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RESEARCH ARTICLE

Flow Patterns of Carotid Atherosclerotic Plaques Using a New Us Technology: Vectorial Doppler Flow Imaging

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ABSTRACT

Cardiovascular disease caused by atherosclerosis is the first cause of mortality and disability around the world. To understand the pathophysiology of the atherosclerotic plaques and specially their hemodynamics, the changes with their evolution or with the therapeutic interventions are essential to develop effective diagnostic and therapeutic interventions to stop the cardiovascular pandemics. Ultrasound is a cheap, simple and accessible methodology that experienced in the last three decades an astounding development in terms of technology and diagnostic precision. In the field of doppler ultrasound, Power doppler and Color doppler enabled a detailed evaluation of cardiac valves, arteries and veins that conducted to impressive diagnostic precision and to enable advances in therapeutic interventions. Recently a new doppler technology based on processing in blocks the ultrasound information and transduce it into a vector representation of the displacement of blood flow in the space, named VFLOW (acronysm of Vector Flow Imaging), conducted to a new field of investigation of complex flow patterns, by means of the instantaneous measurement of speed, flow gradients, wall shear stress and vessel wall stiffness. We conducted an investigation of different hemodynamic patterns according to plaque structure and vascular regional hemodynamics. Two main groups of plaques have been described, the soft ones ("expanding") and the stiff or hard ones ("non expanding"). The first type has been associated with acute cardiovascular complications and increased cardiovascular risk and just the opposite, chronic lesions in stable disease to the later. We analyze in this paper the characterization of both types of plaques, their hemodynamic patterns and in particular, for the first time, the behavior of the wall shear stress at different sectors of the plaques which may be linked to their development and/or complications. This technology deserves further development face to future applications in the diagnosis and treatment of atherosclerotic vascular disease.

Keywords: Atherosclerosis, Atherosclerotic plaques, Arterial Ultrasound, Carotid ultrasound, Vectorial Doppler, Wall Shear Stress, Pulse Wave Velocity, Arterial Stiffness.

Introduction

Cardiovascular disease caused by atherosclerosis is the first cause of mortality and disability around the world. This pandemic is far to be controlled as the focus is more in reacting rather after the arteries are occluded than in the early stages of the disease through cardiovascular prevention on the main risk factors. The link between the genetics, the risk factors and environmental conditioners with the atherosclerotic plaques is complex, and a deep and comprehensive approach to these mechanisms and interactions is urgently needed to give a rationale to preventive interventions and develop appropriate prevention measures. [1, 2]

However, understanding the pathophysiology of the atherosclerotic plaques and specially their hemodynamics, the changes with their evolution or with therapeutic interventions are essential to develop effective diagnostic and therapeutic interventions to stop the cardiovascular pandemics.[3]

Atherosclerotic plaques can be evaluated in every territory and using a wide number of techniques namely ultrasound, X rays, NMR, thermography, and so on. The choice of the method is based on its availability and its precision to diagnose the vascular pathology and in particular face to use it in the clinical practice easily, non-invasively and friendly to be understood by the referring physicians.[4, 5]

Ultrasound is a cheap, simple and accessible methodology that experienced in the last three decades an astounding development in terms of technology and diagnostic precision. In the field of doppler ultrasound, Pulsed doppler (PWD) and Color doppler (CD) enabled a detailed evaluation of cardiac valves, arteries and veins that conducted to impressive diagnostic contributions and to enable advances in therapeutic interventions. [6]

However, the examination of plaques is mainly limited to the degree of obstruction, structural instability or possibility of thrombosis, face to the need of revascularization therapy. For a long time, the analysis of vulnerability or the hemodynamic patterns of plaques remained in the experimental and theoretical domain, limited to CD assumptions of flow direction or turbulence.[7 8]

Recently a new doppler technology based in processing ultrasound information in blocks and a vector representation of the displacement of blood flow in the space, named VFLOW® led to a new field of investigation and quantification of complex flow patterns, by means of the instantaneous measurement of speed and flow gradients, the wall shear stress (WSS) and the vessel wall stiffness. (Figure 1)[9, 10 11 12]

