

ROLE OF CARDIAC MAGNETIC RESONANCE IMAGING IN ASSESSMENT OF RIGHT VENTRICULAR DYSFUNCTION IN PATIENTS WITH PULMONARY HYPERTENSION

By

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ABSTRACT

Background: Patients with pulmonary hypertension (PH) can develop cor pulmonale later which is the leading cause of death in those patients. Although echocardiography is the mainstay in the assessment of hemodynamic and ventricular function in PH, magnetic resonance imaging (MRI) has emerged as the gold standard for quantifying volume, function, and flow in the right side of the heart.

Objective: To evaluate the role of cardiac magnetic resonance image in assessment of right ventricular functions in patients with pulmonary hypertension and to explore the relation between pulmonary artery pressures obtained from echocardiography and MRI -derived ventricular functions parameters.

Patients and methods: A total number of 27 patients scheduled for elective conventional echocardiography with known or suspected pulmonary hypertension were enrolled for MRI examination of the heart between October 2018 and March 2020, in Radiology Department, Al-Hussein University Hospital. All patients underwent cardiac MRI and echo within one week.

Results: We found that the mean value of left ventricular end systolic volume (LVESV) and left ventricular end systolic volume index (LVESVI) increased in pulmonary hypertensive patients. On assessment of the right ventricular mass index, the mean value was increased in our study compared to normal value. Right ventricular (RV) ventricular mass index (VMI) increased due to increased RV mass and decrease left ventricular (LV) mass. We visually assessed the position of the interventricular septum (IVS) during systole and diastole and had an abnormal position in some patients (nearly 38% of our cases either flattening or bowing toward the left ventricle). All of patients with abnormal septal position had systolic pulmonary pressure above 60 mmHg. We found that 79% of our patients had pulmonary regurge and the average velocity in the main pulmonary was 9.024 ± 6 cm/sec which was less than normal average velocity (13.6 ± 7 cm/sec).

There was a significant positive correlation between the mean pulmonary arterial pressure (mPAP), and the RV mass ($r=0.4$), and a significant positive correlation between mPAP and VMI ($r=0.52$).

Conclusion: Cardiac MRI is a valuable tool to assess the effect of pulmonary hypertension on the ventricular functions and also valuable for assessment of treatment response, follow up and prognosis of such patients.

Keywords: Cardiac MRI, Right ventricular dysfunction, Pulmonary hypertension.

INTRODUCTION

Pulmonary arterial hypertension (PAH) is one of the most important and potentially fatal alterations to the pulmonary circulation; if it goes untreated. It is associated with high morbidity, high mortality, and a poor prognosis. An increase in pulmonary artery pressure (PAP) leads to secondary right ventricular (RV) failure. In patients with PAH, the development of heart failure is an indicator of a poor prognosis. In the last decade, various studies have highlighted the importance of imaging methods other than angiography in the evaluation of pulmonary artery disease. One of the most widely used of such methods is cardiac magnetic resonance imaging (MRI) (*Junqueira and Neto, 2019*).

Pulmonary hypertension (PH) is a disease characterized by progressive rise of pulmonary artery (PA) pressure, which can lead to right ventricular (RV) failure. It is usually diagnosed late because of the nonspecificity of its symptoms. RV performance and adaptation to an increased afterload, reflecting the interaction of the PA and RV as a morphofunctional unit, constitute a critical determinant of morbidity and mortality in these patients. Therefore, early detection of dysfunction may prevent treatment failure (*Broncano et al., 2020*).

