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CARDIAC IMAGE UPDATED

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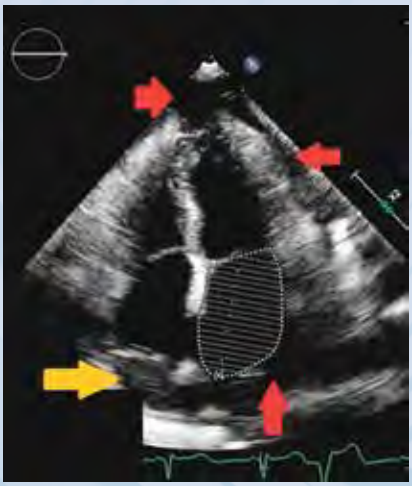

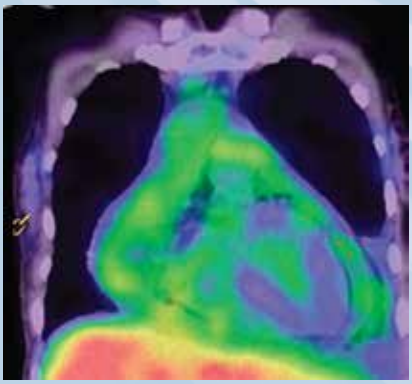

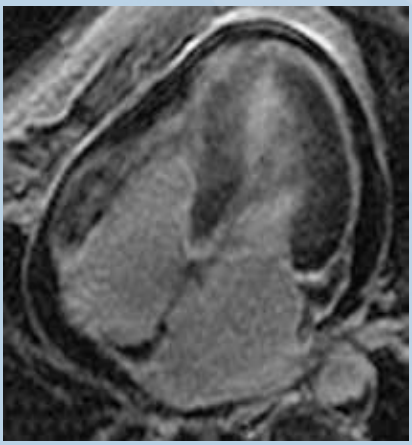

Original Research

Review

Volume 1, No. 1
January-March, 2019

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Contenido

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Bienvenidos

Queridos lectores:

La segunda década del siglo XXI nos ha dejado avances jamás soñados en el mundo de la imagen cardiaca y, de manera fascinante, hemos descubierto que los diversos métodos no se sustituyen, sino que se complementan.

La resonancia magnética es cada día más accesible a la población general, proporcionando al clínico precisión y abriendo otro panorama en la comprensión de la patología cardiaca.

El ecocardiograma se ha extendido a través de las técnicas especiales como la deformación y las imágenes tridimensionales. Al mismo tiempo, el ecocardiograma bidimensional es accesible al personal de urgencias a través de aplicaciones y de dispositivos portátiles y de bajo costo.

Todo ello en un mundo que cada día es más cercano gracias a la web. Cambios exigen cambios, y han comenzado a surgir preguntas: ¿debe aumentar el tiempo de formación del ecocardiografista?, ¿quién, y bajo qué formación y regulación puede realizar ecocardiografía en un Servicio de Urgencias o de Terapia Intensiva?, ¿cuánto vamos a tardar en emitir el diagnóstico a distancia?

Dichas preguntas ameritan un foro donde todos los que nos dedicamos a la imagen cardiovascular reflejemos nuestra experiencia, nuestro trabajo, y compartamos las diferentes perspectivas en un mundo que, gracias a la tecnología, avanza de forma vertiginosa.

Con gran ilusión y esperanza relanzamos ahora la revista de nuestra asociación con el propósito de que se transforme en ese foro necesario en nuestro país. La revista que tienen en sus manos será un órgano plural, incluyente y abierto. Son éstas las características que creemos son esenciales para que todos nos unamos, y podamos ver la realidad desde un solo frente, esa realidad llamada paciente.

Bienvenidos entonces, bienvenidos todos.

Atentamente

Dr. León Gerardo Aello Reyes

Dr. Enrique Berríos Bárcenas

Editores en jefe

CARDIAC IMAGE UPDATED
Vol. 1, No. 1, January-March 2019

Editorial

Welcome

Dear readers:

The second half of the XXI century provided great advances never imagined in the field of cardiac imaging, and, in a fascinating manner, we have discovered that these imaging methods are not mutually exclusive but rather complementary.

Magnetic resonance imaging for instance is now more accessible to the general population; it provides precise images at the same time that it aids the clinician in the understanding of the cardiac pathology. The echocardiogram on the other hand, has broadened its usefulness with special techniques such as strain and tridimensional echocardiography, whereas the two-dimensional echocardiography is accessible due to its low cost in the Emergency departments using special apps and portable devises.

All this is possible today in a world where the internet shortens distances. Changes demand changes, and several questions have risen: What is the optimal length for echocardiograpy training?; Who, and under what supervision is allowed to perform echocardiograms in the Emergency departments or Intensive Care Units?; How long would it take to make a diagnosis in a remote laboratory?

These questions deserve a forum where all of us who are interested in the field of the cardiovascular imaging can share our experience, our work and our different perspectives in a world that is moving fast due to technology.

It is with great illusion and hope that today we relaunch the journal of our Association, with the purpose of transforming it in that forum that our country needs. This journal represents a plural, inclusive and open opportunity. These are the characteristics that we think, are essential for all of us to stay together in order to analyze reality from a common place, that reality represented by the patient.

Welcome all.

Sincerely

León Gerardo Aello Reyes, MD.

Enrique Berríos Bárcenas, MD.

Editors in Chief

CARDIAC IMAGE UPDATED
Vol. 1, No. 1, January-March 2019

Editorial

Cardiac Image Updated, Marie Curie and the origin of cardiovascular image technics

Actualización de la imagen cardiaca, Marie Curie y el origen de las técnicas cardiovasculares de imagen

Francisco Javier Roldán,* Jessica Canseco**

* Member of the National Academy of Medicine in Mexico.
** Medical student at Social Service Program.

Francisco Javier Roldán

The effort of many enthusiastic professionals that dreamed of the first scientific journal of cardiovascular imaging in Latin-America culminates with this first number of Cardiac Image Updated (CIU), now in your hands. CIU seeks to be a common humanistic space for exchange of knowledge, promoting development of the specialty and also the correct use of technology at service of health.

As in every birth the recognition to our history is an obligation. For this reason, Editorial Committee has decided pay tribute to Maria Salomea Skłodowska, better known as Marie Curie. In hard times, her sacrifices, efforts and vision changed gender paradigms and started a technological revolution that settle the fundamentals for cardiovascular image techniques.

Marie Curie was born in Warsaw (Poland) at November 7, 1867. Since she was a child she felt special attraction to physical and chemical sciences that learned from her father. At that time, in the Russian empire of which Kingdom of Poland was a part, women were not allowed to get university degrees, for that reason Marie had to study clandestinely and then emigrate. In 1981 she arrived to Paris, completed her university studies and she started to work on different researches of the magnetic properties of steels. She married Pierre Curie and they conducted pioneering researches on radioactivity based on Becquerel´s studies. In 1880 Pierre and his brother Paul Jacques had discovered piezoelectric effect and they made a device called electrometer. Using these tools, Marie was able to demonstrate that the air around uranium salts conducted electricity and she conjectured that radiation came from the atom itself and not for interaction between molecules. Radioactivity theory was born.

Throughout her life Marie had to face many professional and personal obstacles that are not the subject of this editorial, but we invite you to investigate more about them for the reason of interest. With the beginning of the First World War, Marie was forced to interrupt her researches but, in her eagerness to help, she made it possible for her to create the first portable diagnostic unit, a radio-diagnostic one, allowing doctors assisted

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soldiers on the battlefield. Later, in 1915, she produced cannulas containing a radioactive gas called radon hopping it would help in the treatment of cancer. Unfortunately, her discoveries, that offer so many benefits to our patients today, led her to dead in 1934 at the age of 67 due to aplastic anemia consequence of radiation exposure. «The road to progress is not quick nor easy», she used to say.

At the 85th anniversary of her death, every image device keeps a close relationship with Marie Curie and her colleagues' works. Piezoelectric effect is the physical tool that animates our ultrasound equipment; X-ray are the fundamentals for tomographic studies; portability made possible diagnosis at the bedside especially in critically ill patients, magnetism and radioactivity let us know anatomy and cellular metabolism by Magnetic Resonance, Nuclear Medicine studies and Positron Emission tomography. For it all, no mentioning therapeutic possibilities and medical contributions, we consider to dedicate Marie these first pages as a grateful duty and as a sing of recognition to her work.

We hope you find in Cardiac Image Updated a place for sharing your researches, knowing your colleagues' work and remaining updated on this fast-evolving medical discipline. It is dedicated to all those who, following the Marie Curie wish, make a permanent contribution to science.

Welcome to this new scientific forum.

CARDIAC IMAGE UPDATED

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Original Research

Radial deformation and left ventricle rotation of the base are mechanisms associated with the severity of primary mitral regurgitation

La deformación radial y la rotación de la base del ventrículo izquierdo son mecanismos asociados con la gravedad de la regurgitación mitral primaria

Nydia Ávila Vanzzini,* Juan Francisco Fritche-Salazar,* Nestor Alejandro Parra Ordoñez,* Héctor Herrera Bello**

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** Fundación Clínica Médica Sur.

ABSTRACT. Introduction: The rotational mechanics of the left ventricle (LV) are part of the function of the mitral annulus; the loss of this mechanism has been associated with the severity of mitral regurgitation (MR). Our objective was to evaluate whether the rotation of the LV base is a mechanism associated with the severity of MR. **Methods:** A cross-sectional analytical study was designed. Patients older than 18 years old with non-ischemic MR were included. A healthy control group was analized. **Results:** Ninety-one participants were included, 57 cases and 34 controls. The patients with MR had worse ventricular strain (longitudinal, radial and circumferential). The case group were divided by severity of regurgitation; longitudinal strain had a progressive decrease from mild to severe MR (-19% [95%CI -22%-15%] and -14% [95%CI -17%-6%]) respectively ($p<0.033$). Twist in patients with mild MR was 16º (13.6-19.4), which decreases to 10.8º (7.3-17.3) in severe MR ($p<0.034$), torsion shows a similar pattern with a value of 2.05º/cm, in the mild MR, and that decreases to 1.1º/cm in severe MR ($p<0.038$). Multivariate analysis shown that the independents mechanisms associated with severity of MR were: radial strain (OR:1.08, 95%CI:1.03-1.13, $p<0.006$), and basal rotation [OR:1.14 95% CI 1.07-1.26, $p<0.02$]. **Conclusion:** In MR of non-ischemic etiology, poor rotation of the base and poor radial strain were associated with MR severity.
Key words: Mitral regurgitation, ventricular rotational mechanics, basal rotation.

RESUMEN. Introducción: *La mecánica de rotación del ventrículo izquierdo (VI) es parte de la función del anillo mitral; la pérdida de este mecanismo se ha asociado con la gravedad de la regurgitación mitral (RM). Nuestro objetivo fue evaluar si la rotación de la base de BT es un mecanismo asociado con la severidad de la RM.* ***Métodos:*** *Se diseñó un estudio analítico transversal. Se incluyeron los pacientes mayores de 18 años con RMN no isquémica. Se analizó un grupo de control sano.* ***Resultados:*** *Se incluyeron 91 participantes, 57 casos y 34 controles. Los pacientes con RM tuvieron peor tensión ventricular (longitudinal, radial y circunferencial). El grupo de casos se dividió por la gravedad de la regurgitación; la deformación longitudinal tuvo una disminución progresiva de leve a severa RM (-19.1% [IC 95% -22-15.7%] a -14% [IC 95% -17.8-6.7%]) respectivamente ($p<0.033$). La torsión en pacientes con RM leve fue de 16º (13.6-19.4), que disminuye a 10.8º (7.3-17.3) en la RM grave ($p<0.034$), la torsión muestra un patrón similar con un valor de 2.05º/cm, en la RM leve, y que disminuye a 1.1º/cm en la RM grave ($p<0.038$). El análisis multivariado mostró que los mecanismos independientes asociados con la gravedad de la RM fueron: deformación radial (OR: 1.08; IC 95%: 1.03 a 1.13; $p<0.006$) y rotación basal (OR: 1.14; IC 95%: 1.07 a 1.26; $p<0.02$).* ***Conclusión:*** *En la RM de etiología no isquémica, la mala rotación de la base y la mala deformación radial se asociaron con la gravedad de la RM.*
Palabras clave: *Regurgitación mitral, mecánica de rotación ventricular, rotación basal.*

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INTRODUCTION

Mitral regurgitation (MR) generates a chronic volume overload, leading to irreversible left ventricular (LV) dysfunction.[1] Several coexisting pathophysiologic mechanisms are independently involved in MR pathogenesis, including LV systolic dysfunction, LV local and global remodeling, LV dyssynchrony, annular shape, and organic alterations.[2,3] In ischemic MR the restricted sphincter motion of the mitral annulus (MA) and the impaired basal rotation, consequence of an inferior-posterior myocardial infarction, have been associated with increased of MR severity.[4] Advances in cardiac imaging techniques have provided new insights into LV mechanics.[5] The rotation obtained by 2-dimensional speckle tracking echocardiography, hold promise to be a more reliable index of «myocardial performance». In chronic MR correlations between disease severity and torsional parameters suggest a potential role of these variables in assessing early signs of ventricular dysfunction;[6] however, it has not been evaluated in different etiologies of MR and the mechanism by which the antero-posterior annular function is loss is not fully understood. In the present study, we sought to investigate whether basal rotational mechanical failure is associated with increased severity of MR and to provide a hypothesis for the pathophysiologic mechanism by which the annular function is lost.

METHODS

A cross-sectional study was designed; patients older than 18 years attending to echocardiographic evaluation from March to August 2017 with the diagnosis of MR were consecutively included. We evaluated 186 patients with functional non-ischemic or organic MR. Were excluded patients with clinical and echocardiographic evidence of other cardiac and/or heart valve disease (45), previous cardiac surgery (6), previous treatment with chemotherapy (2), acute o chronic myocardial infarction (28), atrial fibrillation (9), inflammatory diseases (4), left bundle branch block (5), and technically inadequate two-dimensional echocardiographic images for speckle-tracking echocardiographic analysis (14). About mechanism of MR were excluded patients with flail (4) and ischemic mitral regurgitation (12). A healthy young control group or 34 people was analyzed. All the patients underwent preliminary cardiologic examinations with comprehensive clinical data collection, including cardiovascular risk factors, electrocardiogram and transthoracic echocardiogram. The study was approved by the local research and ethics committee and follows the Helsinki guidelines.

Two-dimensional echocardiography. Each subject underwent standard transthoracic echocardiogram using Vivid 9 XD clear echocardiography equipment (GE Vingmed Ultrasound AS, Horten, Norway) according to the American Society of Echocardiography guidelines.[7] The parameters of LV global remodeling, including LV end-diastolic volume (EDV), end-systolic volume (ESV) and biplane Simpson's ejection fraction, were measured. LV volumes were indexed.

Global LV strain and rotational mechanics. Ventricular mechanics were performed according to actual recommendations.[8] For offline analysis of strain and rotation, LV short-axis views acquired at the basal, mid and apical levels, and standard LV apical four-chambers, three chamber and two-chamber views were recorded with a mean frame rate or ≥70 frames/sec. Two-dimensional strain and rotation data were analyzed by frame-to-frame tracking of the grayscale patterns using dedicated software of Echopac.

Evaluation of mitral regurgitation. In the case of a single jet, the contract vein (CV) was measured on the perpendicular axis to the regurgitant jet; a CV <0.3 cm was equivalent to mild insufficiency, a CV ≥0.7 cm was equal to severe insufficiency, the intermediate points were evaluated with quantitative methods such as the flow convergence method (PISA). If two or more jets were found, the severity assessment was made using the Doppler continuity method, in accordance with the recommendations for the assessment of valvular regurgitation.[9] Both the PISA method and the continuity method considered a regurgitant orifice area (AORE) ≥0.40cm^2 and regurgitant volume (RVol) ≥60mL as severe; AORE of 0.20-

0.39cm^2 and RVol of 30-59mL was considered moderate, and AORE <20cm^2 and <30mL of RVol, was considered mild.[9]

Statistic analysis

Normality of the continuous variables was sought with the Shapiro Wilk test. The parametric variables are expressed as mean and standard deviation and their comparison was made with Student's t-test; the non-parametric variables are expressed in median and interquartile range (25-75); their comparison was made with the Wilcoxon sum-rank test. The comparison of more than two groups of continuous variables was performed with the one-way analysis of variance (ANOVA) or the Kruskal Wallis test as appropriate. The categorical variables are expressed as a percentage and their comparison between groups was performed with the χ^2 test. A value of $p<0.05$ was considered as statistical significant.

