ORIGINAL ARTICLE

Right Ventricle: Echocardiographic Evaluation of Pressure and Volume Overload

Ventrículo derecho: evaluación ecocardiográfica de las sobrecargas de presión y de volumen

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ABSTRACT

Background: New echocardiographic techniques have renewed the interest in the assessment of right ventricular function; however, there are still not clearly defined reference values or large-scale analyses on the behavior of this chamber when submitted to pressure (RVPO) or volume (RVVO) overload.
Objective: The aim of this study was to analyze echocardiographic abnormalities and right ventricular longitudinal and transverse strain in patients with RVPO and RVVO and in healthy subjects (CTRL).
Methods: Fifty CTRL subjects (mean age 47±18 years, 31 females), 50 RVPO patients (mean age 48±12 years, 32 females) and 30 RVVO patients (mean age 43±14 years, 19 females) were included in the study. Left ventricular, left atrial, right ventricular and right atrial dimensions, and left and right ventricular function were analyzed. Two-dimensional left and right ventricular longitudinal strain and right ventricular transverse strain were evaluated. Analysis of variance was used to compare means and standard deviations.
Results: Demographic data, left ventricular ejection fraction and adjusted left atrial volume showed no significant differences among groups. Diameters, right ventricular wall thickness, tricuspid regurgitation gradient and adjusted right atrial volume were higher in RVPO patients. Right ventricular function parameters were decreased in RVPO and normal in RVVO patients. The right ventricular lateral transverse strain was increased both in RVPO and RVVO.
Conclusions: Right ventricular echocardiographic parameters and strain showed signs of remodeling, both in RVPO and RVVO, and decreased systolic function in RVPO. Increased right ventricular transverse strain would probably be due to remodeling, and decreased longitudinal strain to systolic dysfunction. Decreased left ventricular longitudinal strain may indicate early biventricular dysfunction.

Key words: Echocardiography - Two-dimensional Strain - Pulmonary Hypertension - Right Ventricle

RESUMEN

Introducción: Con las nuevas técnicas ecocardiográficas se ha renovado el interés en la evaluación de la función del ventrículo derecho (VD); no obstante, aún no se cuenta con valores de referencia definidos ni análisis a gran escala sobre el comportamiento de esta cavidad cuando es sometida a sobrecarga de presión (SPVD) y de volumen (SVVD).
Objetivo: Analizar las alteraciones ecocardiográficas y el strain longitudinal y transversal del VD en pacientes con SPVD y con SVVD y en individuos saludables (CTRL).
Material y métodos: En 50 pacientes CTRL (edad 47 ± 18 años; 31 femeninos), 50 pacientes con SPVD (edad 48 ± 12 años; 32 femeninos) y 30 pacientes con SVVD (edad 43 ± 14 años; 19 femeninos) se analizaron las dimensiones del ventrículo izquierdo (VI), de la aurícula izquierda (AI), del VD y de la aurícula derecha (AD), la función del VI y del VD, el strain bidimensional longitudinal del VI y del VD y transversal del VD. Las medias y desviaciones estándar se compararon mediante el análisis de la varianza.
Resultados: Los datos demográficos no mostraron diferencias significativas entre los grupos, como tampoco la fracción de eyección del VI y el volumen indexado de la AI. En la SPVD, los diámetros, el espesor de la pared del VD, el gradiente tricuspídeo y el volumen indexado de la AD fueron mayores. Los parámetros de función del VD estaban disminuidos en la SPVD y normales en la SVVD. El strain transversal lateral del VD se encontraba aumentado en la SPVD y en la SVVD.
Conclusiones: Los parámetros ecocardiográficos y el strain del VD mostraron señales de remodelación en la SPVD y en la SVVD y disminución de la función sistólica en la SPVD. El aumento del strain transversal del VD se debería a la remodelación y la disminución del strain longitudinal, a la disfunción sistólica. La disminución del strain longitudinal del VI puede indicar precozmente la disfunción biventricular.