For the evaluation of systolic function and the quantification of cavity volumes, as well as of the myocardial mass, cardiac MRI is considered the gold standard. It has several advantages over other methods, mainly due to its noninvasive nature and its capacity to evaluate, in only one examination, morphology, RV function, left ventricular (LV) function,

and tissue characteristics, as well as to provide functional information by perfusion imaging at rest, pharmacological stress testing, and flow studies. Flow cardiac MRI studies can provide several noninvasive measurements that reflect the hemodynamics of the pulmonary arterial system. For example, curvature of the ventricular septum is strongly correlated with an $RV > LV$ pressure gradient and is comparable to RV systolic pressure determined by catheterization. The maximum angle of septal excursion toward the LV in ventricular systole—the interventricular septal angle (α)—also shows a strong correlation with PAP determined by invasive techniques (*Junqueira and Neto, 2019*).

The mean PAP and pulmonary vascular resistance can also be estimated by cardiac MRI with regression equations. The mean PAP estimated thusly has a sensitivity of 87% and a specificity of 90% for the diagnosis of $PAP > 32$ mmHg. Pulmonary artery flow velocity has also been shown to correlate with the mean PAP. Other variables evaluated in PAH include the relative change in area; increase in the maximum peak velocity; the time to maximum velocity; the maximum change in flow at ejection time; increase in the oscillatory shear index; increase in the shear interval; the transpulmonary gradient in the pulmonary artery; transmitral flow; myocardial tissue velocity; left atrial volume; and left atrial flow. In addition to all the information provided above, cardiac MRI allows prognostic factors to be estimated and risk to be stratified. For example, delayed myocardial enhancement volume correlates with RV mass, RV volume, RV

dysfunction, RV remodeling, and septal curvature, indicating a poorer prognosis (*Junqueira and Neto, 2019*).

Cardiac magnetic resonance imaging (CMRI) is a noninvasive tool that provides high-resolution, three dimensional images of the heart. It provides information about right heart structure, volumes and function that is not readily obtained via other methods, such as echocardiography and RHC (*Andrew et al., 2013*).

This study aimed to evaluate the role of cardiac magnetic resonance image in assessment of right ventricular functions in patients with pulmonary hypertension and to explore the relation between pulmonary artery pressure obtained from echocardiography and MRI -derived ventricular functions parameters.

PATIENTS AND METHODS

A total number of 27 patients scheduled for elective conventional echocardiography with known or suspected pulmonary hypertension were enrolled for MRI examination of the heart between October 2018 and March 2020, in Radiology Department, Al-Hussein University Hospital. All patients underwent cardiac MRI and echo during the same week.

Three of the 27 patients were excluded from the study because of failure of data acquisition (patient orthopnic, could not lie along the time of examination and could not obey breathing instructions). So, 24 patients were included in the study. The age of the patients ranged between 10 and 65 years old with average of 41 years. The number of females included in the

study was 14, whereas the number of males was 10 patients.

Those patients presented with dyspnea (defined as a shortness of breathing, some of them suffering from exertional, less than ordinary effort and others dyspnea at rest), orthopnea and weakness. Patients enrolled in this study had to fulfill sinus heart rhythm and able to hold breath for accepted time (10-20 seconds). When the breath-hold technique was not possible because of dyspnea (some patients with severe dyspnea and for phase-contrast sequence only), we asked them to breath as slowly as possible. The total duration of the MR examination was around 30–40 min.

Exclusion criteria were hemodynamic instability, severe dyspnea, atrial fibrillation, and contra-indications for MR imaging as claustrophobia, patients with pacemaker and metal implants.

No special instructions were required prior to the examination. Medications were not to be discontinued. First, a short medical history was taken. Patients were then screened for contraindication to MR imaging. All undergarments containing metal were removed. Before the examination; the heart rate and rhythm were evaluated. All steps of the study were explained in details for each patient. To evaluate patients ability of breath-withholding for relatively long time; they were required to perform a deep inspiration and to continue to hold their breath without pushing (i.e Valsalva maneuver). A Philips Achiva, Netherland (1.5 Tesla) superconducting magnet was used in Radiology Department, AL Hussein University Hospital.

All patients were examined in the supine position, head first. The patient's knees and legs can be elevated to help relieve back strain and secure the patient's comfort. Head phones supplied with the MRI machine were used to reduce repetitive gradient noise and in the same time allowed the patient to hear the breath hold instructions.

