycated hemoglobin (%)	5.23 ± 0.54	5.74 ± 0.51	**0.040**
HOMA-IR	7.43 ± 1.98	8.13 ± 2.07	0.321
Total cholesterol (g/l)	2.09 ± 0.46	2.28 ± 0.36	0.251
HDL cholesterol (g/l)	0.59 ± 0.15	0.65 ± 0.12	0.299
LDL cholesterol (g/l)	1.40 ± 0.38	1.52 ± 0.29	0.354
Triglycerides (g/l)	0.70 ± 0.27	0.98 ± 0.31	**0.050**
Apolipoprotein B / A	0.66 ± 0.22	0.77 ± 0.27	0.355

HOMA-IR : Homeostatic Model Assessment of Insulin Resistance ; ft-PWV : finger-toes Pulse Wave Velocity

Evaluation of the parameters associated with the ft-PWV

The age of the subjects (rho=0.412 p=0.001), the percentage of glycated hemoglobin (rho=0.269 p=0.043), the triglyceride level (rho=0.320 p=0.015) and the apolipoprotein B/A ratio (rho=0.330 p=0.012) are significantly and positively correlated with ft-PWV.

Table IV : determination of the parameters correlated to the ft-PWV

Variables	Correlation coefficient	
	rho	p-value
Age (years)	**0.412**	**0.001**
Body mass index (kg/m²)	-0.039	0.774
Waist circonference (cm)	0.072	0.567
Waist-Hip Ratio	0.228	0.088
Body mass fat (%)	0.002	0.988
Visceral fat level	0.077	0.570
Systolic blood pressure (mm Hg)	0.178	1.900
Diastolic blood pressure (mm Hg)	0.225	0.096
Mean blood pressure (mm Hg)	0.227	0.090
Pulse pressure (mm Hg)	0.040	0.768
Heart rate (bpm)	0.112	0.434
Glycated hemoglobin (%)	**0.287**	**0.034**
HOMA-IR	0.131	0.330
Total cholesterol (g/l)	0.233	0.081
HDL cholesterol (g/l)	0.058	0.668
LDL cholesterol (g/l)	0.205	0.127
Triglycerides (g/l)	**0.320**	**0.015**
Apolipoprotein B/A	**0.330**	**0.012**

Discussion

Arterial stiffness is a loss of arterial compliance, a marker for changes in viscoelastic characteristics that predispose to the development of cardiovascular diseases. To screen for subclinical arterial stiffness, we conducted a cross-sectional and prospective study from a cohort, apparently healthy, made of 57 young adult women with a mean age of 50.63 ± 8.10 years, with a predominance of the age group 40 to 60 years which concerned 89.47% of the study population.

Among the study subjects, 7 women (12.28%) had a ft-PWV higher than the normal value (10 m/s). The prevalence of arterial stiffness is relatively low in our study population. This finding goes in the same direction as the literature data. Indeed, a low prevalence of arterial stiffness in young adult women has been reported by several studies **[18]**. Authors suggest that female sex steroids may exert their actions to reduce arterial stiffness in young adult women **[19]**. Estrogens exert a major vasoprotective effect by stimulating the production of nitric oxide (NO) by the endothelium, promoting vasodilation and arterial compliance **[18]**. Estrogens inhibit smooth muscle cell proliferation and collagen degradation in the media **[20]**. They also promote a balanced distribution of collagen and elastin, maintaining the flexibility of the arteries **[20]**.

The mean age was significantly higher in the group of subjects with high ft-PWV when compared to the value of subjects with normal high ft-PWV (respectively, 56.43 ± 7.25 & 49.82 ± 7.95; p=0.050).

In addition, ft-PWV was significantly and positively correlated with age (rho=0.412 p=0.040). Our results show that age is the source of arterial stiffness. This corroborates the data from the literature. It is admitted that arterial stiffness increases with age **[21]**. Age is the main determinant of arterial stiffness. With advancing age, the elastic fibers of the arteries gradually degrade and are replaced by more rigid collagen, reducing arterial compliance. These degenerative changes would contribute to early and increased arterial stiffening over time **[22]**. Although chronological age is a major determinant of arterial stiffness, genetic and environmental determinants can also accelerate arterial stiffness.