Palabras clave: Ecocardiografía - Strain bidimensional - Hipertensión pulmonar - Ventrículo derecho

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Abbreviations

ASD	Atrial septal defect	**MRI**	Magnetic resonance imaging
CT	Computed tomography	**PHT**	Pulmonary hypertension
CTRL	Control group	**RV**	Right ventricle
LA	Left atrium	**RVPO**	Right ventricular pressure overload
LV	Left ventricle	**RVVO**	Right ventricular volume overload
RA	Right atrium	**TAPSE**	Tricuspid annulus plane systolic excursion

INTRODUCTION

In his brilliant description of the circulatory system, Sir William Harvey defined, in 1628, the right ventricle (RV) as an "organ intended to eject blood to the lungs", contradicting Galen, who had described this cavity as a mere duct for the passage of blood. (1) The analysis of this ventricular chamber was postponed until the end of the last century due, among other reasons, to its irregular geometry hampering volume calculation, and hence, ejection fraction.

The emergence of imaging methods such as angiocardiography, computed tomography (CT) scan and magnetic resonance imaging (MRI) raised great interest in the RV in recent years. The anatomical evidences of the helical heart demonstrated by Torrent Guasp in 1980 (2) and the investigations of LeGrice on the laminar myocardial structure, (3) demonstrating the mechanism of torsion, increased the anatomo-physiological basis needed for these studies.

The first methods able to measure myocardial wall deformation appeared in the 90s. The technique, named "cardiac strain" was applied to the analysis of the left ventricle (LV) by MRI and echocardiography, using tissue Doppler, which was greatly influenced by the angle of insonation, delaying the analysis of the RV. (4) The two-dimensional strain technique (speckle tracking), relatively independent of the angle of insonation and three-dimensional echocardiography, renewed the interest for the RV. (5, 6)

Having established left ventricular deformation parameters, (7) studies on right ventricular deformation are currently performed. However, although there are still not clearly defined reference values or a large scale analysis on the behavior of this chamber when submitted to pressure or volume overload, (8) its use is recommended in the most recent guidelines. (9)

The present work analyzed right ventricular deformation vectors in healthy subjects, and in patients with pressure overload [pulmonary hypertension (PHT)] or volume overload [atrial septal defect (ASD)].

Morphological-functional analysis of the right ventricle

Studies on myocardial architecture obtained with diffusion tensor magnetic resonance imaging validated the anatomical model of the single double helical ventricular myocardial band described by Torrent-Guasp, recurrent in the left ventricular apex and anchored at one end in the pulmonary annulus and at the other in the aortic annulus. (10). The basal band, anchored in the pulmonary annulus, with predominantly circular fibers, can be seen enveloping the ventricles, with a slightly oblique direction towards the apex. In the left ventricular anterior superior border, these fibers bend towards the interior of the chamber forming the descending band, which extends from the septal endocardium to the whole left ventricular subendocardial region. This band turns in the apex forming the ascending band, with more disordered fibers, some mainly ascending on the right side of the interventricular septum and on the subepicardium of the left ventricular free wall to anchor in the aortic annulus. Other fibers are directed to the right to form part of the right ventricular anterior wall and others form the right ventricular outflow tract. (11) The descending and ascending bands cross at an angle of 70-80 degrees, an angulation responsible for left ventricular torsion and counter-torsion. The thinner right ventricular free wall is formed by circular fibers originating in the basal band, internal fibers derived from the descending band and some external, oblique fibers, arising from the ascending band, which cross the anterior interventricular sulcus, called aberrant fibers. There is practically no angulation between the descending and ascending fibers, and hence no torsion mechanism. The interventricular septum, with more complex architecture, has fibers with different orientation in the right and left side. (12) The oblique, anterior posterior fibers of the left septum correspond to the descending band. The posterior ascending fibers of the right septum are derived from the descending band and the anterior fibers from the ascending band, contributing to form the right ventricular outflow tract. (13) Due to these characteristics, the RV has mainly longitudinal deformation. Transverse deformation, in a radial sense, is less evident due to a lower number of circular fibers.

The sequential depolarization of the myocardial band has anisotropic characteristics, with almost simultaneous activation of the right and left side of the basal band through the bundle of His branches. (14) The activation of the basal band is responsible for the pre-ejective, isovolumic period. The activation of the descending band and immediately of the ascending band produces the torsion mechanism. The progress of the ascending band depolarization with descending band relaxation originates the counter-torsion

mechanism, creating a negative pressure gradient between the base and the apex of the LV, forming the post-ejective (isovolumic) and fast ventricular filling periods. The negative gradient sucks blood from the left atrium (LA). During diastasis, myofibrils return to their initial state, completing the cycle with atrial contraction.