Four carbon fibers ECG pads were placed on the anterior chest wall, the first is placed 1 cm to the left of the xyphisternum, the second and the third are places in such a way that they were aligned at 90° to each other where the first electrode forms the right angle and the distance between the electrodes 15 cm. The fourth electrode was placed below the first electrode. The ECG leads were attached. The green lead to the first pad, the red lead to the second pad, the white lead to the third pad, and the black lead to the fourth pad.

The SENSE (sensitivity encoding) cardiac coil (6 element phased-array coil, receive only) was used. It has a rigid lower part and flexible upper part. The lower part contains two phased array coil elements and the upper part contains four phased array elements. The coil is positioned on the chest, so that the midline of its upper part lies just below the sternoclavicular notch and the lower part of the coil lies underneath the patient. It is carefully strapped into the patient by four straps. The connection to the magnet was checked.

The respiratory sensor was placed over the maximum area of respiratory movement (abdomen or thorax) under the coil. A strap was used to fix the sensor. The respiratory signal was then checked

as the respiratory wave appears on the monitor and used to detect the patient's respiratory rhythm and synchronize breath hold instructions to the patient abilities.

Fast imaging with steady state precession sequences were used to generate the initial axial, sagittal and coronal scout images required to localise the heart within the thoracic cavity and plan all subsequent cine images. Axial zero angle images were then taken, then vertical- and horizontal-long axis (VLA and HLA) cines image were planned and acquired based on the scout images.

The SA imaging plane was then propagated apically, covering both ventricles with 8-mm SA imaging slices, separated by a 2-mm inter-slice gap, imaging parameters, which were standardized for all subjects, included: TR/TE: 3/2ms, Bandwidth: 125 kHz FOV: 320, Phases: 25, NSA: 1, matrix: 128x128, flip angle: 60°, slice thickness:8mm, slice number: 12-16.

Flow measurements were performed to pulmonary arteries and ascending aorta at through plane using a velocity-encoded phase contrast (PC) imaging sequence with the following image parameters (repetition time 5 ms, echo time 3 ms, field of view 320mm, matrix=128x126, slice thickness 8 mm, flip angle=12 and velocity encoding=200cm/sec). Images were acquired during periods of breath holding, or with signal averaging for those subjects unable to cooperate with breath-hold instructions. Retrospective gating was used predominantly for PC imaging.

Qualitative flow of the Pulmonary artery: got it through imaging planes perpendicular to the main pulmonary artery in the sagittal scout image and axial

cine image. The resulting view is pulmonary artery in circular cross-sectional manner.

Qualitative flow of the Right Pulmonary Artery: got it through imaging planes perpendicular to the right pulmonary artery in the coronal scout image and axial cine image. The resulting view is right pulmonary artery in circular cross-sectional manner.

Qualitative flow of the Left Pulmonary Artery: got it through imaging planes perpendicular to the left pulmonary artery in the coronal scout image and axial cine image. The resulting view is right pulmonary artery in circular cross sectional manner.

Qualitative flow of the Ascending Aorta: got it through imaging planes perpendicular to the ascending aorta in the coronal and sagittal scout images. The resulting view is ascending aorta in a circular cross-sectional manner.

Image analysis:

Images (DICOM) were transferred to a workstation equipped with a dedicated cardiac software package (Brilliance 170 P workstation). The axial plane was used for evaluation of the anatomy and the short-axis planes to assess the ventricular functions. The endocardial borders of both ventricles were traced manually from the short axis images during end systole phase and end diastole phase. RV and LV end-diastolic volume (EDV) and end-systolic volume (ESV) were calculated. Subsequently, stroke volume (SV) and ejection fraction (EF) was calculated using EDV and ESV values. The ventricular ejection fraction was calculated as (end-diastolic volume – end-

systolic volume) /end-diastolic volume X 100%. Right and left Ventricular masses were measured by manually tracing the epicardial and endocardial borders. Ventricular mass index was measured by dividing right ventricular mass over left ventricular mass.