Twenty-nine among the subjects (50.88%) had high blood pressure readings at the time of blood pressure measurements. Twelve of them (21.05%) were not known to be hypertensive. The other hypertensives subjects were known to be hypertensive, and they were all on antihypertensive medication, justifying the normal blood pressure observed in 6 known and followed hypertensive women. Among the subjects, 63.16% (36 subjects) had an average blood pressure greater than 96 mm Hg. In contrast, 10.53% of subjects (6 women) had a pulsed pressure greater than 60 mm Hg. Hypertension is also considered as a contributing factor promoting arterial stiffness **[23,**

24]. It is both a consequence and an aggravating factor of arterial stiffness. A meta-analysis demonstrated that arterial stiffness is significantly higher in hypertensive patients compared to normotensive patients, and that blood pressure control reduces this phenomenon of arterial stiffness **[25]**. In a longitudinal study, it was also demonstrated that the evolution of arterial stiffness with age was twice as significant in hypertensive subjects than in normotensive subjects **[26]**. Furthermore, it is accepted that the prolonged decrease in blood pressure is a necessary condition for the normalization of ft-PWV **[27]**. However, we did not observe any association between blood pressure and ft-PWV in our study population. These results are not contradictory to those found in the literature. We have a cohort of 57 women and just 7 subjects with abnormally high ft-PWV, therefore presenting arterial stiffness. In addition, they are in the vast majority of young adult women who have recently been hypertensive or even have high blood pressure numbers under the effect of the white coat. However, the intrinsic elastic properties of the arterial wall are little or not modified in young hypertensive subjects compared to normotensive patients of the same age. The persistence of chronic hypertension will progressively alter the arterial structure and will result in an increase in the alteration of the elastic modulus and a worsening of arterial stiffness in aging hypertensive patients. The phenomenon is explained both by the increase in distension pressure and by the modification of the intrinsic elastic properties by alteration of the mechanical properties of the arterial wall. The consequence is an accentuation of the increase in systolic blood pressure as well as a relative decrease in diastolic blood pressure and therefore an increase in pulse pressure. An analysis of the Framingham cohort showed that the increase in systolic blood pressure and the decrease in diastolic blood pressure with age were much more marked in subjects with previous systolic-diastolic hypertension **[28]**. High blood pressure values accelerate the degradation of arterial walls. The increase in blood pressure directly decreases arterial distensibility, increasing the distension tension.

The consequence is an accentuation of the increase in systolic blood pressure as well as a relative decrease in diastolic blood pressure and therefore an increase in pulse pressure. An analysis of the Framingham cohort showed that the increase in systolic blood pressure and the decrease in diastolic blood pressure with age were much more marked in subjects with previous systolic-diastolic arterial hypertension.

According to BMI, we had 37 obese women (64.9% of subjects) while according to the percentage of total body fat mass, 27 women were obese (47.4% study population). Compared to the waist circumference (WC), 78.9% of subjects (45 women) had an abnormally high WC (i.e abdominal obesity). According to the waist-hip ratio (WHR), 56.1% subjects of the study (32 women), had an abnormally high WHR (i.e abdominal obesity). According to the visceral fat level, we had abdominal obesity in 29.8% of subjects (17 women). The group of subjects with abnormally high ft-PWV, compared to the group of subjects with normal ft-PWV, had higher waist circumference values (respectively, 103.1 ± 8.4 cm & 92.7 ± 15.3 cm; p=0.018), the visceral fat level (respectively, 11.8 ± 2.7 & 9.1 ± 3.4; p=0.042) and of the total fat mass (respectively, 45.8 ± 4.2% & 40.3 ± 8.0%; p=0.014). This finding suggests a probable association between obesity, especially in its abdominal form, and increased arterial stiffness. Obesity is strongly associated with a rapid increase in arterial stiffness. It is accepted that individuals with obesity have a high risk of arterial stiffness, independent of blood pressure, ethnic origin, and age **[29]**. Similarly, it has been shown that cf-PWV is also significantly associated with obesity through significantly and positive correlations between BMI and PWV **[30]**. Wildman et al. reported that the median cf-PWV was 4 m/s higher in obese individuals than in normal-weight individuals **[30]**. Through the production of adipokines, there is an inflammatory reaction **[31]** which contributes to the acceleration of fibrosis and the formation of atheroma, with an increase in cardiovascular risk **[32]**, having among other things an independent effect of increasing arterial stiffness **[33, 34]**. High ft-PWV affected 27.8% of subjects with a high glycated hemoglobin percentage and 18.2% of subjects with a high insulin-resistant index. In addition, the only subject with fasting blood glucose exceeding 1.10 g/l had a high ft-PWV. The value of the percent glycated hemoglobin was significantly higher in the group of subjects with abnormally high ft-PWV when compared to the value found in subjects with normal ft-PWV value (respectively, 5.7 ± 0.5 & 5.2 ± 0.5; p=0.050). In addition, ft-PWV was significantly and positively correlated with the glycated hemoglobin percentage (rho=0.269 p=0.043). Glycated hemoglobin is a reflection of chronic hyperglycaemia (provides information on glycemic control over at least 3 months). Chronic hyperglycemia is an aggravating factor of arterial stiffness. It is a major determinant of an increase in arterial stiffness **[35-37]**. The mean blood glucose