The interventricular septum does not have a predominant side, becoming the central structure between both ventricular chambers, characterized by the same type of fibers forming the left ventricular free wall. (15)

The aim of this prospective, observational, non-randomized study was to evaluate the right ventricular free wall and interventricular septum myocardial deformation or strain pattern in healthy subjects and in patients with right ventricular pressure overload (RVPO) [pulmonary hypertension (PHT)] and right ventricular volume overload (RVVO) [atrial sepal defect (ASD)].

METHODS

Three study groups were formed:

- CTRL group: Control group formed by 50 healthy subjects (31 females), with mean age of 47±18 years (range between 17 and 70 years).
- RVPO group: Group formed by 50 patients (32 females) with RVPO, with mean age of 48±12 years (range between 21 and 66 years). These patients had chronic PHT due to schistosomiasis by S. mansoni.
- RVVO group: Group formed by 30 patients (19 females) with RVVO, with mean age of 43±14 years (range between 23 and 65 years). These patients had ASD with moderate to severe left to right shunt, (16) without evidence of major PHT, as seen from the analysis of tricuspid regurgitation velocity and the inferior vena cava diameter. Twenty-six patients had ostium secundum ASD and four sinus venosus ASD.

Left ventricular dimensions, wall thickness, ejection fraction (Simpson method) and left atrial dimensions were assessed with conventional echocardiography. Right ventricular inflow dimensions, the percent area changes, tricuspid annular plane systolic excursion (TAPSE), and systolic wave velocity (s') of the lateral tricuspid annulus were assessed with tissue Doppler.

Global left ventricular longitudinal strain, right ventricular lateral wall longitudinal strain, right ventricular septal longitudinal strain and right ventricular free wall and septal transverse (radial) strain were analyzed with two-dimensional strain. The septum was measured including the entire wall thickness (Figure 1). In all cases, studies with more than two non-measurable segments by speckle tracking were excluded.

Studies were performed with HD15, CX50 and IE33 (Philips Healthcare, Andover, MA) ultrasound systems and the digitized images were analyzed with Qlab 9.0™ software.

Statistical analysis

The data were expressed as mean and standard deviation and were compared using ANOVA followed by Tukey post-hoc test. A p value <0.05 was considered statistically significant.

Ethical considerations

This was a research project registered in the Postgraduate and Research Information System of the University of Pernambuco, Brazil on November 27, 2013, authorized by the Research Ethics Committee of the HUOC/PROCAPE Hospital Complex under number 5192, following the recommendations of the Declaration of Helsinki.

RESULTS

Age, weight, height and body surface area showed no significant differences among CTRL, RVPO and RVVO groups. Left ventricular diameters were significantly larger in the CTRL group, without significant difference in the left ventricular free wall thickness. Septal thickness was larger in the RVPO group and mass and the mass index were significantly lower in the RVVO group. Left ventricular ejection fraction did not show significant differences among groups. These results are detailed in Table 1.

No significant differences among groups were found for left atrial volume adjusted by body surface area.

The right ventricular free wall thickness was significantly increased in RVPO while diameters were increased in both RVPO and RVVO groups. The variation in right ventricular areas was decreased in the RVPO group, but was normal in the RVVO group.