The index of the result parameters was calculated by dividing the parameter on the Body Surface Area which calculated from The DuBois and DuBois formula2: **BSA (m²) = 0.007184 x Height (cm) 0.725 x Weight (kg) 0.425**

The motion of the septum in the two cardiac phases was visually analyzed on short-axis and four-chamber views and its position was classified into one of two groups: normal and flattened or left bowing (convex towards LV).

To quantify flow within the proximal pulmonary trunk, right pulmonary artery, left pulmonary artery and ascending aorta by PC imaging, a region of interest was drawn manually around the vessel lumen in each phase of the dataset using the gradient echo image; contours were then correlated with the corresponding phase image. The workstation calculated flow volumes within the vessel by integrating the instantaneous velocity within the region of interest over the cardiac cycle.

Calculation of tricuspid valve regurgitation fraction combined data from volumetry and flow. Tricuspid regurgitation fraction equals RV stroke volume minus pulmonary artery forward volume (measured by velocity encoded phase contrast MR) divided by RV stroke volume multiplied by 100%. Or from RV stroke volume minus LV stroke volume divided by RV stroke volume multiplied by 100% and the degree of regurgitation

could be classified according to the regurgitation fraction in to : Mild <30% , Moderate 30—about 40%, and Severe >40% (Devos and Kilner, 2010).

Statistical Analysis:

The numerical variables were initially given as the mean and standard deviations for each parameter. Pearson correlation coefficients (r) between MRI and systolic or mean pulmonary artery pressure obtained from echo were calculated. P < 0.5 was considered significant.

RESULTS

The study included 24 patients with pulmonary hypertension. Their demographic criteria are shown in **Table (1)**.

Table (1): Demographic Criteria

Variable	Category	Value
Gender	Male	10
	Female	14
Age (years)	Mean	41
	Standard Deviation	17
	Minimum	10
	Maximum	65
Weight (kg)	Mean	61
	SD	18
Height (cm)	Mean	159
	SD	15
Body Surface Area (m ²)	Mean	1.83
	SD	0.38

In our study, the quantitative and qualitative flow parameters were obtained through accurate measurements of Ventricular volumes, masses and functions through assessment of Ventricle Stroke Volume, Ventricle End Diastolic

Volume, Ventricle End Systolic Volume, Ventricle Stroke Volume Index, Ventricle End Diastolic Volume Index, Ventricle End Systolic Volume Index, all parameters for the right and left ventricles were displayed at **Table (2)**.

Table (2): Right and left ventricular volumes

Variable Data	EDV (ml)		ESV (ml)		SV (ml)		EF (%)		EDVI (ml/m ²)		ESVI (ml/m ²)		SVI (l/min/m ²)	
	RV	LV	RV	LV	RV	LV	RV	LV	RV	LV	RV	LV	RV	LV
Mean	164	128	84	64	74	64	47	55	98	78	51	39	47	39
Maximum	321	279	242	193	195	110	85	78	209	155	130	106	79	49
Minimum	67	55	11	13	27	42	21	28	39	35	6	8	33	27
Standard deviation.	69	58	49	52	42	18	17	15	42	32	28	28	29	28

EDV: end-diastolic volume, ESV: end-systolic volume, SV: stroke volume, EDVI: end-diastolic volume index, ESVI: end-systolic volume index, EF: ejection fraction.

In our study, stroke volume of the main pulmonary was correlated well with the stroke volume of the right

ventricle($r=0.723$), the obtained data were demonstrated at **Table (3)**.