value is correlated with PWV **[38]**. Diabetes significantly and early accelerates arterial stiffness **[39]**. This acceleration occurs independently of other vascular risk factors, which partly explains the presence of an almost constant increase in systolic blood pressure and pulse pressure in diabetic subjects **[40]**. Authors have demonstrated that diabetic patients have higher arterial stiffness than non-diabetic subjects, thus increasing their cardiovascular risk **[41]**. Chronic hyperglycemia leads to arterial remodeling which will be partly responsible for a thickening of the intima and the media with, consequently, an increase in arterial stiffness **[42]**. Chronic hyperglycemia leads to the cross-linking of adjacent collagen fibers through advanced glycation end products (AGEs). Indeed, the formation of AGEs on vascular collagen causes intermolecular covalent bonds "cross-links", which induces a loss of elasticity in the arterial and myocardial walls **[43, 44]**. In other words, AGEs, through collagen cross-linking and interactions with RAGE receptors, contribute to vascular rigidity, diastolic dysfunction, and isolated systolic hypertension **[44, 45]**.

High ft-PWV affected 15.8% of subjects with high total cholesterol, 13.3% of subjects with high LDL cholesterol, 50% of subjects with high triglycerides and 20% of subjects with high Apolipoprotein B/A ratio. In contrast, high ft-PWV did not concern any of the subjects with abnormally low HDL cholesterol levels. The triglyceride level was significantly higher in the group of subjects with high ft-PWV when compared to that of subjects with normal ft-PWV (respectively, 0.98 ± 0.31 & 0.70 ± 0.27; p=0.050). The ft-PWV was significantly higher in the group of subjects with abnormally high total cholesterol levels when compared to the value found in subjects with normal total cholesterol levels (respectively, 7.9 ± 2.1 & 6.7 ± 1.6 ; p=0.028). In addition, ft-PWV was significantly and positively correlated with triglyceride levels (rho=0.320 p=0.015) and Apolipoprotein B/A index (rho=0.330 p=0.012). Several investigations have shown a positive associations between lipid parameters and arterial stiffness **[46-50]**. The biological mechanisms are multiple, varied and often intertwined. The accumulation of lipids in the arterial wall promotes plaque formation, and consequently, local inflammation, which alters arterial wall elasticity. Through oxidative stress, oxidized LDL (ox-LDL) causes endothelial damage and triggers inflammatory responses favoring collagen production and elastin degradation **[51]**. Some authors even recommend a good management of lipid parameters in primary prevention of increased arterial stiffness, and consequently, cardiovascular diseases **[52]**. The loss of elasticity in the arteries contributes to reducing the movement of blood in the artery rather than being pushed out by the elastic return **[28]**. The result is a drop in diastolic blood pressure with some "stagnation" of blood in diastole.

These hemodynamic alterations, which occur during arterial stiffness, have major clinical consequences, being responsible for several cardiovascular diseases whose prevalence is increasing very significantly : systolic arterial hypertension, left ventricular hypertrophy, coronary artery disease, cardiac rhythm disorders, heart failure, strokes and kidney failure **[53, 54]**.