FIGURE 1: Vector Flow Imaging

Atherosclerotic plaques are evaluated according to the atherosclerotic load (surface or volume of plaques) and their vulnerability by means of Grey Scale grading, chromatic pixeling. Considering the behavior of the plaque in response to the pulsatile forces and flow, two main groups of plaques have been described, the **soft** ones ("expanding plaques") and the **stiff or hard** ones ("non expanding plaques"). The first type has been associated with acute cardiovascular complications

and increased cardiovascular risk and just the opposite, the chronic lesions in stable disease, to the later.[13, 14]

The objective of this study was to analyze different hemodynamic patterns according to plaque structure and behavior and the relation of these parameters with the plaques and near vascular wall regional hemodynamics.

As an exploratory study, we evaluated patients referred to the Non-Invasive Vascular Laboratory during one year, and selected among them, those with plaques in the Left Carotid Artery (LCA) bifurcation, suitable for VFLOW analysis (type of bifurcation, plaque size and composition) and simultaneously, the hemodynamic structural, functional and VFLOW data of the Left Common Carotid Artery (LCCA) must be available. We decided to analyze LCCA because we performed currently LCCA VFLOW hemodynamic evaluations to compare different types of patients or pathologies (hypertensives, diabetics, coronary artery disease of kidney chronic disease patients)

In addition to the characterization of both types of plaques and their hemodynamic patterns, for the first time, we analyzed the behavior of the wall shear stress at different sectors of the plaques. The difference between a **soft** and a **stiff** plaque could be linked to their development and/or complications and may be useful to design therapeutic strategies or improve those implemented, thus conducting to a more effective prevention of cardiovascular disease.

Methods

PATIENTS:

This is a descriptive Case Reports and Series article. Fifteen patients (3,7%) were evaluated out of 398 consecutive noninvasive vascular evaluations (NIVE) performed by the same operator (PF) from July 2022 to June 2023. Written informed consent was obtained of each patient included and the protocol of NIVE and data management was approved by an independent Ethics Committee.

To be included, a patient must be a male of female between 18 and 80 years old, with a complete NIVE and presenting plaques in the Left Common Carotid Artery (LCCA) Bifurcation with structural and hemodynamic evaluation of de LCCA and the plaque, including VFLOW determinations at both places and details of the plaque composition. Only two cases are presented, due to space constraints for the presentation, showing examples of a "**soft expanding plaque**" and other a "**stiff non expanding plaque**".

Equipment: We used a Resona 7 colour Doppler ultrasound device (MINDRAY, Shenzhen, China) with a high-sensitive 4-13 MHz multi-frequency linear array probe transducer. The patient was in the supine position with his or her head 20°deviated to the opposite side. The device has built-in digital system processing software. A series of carotid artery parameters such as Vector Flow Imaging (VFI or VFLOW), Wall Shear Stress (WSS), Radio Frequency (RF), Quality Intima-Media Thickness (RFQIMT), RF Quality Arterial Stiffness (RFQAS), to determine local PWV and the hardness coefficient were measured automatically in standardized fashion in the Left Common Carotid Artery (LCCA) and in those patients presenting plaques in the LCCA bifurcation the VFLOW parameters were measured at different parts of the plaque, namely the WSS in proximal and distal shoulders and at the tip of the plaque and also, the speed and flow gradients across the plaque.

HIGH-FRAME RATE VECTOR FLOW IMAGING

The high-frame rate Vector Flow Imaging (VFI) is based on Pulsed Wave Imaging (PWI). Acquisition of flow vector information at high frame rates was obtained by performing multi-directional transmissions of plane waves; after a single plane wave transmission, multiple images receiving lines were obtained. It allowed calculation of the true velocity vectors at any location in a vessel. The dynamic flow was obtained by continuously updating the target's position according to the calculated velocity. The interleaved transmissions ensure both a highly sensitive vector flow image and a high-resolution B-mode images. The flow was analyzed by the system for 1.5 s at a pulse repetition frequency (PRF) of 10–15 kHz and at a very high frame rate of 400–600 Hz, depending on the used PRF, allowing to study at least one cardiac cycle. The data were reprocessed automatically by the US system in a 35–36-s clip, generating a sequence of about 600–900 images that could be displayed at a frame rate of 20–30 Hz. The acquired data could be further evaluated in the saved video. Such a high frame rate allowed a detailed analysis of hemodynamics.