Table (3): Stroke volume of aorta and pulmonary arteries

Variable Data	SV AA (ml)	SV PU (ml)	SV RPA (ml)	SV LPA (ml)
Mean	50	61	35	23
Maximum	96	235	150	70
Minimum	22	18	0	1
Standard deviation.	16	44	31	17

SV: Stroke Volume, PU: Pulmonary Artery, RPA: Right Pulmonary Artery, A: Ascending Aorta and LPA: Left Pulmonary Artery.

In our study we found a significant positive correlation between the mPAP, and the RV mass($r=0.4$). A significant negative correlation between the SPAP, and the RVEF ($r=-0.48$) Also, a significant negative correlation between

the mPAP, and the RVSV, RVSVI($r=-0.34$ and -0.39) respectively. RV mass, VMI and RVEF could be predictors of the degree of pulmonary hypertension and valuable parameters for follow up and prognosis of such patients (**Table 4**).

Table (4): Pulmonary artery flow data

Variable Data	Forward Flow(ml)	Backward Flow (ml)	Net Flow(ml)	PR %	Mean velocity (cm/sec)	Acceleration time(ms)	Ejection time(ms)
Average	71	10	61	14	9.02	101	317
Maximum	240	61	235	76	33.2	180	420
Minimum	24	0	11	0	0.4	40	250

DISCUSSION

All patients in the present work were either diagnosed to have PH by right heart catheter or by echo. SPAP and mPAP measured by echo have strong correlation with RHC results (*Swift et al., 2012 and 2014*).

On assessment of right ventricular mass and mass index, our study revealed that, the mean values of the right ventricular mass and mass index increased compared to previously published normal. This was in agreement with *Swift et al. (2012)* who stated that the RV mass intuitively increases over time due to

elevated RV after load in patients with PH. They postulate that RVH is an adaption mechanism of the RV in order to preserve function.

Our study revealed that the mean values of (RVEDV-RVEDVI) increased among pulmonary hypertensive patients compared to previously publish normal reference. The mean values (RVESV-RVESVI) increased pulmonary hypertensive patients compared to previously publish normal reference. Similar results were obtained by *Wolferen et al. (2011)*.

An initial adaptive response of myocardial hypertrophy is followed by progressive contractile dysfunction. Chamber dilation ensues to allow compensatory preload and maintain stroke volume despite reduced fractional shortening. As contractile weakening progresses, clinical evidence of decompensated right ventricular failure occurs, characterized by rising filling pressures, diastolic dysfunction, and diminishing stroke volume (Voelkel *et al.*, 2010).

The mean of right ventricular SV and SVI reduced in pulmonary hypertensive patients compared to previously published normal reference. Andrew *et al.* (2013) stated similar results. Reduced stroke volume in pulmonary hypertensive patients was due to decreased contractility and increased afterload (Noordegraaf and Galie, 2011). Reduced RSV after 1-year follow-up have been shown to correlate with worse survival (Wolferen *et al.*, 2011).

In our study, stroke volume of the main pulmonary was correlated well with the stroke volume of the right ventricle and it was less than the correlation in healthy people as stated by Marrone *et al.* (2010). This difference was due to the presence of valves regurgitation either pulmonary or tricuspid valve. Noordegraaf *et al.* (2015) stated that RSV calculated from flow measurements in the PA and those derived from volumetric analysis correlate well in the absence of valvular regurgitation. However, both tricuspid and pulmonary regurgitation are common in patients with PH thus volumetric analysis overestimates 'true' or 'effective' stroke volume.

Our study showed that the mean value of right ventricular ejection fraction reduced compared to previously published normal reference. Similar results were found by Swift *et al.* (2012). RVEF reduced due to decreased RSV and increased RVEDV as $EF = \text{stroke volume} / \text{end diastolic volume} \times 100$ (Raieszadeh *et al.*, 2013). It was found that $RVEF < 35\%$ predicts increased mortality and $RVEDVI \leq 84 \text{ mL/m}^2$, $LVEDVI \geq 40 \text{ mL/m}^2$, and a $SVI \geq 25 \text{ mL/m}^2$ were associated with better survival in patients with idiopathic PH (Swift *et al.*, 2014).