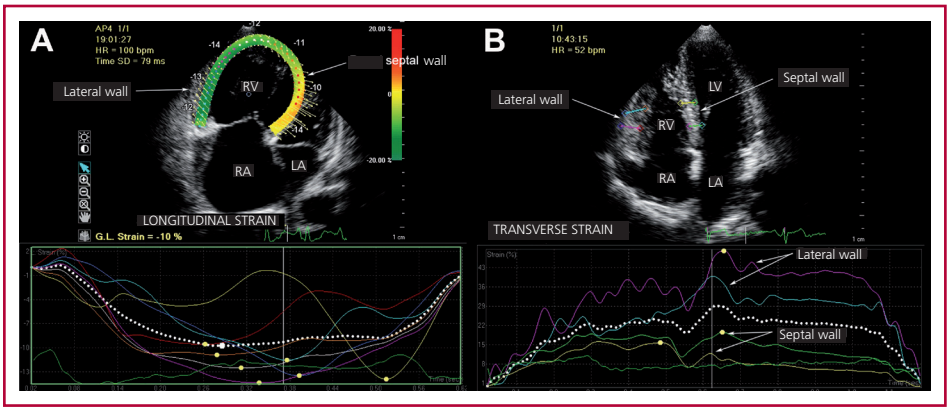

Fig. 1. A: Right ventricular longitudinal strain. Lateral wall strain is calculated as the average strain of the four lateral segments and septal wall strain is calculated as the average strain of septal segments. **B:** Right ventricular transverse strain. Lateral and septal strains are obtained as the average of the corresponding segments. RV: Right ventricle. RA: Right atrium. LV: left ventricle. LA: left atrium.

Tricuspid annular plane systolic excursion was lower in RVPO but still within normal limits. Systolic wave velocity (s') of the lateral tricuspid annulus obtained with tissue Doppler was decreased only in the RVPO group. Pulmonary artery diameter and systolic gradient through the tricuspid valve were larger only in the RVPO group, but the right atrial (RA) volume adjusted by body surface area was increased in RVPO and RVVO groups (Tables 2 and 3). Mean left to right shunt due to ASD, estimated as the ratio between pulmonary output and systemic output (Qp/Qs) was 2.1±0.4 in the RVVO group, with range between 1.5 and 2.7.

Global left ventricular longitudinal strain was significantly decreased in the RVPO group, but normal in RVVO. Right ventricular lateral, septal, and global longitudinal strain was significantly decreased in RVPO, while no differences were found between RVVO and CTRL groups. Lateral right ventricular transverse strain was significantly increased in RVPO and RVVO groups, whereas septal transverse strain was increased only in RVVO (Table 4).

DISCUSSION

Despite belonging to different groups, age, weight, height and body surface area presented no statistically significant differences between patients and healthy control subjects.

Differences in left ventricular dimensions between patients and control subjects were probably due to

Table 1. Left ventricular dimensions, mass and function in CTRL, RVPO and RVVO groups

Group		LVDd (mm)	LVDs (mm)	SThd (mm)	WThd (mm)	Mass (g)	Mass index (g/m²)	Relative thickness	EF (%)
CTRL	M	49.4	32.0	8.3	8.1	145.0	83.5	0.33	58.7
	SD	3.8	2.9	1.8	1.4	39.9	24.4	0.07	5.1
RVPO	M	44.5	27.0	9.0	7.8	126.5	75.1	0.38	60.9
	SD	6.2	5.6	1.4	0.9	39.4	19.3	0.06	6.3
RVVO	M	42.9	27.0	7.6	7.4	105.2	59.6	0.35	64.4
	SD	5.9	5.4	1.5	1.4	43.6	23.7	0.05	7.9
p		0.001	0.001	0,09	0.15	0.01	0.006	0.004	0.26
Tukey	1-2	<0.01	<0.01	ns	ns	ns	ns	<0.01	ns
	1-3	<0.01	<0.05	ns	ns	<0.05	<0.01	ns	ns
	2-3	ns	ns	<0.05	ns	ns	ns	ns	ns

CTRL: Control group. RVPO: Right ventricular pressure overload group. RVVO: Right ventricular volume overload group. LVDd: Left ventricular diastolic diameter. LVDs: Left ventricular systolic diameter. SThd: Diastolic septal thickness. WThd: Diastolic wall thickness. EF: Ejection fraction. M: mean. SD: Standard deviation. p: p value. Tukey: Tukey test. ns: Non-significant.

Table 2. Right ventricular dimensions and thickness, pulmonary artery dimension and right ventricular volume adjusted by body surface area in CTRL, RVPO and RVVO groups

Group		RV1 (mm)	RV2 (mm)	RV3 (mm)	RVDD (mm)	PAD (mm)	RA/BSA volume (g/m²)
CTRL	M	32.5	25.9	68.7	5.1	21.4	26.7
	SD	4.4	3.9	7.6	0.6	4.1	4.6
RVPO	M	43.7	39.6	78.8	9.2	41.0	58.0
	SD	5.8	8.2	8.7	1.9	10,5	34.9
RVVO	M	39.7	33.2	70.6	5.4	26.7	42.3
	SD	6.6	6.6	11.6	0.7	5.1	24.7
p		<0.0001	<0.0001	0.0001	<0.0001	<0.0001	0.009
Tukey	1-2	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
	1-3	<0.01	<0.05	ns	ns	ns	<0.01
	2-3	<0.05	<0.01	<0.01	<0.01	<0.01	ns