VFLOW detects the speed and direction of all blood cells flowing through every point of the region of interest (ROI). There are low-speed cells, high-speed blood cells, and reverse cells flowing through a point in a short moment. It means that the speed measured and displayed by VFLOW in a point is the average speed of all blood cells in a precise short moment. Spatiotemporal characteristics of flow was evaluated visually and quantitatively to assess the specific flow patterns.

VFI shows velocity vectors, streamline distribution and vorticity distribution.[15] (Figure 2)

The streamline distribution uses arrows to indicate the flow direction. The color and length of the arrows showed the flow velocity, magnitude and direction (green means low velocities, yellow and orange medium velocities and red higher velocities; the longer the arrows the faster the flow). For quantitative evaluation, velocity curves were available: the maximum velocity vector point curve, automatically detected by the system, and the user-defined vector point curve. Both were displayed at the bottom of the image and showed the fluctuating velocities of the flow varying in subsequent cardiac cycles.[16]

WALL SHEAR STRESS

Both estimates of viscosity and wall shear rate (WSR) are needed to quantify WSS. Because viscosity can be challenging to measure noninvasively, many ultrasonic techniques to date have instead focused on measuring the Wall Shear Rate (WSR). One proposed approach is to measure the peak flow velocity at a single depth within the artery using conventional spectral Doppler (typically near the center of the lumen) and then estimate the WSR from the velocity data using a flow model. Alternatively, WSR can be estimated by measuring the flow velocity at several depths in the artery and then computing the spatial derivative of the measured velocity profile near the wall as we did as these approaches need multi-gated spectral Doppler or VFI to characterize the flow velocity profile within the artery.[17]

RADIOFREQUENCY PARAMETERS

A series of carotid artery parameters (named in this study "local" parameters) such as RF quality intima-media thickness (RFQIMT), RF quality arterial stiffness (RFQAS), real-time carotid IMT and elasticity can be measured automatically. The bilateral long axes of the carotid arteries were explored, then the probe was adjusted so that the ultrasound beam was perpendicular to the anterior and posterior wall of the carotid artery and to get a clear image of the front and rear walls of the blood vessels. Then RFQIMT was initiated, the carotid artery IMT was measured at 1.0–1.5 cm at the posterior wall of the carotid artery enlargement proximal end, quantitative real-time detection of six cardiac cycles of carotid artery IMT was performed, then their mean values were calculated.[18]

The system can automatically calculate RAVQRS and then the local PWV, hardness coefficient (coefficient) an index of arterial compliance (CC).
Parameter definitions:

$$\beta = \ln (Ps/Pd)/[(Ds - Dd)/Dd] \quad (1)$$

$$CC = \pi (Ds \,.\, Ds - Dd \,.\, Dd)/ [4 (Ps - Pd)] \quad (2)$$

$$PWV = \sqrt{} (\beta\, Pd/2\rho). \quad (3)$$

Among them, *Ds* is the largest internal diameter of the carotid artery, *Dd* is the minimum inside diameter of the carotid artery, and ρis the density of blood.[19]

Results

We considered a plaque those protrusions of IMT through the lumen of more than 1,2 mm as it is more than 25% of the upper limit of IMT at 80 years old age. We defined as a "soft expanding plaque" those with a Grey Scale Mean (GSM) below 80 shades of grey (SG) and a stiff non expanding plaque those with more than 100 SG. Six patients presented stiff not expanding plaques (fibrous/fibrocalcic) and five soft expanding plaques (lipidic). Four patients presented GSM between 80 and 100 and classified as fibrolipidic plaques. (Mean 57,6 $\pm$ 16 vs 108.8 $\pm$ 5,9 GSM $p < 0.001$) (Table 1).