On assessment of the LV volumes and index volumes, the mean value of LVEDV reduced in pulmonary hypertensive patients compared to previously published normal reference. Similar results were published by Alunni *et al.* (2010).

Noordegraaf and Galie (2011) stated that, as right ventricular function declines, an increase in right ventricular contraction time and subsequent ventricular asynchrony, together with a decrease in RSV, leads to under filling of the LV. Filling of the left ventricle is also impaired due to the development of leftwards ventricular septal bowing, resulting from prolonged right ventricular contraction time reducing LV volume during early diastole.

We found that the mean value of LVESV and LVESVI increased in pulmonary hypertensive patients compared to previously published normal references. Gan *et al.* (2010) stated that in primary pulmonary hypertension, the increased pulmonary vascular resistance limits the RV stroke volume, and thus limits automatically the volume available

for LV filling also leftward ventricular septal bowing reduces the LV volume in early diastole, and thereby might present a secondary, additive mechanism that further impairs the LV-filling process just in the most important phase of rapid filling. The resulting impaired LV filling interferes with LV pump function: the LV blood volume is reduced, and also the contractile force of the LV myocardial muscle fibers is reduced due to the Frank-Starling law.

On assessment of the right ventricular mass index, the mean value increased in our study compared to normal value. RV VMI was increased due to increased RV mass and decrease LV mass. *Swift et al. (2012)* stated that RV mass index of greater than 0.6 had a sensitivity of 84% and specificity of 71% for detecting PH.

We visually assessed the position of the IVS during systole and diastole, we found that it had an abnormal position in some patients nearly 38% of our cases either flattening or bowing toward the left ventricle. All of patients with abnormal septal position had systolic pulmonary pressure above 60 mmHg. These findings were close to that published by *Roeleveld et al. (2010)* who stated that it has been suggested that a systolic PAP higher than 67 mm Hg is to be expected if leftward ventricular septal bowing is seen. *Swift et al. (2014)* stated that elevated RV pressure causes the IVS to bow to the left in patients with PH. This leftward motion of the IVS causes the deformation of the LV into a "D shape" as a result of the pressure differential between LV and RV chambers.

In our study, we found that 79% of our patients had pulmonary regurge. *Rao et al.*

(2012) stated that pulmonary hypertension is a cause of pulmonary regurgitation due to dilatation of the pulmonary artery and the pulmonary annulus. *Bogaert et al. (2012)* stated that pulmonary valve regurgitation is prevalent in patients with PH and its severity (regurgitation fraction) correlates with functional status of patients. Transcatheter re-valuation has been associated with improved functional status and reduction in RV volume.

The average velocity in the main pulmonary was 9.024 ± 6 cm/sec which is less than normal average velocity (13.6 ± 7 cm/sec). *Swift et al. (2012)* stated that patients with PH have significantly more retrograde PA flow and significantly reduced average PA velocity than patients without PH. *Sanz et al. (2011)* found that average velocity within the main pulmonary artery correlated with pulmonary artery pressure and resistance. The threshold value for average flow velocity of 11.7 cm/s revealed PH with a sensitivity of 92.9% and a specificity of 82.4%.

In our study, we found that the mean values of AT, ET and AT/ET decreased compared to previously publish normal people. Similar results were found by *Sanz et al. (2011)*. A significant positive correlation between the mPAP, and the RV mass. Significant positive correlation was also found by *Saba et al. (2012)* between the mPAP and RV mass. We found a significant positive correlation between mPAP and VMI. That was similar to results obtained by *Guo et al. (2015)*. Also, a significant negative correlation between the SPAP, and the RVEF. There was EF was the best parameter to reflect RV function which

could be used to exactly evaluate RV function in patient with PH (Guo *et al.*, 2015). Alunni *et al.* (2010) also found significant negative correlation between the SPAP, and RVEF.