RESULTS

A total of 91 participants were included, 57 were cases and 34 controls. Differences in age, height, systemic arterial hypertension (SAH) and dyslipidemia were observed between cases and controls; in the echocardiographic findings there was a significant difference in: left ventricular ejection fraction (LVEF), left atrium volume (LAV), systolic mitral annulus dimension, fractional shortening of the mitral annulus, tenting area and depth of coaptation. In the analysis of ventricular mechanics, the case group showed a significant decrease in the overall longitudinal, radial and circumferential deformation, as well as a decrease in basal rotation, twist and torsion in relation to the control group *(Table 1)*.

Analysis by subgroups according to the severity of mitral regurgitation. Differences were found in EDV, ESV and LAV, they shown a progressive increased in relation to the severity of MR; $p<0.0027$; $p<0.0085$ and $p<0.001$; mild, moderate and severe respectively). The same behavior was observed in the anterior-posterior diameter of the mitral annulus, which was larger the more severe the MR was ($p<0.001$; $p<0.0001$ diastolic and systolic annulus respectively). The shortening fraction of mitral annulus was clearly compromised in severe MR, $p<0.0008$. A progressive decrease in the LVEF was observed $p<0.013$ *(Table 2)*. Tenting area and depth of coaptation were greater among the more severe was the regurgitation $p<0.0009$; $p<0.045$ respectively. The longitudinal global strain showed a significant progressive decrease from mild MR (-19.1% [-22%-15.7%], moderate MR -18.1% [-21%-14.7%] and severe MR -14% [-17.8%-6.7%]) ($p<0.033$) (the rest of the deformation vectors are shown in *Table 2)*. The basal rotation was statistically different between the mild and severe MR groups; $p<0.043$. The twist in mild MR was 16º (13.6-19.4), this decreased to 10.8º (7.3-17.3) in severe MR; $p<0.034$; the torsion shows a similar pattern with a value of 2.05º/cm (1.6-2.4) in mild MR, and 1.1º/cm (0.8-2.1) in severe MR; $p<0.038$. *(Figure 1 and Table 2)*. A multivariate analysis (logistic regression) was performed to search variables associated with moderate or severe MR; global radial strain (GRS) [OR 1.08, 95%CI 1.03-1.13, $p<0.006$] and basal rotation [OR: 1.14, 95%CI 1.07-1.26, $p<0.02$] were independently associated with a higher MR severity *(Table 3)*.

Interobserver variability. Intraclass correlation coefficients were performed for longitudinal, circumferential, radial and rotational profiles, according to what was reported in previous studies, a variability of 5 and 3% was obtained for longitudinal and circumferential deformation respectively, the ventricular mechanics parameters had the best reproducibility *(Table 4)*.

DISCUSSION

This study shows that the decrease in rotation of the base of the LV and the decrease in the radial deformation are associated with a greater severity of MR in patients with MR of degenerative and non-ischemic functional etiology. This phenomenon had already been observed previously in patients with MR of ischemic etiology; Zito et al[4] reported the impaired basal rotational mechanics occurring after an inferior-posterior myocardial infarction is associated with increased MR. In another study of patients with different MR etiology

Table 1: Clinical and echocardiographic findings in both groups.

	Control group n = 34	MR Group n = 57	p
Age (years)	26 (21-33)	54 (47-62)	0.00001
Female (%)	19 (55.8)	32 (56.1)	0.981
Height (cm)	167 ± 10	170 ± 11	0.009
Weight (kg)	67 (56-78)	68 (59-76)	0.457
SAH (%)	0	21 (36.8)	0.0001
DL (%)	0	14 (24.5)	0.002
Echocardiographic findings			
EDV (mL/m^2)	50 (42-60)	55 (49-60)	0.067
ESV (mL/m^2)	21 (16-25)	23 (18-39)	0.058
LAV (mL/m^2)	15 (11-19)	23 (19-35)	0.0001
LVEF (%)	60.5 (60-65)	55 (49-60)	0.0001
Measurement of the mitral valve			
MAD (mm)	31 ± 3.8	33 ± 5.3	0.081
MAS (mm)	24 (20-28)	28 (23-33)	0.024
SFMA (%)	21 (15-28)	12 (6-21)	0.018
Tenting area (cm^2)	0.9 (0.7-1.4)	1.9 (1.6-2.5)	0.0001
Depth of coaptation (mm)	0.7 (0.6-0.9)	1 (0.7-1.2)	0.0015
Ventricular mechanics			
GLS (%)	-22 (-23 to -20.5)	-17.4 (-21.8 to -12.7)	0.0001
GCS (%)	-25 (-29 to -20)	-16.3 (-21 to -13)	0.0001
GRS (%)	42 (33-47)	22 (15.9-28)	0.0001
Basal rotation (º)	-6.7 (-7.3 to -5)	-4.2 (-6.7 to -3)	0.041
Apical rotation (º)	11 (9-11)	10 (6-13)	0.05
Twist (º)	17 (15-18)	14 (10-18)	0.007
Torsion (º/cm)	2.0 (1.8-2.2)	1.8 (1.3-2.3)	0.047

SAH = systemic arterial hypertension; DL = dyslipidemia; EDV = end diastolic volume; ESV = end systolic volume; LAV = left atrial volume; LVEF = left ventricular ejection fraction; MAD = diameter of mitral annulus in diastole; MAS = diameter mitral annulus in systole; SFMA = shortening fraction of mitral annulus; GLS = global longitudinal strain, GCS = global circumferential strain; GRS = global radial strain.

(mitral valve prolapse), Zito et al[10] found that the basal rotation increased progressively in proportion to the severity of the MR, and the torsion reached the maximum peak in patients with moderate MR and decreased in those with severe MR; these results are different from our study; while Zito el al[10] were looking for predictors of myocardial function, we seek to associate the mechanics of rotation of the base of LV to the degree of MR, Zito included only patients with LVEF ≥60% and the decrease in LVEF was not an exclusion criterion for us; thus our patients had more altered rotation patterns.

Under normal conditions, the twisting motion of the heart (LV twist is the preferred nomenclature for the measurement defined as the difference in the rotation of the apex relative to that of the base of the heart), arises from transmural differences in the local myofiber orientation, which is thought to minimize transmural stress gradients.[11] On the other hand, basal rotation shortens the distance between the MV and the head of papillary muscles, this mechanism might contribute to MV leaflet closure and counterbalancing the tethering forces. In relation to the mitral

Table 2: Analysis by subgroups according to the severity of mitral regurgitation.

	Mild MR n = 26	Moderate MR n = 12	Severe MR n = 19	p
Age (years)	51 (44-62)	54 (46-60)	55 (47-62)	0.824
Female (%)	15 (58)	9 (75)	8 (42)	0.089
Height (cm)	162 ± 12	159 ± 10	161 ± 9	0.701
SAH (%)	8 (31)	5 (42)	8 (42)	0.684
DL (%)	6 (23)	3 (25)	5 (26)	0.969
Echocardiographic findings				
EDV (mL/m^2)	44 (44-58)	57 (44-87)	79 (51-97)*	0.0027
ESV (mL/m^2)	19 (13-31)	25 (16-39)	32 (23-62)*	0.0085
LAV (mL/m^2)	40 (32-50)	61 (51-68)‡	79 (74-128)*	0.001
LVEF (%)	60 (55-61)	58 (53-63)	50 (26-55)*	0.013
Measurement of the mitral valve				
MAD (mm)	30 ± 3.8	31.6 ± 4.6	36.4 ± 5.1*	0.001
MAS (mm)	25 ± 4	26 ± 6	34 ± 5*	0.0001
SFMA (%)	17 (7-24)	17 (11-29)	6 (2-12)*	0.0008
Tenting area (cm^2)	1.6 (1.1-2.1)	1.8 (1.4-2.1)	2.5 (2.3-3.1)*	0.0009
Depth coaptation (mm)	0.9 (0.6-1)	0.95 (0.8-1.1)	1.1 (0.9-1.4)*	0.045
Ventricular mechanics				
GLS (%)	-19 (-22 to -15)	-18 (-21 to -14)	-14 (-17 to -6)*	0.033
GCS (%)	-17 (-21 to -15)	-16 (-20 to -13)	-15 (-17 to -8)	0.141
GRS (%)	25.5 (19-31)	17 (13.5-24.5)	19 (14-27)	0.099
Basal rotation (º)	-6 (-8.2 to -3.7)	-4.5 (-6 to -2.4)	-3.4 (-6 to-1.9)*	0.043
Apical rotation (º)	10.8 (7.3-13)	9.5 (7.6-13)	6.5 (4.2-14.5)	0.304
Twist (º)	16 (13.6-19.4)	14.5 (11.4-17)	10.8 (7.3-17.3)*	0.034
Torsion (º/cm)	2.05 (1.6-2.4)	1.65 (1.35-2.2)	1.1 (0.8-2.1)*	0.038

Abbreviations in previous table.
*Differences between mild and severe MR. ‡Differences between mild and moderate MR.

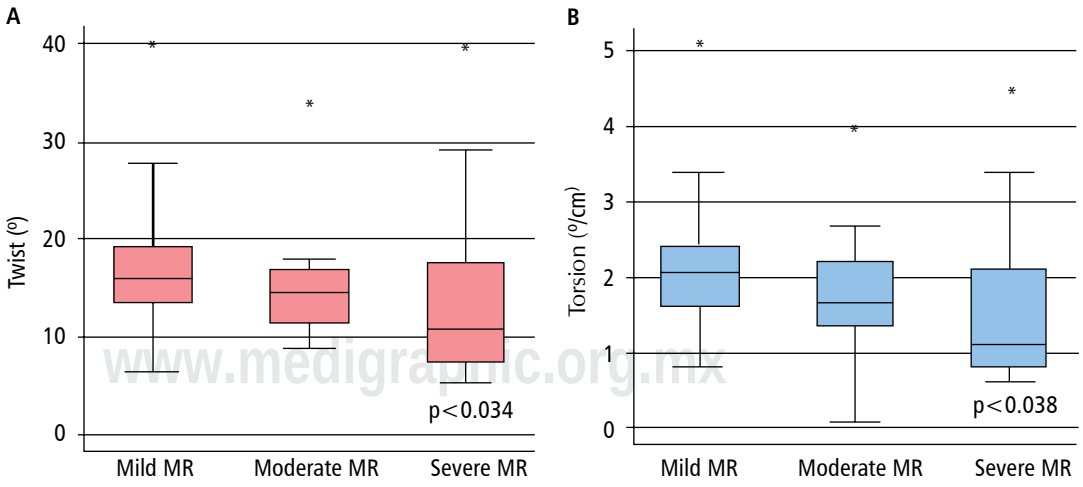

Figure 1: Evaluation of twist and torsion in different degrees of severity of mitral regurgitation.
Analysis of twist and torsion by speckle tracking 2D. **A.** Show that the twist is smaller as the severity of the mitral regurgitation increases. **B.** The same behavior is observed in the analysis of the torsion, show statistic differences.

Table 3: Multivariate analysis, association with moderate to severe mitral regurgitation.

Variable	OR	z	p (> Z)	95% CI
GLS	1.18	1.95	0.052	0.99-1.41
GCS	1.11	1.43	0.152	0.96-1.29
GRS	1.08	2.74	0.006	1.03-1.13
AR	0.85	-1.4	0.163	0.68-1.06
BR	1.14	2.96	0.02	1.07-1.26
Twist	1.11	0.62	0.53	0.79-1.55
Torsion	1.40	0.36	0.72	0.21-9.10

AR = apical rotation; BR = basal rotation. Rest of abbreviations in Table 1.

Table 4: Interobserver variability.

	Intraclass coefficient	p
GLS	0.955	0.0001
GRS	0.796	0.0150
GCS	0.975	0.0001
Basal rotation	0.892	0.0010
Apical rotation	0.738	0.0290
Twist	0.778	0.0180
Torsion	0.821	0.0090

annulus, its mechanism is useful to maintain MV competence; contraction of the sphincter of the posterior annulus caused by the shortening of the basal helical fibers of the ventricle, creates a competent superimposed coaptation.

Primary MR is characterized by an incomplete closure of the mitral valve, which permits the flow of blood across the mitral valve during systole (decreased afterload) and leads to increased EDV (increased preload). In chronic MR with preserved myocardial structure (without loss of the helical architecture of the fibers) the increase of the preload increases the rotation of the base and apex of the LV[12] as was described in the Zito Study;[10] but if it already exists increased LV sphericity and loss of the oblique architecture of the apical and basal fibers, the rotational mechanisms of the ventricle become affected, which in turn leads to a deficient function of sphincter of the mitral annulus and this is what can explain a possible added mechanism in the severity of the MR. Hence LV torsional parameters corelate with the degree of LV remodeling and the severity of MR.[13]

CONCLUSION

In patients with MR of degenerative and non-ischemic etiology, poor rotation of the base and poor radial strain were associated as independently variables with MR severity.

Limitations of study

Our results have to be considered within the context of some limitations. The study population was small and came from a single tertiary center. It is a cross-sectional study focused basically on the evaluation of the rotational mechanics and MR, so the LVEF was not taken into account as a selection criteria of patients.

REFERENCES

1. Avierinos JF, Gersh BJ, Melton LJ 3rd et al. Natural history of asymptomatic mitral valve prolapse in the community. Circulation. 2002; 106: 1355-1361.
2. Agricola E, D'Amato R, Stella S et al. Effects of mild ischemic mitral regurgitation on ventricular remodeling and its contribution to congestive heart failure. J Am Soc Echocardiogr. 2011; 24: 1376-1382.
3. Agricola E, Oppizzi M, Pisani M, Meris A, Maisano F, Margonato A. Ischemic mitral regurgitation: mechanisms and echocardiographic classification. Eur J Echocardiogr. 2008; 9 (2): 207-221.
4. Zito C, Cusma-Piccione M, Oreto L et al. In patients with post-infarction left ventricular dysfunction, how

does impaired basal rotation affect chronic ischemic mitral regurgitation? J Am Soc Echocardiogr. 2013; 26: 1118-1129.
5. Sengupta PP, Tajik AJ, Chandrasekaran K, Khandheria BK. Twist mechanics of the left ventricle: principles and application. JACC Cardiovascular Imaging. 2008; 1: 366-376.
6. Borg AN, Harrison JL, Argyle RA, Ray SG. Left ventricular torsion in primary chronic mitral regurgitation. Heart (British Cardiac Society). 2008; 94: 597-603.
7. Lang RM, Badano LP, Mor-Avi V et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2015; 16 (3): 233-270.
8. Mor-Avi V, Lang RM, Badano LP et al. Current and evolving echocardiographic techniques for the quantitative evaluation of cardiac mechanics: ASE/EAE consensus statement on methodology and indications endorsed by the Japanese Society of Echocardiography. J Am Soc Echocardiogr. 2011; 24 (3): 277-313.
9. Lancellotti P, Moura L, Pierard LA et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 2: mitral and tricuspid regurgitation (native valve disease). Eur J Echocardiogr. 2010; 11 (4): 307-332.
10. Zito C, Carerj S, Todaro MC et al. Myocardial deformation and rotational profiles in mitral valve prolapse. Am J Cardiol. 2013; 112 (7): 984-990.
11. Bovendeerd PH, Arts T, Huyghe JM, van Campen DH, Reneman RS. Dependence of local left ventricular wall mechanics on myocardial fiber orientation: a model study. J Biomech. 1992; 25 (10): 1129-1140.
12. Omar AM, Vallabhajosyula S, Sengupta PP. Left ventricular twist and torsion: research observations and clinical applications. Circ Cardiovasc Imaging. 2015; 8 (6). pii: e003029.
13. Reyhan M, Wang Z, Li M et al. Left ventricular twist and shear in patients with primary mitral regurgitation. J Magn Reson Imaging. 2014; 42 (2): 400-406.