In addition, since the ventricle provides a greater effort to counter the reflected wave (post-charge), the myocardial oxygen consumption is greater. If this phenomenon is combined with a decreased coronary perfusion, an imbalance will occur between oxygen supply and demand. This increases the risk of ischemic complications. In response to the chronic increase in ventricular afterload, left ventricular hypertrophy may develop **[55]**.

The summation of changes in the myocardium and the vicious circle they generate can lead to diastolic dysfunction or cardiomyopathy **[55]**.

Conclusion

The prevalence of arterial stiffness would be relatively low in young adult women. Cardiovascular and metabolic risk factors such as obesity, chronic hyperglycemia, and dyslipidemia are reported to be associated with the onset and the increase of arterial stiffness in young adult women. A larger-scale study is planned to better establish these findings. Thus, several factors influence the increase in arterial stiffness, and their identification allows adapting strategies to prevent arterial stiffness.

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REFERENCES

1. **Laurent S, Boutouyrie P, Lacolley P. Structural and genetic bases of arterial stiffness. Hypertension. 2005; 45(6): 1050-5.**
2. **Avolio A. Arterial Stiffness. Pulse. 2013; 1(1): 14-28.**

3. **Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension. 2001; 37(5): 1236-41.**
4. **Demir S, Akpinar O, Akkus O, Nas K, Unal I, Molnar F et al. The prognostic value of arterial stiffness in systolic heart failure. Cardiology journal. 2013; 20(6): 665-71.**
5. **Bichali S, Bruel A, Boivin M, Roussey G, Romefort B, Roze JC et al. Simplified pulse wave velocity measurement in children: Is the pOpmetre valid? PloS one. 2020; 15(3): e0230817.**
6. **Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. European heart journal. 2006; 27(21): 2588-605.**
7. **Pannier B, Vermeersch S, Collin C, Briet M, Boutouyrie P. Assessment of pulse wave velocity. Artery Research. 2008; 3(1): 3.**
8. **Hallab M, Pichierri S, Boin JM, Tramblay M, Chevalet P, Berrut G. Un nouvel index pour évaluer le vieillissement artériel indépendamment de la pression artérielle : pOpscore®. Annales de cardiologie et d'angeiologie. 2012 ; 61(3) : 184-7.**
9. **National Cholesterol Education Program Expert Panel on Detection E. Treatment of High Blood Cholesterol In A. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation. 2002; 106(25): 3143-421**
10. **Messaï E. Guide des chiffres et formules utiles en pratique médicale préf. de Claude Saint-Maurice. Paris: Arnette Blackwell; 1995. XIII-216 2-7184-0770-0 br. 225 F**
11. **Blacher J, Staessen JA, Girerd X, Gasowski J, Thijs L, Liu L et al. Pulse pressure not mean pressure determines cardiovascular risk in older hypertensive patients. Archives of internal medicine. 2000; 160(8): 1085-9.**
12. **American Diabetes Association Professional Practice C. 9. Pharmacologic Approaches to Glycemic Treatment: Standards of Care in Diabetes-2024. Diabetes care. 2024; 47(1): S158-S78.**
13. **Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2019; 73(24): e285-e350.**
14. **Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. European heart journal. 2020; 41(1): 111-88 10.1093/eurheartj/ehz455**
15. **Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019; 139(25): e1082-e143.**
16. **Fonck E, Feigl GG, Fasel J, Sage D, Unser M, Rufenacht DA et al. Effect of aging on elastin functionality in human cerebral arteries. Stroke. 2009; 40(7): 2552-6.**
17. **Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Bohm M et al. 2013 ESH/ESC guidelines for the management of arterial hypertension: the Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). European heart journal. 2013; 34(28): 2159-219.**
18. **Ahimastos AA, Formosa M, Dart AM, Kingwell BA Gender differences in large artery stiffness pre- and post puberty. The Journal of clinical endocrinology and metabolism. 2003; 88(11): 5375-80.**
19. **Peter I, Kelley-Hedgepeth A, Huggins GS, Housman DE, Mendelsohn ME, Vita JA**