The stiffness of the LCCA represented by Pulse Wave Velocity of the LCCA and the Hardness Coefficient (HC) was higher in the group of "stiff plaques" showing a connection of the artery structure disarrangements and the plaque structure. (PWV p 0,2, HC p 0,14)

There were only slight differences in the WSS at the proximal shoulder of the plaques (p0,06) that noticeably was lower in stiff plaques. In both types of plaques there was a drop of the WSS at the tip of the plaque (p NS). With similar hemodynamic patterns, stiff plaques presented lower WSS in the proximal shoulder and a relatively higher WSS at the distal shoulder, like the "afterburner" effect in the turbines (p 0,67). Two typical cases are compared in Figures 2, 3 and 4 showing plaques composition, hemodynamic comparison, plaques expansion and LCCA hemodynamic and stiffness comparison.

Table 1: Main parameters of the whole population and comparing Soft vs. stiff plaques groups. At the top of the table the entire group, second group: soft plaques and bottom group: stiff plaques, final line: statistical p signification. Ref: CV Risk F: Cardiovascular Risk Factors, CV Dis: Cardiovascular Disease, CV Treat: Cardiovascular Treatment, SBP: Systolic Blood Pressure DBP: Diastolic Blood Pressure, LCCA Diam: Left Common Carotid Artery Diameter, LCCA PWV: LCCA Pulse Wave Velocity, LCCA Hard: LCCA Hardness Coefficient, PLQ Area: Plaque Area PLQ Grad: Plaque Gradient, Prox WSS: Proximal Wall Shear Stress, Dist WSS: Distal Wall Shear Stress.