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Original Research

Flow ejection dynamics as echocardiographic diagnostic parameters of severity in aortic stenosis

Utilidad de la dinámica de eyección como parámetros ecocardiográficos diagnósticos de la severidad de la estenosis aórtica

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ABSTRACT. Introduction: Aortic stenosis (AS) is a progressive disease whose final stage results in an inadequate cardiac output and death from cardiovascular causes. Its severity can be assessed by non-invasive methods such as echocardiography; however, discrepancies have been observed between severity estimated by the continuity equation and transvalvular gradients in up to a third of patients with preserved LVEF when compared with hemodynamic parameters obtained by cardiac catheterization. Recent studies have evaluated the usefulness of ejection dynamics, such as acceleration time (AT) and acceleration time/ejection time (AT/ET) ratio, as diagnostic and prognostic parameters. **Objective:** To assess whether AT and AT/ET ratio measured in the continuous Doppler curve of aortic flow have a direct relationship with severity of AS. **Material and methods:** Cross-sectional, analytical and predictive study. Patients with AS (aortic peak velocity >2m/s) were included. General characteristics, clinical presentation and echocardiographic parameters were analyzed in different stages of AS. A ROC curve was plotted to determine the best cutoff value of AT, ET and AT/ET ratio to identify severe AS. **Results:** 75 patients were included (mean age 64.7±16.7 years, 48% women); of whom 8 had mild AS (10.7%), 16 had moderate AS (21.3%) and 51 had severe AS (68%). A cutoff value of 104.5ms for AT had a sensitivity of 92.2% and a specificity of 83.3% for severe AS; a cut-off value of 323.5ms for ET had a sensitivity of 80.4% and a specificity of 70.8%, and a cut-off value of 0.345 for AT/ET ratio had a sensitivity of 84.3% and a specificity of 91.7%. **Conclusion:** AT, ET and AT/ET ratio are useful to identify severe AS.
Key words: Aortic stenosis, acceleration time, ejection time, acceleration time/ejection time ratio.

RESUMEN. Introducción: *La estenosis aórtica (EAo) es una enfermedad progresiva cuyo estadio final resulta en un gasto cardiaco inadecuado y muerte por causas cardiovasculares. Su severidad puede evaluarse por métodos no invasivos como la ecocardiografía; sin embargo, se han observado discordancias entre la severidad estimada por ecuación de continuidad y los gradientes transvalvulares hasta en una tercera parte de los pacientes con FEVI conservada cuando se compara con cálculos hemodinámicos durante el cateterismo. Estudios recientes han evaluado la utilidad de la dinámica de eyección, como lo es del tiempo de aceleración (TA) y el índice tiempo de aceleración/tiempo de eyección (TA/TE), como parámetros diagnósticos y pronósticos.* ***Objetivo:*** *Evaluar si el TA y el índice TA/TE medidos en la curva Doppler continuo del flujo aórtico tienen una relación directa con la severidad de la estenosis aórtica.* ***Material y métodos:*** *Estudio transversal, analítico y predictivo. Se incluyeron pacientes con EAo de válvula nativa (velocidad máxima del jet aórtico >2m/s). Se analizaron las características generales, presentación clínica y parámetros ecocardiográficos en los diferentes estadios de la EAo. Se realizó una curva ROC para determinar el mejor valor de corte del TA, TE e índice TA/TE para identificar la EAo severa.* ***Resultados:*** *Se incluyeron 75 pacientes con diagnóstico de EAo (edad media de 64.7±16.7 años, 48% mujeres); 8 de grado ligero (10.7%), 16 moderado (21.3%) y 51 severo (68%). El mejor valor de corte para la detección de EAo severa para el TA fue de 104.5ms (S 92.2%, E 83.3%), para el TE fue de 323.5ms (S 80.4%, E 70.8%) y para el índice TA/TE fue de 0.345 (S 84.3%, E 91.7%).* ***Conclusión:*** *El TA, TE y el índice TA/TE son útiles para identificar la EAo severa.*
Palabras clave: *Estenosis aórtica, tiempo de aceleración, tiempo de eyección, índice tiempo de aceleración/tiempo de eyección.*

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INTRODUCTION

Aortic stenosis (AS) is the most frequent native valve disease followed by mitral regurgitation.[1] Anatomic, genetic and clinical factors contribute to the pathogenesis of AS.[2-4] It has three main causes: a congenital bicuspid valve with overlapping calcification, a progressive calcification of a normal trileaflet aortic valve and rheumatic diseases.[2,5]

Echocardiography is the gold standard method to assess the severity of AS.[6] Although current American guidelines recommends specific parameters for the diagnosis,[7,8] discrepancies have been observed between the severity of AS estimated by the continuity equation and the transvalvular gradients in up to 30% of patients with preserved left ventricular ejection fraction (LVEF), when compared with catheterization.[7,9]

Clinical practice guidelines recommend the use of ejection dynamics parameters in the assessment of obstruction of prosthetic aortic valves;[10,11] however, few studies have evaluated these parameters in native aortic valve disease.[12,13] It has been observed that the length of acceleration time (AT) and ejection time (ET) have a significant relationship with the severity of AS, with prolongation of its duration and therefore of AT/ET ratio (not flow dependent parameter). In addition, a change in the aortic waveform shape is described, being more rounded in the case of a severe AS *(Figure 1)*.[10,12] Moreover, AT/ET ratio has shown utility as a prognostic marker.[9]

Therefore, these parameters can be useful to confirm the diagnosis of severe AS when there are discrepancies in the usual measurements. Our objective was to assess whether AT and AT/ET ratio have a direct relationship with severity of AS.

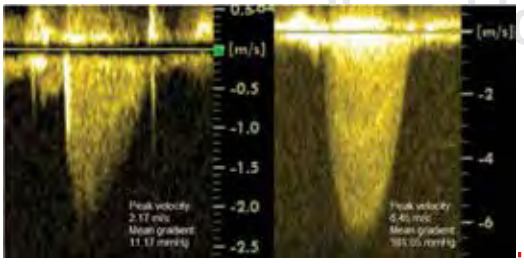

Figure 1:

Aortic waveform shape measured by continuous Doppler and its relationship with the severity of aortic stenosis.

MATERIAL AND METHODS

A cross-sectional, analytical and predictive study was conducted. We prospectively included 75 patients between January and October 2018, both genders, age ≥18 years and valvular native AS (peak velocity >2m/s) diagnosed by transthoracic echocardiogram (TTE). The exclusion criteria were subvalvular or supravalvular AS, moderate or severe aortic regurgitation, ascending thoracic aorta diameter <25 mm, mitral or tricuspid valvular disease more than mild, hyperdynamic states and suboptimal window.

2D and Doppler TTE was performed using a General Electric Healthcare Vivid S6 version 12.2 equipment. All tests were performed by one experienced cardiologist. The parasternal long-axis view was used to measure aortic annular diameter in early systole. The time-velocity integral (TVI) was obtained by using pulsed Doppler in the LVOT, placing the sample volume 0.5 cm below the aortic valve. Stroke volume was calculated assuming a circular shape of the LVOT. Maximal and mean pressure gradients through aortic valve was performed from the five-chamber windows, using the modified Bernoulli equation. Effective orifice area (EOA) was calculated using the continuity equation, and then indexed by body surface area. Doppler velocity index (DVI) was calculated as the TVI of the LVOT divided by the TVI of the aortic jet. All measurements were made in an average of 3 cardiac cycles for patients with sinus rhythm and 6 for patients with a rhythm other than sinus rhythm. The inclusion of an extrasystolic heartbeat was avoided. The Doppler records were made at a scanning speed of 100mm/s.

No patient required a dobutamine echocardiogram, because no patient with aortic stenosis included during the study period met the criteria to carry out said evaluation.[8]

The ejection dynamics parameters were measure using the velocity curve of the continuous Doppler recording in the apical 5-chambers axis. The ET was measured as the time from beginning to the end of systolic flow. The AT was obtained as the time interval between the onset of systolic flow to its

maximum velocity. Finally, the AT/ET ratio was calculated as well as the DVI.

The aortic waveform shape measured by continuous Doppler was performed by an by an expert echocardiography cardiologist, classifying it as rounded or triangular.

Statistical management of the information was analyzed with the IBM SPSS Statistics version 20 program. Quantitative variables are presented as mean and standard deviation (SD), and were compared using Kruskal-Wallis or ANOVA one-way. Categorical variables are reported as percentages and were compared using the χ^2 test or the Fisher's exact test. A ROC curve was plotted to determine the best cutoff value of AT, ET and AT/ET ratio that identified severe AS. Comparison between ejection dynamics parameters and other echocardiographic parameters was calculated by Pearson or Spearman correlation. Analysis of the aortic waveform shape was performed using a χ^2 test and subsequent subanalysis with Fisher's exact test. Differences were considered significant at p values <0.05 with a confidence interval (CI) of 95%.

RESULTS

A total of 75 patients were included with a mean age 64.7±16.7 years (48% women), of whom 10.7% had mild AS, 21.3% had moderate AS and 68% had severe AS. General patient characteristics are summarized in *Table 1*. 37.9% of the patients presented other diseases such as Parkinson's disease, hypothyroidism, COPD, rheumatoid arthritis, epilepsy and depressive disorder. Two patients were carriers of a definitive pacemaker, 5 patients had atrial fibrillation and one patient had a history of EVC.

Clinical presentation is shown in *Table 2*. Of the symptomatic patients (n=53), the average in

Table 1: General patient characteristics.[¥]

	Mild AS (n = 8)	Moderate AS (n = 16)	Severe AS (n = 51)	p
Age (y)	69.0 ± 16.6	68.6 ± 16.6	62.8 ± 16.7	NS
Women (%)	62.5	37.5	49.0	NS
Weight (kg)	69.0 ± 9.9	76.5 ± 11.1	72.6 ± 13.4	NS
BMI (kg/m^2)	27.10 ± 3.63	29.16 ± 3.27	28.03 ± 4.64	NS
BSA (m^2)	1.72 ± 0.15	1.81 ± 0.16	1.76 ± 0.18	NS
HR (bpm)	71.0 ± 10.7	70.4 ± 13.8	66.8 ± 13.0	NS
SBP* (mmHg)	125.1 ± 20.3	123.1 ± 19.0	126.2 ± 20.4	NS
DBP* (mmHg)	72.9 ± 9.5	79.0 ± 9.8	80.3 ± 11.0	NS
MBP* (mmHg)	90.3 ± 10.4	93.7 ± 12.2	95.6 ± 13.5	NS
Physical inactivity* (%)	85.7	69.2	91.3	NS
Smoking* (%)	57.1	53.8	47.8	NS
Hypertension* (%)	85.7	69.2	69.6	NS
Diabetes* (%)	71.4	30.8	26.1	NS
Dyslipidemia* (%)	42.9	46.2	34.8	NS
IHD* (%)	42.9	30.8	8.7	0.024[§]
CKD* (%)	28.6	15.4	2.2	0.024[§]
Other* (%)	57.1	38.5	34.8	NS

[¥] Data are expressed as mean ± SD or as percentages.
* Data of 9 patients were not included because they were not recorded during the study.
[§] Mild AS vs. severe AS.
AS = aortic stenosis; BMI = body mass index; BSA = body surface area; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; MBP = mean blood pressure; IHD = ischemic heart disease; CKD = chronic kidney disease; NS = not significant.

months of the onset of symptoms was 23.53±22.47, without statistically significant difference.

As expected, a statistically significant difference was observed between the usual echocardiographic parameters and the severity of AS, with the exception of the indexed stroke volume and LVEF *(Table 3)*. 70.7% patients presented mild aortic insufficiency.

It can be observed that ejection dynamics parameters were higher in patients with more severe AS. ROC curve analysis shows the best cutoff value to detect severe AS for AT (104.5ms), ET (323.5ms) and AT/ET ratio (0.345), with good sensitivity and specificity *(Table 4)*. The largest AUC was for AT (0.968), followed by AT/ET ratio (0.944), and finally by ET (0.726) *(Figure 2)*.

Table 2: Clinical presentation of patients.¥

	Mild AS	Moderate AS	Severe AS	p
Heart failure	42.9	46.2	69.6	NS
NYHA I-II	66.7	16.7	31.3	
NYHA III-IV	33.3	83.3	68.7	
Syncope	14.3	15.4	19.6	NS
Angor pectoris	14.3	38.5	60.9	0.041§
CCS I-II	100.0	40.0	32.2	
CCS III-IV	0.0	60.0	67.8	
Asymptomatic	57.1	30.8	10.9	0.009§
Other	0.0	7.7	10.9	NS

¥ Data are expressed as percentages. Data of 9 patients were not included because they were not recorded during the study.
§ Mild AS vs. Severe AS.
AS = aortic stenosis; NYHA = New York Heart Association; CCS = Canadian Cardiovascular Society; NS = not significant.

Table 3: Echocardiographic parameters in the different stages of AS.¥

	Mild AS	Moderate AS	Severe AS	p
Peak velocity (m/s)	2.50 ± 0.47	3.22 ± 0.46	4.94 ± 0.71	< 0.001‡*
Mean gradient (mmHg)	14.61 ± 6.19	23.64 ± 7.15	63.96 ± 21.19	< 0.001‡*
Maximal gradient (mmHg)	26.04 ± 9.04	41.99 ± 11.58	99.64 ± 30.53	< 0.001‡*
EOA (cm^2)	1.66 ± 0.16	1.30 ± 0.19	0.69 ± 0.19	< 0.001
Indexed EOA (cm^2/m^2)	0.98 ± 0.13	0.72 ± 0.11	0.40 ± 0.10	< 0.001
DVI	0.47 ± 0.14	0.35 ± 0.08	0.20 ± 0.05	< 0.001
Indexed stroke volume (ml/m^2)	46.29 ± 14.06	50.47 ± 12.45	48.83 ± 9.92	NS
LVEF (%)	61.0 ± 6.0	59.4 ± 10.6	62.9 ± 7.3	NS
AT (ms)	86.38 ± 14.81	90.06 ± 14.65	135.49 ± 22.30	< 0.001‡*
ET (ms)	304.13 ± 51.61	311.06 ± 51.16	343.37 ± 35.92	0.021‡, 0.038*
AT/ET ratio	0.29 ± 0.04	0.29 ± 0.04	0.40 ± 0.05	< 0.001‡*
Indexed ventricular mass (g/m^2)	86.44 ± 25.59	102.10 ± 42.04	126.06 ± 49.30	0.025

¥Data are expressed as mean ± SD or as percentages. (To evaluate the differences between the groups, the Tukey *post hoc* test was used).
‡Severe AS vs. mild AS.
*Severe AS vs. moderate AS.
AS = aortic stenosis; EOA = effective orifice area; DVI = Doppler velocity index; LVEF = left ventricular ejection fraction;
AT = acceleration time; ET = ejection time; NS = not significant.

Table 4: Diagnostic tests: optimal cutoff values of AT, ET and AT/ET ratio to differentiate severe AS.

Variable	AUC	Optimal cutoff	Sensitivity (%)	Specificity (%)	Accuracy (%)	PPV (%)	NPV (%)
AT	0.968	104.5 ms	92.2	83.3	89.3	92.2	83.3
ET	0.726	323.5 ms	80.4	70.8	77.3	85.4	63.0
AT/ET ratio	0.944	0.345	84.3	91.7	86.6	95.6	73.3

AT = acceleration time; ET = ejection time; AS = aortic stenosis; AUC = area under the curve; PPV = positive predictive value; NPV = negative predictive value.

Table 5: Correlation between AT and AT/ET ratio with usual echocardiographic measures to evaluate the degree of AS.

Variable	AT	p	AT/ET ratio	p
Peak velocity	r = 0.735	< 0.001	r = 0.719	< 0.001
Mean gradient	r = 0.718	< 0.001	r = 0.744	< 0.001
Maximal gradient	r = 0.705	< 0.001	r = 0.699	< 0.001
EOA	r = -0.732	< 0.001	r = -0.666	< 0.001
Indexed EOA	r = -0.767	< 0.001	r = -0.704	< 0.001
DVI	r = -0.754	< 0.001	r = -0.724	< 0.001

AT = acceleration time; ET = ejection time; AS = aortic stenosis; EOA = effective orifice area; DVI = Doppler velocity index.

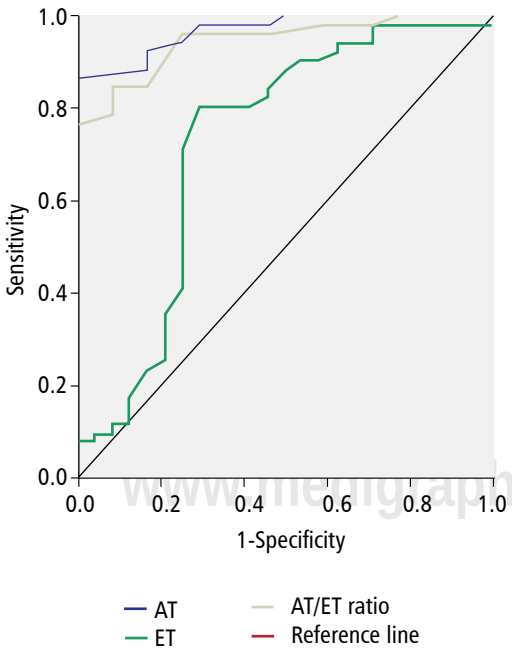

Figure 2: ROC curve to detect severe AS based on AT, ET and AT/ET ratio.