et al. Association between arterial stiffness and variations in oestrogen-related genes. Journal of human hypertension. 2009; 23(10): 636-44.
20. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. The New England journal of medicine. 1999; 340(23): 1801-11.
21. Lee HY, Oh BH. Aging and arterial stiffness. Circulation journal : official journal of the Japanese Circulation Society. 2010; 74(11): 2257-62.
22. Halsey G, Sinha D, Dhital S, Wang X, Vyavahare N. Role of elastic fiber degradation in disease pathogenesis. Biochimica et biophysica acta Molecular basis of disease. 2023; 1869(5): 166706
23. O'rourke M. Systolic blood pressure: arterial compliance and early wave reflection, and their modification by antihypertensive therapy. Journal of human hypertension. 1989; 3 (1): 47-52
24. Safar ME, Frohlich ED. The arterial system in hypertension. A prospective view. Hypertension. 1995; 26(1): 10-4.
25. Safar ME, London GM, Plante GE. Arterial stiffness and kidney function. Hypertension. 2004; 43(2): 163-8.
26. Benetos A, Adamopoulos C, Bureau JM, Temmar M, Labat C, Bean K et al. Determinants of accelerated progression of arterial stiffness in normotensive subjects and in treated hypertensive subjects over a 6-year period. Circulation. 2002; 105(10): 1202-7.
27. Boutouyrie P, Alivon M, Herbert A. Precision, accuracy and added value of central pressure measurement. Journal of hypertension. 2011; 29(6): 1059-60.
28. Franklin SS, Gustin WT, Wong ND, Larson MG, Weber MA, Kannel WB et al. Hemodynamic patterns of age-related changes in blood pressure. The Framingham Heart Study. Circulation. 1997; 96(1): 308-15.
29. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX et al. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. Circulation. 2006; 113(6): 898-918.
30. Wildman RP, Mackey RH, Bostom A, Thompson T, Sutton-Tyrrell K. Measures of Obesity Are Associated With Vascular Stiffness in Young and Older Adults. Hypertension. 2003; 42(4): 468-73.
31. De Jongh RT, Ijzerman RG, Serné EH, Voordouw JJ, Yudkin JS, De Waal HaD-V et al. Visceral and Truncal Subcutaneous Adipose Tissue Are Associated with Impaired Capillary Recruitment in Healthy Individuals. The Journal of Clinical Endocrinology & Metabolism. 2006; 91(12): 5100-6.
32. Tsai WC, Lin CC, Chen JY, Huang YY, Lee CH, Li WT et al. Association of adiponectin with procollagen type I carboxyterminal propeptide in non-diabetic essential hypertension. Blood pressure. 2009; 17(4): 233-8.
33. Kim J-S, Kang TS, Kim J-B, Seo H-S, Park S, Kim C et al. Significant association of C-reactive protein with arterial stiffness in treated non-diabetic hypertensive patients. Atherosclerosis. 2007; 192(2): 401-6.
34. Vlachopoulos C, Pietri P, Aznaouridis K, Vyssoulis G, Vasiliadou C, Bratsas A et al. Relationship of fibrinogen with arterial stiffness and wave reflections. Journal of hypertension. 2007; 25(10): 2110-6 10.1097/HJH.0b013e3282dc25da
35. Cruickshank K, Riste L, Anderson SG, Wright JS, Dunn G, Gosling RG. Aortic Pulse-Wave Velocity and Its Relationship to Mortality in Diabetes and Glucose Intolerance. Circulation. 2002; 106(16): 2085-90.
36. Henry RM, Kostense PJ, Spijkerman AM, Dekker JM, Nijpels G, Heine RJ et al. Arterial stiffness increases with deteriorating glucose tolerance status: the Hoorn Study. Circulation. 2003; 107(16): 2089-95.
37. Alecu C, Gueguen R, Aubry C, Salvi P, Perret-Guillaume C, Ducrocq X et al. Determinants of arterial stiffness in an apparently healthy population over 60 years. Journal of human hypertension. 2006;20(10):749-56.
38. Gordin D, Ronnback M, Forsblom C, Heikkila O, Saraheimo M, Groop PH. Acute hyperglycaemia rapidly increases arterial stiffness in young patients with