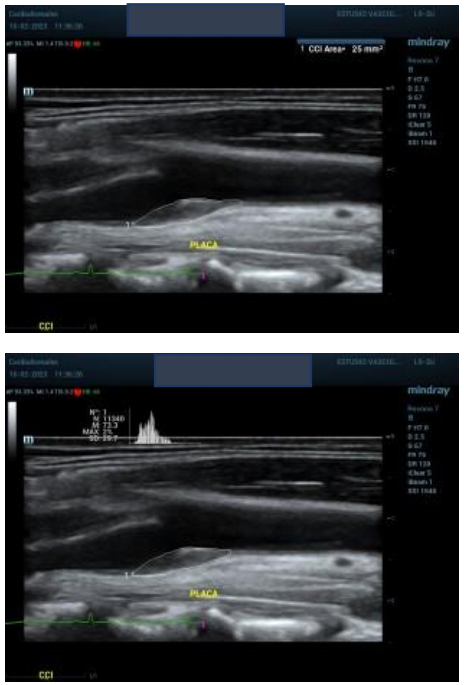
PLAQUES HEMODYNAMICS STUDY																						
PATIENT	ID	SEX	AGE	CV RISK F	CV DIS	CV TREAT	SBP	DBP	LCCA DIAM	LCCA FLOW	LCCA PWV	LCCA HARD	PLQ AREA	GREY MEAN	PLQ GRAD	PROX WSS	TIP WSS	DIST WSS	FLOW	MEAN SPEED	DIM SIST	DIAM DIAST
1	OI	F	68	Y	N	Y	134	80	5.2	275	9.6	7.6	14	111	0.9	0.68	0.66	1.31	29	12	7	7
2	SA	F	40	Y	N	N	140	86	4.7	230	5	1.9	25	73	1.1	1.41	0.9	1.64	161	35		4.4
3	IG	M	61	Y	N	Y	135	80	5.6	238	8	5.4	7	43	1.8	1.66	1.88	0.29	295	32		
4	TJ	M	63	Y	N	Y	130	70	4.5	400	6.1	3.5	24	93	0.6	0.86	1.94	0.85	392	44		
5	HG	M	62	Y	N	Y	150	70	4.7	308	9.8	8.9	9	107	2.1	0.47	0.4	1.89	79	32		
6	FJ	M	65	Y	Y	Y	170	100	5	253	9	6.2	20	100	3.3	0.65	0.29	0.9	218	28		
7	LM	F	34	Y	N	Y	134	72	5.3	399	5.9	3.2	17	80	1.3	2.6	2.2	1.9	299	52		
8	CF	M	42	Y	N	Y	140	80	5.9	459	7.6	4.7	23	46	1.2	0.49	0.34	2.9	249	35		
9	PT	F	72	Y	N	Y	148	75	6.2	442	8.5	6.3	27	49	0.7	2.1	0.66	0.57	378	32		
10	AR	M	70	Y	N	Y	167	98	6	405	5	1.7	17	118	1.9	0.8	1.8	0.45	507	50		
11	HH	M	64	Y	Y	Y	135	73	6.4	368	10.4	9.8	31	107	1.1	1.1	1.9	1	313	33		
12	FM	M	57	Y	N	Y	160	90	4.6	217	9.1	6	30	77	0.8	0.9	0.8	1	77	38		
13	BB	F	66	Y	N	Y	170	93	5.7	300	11.2	8.8	10	92	0.3	1	0.3	1.1	125	34		
14	CC	F	72	Y	N	Y	127	54	6.1	382	6.2	4.6	24	80	1.4	2.1	2.2	0.3	340	43		
15	BL	M	79	Y	Y	Y	157	72	7.1	451	11.4	12	29	110	1	0.3	0.3	1	451	49		
MEAN			**61**				**146.47**	**79.53**	**5.53**	**341.80**	**8.19**	**6.04**	**20.47**	**85.73**	**1.30**	**1.14**	**1.10**	**1.14**	**260.87**	**36.60**		
SD			**12.8**				**15.0**	**12.3**	**0.8**	**85.7**	**2.2**	**3.0**	**7.8**	**24.7**	**0.7**	**0.7**	**0.8**	**0.7**	**144.0**	**10.1**		
SOFT PLAQUES																						
PATIENT	ID	SEX	AGE	CV RISK F	CV DIS	CV TREAT	SBP	DBP	LCCA DIAM	LCCA FLOW	LCCA PWV	LCCA HARD	PLQ AREA	GREY MEAN	PLQ GRAD	PROX WSS	TIP WSS	DIST WSS	FLOW	MEAN SPEED		
2	SA	F	40	Y	N	N	140	86	4.7	230	5	1.9	25	73	1.1	1.41	0.9	1.64	161	35		
3	IG	M	61	Y	N	Y	135	80	5.6	238	8	5.4	7	43	1.8	1.66	1.88	0.29	295	32		
8	CF	M	42	Y	N	Y	140	80	5.9	459	7.6	4.7	23	46	1.2	0.49	0.34	2.9	249	35		
9	PT	F	72	Y	N	Y	148	75	6.2	442	8.5	6.3	27	49	0.7	2.1	0.66	0.57	378	32		
12	FM	M	57	Y	N	Y	160	90	4.6	217	9.1	6	30	77	0.8	0.9	0.8	1	77	38		
MEAN			**54.4**				**144.60**	**82.20**	**5.40**	**317.20**	**7.64**	**4.86**	**22.40**	**57.60**	**1.12**	**1.31**	**0.92**	**1.28**	**232.00**	**34.40**		
SD			**13.4**				**9.8**	**5.8**	**0.7**	**122.1**	**1.6**	**1.8**	**9.0**	**16.1**	**0.4**	**0.6**	**0.6**	**1.0**	**116.9**	**2.5**		
STIFF PLAQUES																						
1	OI	F	68	Y	N	Y	134	80	5.2	275	9.6	7.6	14	111	0.9	0.68	0.66	1.31	29	12		
5	HG	M	62	Y	N	Y	150	70	4.7	308	9.8	8.9	9	107	2.1	0.47	0.4	1.89	79	32		
6	FJ	M	65	Y	Y	Y	170	100	5	253	9	6.2	20	100	3.3	0.65	0.29	0.9	218	28		
10	AR	M	70	Y	N	Y	167	98	6	405	5	1.7	17	118	1.9	0.8	1.8	0.45	507	50		

11	HH	M	64	Y	Y	Y	135	73	6.4	368	10.4	9.8	31	107	1.1	1.1	1.9	1	313	33		
15	BL	M	79	Y	Y	Y	157	72	7.1	451	11.4	12	29	110	1	0.3	0.3	1	451	49		
MEAN			**68.0**				**152.2**	**82.2**	**5.7**	**343.3**	**9.2**	**7.7**	**20.0**	**108.8**	**1.7**	**0.7**	**0.9**	**1.1**	**266.2**	**34.0**		
SD			**6.1**				**15.4**	**13.5**	**0.9**	**77.6**	**2.2**	**3.5**	**8.6**	**5.9**	**0.9**	**0.3**	**0.8**	**0.5**	**193.9**	**14.2**		
p value			**0.05**				**0.37**	**1**	**1**	**0.68**	**0.2**	**0.14**	**0.66**	**<,001**	**0.2**	**0.06**	**1**	**0.67**	**0.74**	**1**		