ROC = receiver operating characteristics; AS = aortic stenosis; AT = acceleration time; ET = ejection time.

We found high correlations between AT and AT/ET ratio with usual echocardiographic measures to evaluate the degree of AS *(Table 5)*. There were no correlations respect these measures and ET. Similarly, there was also no correlation between indexed stroke volume, LVEF and indexed ventricular mass with ejection dynamics parameters.

Respect the aortic waveform shape, we found that a redound contour of the Doppler jet velocity profile is more related to severe AS compared with moderate AS ($p<0.001$) and mild AS ($p<0.001$), stages in which it is more frequent to find a triangular spectrum.

DISCUSSION

Despite the fact that echocardiography remains the cornerstone in the diagnosis of AS, there is sometimes a discrepancy between clinical evaluation and echocardiographic data. The main limitations in calculating the valvular area by the continuity equation, lie in their

complexity, variability in the measurement of aortic valve annulus, flow dependence, state of volume and in some cases ventricular function. Therefore, it's considered necessary to find new parameters that demonstrate greater independence and reproducibility, in order to confirm the diagnosis, stratify the pathology, predict the outcomes and/or the evolution of the disease to choose appropriate treatment on time.

Study groups such as those of Hatle et al[14] and Nakamura et al, have previously reported a good correlation between the prolongation of AT and AT/ET ratio with the peak and mean transvalvular gradient evaluated by catheterization. Only works from a group of researchers in Spain,[12,13] France[15] and South Korea[6] have been published, who report a direct relationship between the duration of AT and AT/ET ratio with the severity of AS, and a strong association with high risk of death during follow-up. After adjusting variables of prognostic importance, such as the mean pressure gradient or indexed stroke volume, patients with a AT/ET ratio >0.36 and a AT >112ms presented an increased risk of global mortality.[15] So, both groups suggest to include these measurements in all echocardiograms of patients with AS.

The above has not been studied yet in the Mexican population, until this moment. The AT was significantly higher in patients with severe AS compared to the group of moderate and mild AS. We found that the best cutoff value to detect severe AS was 104.5ms, compared to Gamaza-Chulian et al, whose cut point reported was lower (94ms), with lower sensitivity and specificity.

The AT/ET ratio was significantly increased in the group of severe AS with respect to the other groups. Our optimal cutoff point was 0.345, while our reference study[12] found a slightly higher cut-off point (0.35), with lower sensitivity and specificity.

When we performed the correlation between the traditionally used parameters for the evaluation of the severity of the AS with the AT, ET and AT/ET ratio, a high correlation was found only with the AT and the AT/ET ratio. In concordance with previous reports,[12] we found no significant correlation with ET.

Our work group similarly found no correlation between the indexed stroke volume and the LVEF with AT nor with the AT/ET ratio. The above is of great importance, since it shows us that such parameters are independent of transvalvular flow and ventricular function.

Finally, regarding aortic waveform shape, coinciding with the other study groups,[13,16] we found that severe AS significantly presented a more rounded spectrum or with a later maximum peak compared to moderate or mild AS.

These novel measurements are obtained in a simpler and more reproducible way, with the advantage of being independent from ventricular function, flow and volume status, unlike most of the traditional parameters, which are subject to a greater margin of error besides increased inter and intra-observer variability. Our study confirms in our population that AT, ET and AT/ET ratio are useful parameters to identify the severity of AS and its usefulness is not limited only to cases in which there is disagreement with the usual measurements.[17]

CONCLUSIONS

The AT and the AT/ET ratio measured in the continuous Doppler curve of the aortic flow have a direct relationship with the severity of the aortic stenosis.

Limitations

The principal limitation of this trial was that it did not include patients with severe low-flow/low-gradient AS with reduced LVEF or paradoxical low-flow severe AS, which would help us confirm the validity of the parameters of the ejection dynamics when there is left ventricular dysfunction. Furthermore, we do not assess intra- and inter-observer variability as in other studies. In any way, to our knowledge until the writing of this work, we are the first group to study the parameters of ejection dynamics in Mexican population and also obtain a cut-off point for severe AS, not only with AT and the AT/ET ratio, but also with ET. A tool that can be easily measured, which

helps to corroborate the diagnosis of severe AS when there are discrepancies with the usual measurements.

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REFERENCES

1. Lung B, Baron G, Butchart EG et al. A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease. Eur Heart J. 2003; 24 (13): 1231-1243.
2. Otto CM, Prendergast B. Aortic-valve stenosis - from patients at risk to severe valve obstruction. N Engl J Med. 2014; 371: 744-756.
3. Joseph J, Nagvi SY, Giri J, Goldberg S. Aortic stenosis: pathophysiology, diagnosis, and therapy. Am J Med. 2017; 130 (3): 253-263.
4. Thaden JJ, Nkomo VT, Enriquez-Sarano M. The global burden of aortic stenosis. Prog Cardiovasc Dis. 2014; 56 (6): 565-571.
5. Otto CM, Bonow RO. BRAUNWALD Tratado de Cardiología. Texto de Medicina Cardiovascular. Capítulo 63: Cardiopatía valvular. 10a ed. Madrid: Editorial Elsevier; 2015. pp. 1446-1458.
6. Kim SH, Kim JS, Kim BS et al. Time to peak velocity of aortic flow is useful in predicting severeaortic stenosis. Int J Cardiol. 2014; 172 (3): e443-e446.
7. Minners J, Allgeier M, Gohlke-Baerwolf C et al. Inconsistent grading of aortic valve stenosis by current guidelines: haemodynamic studies in patients with apparently normal left ventricular function. Heart. 2010; 96 (18): 1463-1468.
8. Nishimura RA, Otto CM, Bonow RO et al. 2014 AHA/ACC Guideline for the management of patients with valvular heart disease: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014; 129: 2440-2492.
9. Gamaza-Chulián S, Ruiz-Fernández D, Díaz-Retamino E et al. Valor pronóstico del ratio tiempo de aceleración/tiempo de eyección en la estenosis valvular aórtica. CARDIOCORE. 2017. doi: 10.1016/j.carcor.2017.12.002.
10. Ben Zekry S, Saad RM, Ozkan M et al. Flow acceleration time and ratio of acceleration time to ejection time for prosthetic aortic valve function. J Am Coll Cardiol Img. 2011; 4 (11): 1161-1170.
11. Lancellotti P, Pibarot P, Chambers J et al. Recommendations for the imaging assessment of prosthetic heart valves: a report from the European Association of Cardiovascular Imaging endorsed by the Chinese Society of Echocardiography, the Inter-American Society of Echocardiography, and the Brazilian Department of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2016; 17 (6): 589-590.
12. Gamaza-Chulián S, Díaz-Retamino E, Camacho-Freire S et al. Acceleration time and ratio of acceleration time to ejection time in aortic stenosis: new echocardiographic diagnostic parameters. J Am Soc Echocardiogr. 2017; 30 (10): 947-955. doi: 10.1016/j.echo.2017.06.001.
13. Gamaza-Chulián S, Camacho-Freire S, Toro-Cebada R et al. Ratio of acceleration time to ejection time for assessing aortic stenosis severity. Echocardiography. 2015; 32: 1754-1761.
14. Hatle L, Angelsen BA, Trombsdal A. Non-invasive assessment of aortic stenosis by Doppler ultrasound. Br Heart J. 1980; 43: 284-292.
15. Ringle-Griguer A, Tribouilloy C, Truffier A et al. Clinical significance of ejection dynamics parameters in patients with aortic stenosis: an outcome study. J Am Soc Echocardiogr. 2018; 31 (5): 551-560.e2.
16. Chambers J, Rajani R, Hankins M et al. The peak to mean pressure decrease ratio: a new method of assessing aortic stenosis. J Am Soc Echocardiogr. 2005; 18 (6): 674-678.
17. Baumgartner H, Hung J, Bermejo J et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. J Am Soc Echocardiogr. 2009; 22 (1): 1-23.

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Original Research

Decreased reservory function of the left atrium is associated with pre-clinical diastolic dysfunction, analysis by sex. A 2D speckle tracking study

La disminución de la función reservoria de la aurícula izquierda se asocia con disfunción diastólica preclínica, análisis por sexo. Un estudio de rastreo de manchas (2D-STE)

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ABSTRACT. Heart failure with preserved ejection fraction, characterized by the presence of diastolic dysfunction, affects more women than men. A decrease in the phasic function of the left atrium has been found in individuals with diastolic dysfunction in pre-clinical stages. It is unknown if such deterioration is greater in women. **Objective:** To determine if there are differences in the phasic function of the left atrium obtained by «strain» analysis by speckle-tracking technique (2D-STE), in individuals with preclinical diastolic dysfunction in relation to sex. **Material and methods:** This is an observational, cross-sectional and analytical study which included 53 subjects of both sexes (62% men) with ejection fraction greater than 50% in stage I of NYHA. **Results:** In individuals with diastolic dysfunction, left atrial reservoir function determined by 2D-STE, was lower compared with individuals with normal diastolic function (37.6% vs 30.3%, p<0.0001). However, no significant differences were found between men and women. **Conclusion:** The decrease in reservoir function was similar for individuals of both sexes, in preclinical stages of diastolic dysfunction. Sex does not seem to be a determining factor for the development of alterations in left atrial function, at least in the reservoir phase.
Key words: Diastolic dysfunction, left atrial phasic function, left atrial strain, 2D-STE.

RESUMEN. *La insuficiencia cardiaca con fracción de expulsión preservada, caracterizada por la presencia de disfunción diastólica afecta más a mujeres que a hombres. Se ha encontrado disminución en la función fásica de la aurícula izquierda en individuos con disfunción diastólica en etapas preclínicas. Se desconoce si tal deterioro es mayor en mujeres.* ***Objetivo:*** *Determinar si existen diferencias en la función fásica de la aurícula izquierda obtenida por análisis de esfuerzo (strain) por técnica de rastreo de manchas (ECO 2D-STE), en individuos con disfunción diastólica preclínica en relación al sexo.* ***Material y métodos:*** *Se trata de un estudio observacional, transversal y analítico, que incluyó a 53 sujetos de ambos sexos (62% hombres) con fracción de expulsión mayor al 50% en etapa I de la NYHA.* ***Resultados:*** *En individuos con disfunción diastólica, la función de reservorio de la aurícula izquierda, determinada por ECO 2D-STE fue menor comparada con individuos con función diastólica normal (37.6% vs 30.3%; p<0.0001). Sin embargo, no se encontraron diferencias entre hombres y mujeres.* ***Conclusión:*** *La disminución en la función de reservorio fue similar para individuos de ambos sexos, en etapas preclínicas de disfunción diastólica. El sexo no parece ser un factor determinante para el desarrollo de alteraciones en la función auricular izquierda, al menos en la fase de reservorio.*
Palabras clave: *Disfunción diastólica, función fásica de aurícula izquierda, esfuerzo de aurícula izquierda, 2D-STE.*

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INTRODUCTION

Diastolic dysfunction (DD) early diagnosis is essential due to the risk of progression to heart failure with preserved ejection fraction (HFpEF), because once that symptoms develop, the 5-year mortality is greater than 70%[1] and no treatment to date has shown benefits in prevention of adverse outcomes. It is well known that HFpEF is more prevalent in women, implying that gender may play an important role in its development.[2] Left atrial enlargement is one of the four echocardiographic criteria used to define DD.[3] However, studies suggest that left atrial (LA) phasic function (reservoir, conduit and pump functions) obtained by 2D speckle tracking strain analysis (2D-STE), are more sensitive tool for the detection of DD in early stages.[4,5]

Many echocardiographic reference values are sex related[6] and even though previous studies have not demonstrated differences in left atrial strain (LAS) between men and women,[7] these have been performed in specific populations and therefore cannot be generalized. The aim of this study is to determine if there is a difference in LA phasic function, obtained by 2D-STE, in individuals with preclinical DD in relation to sex. This would allow us to understand if LA dysfunction is more prevalent in women with DD[6] and according to this establish gender specific cut-off points.

MATERIAL AND METHODS

Study population: This is an observational, cross-sectional and analytical study, carried out in individuals of both sexes, aged 18 years or older, with a left ventricular ejection fraction (LVEF) ≥50% determined by transthoracic echocardiography (TTE) and New York Heart Association (NYHA) functional class I. This study was conducted at Hospital Español of México, from May to December 2017. It was a convenience, non-probability and sequential sampling. Patients with decompensated chronic heart failure, significant valvular disease (moderate or severe aortic stenosis, any degree of mitral stenosis, moderate to severe regurgitation), previously diagnosed cardiomyopathies, prevalent or corrected congenital heart diseases, pericardial diseases, cardiac stimulation devices, atrial flutter or fibrillation and poor image quality were excluded. Data was collected in a capture sheet, created by the investigator, with patient identification, clinical and echocardiographic data. The protocol was approved by the local ethics and research committees, follows the guidelines of the Helsinki agreement and all participants gave informed written consent.

Echocardiography measurements: Standard echocardiography was performed using a Philips Epiq 7 ultrasound system with a 1-5 MHz transducer. LV function analysis was assessed by Simpson's modified rule.[6] LA volume index (LAVI) was obtained automatically using the biplane disk summation technique. Diastolic function was analyzed acquiring peak transmitral inflow velocities in early diastole (E), atrial systole (A) and E wave deceleration time (DTE) with pulsed Doppler. Tissue Doppler analysis of mitral annular septal and lateral velocities in early diastole (e'), their average and the average E/e' ratio were used for estimation of LV filling pressures. Maximum velocity of the tricuspid regurgitation (TR) jet was obtained in the apical four chamber, right ventricle entrance chamber or parasternal short axis at the level of the great vessels views, recording the highest value. DD was diagnosed based on guideline recommendations.[3] For 2D speckle-tracking LAS analysis images were acquired at 60-80 frames per second with manual tracing of atrial endocardial borders. An average of 3 measurements from 2 cardiac cycles in the apical 2 and 4 chamber axis views was used, for a total of six measurements per each atrial function. Strain curve analysis was QRS-triggered. Reservoir function was obtained from peak systolic deformation of the plotted curve, conduit function during passive LV filling and pump function at peak of atrial contraction. Images were processed with QLAB Cardiac software (Philips Healthcare).

Statistical analysis: Normality's assessment was based on kurtosis and skewness values. Data are expressed as mean±SD for continuous variables or interquartile ranges according to distribution and percentages for categorical variables. The primary objective was evaluated with Student's T test for parametric variables or Mann-Whitney U test for nonparametric

ones. Proportion differences were evaluated with the χ^2 test. A ROC curve analysis was used to compare sensitivity and specificity of atrial function against. P values<0.05 at two tails were considered significant.

RESULTS

Clinical and echocardiographic characteristics

Were included 53 patients, 34 (64%) with normal diastolic function and 19 (35%) with diastolic dysfunction. Amongst the known risk factors for DD, we observed that 61% of the subjects with systemic arterial hypertension presented with some degree of DD (p=0.008) and prevalence was higher in older individuals (p<0.001). Individuals with DD had lower LVEF (p=0.005) and higher LAVI (22.6mL/m^2 vs 30.9mL/m^2, p=0.002). The average e' velocity was significantly lower in individuals with DD (p<0.0001). However, no difference was found between the E/e' ratio and the presence or absence of DD (p=0.151). There were no significant differences in DD between men and women (p=0.279). Only in five cases had evidence of increased LV filling pressures determined by E/e' ratio or significant dilatation of the LA was found (LAVI>42mL/m^2) *(Table 1)*.

Left atrial function and diastolic dysfunction

Reservoir function was significantly lower in subjects with DD (37.6 vs 30.3%, p<0.0001). However, we found no significant differences between men and women (p=0.298). There was no deterioration in conduit and pump functions in individuals with DD compared with those with normal diastolic function (p=0.383 y p=0.439, respectively) *(Table 1)*.

ROC curve analysis of LA reservoir function and LAVI

We assessed sensibility and specificity for DD diagnosis by ROC curve analysis. Reservoir function showed better performance with an area under the curve (AUC) of 0.83 compared to LAVI (AUC=0.77, p<0.001).

DISCUSSION

Our results were consistent with previously published studies in which a decrease in LA

Table 1: Echocardiographic variables in total population and group differences.