type 1 diabetes. Diabetologia. 2007; 50(9): 1808-14.
39. Rahman S, Ismail AA, Ismail SB, Naing NN, Rahman AR. Early manifestation of macrovasculopathy in newly diagnosed never treated type II diabetic patients with no traditional CVD risk factors. Diabetes research and clinical practice. 2008; 80(2): 253-8.
40. Strain WD, Chaturvedi N, Dockery F, Shiff R, Shore AC, Bulpitt CJ et al. Increased arterial stiffness in Europeans and African Caribbeans with type 2 diabetes cannot be accounted for by conventional cardiovascular risk factors. American journal of hypertension. 2006; 19(9): 889-96.
41. Cruickshank K, Riste L, Anderson SG, Wright JS, Dunn G, Gosling RG. Aortic pulse-wave velocity and its relationship to mortality in diabetes and glucose intolerance: an integrated index of vascular function? Circulation. 2002; 106(16): 2085-90.
42. O'leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. Cardiovascular Health Study Collaborative Research Group. The New England journal of medicine. 1999; 340(1): 14-22.
43. Aronson D. Cross-linking of glycated collagen in the pathogenesis of arterial and myocardial stiffening of aging and diabetes. Journal of hypertension. 2003; 21(1): 3-12.
44. Zieman S, Kass D. Advanced glycation end product cross-linking: pathophysiologic role and therapeutic target in cardiovascular disease. Congestive heart failure. 2004; 10(3): 144-9; quiz 50-1.
45. Sell DR, Monnier VM. Molecular basis of arterial stiffening: role of glycation - a mini-review. Gerontology. 2012; 58(3): 227-37
46. Vallee A, Lelong H, Lopez-Sublet M, Topouchian J, Safar ME, Blacher J. Association between different lipid parameters and aortic stiffness: clinical and therapeutic implication perspectives. Journal of hypertension. 2019; 37(11): 2240-6.
47. Wen J, Zhong Y, Kuang C, Liao J, Chen Z, Yang Q. Lipoprotein ratios are better than conventional lipid parameters in predicting arterial stiffness in young men. The Journal of Clinical Hypertension. 2017; 19(8): 771-6.
48. Wang L, Zhi F, Gao B, Ni J, Liu Y, Mo X et al. Association between lipid profiles and arterial stiffness: A secondary analysis based on a cross-sectional study. Journal of International Medical Research. 2020; 48(7).
49. Miao DM, Ye P, Xiao WK, Gao P, Zhang JY, Wu HM. Influence of low high-density lipoprotein cholesterol on arterial stiffening and left ventricular diastolic dysfunction in essential hypertension. J Clin Hypertens (Greenwich). 2011; 13(10): 710-5.
50. Baba M, Maris M, Jianu D, Luca CT, Stoian D, Mozos I. The Impact of the Blood Lipids Levels on Arterial Stiffness. Journal of cardiovascular development and disease. 2023; 10(3).
51. Brinkley TE, Nicklas BJ, Kanaya AM, Satterfield S, Lakatta EG, Simonsick EM et al. Plasma oxidized low-density lipoprotein levels and arterial stiffness in older adults: the health, aging, and body composition study. Hypertension. 2009; 53(5): 846-52.
52. Vallée A. Association Between Lipids and Arterial Stiffness for Primary Cardiovascular Prevention in a General Middle-Aged European Population. Frontiers in cardiovascular medicine. 2022; 9.
53. Safar ME, Levy BI, Struijker-Boudier H. Current perspectives on arterial stiffness and pulse pressure in hypertension and cardiovascular diseases. Circulation. 2003; 107(22): 2864-9.
54. Struijker Boudier HA, Cohuet GM, Baumann M, Safar ME The heart, macrocirculation and microcirculation in hypertension: a unifying hypothesis. Journal of hypertension Supplement : official journal of the International Society of Hypertension. 2003; 21(3): S19-23
55. O'rourke MF, Kelly RP. Wave reflection in the systemic circulation and its implications in ventricular function. Journal of hypertension. 1993; 11(4): 327-37.

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