Figure 2: Comparison of structure of a soft plaque (left “A”) and a stiff plaque (right “B”)

FIGURE 2: Comparison of structure of a soft plaque (left “A”) and a stiff plaque (right “B ”)

A Soft plaque



B Stiff plaque

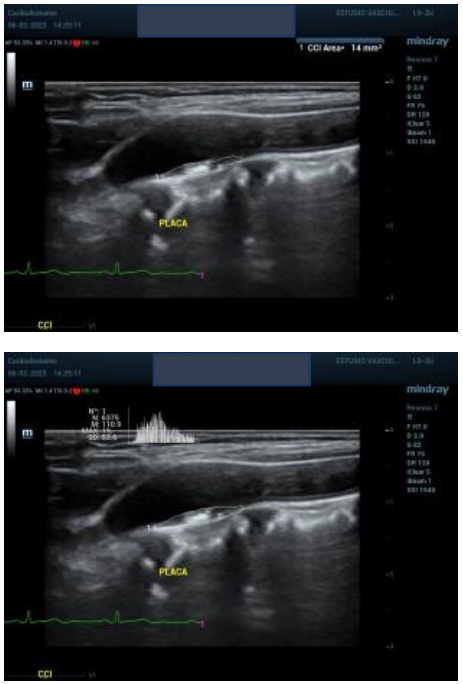


Figure 3: Hemodynamic comparison of a soft Plaque (Left "A") and a stiff plaque. In pictures from top to bottom and left to right: Speed gradient, Speed panel (T-Max,Mean, Median), Wall Shear Stress at different sites of the plaques and Flow)

FIGURE 3: Hemodynamic comparison of a soft Plaque (Left "A") and a stiff plaque. In pictures from top to bottom and left to right: Speed gradient, Speed panel (T-Max,Mean, Median), Wall Shear Stress at different

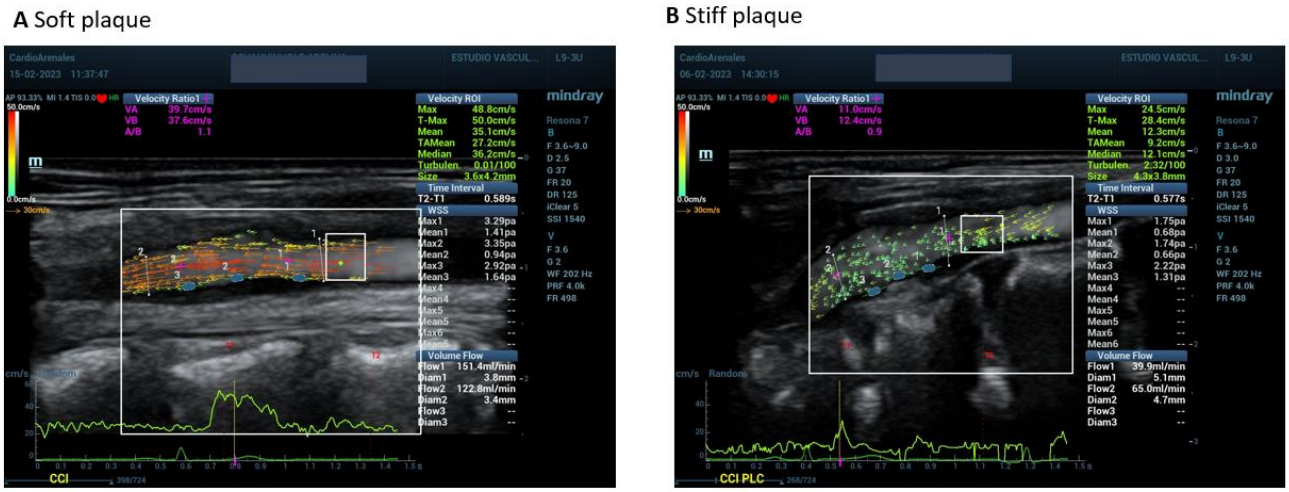


FIGURE 4: Hemodynamic comparison of a soft Plaque (Top Line "A") and a stiff plaque (Bottom line "B") showing the expansion from diastole to systole in the center of the plaque in "A" and no expansion in "B".