	Total population (n = 53)	Normal diastolic function (n = 34)	Diastolic dysfunction (n = 19)	p
LVEF (%)**	63.1 (60-65)	65 (61.9-66.9)	60.7 (59.8-63.8)	0.005
LAVI (mL/m^2)*	25.6 ± 7.8	22.6 ± 4.5	30.9 ± 9.6	< 0.0001
Em (cm/s)**	76 ± 22.7	79.1 (68.6-94.6)	65.5 (49.5-82)	0.047
Am (cm/s)**	66.4 ± 23.7	59.9 ± 20.2	78 ± 25.5	0.009
DT (ms)*	226.7 ± 65.6	195.8 ± 43.6	282.1 ± 62.5	< 0.0001
Septal e' (cm/s)**	8.9 ± 2.6	9.8 ± 2.2	7.3 ± 2.6	< 0.0001
Lateral e' (cm/s)*	11 ± 3.3	12.4 ± 2.6	8.5 ± 2.7	< 0.0001
Average e' (cm/s)**	9.9 ± 2.7	10.9 (9.8-12.3)	8.16 (6.6-9.2)	< 0.0001
E/e'*	8.2 ± 3.1	7.6 ± 2	9.2 ± 4.4	0.077
TR jet velocity (m/s)**	2.1 ± 1.2	2.06 (1.8-2.4)	2.34 (1.3-2.6)	0.409
Reservoir function (%)*	35 ± 6.6	37.6 ± 5.5	30.3 ± 5.9	< 0.0001
Conduit function (%)**	16.1 (14.3-18.5)	16.7 (14.7-18.5)	16 (13.8-18.6)	0.860
Pump function (%)*	-2.5 (-3.5, -1.2)	-2.4 (-4.1, -1.6)	-1.6 (-3.1, -1.04)	0.084

LVEF = left ventricle ejection fraction; LAVI = left atrium index volume; Em = mitral E velocity; Am = mitral A velocity; TR = tricuspid regurgitation; DT = mitral deceleration time.
* Student 's T test. ** Mann-Whitney U test.

reservoir function is observed in patients in preclinical stages of DD, who may not even present LA structural changes or other data suggesting elevated LV filling pressures.[4,5] Reservoir function decline was similar for both men and women, which suggests that sex does not seem to be an independent risk factor for the development of LA dysfunction, at least not in the reservoir phase. The foregoing should be considered when analyzing traditional risk factors for DD since male individuals may present with the same subclinical alterations as women. Similarly, ROC curve analysis suggests that LA dysfunction alterations, in this case reservoir function, could be a more sensitive and specific tool for DD detection in early stages compared with volumetric analysis, which has been previously reported.[8] Therefore, its routine evaluation in patients with known risk factors, the definition of cut-off points and echocardiographic surveillance could result in early diagnosis, allowing for a more efficient prevention strategy. Unlike data reported in other studies, no significant decrease was found in conduit and pump functions, which could be due to the number of subjects included in our study, or to the lack of standardization of measurement techniques and cut-off points. Furthermore, this may be explained because of maintenance of conduit and pump functions until a greater degree of DD develops. Limitations of the present study were our sample size, the lack of established cut-off points and the absence of software specifically dedicated to LA strain analysis.

CONCLUSIONS

Left atrial reservoir function declines in individuals with pre-clinical diastolic dysfunction. However, sex does not seem to be an independent variable for the development of LA dysfunction. LA function strain analysis could be a diagnostic tool with better performance than LAVI for the detection of diastolic dysfunction in preclinical stages. Further studies are needed in our population to establish specific cut-off points and an association with LA dysfunction and clinical adverse outcomes.

REFERENCES

1. Shah KS, Xu H, Matsouaka RA et al. Heart failure with preserved, borderline, and reduced ejection fraction. J Am Coll Cardiol. 2017; 70 (20): 2476-2486.
2. Duca F, Zotter TC, Kammerlander AA et al. Gender-related differences in heart failure with preserved ejection fraction. Sci Rep. 2018; 8: 1080.
3. Nagueh SF, Smiseth OA, Appleton CP et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2016; 29: 277-314.
4. Brecht A, Oertelt-Prigione S, Seeland U et al. Left atrial function in preclinical diastolic dysfunction: two-dimensional speckle-tracking echocardiography–derived results from the BEFRI trial. J Am Soc Echocardiogr. 2016; 29 (8): 750-758.
5. Khan UA, de Simone G, Hill J et al. Depressed atrial function in diastolic dysfunction: a speckle tracking imaging study. Echocardiography. 2013; 30 (3): 309-316.
6. Lang RM, Badano LP, Mor-Avi V et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015; 28: 1-39.
7. Nikitin NP, Witte KK, Thackray SD et al. Effect of age and sex on left atrial morphology and function. Eur J Echocardiogr. 2003; 4: 36-42.
8. Carluccio E, Biagioli P, Mengoni A et al. Left atrial reservoir function and outcome in heart failure with reduced ejection fraction the importance of atrial strain by speckle tracking echocardiography. Circ Cardiovasc Imaging. 2018; 11.

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Original Research

Assessment of endothelial function and intima-media thickness in Mexican children with metabolic syndrome: a cross sectional study

Evaluación de la función endotelial y del grosor de la íntima-media en niños mexicanos con síndrome metabólico: un estudio transversal

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ABSTRACT. Introduction: Metabolic syndrome (MS) is a well-known risk factor for the development of cardiovascular disease. **Objective:** The aim of this study was to compare the endothelial function and intima-media thickness, in children with MS vs control group to determine the existence of mechanisms of atherosclerosis. **Material and methods:** Cross sectional study that included children with MS and controls (15 patients in each group) were studied. Systolic pulmonary artery pressure (SPAP), flow-mediated vasodilation (FMV) and intima-media thickness (IMT) were assessed. **Results:** The FMV in MS was diminished compared with control (8.7%±1.6 vs 12.9%±1.2; p<0.0001). FMV was less than 10% in 73% of MS group vs 13% of controls. IMT was increased in MS (0.06 ± 0.002 vs 0.034 ± 0.005; p < 0.001). In the MS group, SPAP was increased (37.8±14.4 vs 23.2±5.31; p<0.002). A negative correlation between FMV and IMT was found (r =-0.562). **Conclusion:** In our study, metabolic syndrome group had and increased of IMT and SPAP and a decreased FMV. These findings should relate with the initial mechanism of the atherosclerosis from the infancy.
Key words: Metabolic syndrome, flow-mediated vasodilation, intima-media thickness.

RESUMEN. Introducción: *El síndrome metabólico (SM) es un factor de riesgo para el desarrollo de enfermedad cardiovascular.* ***Objetivo:*** *Comparar la función endotelial y el grosor íntima-media en niños con SM y niños sanos, para determinar la existencia de mecanismos de aterosclerosis temprana.* ***Material y métodos:*** *Estudio transversal que incluyó dos grupos: 15 niños con SM y 15 sanos. Se determinó: presión sistólica de la arteria pulmonar (PSAP), vasodilatación mediada por flujo (VMF) y grosor íntima-media (GIMc).* ***Resultados:*** *La VMF en niños con SM fue inferior a la de los sanos (8.7±1.6% vs 12.9±1.2%, p<0.0001). En el 73% de niños con SM y en el 13% de sanos la VMF fue menor al 10%. El GIMc fue mayor en SM (0.06±0.002 vs 0.034±0.005, p<0.001). En SM la PSAP fue mayor (37.8±14.4 vs 23.2±5.31, p<0.002). Existió correlación negativa entre la VMF y el GIMc (r:-0.562).* ***Conclusiones:*** *Los niños con SM tuvieron una VMF disminuida, incremento en el GIMc y PSAP, lo cual podría relacionarse con aterosclerosis temprana.*
Palabras clave: *Síndrome metabólico, vasodilatación mediada por flujo, grosor íntima-media.*

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INTRODUCTION

Metabolic syndrome (MS) is a global epidemic that also affects children. Prevalence according to panel III (ATP III) modified version of MS for children and teenagers (which includes abdominal obesity, atherogenic dyslipidemia, high blood pressure, glucose intolerance, proinflammatory and prothrombotic state), is approximately 4.2% and continues to rise.[1]

Endothelial dysfunction is one of the initial features of MS and characterizes by

diminished vasodilatation and/or paradoxical vasoconstriction of distal or coronary arteries in response to a nitric oxide (NO) liberating stimuli. Such response is observed in the early stages of the atherosclerotic process and may be quantified through the measurement of brachial artery flow mediated dilatation.

Endothelial dysfunction has been documented in children with at least one of the MS components: insulin resistance,[2,3] central obesity,[4-10] arterial hypertension,[10] atherogenic dyslipidemia[11,12] or a combination of the anterior;[5] but few studies have evaluated endothelial function directly in this group of patients. Therefore, the aim of this study is to assess endothelial function and intima-media thickness in children with MS compared with a control group.

MATERIAL AND METHODS

Cross sectional study include children between the ages 3 to 17 years old were enrolled, in a period from November 2005 to July 2007, in the *Instituto Nacional de Cardiología «Ignacio Chávez»*. Two groups were compared: 15 participants in the MS group (body mass index ≥25kg/m^2) vs. 15 in the control group, paired by age and sex. ATP III criteria was used to define MS. Children were evaluated by two expert pediatric cardiologists and an echocardiographic comprehensive evaluation was carried out. Exclusion criteria were children with congenital or acquired heart disease.

Echocardiographic evaluation was performed with a Phillips Sonos 5500 ultrasound system, with an S3 transducer. Left ventricular systolic function was determined with the biplane method of disks. Systolic pulmonary artery pressure (SPAP) was estimated with Bernoulli equation using maximal velocity of tricuspid regurgitation jet acquired with continuous Doppler in the apical four chamber view.

Endothelial function evaluation was performed with a Phillips Sonos 5500 high resolution vascular ultrasound and a linear transducer of 3 to 11MHz. Brachial artery dilatation was measured according to previously established rules.[13] Left arm extension and immobilization is required to allow access to the brachial artery. Image acquisition was performed in a longitudinal plane, including 5cm of the artery, located 4-5cm above the antecubital fold. Mean diameter was obtained from 3 measurements in end-diastole (end of the T wave in the electrocardiogram). Blood pressure cuff was inflated to 150mmHg (systolic pressure) for 5min and arterial diameter was registered one and five minutes after deflating. Flow mediated vasodilatation (FMV) was defined as the percentage difference between the internal diameter of the brachial artery during reactive hyperemia in the first minute and its initial diameter. Any change less than 10% was considered abnormal.

Intima-media thickness (IMT) of the left common carotid artery was evaluated. An average of three measurements from the media-adventitia interface to the adventitia-intima interface was obtained. Such measurements were performed in the longitudinal plane, 2cm before the carotid bifurcation in three different points, with biplane technique. Values were considered normal when IMT was ≤0.04mm.

Statistical analysis. All variables were expressed as means and standard deviation according to their distribution. Primary objective was analyzed with a Student's T test. A p≤0.05 was considered significant. A Pearson correlation coefficient was obtained between FMV and IMT.

RESULTS

Table 1 shows there was a statistically significant difference among all clinical variables between groups. Mean age for the whole sample was 10.6±3.3 years. Both FMV and reactive hyperemia

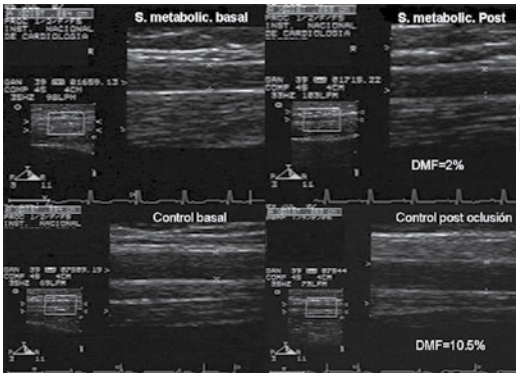

Figure 1:

Brachial artery 2D and m-mode images from a child with MS (upper images) and a healthy participant (lower images). This shows that FMV values are lower in MS group (2%), compared to control group (10.5%).

showed significant differences between children with MS vs. controls ($p<0.001$) *(Figure 1)*. FMV values were below 10 in 73% children with MS and only in 13% of the healthy group. Children with MS had significantly higher IMT and PAP values ($p\leq0.001$ and $p\leq0.002$ respectively).

There was a negative moderate correlation between FMV and IMT ($r=-0.562$).

Children with BMI of 25 to 32kg/m^2 had an IMT of 0.07±0.002 mm vs. children with BMI <25kg/m^2 (0.03±0.001 mm).

DISCUSSION

Our study results in terms of values of FMV and IMT in children with any components of the metabolic syndrome were similar to those reported in other populations.[4,14-20] To be noted are the low values of FMV found in the control group, which may be explained by higher altitude conditions.[21]

Apart from cardiovascular risk factors, other patient specific conditions may influence the IMT and FMV values, among which are: gene polymorphisms of endothelial nitric oxide synthase and other genes involved in lipid metabolism,[22,23] MS family history,[24] ethnic group,[1] birth weight,[25] mother's diet during pregnancy and lactation[26] and socioeconomic level.[27]

There's a positive correlation between IMT and age, in part due to increase in blood volume, blood pressure and body mass index.[28-31] Arterial hypertension is the single most important component of the MS in relation to great arteries structure and function.[32]

Elevated values of IMT and low values of FMV correlate with obesity, high body fat percentage,[7] high levels of plasma adiponectin and glucose,[7] glucose intolerance, hyperinsulinemia, high levels of thrombomodulin, increase in proinflammatory adhesins such as intercellular adhesion molecule 1, selectins, and C reactive protein levels,[5] which indicates a positive correlation between levels of systemic inflammation and endothelial dysfunction degree.[5,19,33-35] In our study IMT values correlate inversely with FMV.

This is the first study that shows SPAP elevation in children with MS that could be explained by vasoconstriction of the pulmonary vascular bed mediated by a reduction in nitric oxide production and endothelin 1 relative increase.

Chronic inflammation is an essential factor in MS pathophysiology. It is well known that children with MS express an increased amount of proinflammatory and antifibrinolytic factors, and thereby develop

Table 1: Clinical and echocardiographic variables in both groups.

Variable	MS	Controls	p
Abdominal perimeter (cm)	96.5 ± 6.7	54.2 ± 6.3	0.001
Cholesterol t (mg/dL)	181 ± 14.8	99 ± 11.7	0.000
HDL (mg/dL)	34 ± 2.9	44 ± 3.8	0.002
LDL (mg/dL)	133 ± 16.1	82 ± 14.5	0.001
Triglicerides (mg/dL)	229 ± 47.8	108 ± 15.1	0.001
Glucose (mg/dL)	144 ± 12.8	85 ± 4.5	0.001
C reactive protein (mg/L)	11.8 ± 2.2	1 ± 0.4	0.000
BMI (kg/m^2)	32 ± 3.01	20 ± 3.66	0.000
FMV (%)	8.7 ± 1.6	12.9 ± 1.2	0.000
IMT (mm)	0.06 ± 0.002	0.03 ± 0.005	0.001
SPAP (mmHg)	37.8 ± 14.4	23.2 ± 5.31	0.002

HDL = high density cholesterol; LDL = low density cholesterol; BMI = body mass index.

insulin resistance which has been associated with endothelial dysfunction, the initial step in atherosclerosis.[34-40] Chronic inflammation represents an important factor in the origin of the MS: stimuli such as over nutrition, physical inactivity, and age, can increase the secretion of cytokines and eventually lead to an increase in insulin resistance.[34,35,41-43] Some authors suggest tha central obesity is the trigger for glucose intolerance, increased pro-inflammatory markers, endothelial dysfunction and prothrombotic state.[44] Various factors secreted by adipocytes such as free fatty acids and tumor necrosis factor alpha have an important role in the development of insulin resistance by interrupting its signaling pathway.[45] Also, the increase in lipase activity is linked to the proinflammatory condition of the syndrome.[46]

IMT values decline in obese children who experience weight loss, suggesting reversibility in the atherosclerotic process.[47] Therefore, the importance in early detection of endothelial dysfunction in children with MS, so that an effective primary prevention strategy could be established. Exercise contributes to improving endothelial function and increasing levels of HDL-cholesterol.[48] Exercise during childhood exerts its protective effect on the cardiovascular system, at the endothelium level.[49] That is why treatment in children with metabolic syndrome should be based on in changes in eating habits and rigorous exercise.

CONCLUSIONS

Our study shows that children with MS have an increase in IMT values, higher PAP a lower VMF values compared with healthy children. All of which could represent signs of early atherosclerosis.