CTRL: Control group. RVPO: Right ventricular pressure overload group. RVVO: Right ventricular volume overload group. RV1: Right ventricular basal diameter. RV2: Right ventricular medial diameter. RV3: Right ventricular longitudinal diameter. RVDD: Right ventricular free wall diastolic diameter. PAD: Pulmonary artery diameter. RA/BSA: Right atrium/body surface area. M: mean. SD: Standard deviation. p: p value. Tukey: Tukey test. ns: Non-significant.

Table 3. RRight ventricular area changes, TAPSE, s' wave velocity of the lateral tricuspid annulus and tricuspid regurgitation gradient in CTRL, RVPO and RVVO groups

Group		RV area changes (%)	TAPSE (cm)	s' wave velocity of the lateral tricuspid annulus (cm/s)	Tricuspid regurgitation gradient (mmHg)
CTRL	M	46.6	2.2	15.4	28.0
	SD	4.6	0.4	1.7	3.1
RVPO	M	28.7	1.9	10.3	75.9
	SD	9.7	0.4	2.2	19.6
RVVO	M	47.6	2.1	15.2	37.8
	SD	12,7	0.6	2.3	15.8
p		<0.0001	0.01	<0.0001	<0.0001
Tukey	1-2	<0.01	<0.05	<0.01	<0.01
	1-3	ns	ns	ns	ns
	2-3	<0.01	ns	<0.01	<0.01

CTRL: Control group. RVPO: Right ventricular pressure overload group. RVVO: Right ventricular volume overload group. TAPSE: tricuspid annular plane systolic excursion. M: mean. SD: Standard deviation. p: p value. Tukey: Tukey test. ns: Non-significant.

Table 4. Left and right ventricular myocardial strain

Group		LVLSglob (%)	RVLSglob (%)	RVLSsep (%)	RVLSlat (%)	RVTSglob (%)	RVTSsep (%)	RVTSlat (%)
CTRL	M	-18.2	-18.4	-20.0	-37.0	14.6	16.2	20.0
	SD	1.0	3.3	7.3	12.4	3.2	4.1	5.6
RVPO	M	-14.5	-15.0	-14.8	-22.1	25.9	18.4	33.0
	SD	3.6	4.8	5.6	10.2	15.0	10.6	24.9
RVVO	M	-18.1	-19.5	-19.6	-26.1	22.4	21.5	32.7
	SD	4.1	8.4	7.1	11.5	6.0	7.8	10.0
p		<0.0001	0.002	0.004	<0.0001	0.002	0.09	0.03
Tukey	1-2	<0.05	<0.05	<0.01	<0.01	<0.01	ns	<0.05
	1-3	ns	ns	ns	<0.01	ns	<0.05	<0.05
	2-3	<0.01	<0.05	<0,05	ns	ns	ns	ns

CTRL: Control group. RVPO: Right ventricular pressure overload group. RVVO: Right ventricular volume overload group. LVLSglob: Global left ventricular longitudinal strain. RVLSglob: Global right ventricular longitudinal strain. RVLSsep: Right ventricular septal longitudinal strain. RVLSlat: Right ventricular lateral wall longitudinal strain. RVTSglob: Global right ventricular transverse strain. RVTSsep: Right ventricular septal transverse strain. RVTSlat: Right ventricular lateral wall transverse strain. M: mean. SD: Standard deviation. p: p value. Tukey: Tukey test. ns: Non-significant.

right ventricular overload, shifting the interventricular septum towards the left with the concomitant decrease in the left ventricular chamber. The increased septal wall thickness in RVPO patients could be related to right ventricular hypertrophy. Left ventricular ejection fraction calculated using the two-dimensional method did not show significant differences among groups.