FIGURE 4: Hemodynamic comparison of a soft Plaque (Top Line "A") and a stiff plaque (Bottom line "B") showing the expansion from diastole to systole in the center of the plaque in "A" and no expansion in"B" .

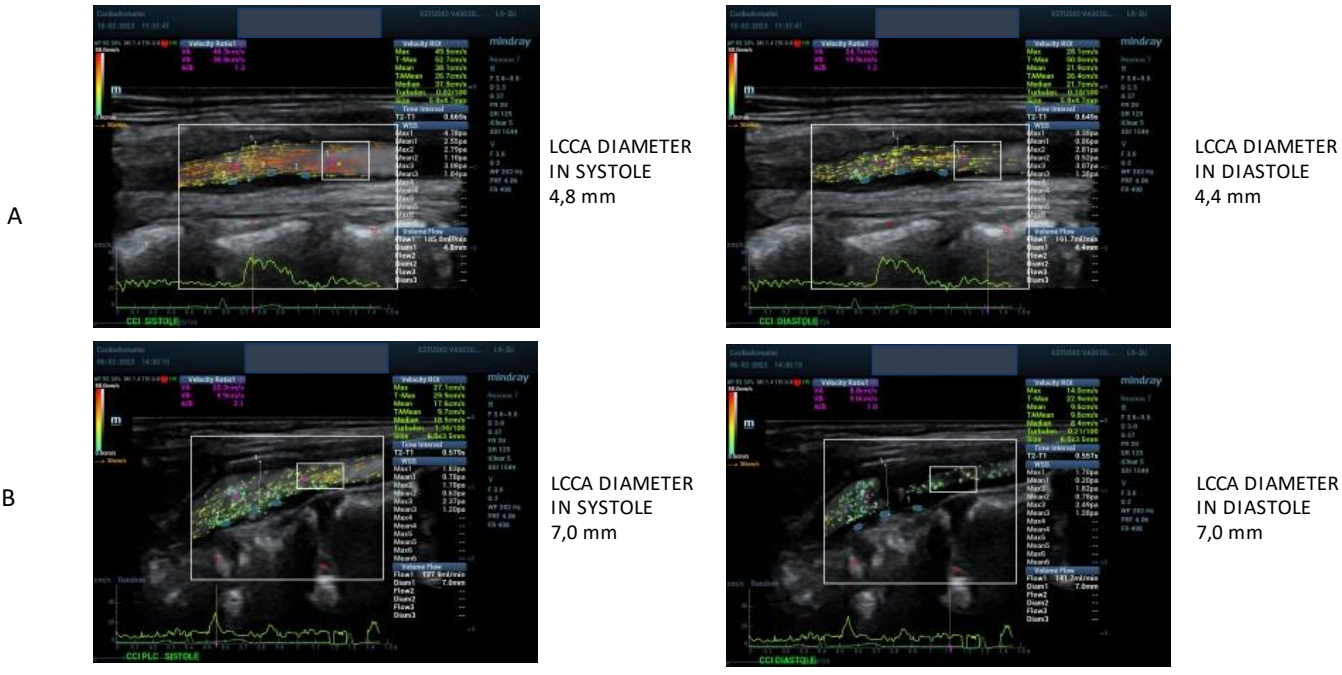


Figure 5: Hemodynamic comparison of LCCA corresponding to the soft plaque and the stiff plaque showing hemodynamics (from top to bottom and left to right: Speed gradient, Speed panel (T-Max,Mean, Median), Wall Shear Stress at different sites of the plaques and Flow). At the bottom stiffness parameters like hardness coefficient and Pulse Wave Velocity of the LCCA.