REFERENCES

1. Barkai L, Paragh G. Metabolic syndrome in childhood and adolescence. Orv Hetil. 2006; 147: 243-250.
2. Del Giudice E, Dilillo A, Tromba L et al. Aortic, carotid intima-media thickness and flow- mediated dilation as markers of early atherosclerosis in a cohort of pediatric patients with rheumatic diseases. Clin Rheumatol. 2018; 37 (6): 1675-1682.
3. Maple-Brown L, Cunningham J, Celermajer DS et al. Increased carotid intima-media thickness in remote and urban Indigenous Australians: impact of diabetes and components of the metabolic syndrome. Clin Endocrinol. 2007; 66: 419-425.
4. Kapiotis S, Holzer G, Schaller G et al. A proinflammatory state is detectable in obese children and is accompanied by functional and morphological vascular changes. Arterioscler Thromb Vasc Biol. 2006; 26: 2541-2546.
5. Gooty VD, Sinaiko AR, Ryder JR et al. Association between carotid intima media thickness, age, and cardiovascular risk factors in children and adolescents. Metab Syndr Relat Disord. 2018; 16 (3): 122-126.
6. Atabek ME, Pirgon O, Kivrak AS. Evidence for association between insulin resistance and premature carotid atherosclerosis in childhood obesity. Pediatr Res. 2007; 61: 345-349.
7. Reinehr T, Kiess W, de Sousa G et al. Intima media thickness in childhood obesity: relations to inflammatory marker, glucose metabolism, and blood pressure. Metabolism. 2006; 55: 113-118.
8. Zhu W, Huang X, He J et al. Arterial intima-media thickening and endothelial dysfunction in obese Chinese children. Eur J Pediatr. 2005; 164: 337-344.
9. Woo KS, Chook P, Yu CW et al. Overweight in children is associated with arterial endothelial dysfunction and intima-media thickening. Int J Obes Relat Metab Disord. 2004; 28: 852-857.
10. El Jalbout R, Cloutier G, Cardinal MR et al. Carotid artery intima-media thickness measurement in children with normal and increased body mass index: a comparison of three techniques. Pediatr Radiol. 2018; 48 (8): 1073-1079.
11. Sorensen KE, Celermajer DS, Georgakopoulos D et al. Impairment of endothelium-dependent dilation is an early event in children with familial hypercholesterolemia and is related to the lipoprotein(a) level. J Clin Invest. 1994; 93: 50-55.
12. Pauciullo P, Llannuzzi A, Sartorio R et al. Increased intima-media thickness of the common carotid artery in hypercholesterolemic children. Arterioscler Thromb. 1994; 14: 1075-1079.
13. Celermajer DS, Sorensen KE, Gooch VM et al. Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis. Lancet. 1992; 340: 1111-1115.
14. Esposito K, Ciotola M, Schisano B et al. Oxidative stress in the metabolic syndrome. J Endocrinol Invest. 2006; 29: 791-795.
15. Tołwińska J, Głowińska-Olszewska B, Urban M et al. Endokrynol Diabetol Chor Przemiany Materii Wieku Rozw. 2006; 12: 200-204.
16. Meyer AA, Kundt G, Steiner M et al. Impaired flow-mediated vasodilation, carotid artery intima-media thickening, and elevated endothelial plasma markers in obese children: the impact of cardiovascular risk factors. Pediatrics. 2006; 117: 1560-1567.
17. Kadono T, Sugiyama H, Hoshiai M et al. Endothelial function evaluated by flow-mediated dilatation in pediatric vascular disease. Pediatr Cardiol. 2005; 26: 385-390.
18. Järvisalo MJ, Lehtimäki T, Raitakari OT. Determinants of arterial nitrate-mediated dilatation in children: role of oxidized low-density lipoprotein, endothelial function,

and carotid intima-media thickness. Circulation. 2004; 109: 2885-2889.
19. Day TG, Park M, Kinra S. The association between blood pressure and carotid intima-media thickness in children: a systematic review. Cardiol Young. 2017; 27 (7): 1295-1305.
20. Liu F, Zhang HY, Liu XN et al. The association between metabolic syndrome and atherosclerosis. Zhonghua Yi Xue Za Zhi. 2003; 83: 1317-1320.
21. Frick M, Rinner A, Mair J et al. Transient impairment of flow-mediated vasodilation in patients with metabolic syndrome at moderate altitude (1,700 m). Int J Cardiol. 2006; 109: 82-87.
22. González-Sánchez JL, Martínez-Larrad MT, Saez ME et al. Endothelial nitric oxide synthase haplotypes are associated with features of metabolic syndrome. Clin Chem. 2007; 53: 91-97.
23. Zannad F, Sass C, Visvikis S. Environmental and genetic determinants of intima-media thickness of the carotid artery. Clin Exp Pharmacol Physiol. 2001; 28: 1007-1010.
24. Yang XZ, Liu Y, Mi J et al. Pre-clinical atherosclerosis evaluated by carotid artery intima-media thickness and the risk factors in children. Chin Med J (Engl). 2007; 120: 359-362.
25. Järvisalo MJ, Raitakari OT. Ultrasound assessment of endotelial function in children. Vasc Health Risk Manag. 2005; 1 (3): 227-233.
26. Das UN. Pathophysiology of metabolic syndrome X and its links to the perinatal period. Nutrition. 2005; 21: 762-773.
27. Kivimäki M, Smith GD, Juonala M et al. Socioeconomic position in childhood and adult cardiovascular risk factors, vascular structure, and function: cardiovascular risk in young Finns study. Heart. 2006; 92: 474-480.
28. Osika W, Dangardt F, Grönros J et al. Increasing peripheral artery intima thickness from childhood to seniority. Arterioscler Thromb Vasc Biol. 2007; 27: 671-676.
29. Ishizu T, Ishimitsu T, Yanagi H et al. Effect of age on carotid arterial intima-media thickness in childhood. Heart Vessels. 2004; 19: 189-195.
30. Empana JP, Zureik M, Gariepy J et al. The metabolic syndrome and the carotid artery structure in noninstitutionalized elderly subjects: the three-city study. Stroke. 2007; 38: 893-899.
31. Sass C, Herbeth B, Chapet O et al. Intima-media thickness and diameter of carotid and femoral arteries in children, adolescents and adults from the Stanislas cohort: effect of age, sex, anthropometry and blood pressure. J Hypertens. 1998; 16: 1593-1602.
32. Czernichow S, Bertrais S, Blacher J et al. Metabolic syndrome in relation to structure and function of large arteries: a predominant effect of blood pressure. A report from the SU.VI.MAX. Vascular study. Am J Hypertens. 2005; 18: 1154-1160.
33. Zambon A, Pauletto P, Crepaldi G. Review article: the metabolic syndrome-a chronic cardiovascular inflammatory condition. Aliment Pharmacol Ther. 2005; 22 Suppl 2: 20-23.
34. Hidvégi T, Szatmári F, Hetyési K et al. Intima-media thickness of the carotid arteries in subjects with hyperinsulinaemia (insulin resistance). Diabetes Nutr Metab. 2003; 16: 139-144.
35. Valle-Jiménez M, Estepa RM, Camacho RM et al. Endothelial dysfunction is related to insulin resistance and inflammatory biomarker levels in obese prepubertal children. Eur J Endocrinol. 2007; 156: 497-502.
36. Han SH, Quon MJ, Koh KK. Reciprocal relationships between abnormal metabolic parameters and endothelial dysfunction. Curr Opin Lipidol. 2007; 18: 58-65.
37. Wassink AM, Olijhoek JK, Visseren FL. The metabolic syndrome: metabolic changes with vascular consequences. Eur J Clin Invest. 2007; 37: 8-17.
38. Ritchie SA, Connell JM. The link between abdominal obesity, metabolic syndrome and cardiovascular disease. Nutr Metab Cardiovasc Dis. 2007; 17: 319-326.
39. Ailhaud G. Adipose tissue as a secretory organ: from adipogenesis to the metabolic syndrome. C R Biol. 2006; 329: 570-577.
40. Beauloye V, Zech F, Tran HT et al. Determinants of early atherosclerosis in obese children and adolescents. J Clin Endocrinol Metab. 2007; 92: 3025-3032.
41. Bahia L, de Aguiar LG, Villela NR et al. The endothelium in the metabolic syndrome. Arq Bras Endocrinol Metabol. 2006; 50: 291-303.
42. Esposito K, Giugliano D. The metabolic syndrome and inflammation: association or causation? Nutr Metab Cardiovasc Dis. 2004; 14: 228-232.
43. Koh KK, Han SH, Quon MJ. Inflammatory markers and the metabolic syndrome: insights from therapeutic interventions. J Am Coll Cardiol. 2005; 46: 1978-1985.
44. Terán-García M, Bouchard C. Genetics of the metabolic syndrome. Appl Physiol Nutr Metab. 2007; 32: 89-114.
45. Lupattelli G, Marchesi S, Ronti T et al. Endothelial dysfunction *in vivo* is related to monocyte resistin mRNA expression. J Clin Pharm Ther. 2007; 32: 373-379.
46. Lamarche B, Paradis ME. Endothelial lipase and the metabolic syndrome. Curr Opin Lipidol. 2007; 18: 298-303.
47. Wunsch R, de Sousa G, Toschke AM et al. Intima-media thickness in obese children before and after weight loss. Pediatrics. 2006; 118: 2334-2340.
48. Kelly AS, Wetzsteon RJ, Kaiser DR et al. Inflammation, insulin, and endothelial function in overweight children and adolescents: the role of exercise. J Pediatr. 2004; 145: 731-736.
49. Abbott RA, Harkness MA, Davies PS. Correlation of habitual physical activity levels with flow-mediated dilation of the brachial artery in 5-10 year old children. Atherosclerosis. 2002; 160: 233-239.

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Original Research

Left atrium function by speckle tracking in cirrhosis

Función de la aurícula izquierda por el rastreo de manchas en la cirrosis

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ABSTRACT. Introduction: Cirrhotic cardiomyopathy, which is defined as systolic and/or diastolic dysfunction of the left ventricle at rest or stress (in the absence of other cardiovascular conditions), worsens prognosis after transplant or other liver surgical procedures. The aim of this study was to characterize left atrial function by speckle tracking in cirrhotic patients. **Methods:** We consecutively included 99 patients with liver cirrhosis of different etiologies. An echocardiographic evaluation with measurement of left ventricular and atrial function was performed using traditional techniques, three-dimensional measurements and speckle tracking. **Results:** The median age was 50.9 years and 40% were men. None had left ventricular systolic dysfunction. Diastolic dysfunction and left atrium enlargement were found in 27%, the latter with a significant increase according to Child Pugh stage. Left atrium pump function showed alterations in 29% of subjects. **Conclusion:** We found left ventricular diastolic dysfunction and alterations in left atrial systolic function measured by speckle tracking in cirrhotic patients.
Key words: Atrial function left, echocardiography, liver cirrhosis.

RESUMEN. Introducción: *La miocardiopatía del cirrótico es la disfunción sistólica y/o diastólica del ventrículo izquierdo en reposo o al estrés, en ausencia de otras condiciones cardiovasculares que la expliquen, y que empeora el pronóstico postrasplante u otros procedimientos quirúrgicos hepáticos. El objetivo del estudio es caracterizar la función auricular izquierda con rastreo de manchas en pacientes cirróticos.* ***Métodos:*** *Se incluyeron 99 pacientes con cirrosis hepática de diferentes etiologías, de forma consecutiva. A todos se les realizó estudio ecocardiográfico en reposo con medición de la función ventricular y auricular izquierda con técnicas tradicionales, mediciones tridimensionales y rastreo de manchas.* ***Resultados:*** *La mediana de edad es 50.9 años, 40% son hombres. No se observaron alteraciones de la función sistólica del ventrículo izquierdo. El 27% presentaron disfunción diastólica y dilatación de aurícula izquierda, esta última con incremento significativo según el estadio Child-Pugh. El 29% presentaron alteraciones de la función de bomba de la aurícula izquierda.* ***Conclusión:*** *Se encontró disfunción diastólica del ventrículo izquierdo y alteraciones en la función sistólica de la aurícula izquierda, medida por rastreo de manchas, en pacientes cirróticos.*
Palabras clave: *Función aurícula izquierda, ecocardiograma, cirrosis hepática.*

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INTRODUCTION

Cirrhotic cardiomyopathy has been defined as the presence of an hyperdynamic state with increased cardiac output at rest, decreased peripheral vascular resistance and splanchnic vasodilation. All this is associated with diastolic dysfunction (DD) in patients with preserved left ventricular (LV) systolic function at rest. These patients have a diminished chronotropic and inotropic response to stress leading to heart failure and QT interval prolongation. This has an impact on prognosis and aggravates the clinical course during surgery, insertion of transjugular intrahepatic portosystemic shunts and liver transplantation. Several studies have shown an improvement in diastolic function, function and size of left atrium (LA), and chronotropic and inotropic response to stress, after liver transplantation. The aim of the study

is to characterize LA and ventricular function with speckle tracking (ST) in cirrhotic patients.

METHODS

Population: This is a cross-sectional study that included patients of both sexes, aged 18 to 70 years, with diagnosis of liver cirrhosis of any etiology, evaluated in the National Institute of Medical Sciences and Nutrition (Mexico), between January 2015 and December 2016. Patient selection was done consecutively at convenience. Cirrhotic patients with diabetes, systemic hypertension, hemoglobin levels less than 10g/dL, acute or chronic renal failure, ischemic heart disease, significant valvular heart disease (at least moderate degree of severity), diagnosis of permanent cardiac arrhythmia (not including Long QT syndrome), thyroid dysfunction, chronic obstructive pulmonary disease, sleep apnea syndrome and/or type I pulmonary hypertension were excluded. Patients with poor acoustic echocardiographic window were eliminated.

Echocardiogram: All studies were performed with VIVID 9 General Electric echocardiography equipment, with two-dimensional sectorial cardiac probe MSS-D (1.5-4.5MHz), as well as three-dimensional sectorial cardiac probe 4D (1.5-4MHz). EchoPAC software was used for processing of myocardial deformation by two-dimensional ST and three-dimensional images for volumetric analysis. The peak strain in the three phases of atrial contraction was determined (εs, εe, εa). LA strain was set to zero at the beginning of the QRS complex (QRS-triggered analysis).

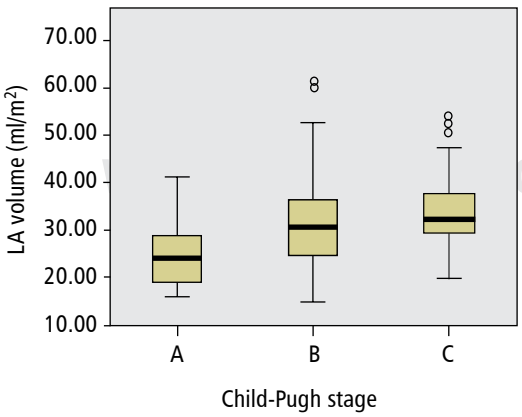

Figure 1:

Indexed volume of LA by Child-Pugh stage, p<0.05.

There are no established cut-off values, however, those proposed by Kim et al[1] were taken as reference. Images were obtained by three experts with interobserver variability of less than 2%. Processing and measurement of the variables was performed by a single expert echocardiographer blinded to clinical data. The processed images fulfilled the quality criteria established in international guidelines regarding to frame rate and volume rate.

Statistical analysis: Data was analyzed using the IBM SPSS Statistics 22 software. Results are presented as medians for quantitative variables and as a percentage for categorical variables. Analysis by groups was carried out according to the Child Pugh classification. χ^2 and Kruskal-Wallis tests were used for comparisons between groups. A value of $p<0.05$ at two tails was considered significant.

RESULTS

Ninety-nine patients were included, with a median age of 50.9 years (minimum of 21, maximum of 81) and 40% were men. The etiology was viral in 27, cryptogenic in 18, primary biliary cirrhosis (PBC) in 16, PBC/autoimmune hepatitis (AIH) in 6, AIH in 13, nonalcoholic fatty liver in 10 and alcoholic in 9 subjects. 24 patients were in Child Pugh stage A, 43 in stage B and 32 in stage C. LA enlargement was found in 27% (15% mild, 4% moderate and 8% severe). All patients had normal systolic function. Left ventricle ejection fraction (LVEF) median was 66.2% and LV global longitudinal deformation was -23%. DD was identified in 27% of patients. A pseudonormal transmitral flow pattern was the most prevalent (15%), followed by slow relaxation in 10% and only 2% with restrictive pattern. 17% of patients presented an increase in LV filling pressures. Regarding LA ST strain analysis, the mean Ɛs/ƐR (reservoir function) was 41.3% with 13% patients displaying lower values. 29.3% and 11% of patients presented alterations in pump and conduit function, respectively. When analyzed by Child stage, no differences were found regarding age, gender, etiology, LVEF or LV global longitudinal deformation. LA volume was progressively larger in relation to Child Pugh stage *(Figure 1*, p=0.001) and a larger

proportion of LA enlargement was observed in more advanced stages (p=0.06). Other LA function parameters did not show significant differences in group analysis. Echocardiographic characteristics are summarized in *Table 1*.