There was a significant negative correlation between the mPAP, and the RSVV, RVSVI. Swift *et al.* (2012) found a less significant negative correlation between the mPAP and the RVSVI.

RV mass, VMI and RVEF could be predictors of the degree of pulmonary hypertension and valuable parameters for follow up and prognosis of such patient.

CONCLUSION

Cardiac MRI is a valuable tool to assess the effect of pulmonary hypertension on the ventricular functions and also valuable for assessment of treatment response, follow up and prognosis of such patients.

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دور الرنين المغناطيسي للقلب في تقييم إختلال وظائف البطين الأيمن في المرضى المصابين بارتفاع ضغط الدم الرئوي

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خلفية البحث: إرتفاع ضغط الشريان الرئوي هو أحد الأمراض التي تؤثر على صحة الانسان بشكل خطير, كما أنه يهدد حياة المرضى المصابين به، الأمر الذي يؤدي إلى قصور وضعف بدني ملحوظ و كذلك يزيد من مخاطر تعرض عضلة القلب للضعف.

الهدف من البحث: تقييم دور صورة الرنين المغناطيسي للقلب في تقييم وظائف البطين الأيمن في المرضى الذين يعانون من إرتفاع ضغط الدم الرئوي وإستكشاف العلاقة بين ضغوط الشريان الرئوي التي تم الحصول عليها من تخطيط صدى القلب ومعلومات وظائف البطين الناتجة عن التصوير بالرنين المغناطيسي.

المرضى وطرق البحث: تم تسجيل 27 مريضاً من المقرر أن يخضعوا لتخطيط صدى القلب التقليدي الاختياري مع ارتفاع ضغط الدم الرئوي المعروف أو المشتبه به لفحص التصوير بالرنين المغناطيسي للقلب بين أكتوبر 2018 ومارس 2020، في قسم الأشعة بمستشفى الحسين الجامعي. وخضع جميع المرضى للتصوير بالرنين المغناطيسي وصدى القلب في غضون أسبوع واحد.

نتائج البحث: زاد متوسط قيمة نبضة البطين الأيمن في مرضى ارتفاع ضغط الدم الرئوي. وعند تقييم مؤشر كتلة البطين الأيمن، حدثت زيادة في متوسط القيمة في دراستنا مقارنة بالقيمة الطبيعية. و زادت قسم ومؤشر الكتلة البطينية بسبب زيادة كتلة البطين الأيمن وتقليل كتلة البطين الأيسر. وعند تقييم وضع الحاجز البطيني بصرياً أثناء الانقباض والانبساط كان غير طبيعي في بعض المرضى ما يقرب من 38 % من الحالات (إما تسطح أو انحناء نحو البطين الأيسر). وكان جميع المرضى من وضع الحاجز غير الطبيعي حيث كان لديهم ضغط رئوي انقباضي

أعلى من 60 مم زئبق. وعاني 79% من مرضانا من قلس رئوي. وكان متوسط السرعة في الرئة الرئيسية هو 6 ± 9.024 سم / ثانية وهو أقل من متوسط السرعة العادي (7 ± 13.6 سم / ثانية). كما كان هناك ارتباطاً إيجابياً مهماً بين متوسط الضغط الشرياني الرئوي وكتلة البطين الأيمن (المعدل = 0.4)، وإرتباطاً إيجابياً كذلك بين متوسط الضغط الشرياني الرئوي ومؤشر الكتلة البطينية (المعدل = 0.52).

الاستنتاج: التصوير بالرنين المغناطيسي للقلب هو أداة قيمة لتقييم تأثير ارتفاع ضغط الدم الرئوي على وظائف البطين الأيمن كما أن له دوراً هاماً في تقييم إستجابة العلاج والمتابعة في هؤلاء المرضى.

الكلمات الدالة على: الرنين المغناطيسي , إختلال وظائف البطين الأيمن , ارتفاع ضغط الدم الرئوي .