Right ventricular remodeling in patients with right ventricular overload is observed in PHT or volume overload, manifested as right ventricular chamber dilation and increased wall thickness, (17) but it can also occur in right ventricular overload without significant PHT and no increase in wall thickness. (18)

Right ventricular dysfunction, due to maladaptive remodeling, is the most important factor associated with worse prognosis. (19) The more severe the PHT, the greater the remodeling, which is observed both in free wall thickness and RV dimensions, as in functional parameters (TAPSE, area changes and longitudinal strain), a dysfunction that tends to become irreversible as long as the cause is not corrected. The boundary between adaptive and maladaptive remodeling is not clearly defined, but the adaptive form is associated with better functional capacity and survival, whereas the maladaptive form is characterized by right ventricular dilation, fibrosis and dysfunction. (20)

Conventional echocardiographic parameters were

altered according to the type and degree of right ventricular remodeling, with greater dilation and hypertrophy in RVPO patients, evidencing significant increase of the RA and pulmonary artery. Functional parameters (s' wave velocity of the lateral tricuspid annulus and area changes), which were decreased in these patients, indicate right ventricular systolic dysfunction. Mean TAPSE in RVPO, despite being significantly decreased compared with CTRL and RVVO groups, was still within normal values, which could be partly due to the incidence of severe tricuspid regurgitation observed in most of these patients. Right ventricular volume overload showed right ventricular dilation, without hypertrophy of the free wall and with lower pulmonary artery and right atrial diameter. Functional parameters (TAPSE, s' wave velocity of the lateral tricuspid annulus and area changes) did not decrease significantly compared with the CTRL group.

The tricuspid regurgitation gradient was increased in RVPO patients and also, though less significantly, in RVVO patients (the study of patients with PHT was the purpose of the present study). The milder PHT in patients with RVVO may be due to chronic ASD still not affecting right ventricular function.

Global left ventricular longitudinal strain was decreased only in patients with RVPO, despite a normal ejection fraction. This decrease could be an early detection parameter of biventricular myocardial dysfunction produced by PHT.

Global right ventricular longitudinal strain was decreased only in the RVPO group, remaining normal in ASD patients. Right ventricular free wall longitudinal strain was decreased in both groups, though it was more enhanced in the RVPO group. The decrease in global strain may be due to the role of septal strain, which was normal in RVVO and decreased in RVPO, probably evidencing the decreased left ventricular strain observed in these patients. These observations confirm the notion that the interventricular septum is part of the LV, so the analysis of the right ventricular free wall is more representative of this chamber's function.

Regarding transverse (radial) deformation, global transverse strain and right ventricular lateral wall strain increased in both groups, which could be the manifestation of right ventricular remodeling, where ventricular hypertrophy is due to the increase in circular fibers, as observed in anatompathological studies. (21, 22) This abnormality, associated with decreased longitudinal strain in the RVPO group could indicate right ventricular maladaptive remodeling.

Abnormal right ventricular free wall longitudinal and transverse deformation shows a change in the deformation pattern in pressure and volume overload, with increased transverse strain. In cases of severe pressure overload (RVPO group) longitudinal strain decreased, suggesting decompensation. These data indicate that increased transverse strain might be an indicator of myocardial remodeling, both in cases of pressure and volume overload, and the decreased longitudinal strain of the right ventricular free wall an indicator of ventricular dysfunction.

CONCLUSIONS

The analysis of right ventricular echocardiographic parameters and strain showed anomalies that indicate remodeling of this chamber in patients with pressure and volume overload, as suggested by the increased transverse strain of the right ventricular free wall. In patients with pressure overload and chronic severe PHT, there is decreased right ventricular systolic function parameters, with reduced area changes, s' wave velocity of the lateral tricuspid annulus and longitudinal strain, mainly of the free wall, which is hypertrophied. These abnormalities do no occur in patients with volume overload without severe PHT, suggesting preserved right ventricular systolic function. The increased transverse strain of the right ventricular free wall would be related with remodeling and the decreased longitudinal strain would be an indicator of systolic dysfunction, also accompanied by decreased parameters of systolic function. (area changes and s' wave velocity of the lateral tricuspid annulus). Left ventricular longitudinal strain, decreased in patients with severe PHT despite normal ejection fraction, may be an early marker of biventricular dysfunction

Conflicts of interest

None declared. (See authors' conflicts of interest forms in the website/Supplementary material).

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