DISCUSSION

Cirrhotic cardiomyopathy is a newly recognized entity in patients with liver cirrhosis of any cause.[2-12] Initially, cardiac changes were attributed to the effects of alcohol, however, in the 1980's,[11,13-15] reports of deaths due to heart failure after liver transplantation, transjugular stent insertion and intrahepatic portosystemic shunts were made. Cirrhotic cardiomyopathy is defined as LV dysfunction (either due impaired contractile response to stress, and/or altered diastolic relaxation) and electrophysiological alterations, in the absence of cardiac disease, in patients with cirrhosis. In this study, we observed a preserved systolic function (both 3D LVEF and LV strain). This data is similar to that reported by Sampaio et al[16] where only 9.2% subjects had LVEF <55%. The absence of LV systolic dysfunction in our population can be explained by two reasons: the measurements used (LVEF-3D and LV strain) and the absence of decompensated subjects, which have been included in other studies. We found LV diastolic dysfunction and LA enlargement in 27% of patients. At this point it is striking that even a large number of patients with advanced clinical stages (Child Pugh B or C) have normal systolic and diastolic function. Even though our study sample was small, we can assume that the majority of patients with cirrhosis do not have cirrhotic cardiomyopathy, which agrees with Sampaio et al., whose population had a 16% prevalence of DD. One of the most recently incorporated measures in echocardiography is the measurement of left atrial function. Sampaio et al reported that reservoir function declines and pump function remains normal in this group of patients. In our study it was observed that reservoir function was only decreased in 13% of patients and pump function was found altered in 29%. This may be due to our greater DD prevalence, which strongly correlates with LA dysfunction. Our study limitations were: (1) its cross-sectional nature, which does not allows to establish causality or prognosis (2) it was performed in a single center with a small sample, (3) cardiac biomarker measurements for

Table 1: Echocardiographic characteristics of the population.

Characteristic	Total (n = 99)	Child A (n = 24)	Child B (n = 43)	Child C (n = 32)
Age in years	50.9 (21-81)	48.5	53	49.9
Male sex	40%	13 (54%)	13 (30%)	16 (50%)
LVEF (3D)	66.2 (56-77)	65.4	66.6	66.3
LV global longitudinal deformation	-23 (-29.8, -17.1)	-22.4	-22.7	-23.9
Increase in LV filling pressures	17%	3 (12%)	6 (14%)	8 (25%)
Diastolic dysfunction	27%	7 (29%)	9 (21%)	11 (35%)
LA volume (mL/m^2)	31.1 (15-61.2)	25.3	31.8	36.7*
LA dilatation	27%	4 (16%)	12 (28%)	11 (35%)
LA reservoir function (εs/εR)	41.3 (19.9-87.8)	39	43.5	40.1
LA reservoir function alteration	13%	4 (16%)	5 (11%)	4 (12%)
LA pump function (εa/εCD)	18 (4.2-51.2)	16.4	19.4	17.3
LA pump function alteration	29%	11 (45%)	10 (23%)	8 (25%)
LA conduit function (εe/εCD)	23.3 (5.9-47.9)	22.6	24	22.8
LA conduit function alteration	11%	3 (12%)	5 (11%)	3 (9%)
E contraction index	43.2 (15.8-79.7)	41.3	44.2	43.4

LVEF = left ventricule ejection fraction; LV = left ventricule; LA = left atrium. * $p < 0.05$.

degree of myocardial dysfunction stratification were not included, (4) not performing a stress test could underestimate our findings and finally (5) invasive measurements to confirm increase in LV filling pressures were not done. In conclusion, our population of cirrhotic patients showed LV diastolic and LA systolic dysfunction. Incorporating new echocardiographic techniques for LA function analysis could help identify cirrhotic patients with a worse prognosis after liver transplantation.

REFERENCES

1. Kim MY, Baik SK, Won CS et al. Dobutamine stress echocardiography for evaluating cirrhotic cardiomyopathy in liver cirrhosis. Korean J Hepatol. 2010; 16 (4): 376-382.
2. Merli M, Calicchia A, Ruffa A et al. Cardiac dysfunction in cirrhosis is not associated with the severity of liver disease. Eur J Intern Med. 2013; 24 (2): 172-176.
3. Wiese S, Hove JD, Muller S. Cardiac imaging in patients with chronic liver disease. Clin Physiol Funct Imaging. 2015: 1-10.
4. Licata A, Novo G, Colomba D et al. Cardiac involvement in patients with cirrhosis: a focus on clinical features and diagnosis. J Cardiovasc Med (Hagerstown). 2016; 17 (1): 26-36.
5. Fede G, Privitera G, Tomaselli T et al. Cardiovascular dysfunction in patients with liver cirrhosis. Ann Gastroenterol Ann Gastroenterol. 2015; 28 (281): 31-40.
6. Milani A, Zaccaria R, Bombardieri G et al. Cirrhotic cardiomyopathy. Dig Liver Dis. 2007; 39 (6): 507-515.
7. Ruiz-Del-Arbol L, Serradilla R. Cirrhotic cardiomyopathy. World J Gastroenterol. 2015; 21 (41): 11502-11521.
8. Zardi EM, Zardi DM, Chin D et al. Cirrhotic cardiomyopathy in the pre and post-liver transplantation phase. J Cardiol. 2016; 67 (2): 125-130.
9. Gassanov N, Caglayan E, Semmo N et al. Cirrhotic cardiomyopathy: a cardiologist's perspective. World J Gastroenterol. 2014; 20 (42): 15492-15498.
10. Rahman S, Mallett SV, Mallett RS. Cirrhotic cardiomyopathy: implications for the perioperative management of liver transplant patients. World J Hepatol. 2015; 7 (3): 507-520.
11. Chayanupatkul M, Liangpunsakul S. Cirrhotic cardiomyopathy: review of pathophysiology and treatment. Hepatol Int. 2014; 8 (3): 308-315.
12. Licata A, Mazzola A, Ingrassia D et al. Clinical implications of the hyperdynamic syndrome in cirrhosis. Eur J Intern Med. 2014; 25 (9): 795-802.
13. Pudil R, Pelouch R, Praus R et al. Heart failure in patients with liver cirrhosis. Cor Vasa. 2013; 55 (4): 391-396.
14. Kazankov K, Holland-Fischer P, Andersen NH et al. Resting myocardial dysfunction in cirrhosis quantified by tissue Doppler imaging. Liver Int. 2011; 31 (4): 534-540.
15. Ripoll C, Yotti R, Bermejo J et al. The heart in liver transplantation. J Hepatol. 2011; 54 (4): 810-822.
16. Sampaio F, Pimenta J, Bettencourt N et al. Left atrial function is impaired in cirrhosis: a speckle tracking echocardiographic study. Hepatol Int. 2014; 8 (1): 146-153.

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Review

Evaluation of inflammatory pericardial syndromes by multimodality imaging: a narrative review

Evaluación de los síndromes pericárdicos inflamatorios mediante imágenes multimodales: una revisión narrativa

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ABSTRACT. Inflammatory pericardial disease is a group of diseases that include acute pericarditis, constrictive pericarditis, pericardial effusion and tamponade. Clinical diagnosis is really a challenge, and multiple imaging tools are needed to complement each other and provide information for diagnosis, treatment and prognosis of diseases. In this narrative review we will discuss the main multimodal imaging techniques (echocardiography, tomography, magnetic resonance and positron emission tomography) and their real applications in the approach of this entity.
Key words: Pericarditis, echocardiography, tomography, magnetic resonance imaging, positron emission tomography.

RESUMEN. *La enfermedad pericárdica inflamatoria es un conjunto de enfermedades que incluyen la pericarditis aguda, pericarditis constrictiva, derrame pericárdico y tamponade. Su adecuado diagnóstico por clínica es realmente un reto, y se necesitan múltiples herramientas de imagen que se complementan y brindan información tanto para el diagnóstico, guiar el tratamiento y pronóstico. En esta revisión narrativa abordaremos las principales técnicas de imagen multimodal (ecocardiograma, tomografía, resonancia magnética y tomografía por emisión de positrones) y sus aplicaciones reales en el abordaje de dicha entidad.*
Palabras clave: *Pericarditis, ecocardiografía, tomografía, resonancia magnética, tomografía emisión positrones.*

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The pericardium is a sac composed of two walls and a virtual space between them, which in normal conditions contains a certain amount of fluid. It surrounds the heart and the great vessels. Its functions, in summary, are fixation, protection and lubrication of the heart. Pericardial syndromes consist of a spectrum of alterations with different pathophysiologies. The most prevalent disease worldwide is acute pericarditis. Despite being a relatively common disease in the cardiology clinic, there are no reliable statistics, due to few epidemiological data. Other disorders included in the pericardial syndromes are chronic pericarditis, constrictive pericarditis, pericardial effusion, cardiac tamponade and pericardial tumors (primary and metastatic). In recent years, the multimodal imaging approach has been standardized and recent management guidelines ultimately recommend it.[1,2] Multimodal imaging includes echocardiogram, computed tomography (CT), cardiac magnetic resonance (CMR) and positron emission tomography (PET). This article will review the uses and indications of each of these diagnostic modalities in the evaluation of inflammatory pericardial syndromes.

ACUTE PERICARDITIS

Acute pericarditis is a multietiological inflammatory pericardial disorder, which can occur as an isolated entity or as part of a systemic disease, it can present with or without a pericardial effusion. Most cases are self-limited, and occasionally, it can turn recurrent or chronic.

Echocardiography: It is recommended to perform an echocardiogram in all patients with suspected pericarditis. Although many patients may have normal results, if pericardial effusion is present, it supports the diagnosis. The amount of effusion may vary between trivial or mild to large, causing the so-called swinging heart. Myocardial ischemia or injury (like myopericarditis) is excluded when no wall motion abnormalities are found. We may also find increased pericardial brightness, thickening of pericardial layers, and in isolated cases, septal bounce, suggestive of constrictive physiology. If fibrinous strands are present, inflammatory etiology should be suspected.

CMR: The pericardium is defined as a hypo intense curvilinear line between the myocardium and the pericardial fat in cine sequences. It is best visualized in the middle third of the free wall of the right ventricle. In acute pericarditis, an increase in the thickness (>2.5mm) and intensity of the pericardium is usually observed. Findings that might be associated with acute inflammation are: 1) increased signal in the pericardium in T2 weighted sequences (edema), 2) post gadolonium enhancement in early cine sequences, and 3) late enhancement *(Figure 1)*.[3,4] Besides its clinical use for diagnosis, CMR has a prognostic value for two reasons: a) diagnosing coexistence of myocarditis, and b) a greater extension of late enhancement, suggests a greater recurrence at six months.[5] Both findings modify the subsequent treatment.

CT: This technique can easily visualize the pericardium. Like CMR, thickness >2.5mm suggest acute pericarditis. In addition, the existence of irregular contours can be observed. In the context of patients with acute chest pain and intermediate risk of coronary artery disease, the diagnosis of pericarditis remains exclusionary, so CT may be the ideal initial approach.

PET: Because it allows direct visualization of the inflammatory activity; PET/CT image using [18F] FDG allows us to use it for diagnosis, risk stratification and therapeutic monitoring of pericarditis.[6]

CONSTRICTIVE PERICARDITIS

Constrictive pericarditis can occur after any pericardial disease process, mostly inflammatory; and by consequence the pericardium thickens, scars, stiffens and even calcifies, turning it into a non-compliant heart, and ultimately limiting its diastolic filling. It is frequently underdiagnosed or confused with restrictive cardiomyopathy due to similar clinical findings. Time from initial pericardial injury up to onset of constriction is variable, specific risk of progression depends on etiology, it rarely occurs after viral or idiopathic pericarditis but is relatively common in bacterial pericarditis, mostly with purulent pericarditis. Tuberculosis is highly related with this process in underdeveloped countries like Mexico. Diagnosis is suggested when data of right heart failure is present along with preserved biventricular function. The physiopathology of constrictive pericarditis is explained as diastolic filling is restricted by an inelastic, non-compliant pericardium after an initial expansion of the myocardium; early diastolic filling is more rapid than normal because the restraining effect happens until mid-diastole, limiting ventricular filling, even with atrial contraction. As constriction becomes deeper, total diastolic volume is

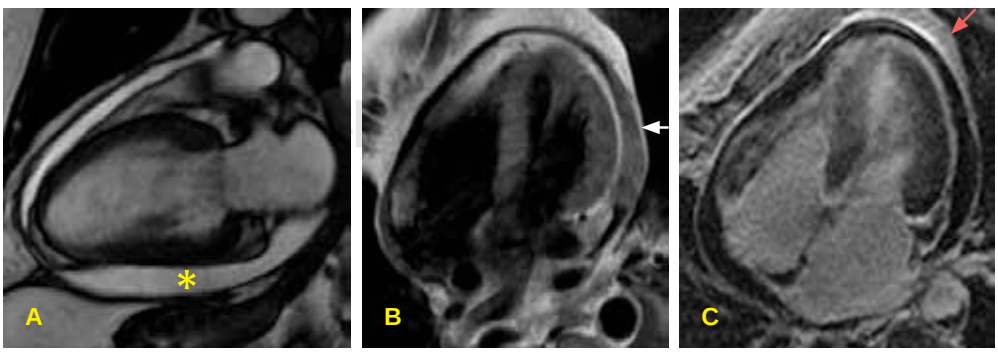

Figure 1: CMR of acute pericarditis. **A.** Cine imaging showing pericardial effusion (*) and thickness of pericardium. **B.** T2w imaging showing increased signal in the pericardium (white arrow), consistent with edema and acute inflammation. **C.** Late gadolinium enhancement of the pericardium (red arrow).

reduced, causing a decrease in cardiac output, and, at the same time, systemic venous pressures increase resulting in blood return to the adjacent inferior vena cava and hepatic veins. As seen with cardiac tamponade, ventricular interdependence becomes exaggerated, manifested as an inspiratory bulging of the interventricular septum to the left, having an additive effect on decreasing the cardiac output during inspiration.

Echocardiography: It is cost effective in these cases. All the different techniques used in echocardiography allow us to differentiate between restrictive cardiomyopathy, constrictive pericarditis and other conditions. It should be noticed that there are not absolute sensitive or specific echocardiographic indicators of constriction, therefore, all data found must be correlated.

With 2D a M modes we can find pericardial thickening and calcification, but it is not mandatory; increased pericardial thickness is suggested by parallel motion of the visceral and parietal pericardium, separated by a relatively echo-free space. If effusion is also present, it makes easier to appreciate the thickened pericardium (>3mm). Shadowing below the pericardium suggests the presence of calcification. The augmented ventricular interdependence appears as exaggerated septal shifts according to the respiratory cycle, or as an abrupt posterior motion of the interventricular septum in early diastole with inspiration (bounce), most evident with M-mode rather than 2D. Also, a diastolic endocardial flattening of the posterior wall endocardium can be present. Imaging of the inferior vena cava shows dilation and a decrease, or even absence, of collapse.

Doppler echocardiography is essential. The classic Doppler finding is an augmented E/A ratio of mitral valve inflow with a short deceleration time, as seen in any disease with restrictive or constrictive physiology, but what makes the difference and establishes the diagnosis is an exaggerated respiratory variability in E-wave velocity (a drop >25% in the first beat after inspiration) with variation >20% in pulmonary vein diastolic forward flow. The propagation velocity of early diastolic trans mitral flow on color M-mode is usually increased as often as >100cm/s.

Doppler tissue imaging is particularly useful. Normally, the lateral mitral annular velocity (e') exceeds the one of the medial annulus in a ratio >1.2, but with constriction, stiff pericardium limits the motion of the lateral annulus with no effect on the medial annulus. There is therefore both preservation of medial velocities and a compensatory augmentation of apex to base shortening of the septum relative to the lateral wall. This increases the medial annulus velocity (>7-8cm/s) situation called «*annulus paradoxus*» and therefore shifting the ratio <1.2 («annulus reverses») which has been documented as a reliable sign of constrictive pericarditis.