FIGURE 5: Hemodynamic comparison of LCCA corresponding to the soft plaque and the stiff plaque showing hemodynamics (from top to bottom and left to right: Speed gradient, Speed panel (T-Max,Mean, Median), Wall Shear Stress at different sites of the plaques and Flow).
At the bottom stiffness parameters like Hardness Coefficient and Pulse Wave Velocity of the LCCA

A Soft plaque

R-VQS Measurement

Vaso	Mean	SD
Diameter	5.376mm	0.092mm
Displacement	581um	169um

Hardness coefficient: 1.90

PWV: 4.99m/s

B Stiff plaque

R-VQS Measurement

Vaso	Mean	SD
Diameter	9.442mm	0.211mm
Displacement	306um	150um

Hardness coefficient: 7.58

PWV: 9.62m/s

With close similar surfaces, soft plaque presents a GSM of 73 SG compared with 110,9 SG in the stiff plaque. (Figure 2)

The speed and gradient are lower in the stiff plaque and the WSS is pronouncedly lower in the proximal shoulder of the stiff plaque and in the tip of the soft plaque depicting the "vulnerable" sites. (Figure 3)
The expansion at the initial systole and the end diastole was 400 microns (approximately 10%) in the soft plaque and no expansion was observed in the stiff plaque. (Figure 4)

Finally, the hemodynamic of the LCCA show a comparable speed and flow in both arteries, lower WSS in the anterior wall of the soft plaque LCCA and the stiffness is clearly low in the soft plaque LCCA (hardness coefficient, and pulse wave velocity) with a compared expansion of the artery of 581 microns $\pm$ 169 in the soft vs 306 microns $\pm$ 150 in the stiff plaque LCCA. (Figure 5)

Discussion

The goal of this communication was to describe new advances in atherosclerotic plaque evaluation using new technologies to go in deep into the physiopathology of plaques and their vulnerability, conducting to develop a better detection approach, more accurate therapeutic interventions and more precise follow up.

This is a small sample of patients, selected from a general population, based on the presence of specific lesions in the LCCA suitable for measurements. To refine the plaque evaluation, we considered to analyze the main artery (namely LCCA) in order to know the speed, flow, WSS and stiffness of the proximal artery and then consider plaque composition, speed and flow gradients, and the WSS at different parts of the plaque and the relation with expanding (soft) and non-expanding (stiff) behavior of the plaque.

The properties of the plaque could be affected by the hemodynamic environment of the artery neighboring walls, as well as by the pressure, flow and speed. So, the plaque composition could be derived from the interaction with blood stream and the near vessel wall. This huge amount of variables need a systematic analysis and find or rule out several interactions.

Plaques usually are complex, sometimes extensive, tortuous, present calcified nucleus, tandem lesions and other have opposite or annular disposition which represent different challenges at the time of analyzing structure and hemodynamics of plaques and their vulnerability. These are common barriers for ultrasound evaluation of plaques, independent if the technique employed is Pulsed Doppler, Color doppler, Power Doppler or VFLOW and prevented from including all the patients with plaques in the LCA bifurcation.

These limitations preclude to make generalization or conclusions but we observed that the patterns of "Soft" and "Stiff" are quite common (two thirds of the population). It is to be stressed that, this is the first time that WSS is measured in the daily clinical practice and was connected with characteristics of the plaque and the neighboring artery structural and functional data. Wall Shear Stress could be connected with plaque vulnerability and zones prone to growth or complications.

The heterogeneity of the plaque seems to be determinant of differences in the distribution of the WSS in the plaque`s surface even when the hemodynamic conditions like speed, flow and stiffness of the LCCA were ruled out as potential confounding factors. Further investigation is required in larger samples of patients, lesions and territories in order to achieve more precise knowledge and comprehension of the determinants of plaques composition and behavior and their potential clinical applications.

As recent guidelines have suggested to seek for subclinical atherosclerosis in younger subjects and in the low-risk population [20, 21], a better knowledge of the physiopathology of the plaques in early stages is urgently needed to direct more precisely therapeutic interventions at early ages, more intensively and appropriate methods to evaluate their impact during the follow up. [22]

Conclusion

We analyzed in this paper the characterization of two types of plaques (soft vs. stiff ones), their hemodynamic patterns and in particular, for the first time, the behavior of the wall shear stress at different sectors of the lesions which may be linked to their development and/or complications.
This technology deserves further development face to future applications in the diagnosis and treatment of atherosclerotic vascular disease.

Conflicts of Interest Statement

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