Novel techniques in echocardiography are promising in this particular scenario and may help distinguishing between constrictive pericarditis and restrictive cardiomyopathy. Circumferential strain, torsion, and early diastolic untwisting reduce, as global longitudinal strain has no change in constriction, whereas longitudinal strain reduces in restrictive cardiomyopathy. Speckle-tracking techniques have shown significant differences between the strain of the right or left ventricle free walls and the septum.

CMR: Calcifications can be observed as an increase in signal intensity in the pericardium. The thickening of the pericardium is suggestive of constriction when it exceeds 4mm, with sensitivity of 88% and specificity of 100%.[7] The abnormal septal motion, which is the septal protodiastolic flattening, showed sensitivity of 81% and specificity of 100% for the diagnosis of contrition.[8] Moderate to intense delayed enhancement (affecting the entire pericardium) has been associated with a greater proportion of progression to constriction.[9] Speaking of findings of functional constriction, CMR can evaluate the respirophasic changes in real-time sequences, observing the septal excursion to the left ventricle at the beginning of inspiration, and trans mitral and trans tricuspid flows can be quantified simultaneously, with cut-off points of its respiratory variation of more than 25% for the mitral (area under curve [AUC]: 1.0) and more than 45% for the tricuspid (AUC: 0.98).[10] Other indirect constriction data include dilation of the inferior vena cava, suprahepatic veins, right atrium, as well as the coexistence of splenomegaly, ascites and pleural effusion.

CT: Is the ideal method to observe pericardial calcifications, either partial or total. Ring calcifications are also observed as a cause

of constriction. Like the CMR, the pericardial thickness >4mm is highly suggestive, with sensitivity of 86% and specificity of 75%. Dilation of the inferior vena cava with a VCI-aortic radius >1.6 showed sensitivity of 86% and specificity of 75% for this diagnosis.[11]

PET: Inflammation of the pericardium can reach a degree that limits the diastolic ventricular filling, with subsequent fibrosis also known as constrictive pericarditis. The importance lies in the natural evolution of acute pericarditis, since, if detected in early stages of inflammation, the natural progression can be stopped, and therefore avoid fibrosis (transient pericarditis). In a study by Chang et al[12] the use of [18F] FDG in PET was hypothesized to predict the reversibility of transient pericarditis with the use of steroids. It was found that after three months of steroid treatment, the standardized maximal uptake value (SUVmax) was reduced compared to the baseline parameters (2.1±0.8, p<0.001). All patients who responded to treatment had SUVmax >3. This determined that if we use a SUVmax cutoff point of 3.0, we will obtain sensitivity of 100%, specificity of 71% to predict treatment response. This would allow us to avoid surgeries and unnecessary steroid treatments with a very high level of certainty *(Figure 2)*.

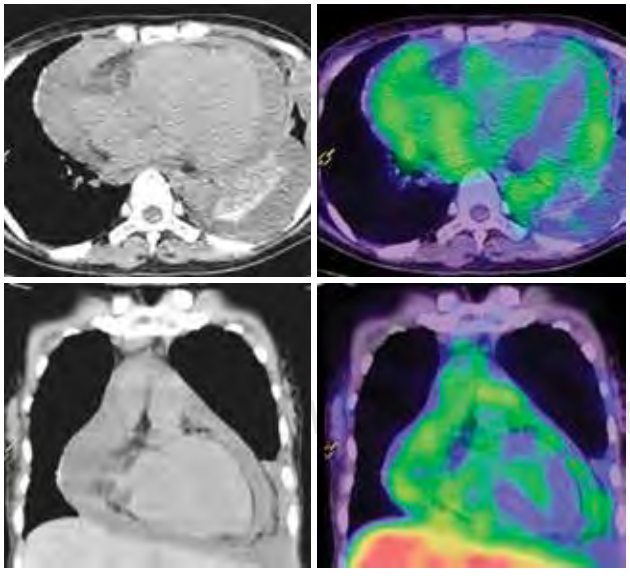

Figure 2: PET of a pericardial effusion with hypermetabolic activity of the fluid, consistent with an exudate.

PERICARDIAL EFFUSION

It is defined as an accumulation of >50mL of fluid inside the pericardial space. As any other pericardial disease it is multietiological. It can be formed either as an increase of production of pericardial fluid, causing an exudate, or by a decrease in the reabsorption, usually generated by an increment of the venous pressure and therefore forming a transudate. Blood in pericardial space may be related with trauma, aortic dissection or with cardiac procedures.

Echocardiography: M and 2D modes show pericardial effusion like an echo free space between both layers of pericardium throughout the cardiac cycle. Using all possible views reliably help to determine the extent of effusion. Left pleural effusion may confused with pericardial effusion; in a 2D parasternal long axis view, if there is fluid between descending aorta and the heart, it should be considered of pericardial origin rather than pleural. 3D echo has shown no difference in this particular case. It's important to note that a specific amount of effusion can't accurately be measured with echocardiography, since there are multiple variations between the effusion and the irregular shape of the heart, and because the possibility of loculations, etc. With echocardiographic data, effusions can be classified according to hemodynamic repercussion (none, mild, severe-cardiac tamponade) and size, measuring the separation between visceral and parietal pericardial layers (mild effusions <10mm, moderate 10-20mm, and large effusions >20mm). When we find an effusion and the heart seems to be dancing in the pericardial space («swinging heart») it is by definition a large effusion. Size also may be an etiological clue, in which small effusions are related with pericarditis while large ones are related with neoplasia or TB. It should be noted that size does not correlate with hemodynamic repercussion, since small increases in an acute onset increase intrapericardial pressure in a short amount of time deriving in hemodynamic instability (cardiac tamponade), whereas, slow increases of fluid elevate the pericardial pressure in the same velocity, allowing cardiac compensatory mechanisms to counteract the hemodynamical effects of the amount of effusion.

CMR and CT: In addition to the uses already described in acute and constrictive pericarditis, CMR and CT contribute little to the approach of these patients. Its use is limited when echocardiography is not diagnostic or in cases where it is necessary to evaluate structures close to the heart, such as trauma or neoplasms *(Figures 3 and 4)*. CT can also suggest the etiology of the effusion, especially when the density is >30 HU, which is very suggestive of hemorrhagic etiology. In cases of significant pericardial effusion, these methods can better visualize the extent of the effusion to guide the pericardiocentesis.

CARDIAC TAMPONADE

Cardiac tamponade is a life threatening condition, with a slow or rapid onset, caused by an accumulation of fluid in the pericardial sac due to inflammation, trauma, rupture of myocardial structures, cardiac devices and procedures, etc., resulting in compression of cardiac chambers and abnormal filling. Considering the pericardial stiffness, as the pressure in the pericardial sac increases due to the effusion, cardiac chambers are compressed and become smaller, reducing diastolic compliance and decreasing systemic venous return. Normally, during inspiration systemic venous return increases distending the right ventricle, however, in tamponade, increased intrapericardial pressure limits this distension and is therefore transmitted to the interventricular septum bulging it to the left, reducing left ventricle compliance and therefore compromising its filling, which at the end results in a decrease of preload, and therefore, of cardiac output and subsequently in hypotension. Ergo, increased intrapericardial pressure and limitation of cardiac volumes causes an exaggerated ventricular interdependence, the physiological basis of clinical pulsus paradoxus. It should be noted that tamponade is the «last drop» of the phenomena of increasing intrapericardial pressure, and it is related with the exhaustion of cardiac compensatory mechanisms. The use of echocardiography is a class I indication in cardiac tamponade and can be also used in guiding pericardiocentesis increasing safety and efficacy.

Even though cardiac tamponade is a clinical diagnosis, there are several helpful echocardiographic findings. Not all of them have to be present to suggest hemodynamic compromise. We must aim to demonstrate an effusion that ultimately causes reduced cardiac output, elevated central venous pressure and chamber collapse. Special considerations must be taken in patients with significant left or right ventricle hypertrophy, situations that mask typical findings. First of all, a pericardial effusion should be present and not necessarily has to be a large one; small but of rapid onset may compromise patients stability further more than those with a slow onset with large pericardial effusion. With M and 2D modes we can find diastolic collapse of the right atrium and ventricle when intrapericardial pressure exceeds intracavitary pressures. Duration of

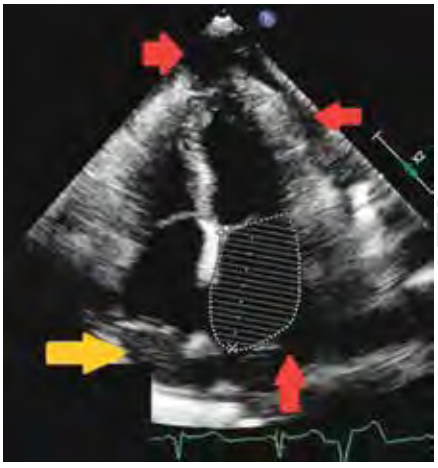

Figure 3:

Female with a global pericardial effusion (red arrows) with no hemodynamic compromise, and an incidental finding of a mass inside the pericardial sac (yellow arrow) which turned to be a malignant mesotelyoma.

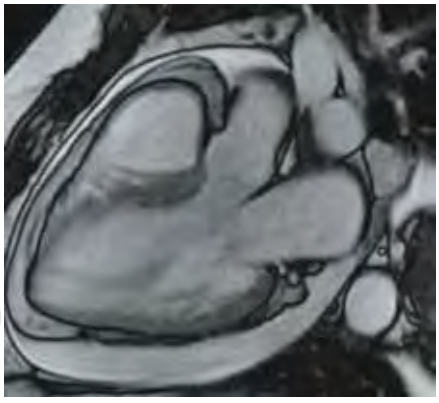

Figure 4: CMR of a patient with asymptomatic pericardial effusion without hemodynamic compromise. Note the normal pericardium thickness and a mass next to left atrium (red arrow).

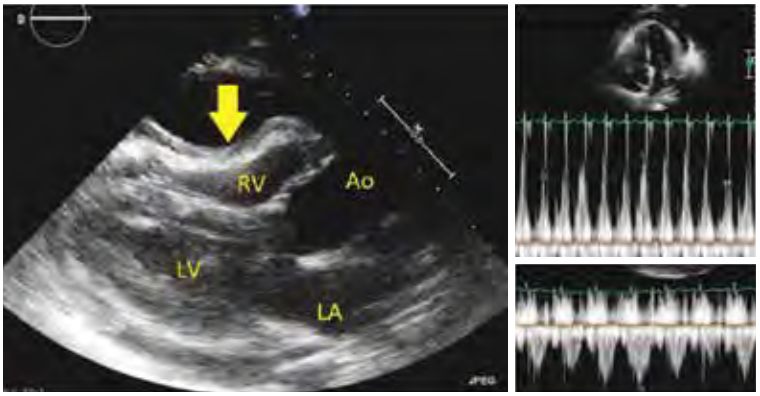

Figure 5: Young male with relapsing pericarditis who presented to the ER with hypotension and tachycardia, pulsus paradoxus, among other clinical manifestations. Echocardiogram performed in the ER, showed diastolic collapse of the right cavities (yellow arrow) and significant respiratory variation in both aortic outflow (down right) a mitral inflow velocities (upper right), echocardiographic findings suggestive of cardiac tamponade. Echo guided pericardiocentesis was performed and a few moments after decompressing the pericardium, patient became hemodynamic stable.

right atrium collapse more than one third of the cardiac cycle is very sensitive and specific. During inspiration we can see bulging to the left of the interventricular septum. This causes a variation of aortic outflow (echocardiographic demonstration of pulsus paradoxus) than can be assessed with Doppler in the left ventricle outflow tract. Also with Doppler we can document exaggerated phasic respiratory variations both in mitral and tricuspid inflow. An exaggerated respiratory variability (>25%) in mitral inflow velocity might be seen, with inspiratory decrease and expiratory increase, also seen in pulmonary vein diastolic forward flow. The opposite happens in the right heart. It can also be appreciated a respiratory variation in ventricular chamber size. Inferior vena cava dilatation with <50% reduction in diameter during inspiration is another very sensitive but poor sensible finding, and with Doppler we can find that its flow into the right atrium becomes predominantly during systole *(Figure 5)*. The use of other imaging techniques in this scenario is exceptional and is not recommended.

As seen above, there is a multimodality diagnostic, and even prognostic approach to evaluate pericardial disease. We must confidently rely in each of them to increase each of their diagnostic value, and as mentioned, before, one must make use of a combination of modalities to increase the diagnostic power of imaging in pericardial disease. As with echocardiographic findings alone, all data found in the different imaging modalities must be correlated.

REFERENCES

1. Cosyns B, Plein S, Nihoyanopoulos P et al. European Association of Cardiovascular Imaging (EACVI) position paper: multimodality imaging in pericardial disease. Eur Heart J Cardiovasc Imaging. 2014; 16: 12-31.
2. Klein AL, Abbara S, Agler DA et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr. 2013; 26 (9): 965-1012.e15.
3. Yared K, Baggish AL, Picard MH et al. Multimodality imaging of pericardial diseases. J Am Coll Cardiol Img. 2010; 3: 650-660.
4. Wang ZJ, Reddy GP, Gotway MB et al. CT and MR imaging of pericardial disease. Radiographics. 2003;23:S167-S180.
5. Kumar A, Sato K, Yzeiraj E et al. Quantitative pericardial delayed hyperenhancement informs clinical course in recurrent pericarditis. Available in: http://dx.doi.org/10.1016/j.jcmg.2016.10.020
6. Treglia G et al. A 'Ring of Fire' around the heart: pericarditis detected by FDG-PET/CT. Cardiovascular Medicine, EMH Media, 19 Sept. 2018. Available in: cardiovascmed.ch/en/article/doi/cvm.2018.00576/
7. Masui T, Finck S, Higgins CB. Constrictive pericarditis and restrictive cardiomyopathy: evaluation with MR imaging. Radiology. 1992; 182: 369-373.
8. Giorgi B, Mollet N, Dymarkowski S et al. Clinically suspected constrictive pericarditis: MR imaging assessment of ventricular septal motion and configuration in patients and healthy subjects. Radiology. 2003; 228: 417-424.
9. Feng D, Glockner J, Kim K et al. Cardiac magnetic resonance imaging pericardial late gadolinium enhancement and elevated inflammatory markers can predict the reversibility of constrictive pericarditis after antiinflammatory medical therapy a pilot study. Circulation. 2011; 124: 1830-1837.
10. Thavendiranathan P, Verhaert D, Walls MC et al. Simultaneous right and left heart real-time, free-breathing cmr flow quantification identifies constrictive physiology. J Am Coll Cardiol Img. 2012; 5: 15-24.
11. Hanneman K, Thavendiranathan P, Nguyen ET et al. Cardiovascular CT in the diagnosis of pericardial constriction: predictive value of inferior vena cava cross-sectional area. J Cardiovasc Comput Tomogr. 2014; 8 (2): 149-157.
12. Chang SA et al. [18F] Fluorodeoxyglucose PET/CT predicts response to steroid therapy in constrictive pericarditis. J Am Coll Cardiol. 2017; 69 (6): 750-751.

No conflict of interest to declare.

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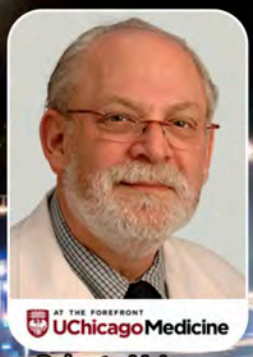

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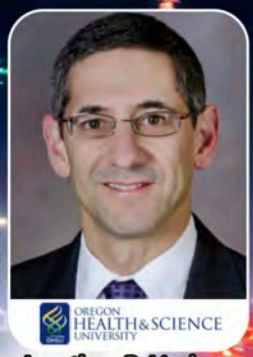

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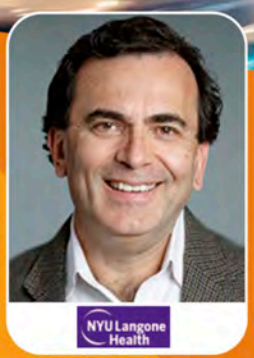

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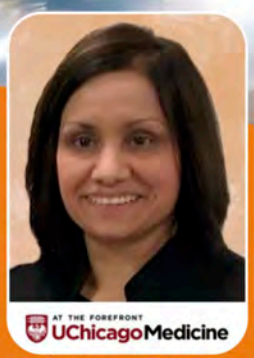

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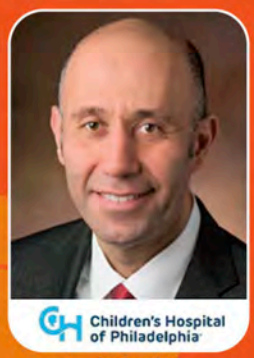

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