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Original Article

Longitudinal Deformation Indices of the Pulmonary Artery in Patients With Pulmonary Venous Hypertension

Atoosa Mostafavi [1], MD; Haniye Hajiali [1], MD;
S. A. Hussein Tabatabaei *[1], MD

ABSTRACT

Background: In this study, pulmonary artery longitudinal deformation indices were evaluated in patients with pulmonary venous hypertension and compared to those in normal subjects to delineate the effects of a high pulmonary pressure on vessel wall characteristics.

Methods: Fifteen patients with a diagnosis of pulmonary venous hypertension and 15 healthy individual were evaluated. In the parasternal short-axis view at the level of the aortic valve, the longitudinal axis of the pulmonary artery was obtained and velocity, strain, and strain rate at different levels of the vessel wall were measured by color-coded Doppler myocardial imaging.

Results: The systolic strain was positive in most segments of both normal and pulmonary hypertension groups. Negative strain was observed only in some parts of the proximal segments in the normal group, but was defined in the other parts of the vessel wall in the pulmonary hypertension group. Velocity, strain, and strain rate were significantly lower in the pulmonary hypertension group, mostly in the medial wall.

Conclusions: This study evaluated the deformation indices of the pulmonary artery in patients with pulmonary venous hypertension and compared them to those in normal subjects. This was the first study by color-coded Doppler myocardial imaging and found a significant reduction in velocity, strain, and strain rate in the pulmonary hypertension group. Our findings can be a basis for future investigations in other types of pulmonary hypertension. ***(Iranian Heart Journal 2017; 18(3):52-57)***

Keywords: Pulmonary artery, Pulmonary artery hypertension, Strain, Strain rate, Doppler echocardiography

[1] Department of Cardiology, Dr. Ali Shariati Hospital, Tehran University of Medical Sciences, Tehran, I.R. Iran

*** Corresponding Author:** S. A. Hussein Tabatabaie, MD; Cardiology Department, Dr. Ali Shariati Hospital, North Karegar Street, Tehran, I.R. Iran.
E-mail: tabatabaieseyedah.gmail.com **Tel:** 09121110106

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Pulmonary hypertension is a common, life-threatening, disabling disorder of the pulmonary circulation that leads to progressive right ventricular failure. Pulmonary hypertension is defined as an increase in the peak arterial pressure equal to or greater than 36 mm Hg or a mean pulmonary artery pressure (PAP) equal to or greater than 25 mm Hg on right-heart catheterization. [1]

The World Health Organization has classified pulmonary hypertension into 5 groups:
Group 1: pulmonary arterial hypertension
Group 2: pulmonary hypertension with left-sided heart disease (pulmonary venous hypertension)
Group 3: pulmonary hypertension associated with lung disease or hypoxemia
Group 4: pulmonary hypertension secondary to chronic thrombotic disease
Group 5: pulmonary hypertension due to miscellaneous causes [1]
There are several tools for the diagnosis of pulmonary hypertension, but right-heart catheterization is the gold-standard to for its confirmation. Echocardiography is the screening tool for the noninvasive diagnosis and assessment of the severity of pulmonary hypertension with reliance on the maximal velocity of the tricuspid regurgitation and calculation of the PAP.
There are also multiple tools to evaluate the effects of pulmonary hypertension on the heart. Phase-contrast magnetic resonance imaging (MRI) is one of these techniques to obtain velocity and flow measurements in pulmonary arteries by using specialized software for the calculation of velocity for each pixel in the region of interest. MRI can measure stroke volume, acceleration time, and strain and distensibility of the pulmonary artery. [2] Nonetheless, this modality is expensive and time-consuming.
In the present study, we sought to determine whether echocardiography could be used as a tool to evaluate the effects of a high PAP on the deformation indices (ie, velocity, strain, and strain rate) of the pulmonary artery.

METHODS

Fifteen individuals (4 men and 11 women) free of overt cardiovascular diseases with an estimated PAP less than 30 mm Hg and 15 patients (4 men and 11 women) with a diagnosis of pulmonary venous hypertension with an estimated PAP more than 45 mm Hg were selected.
Images were obtained using a Vingmed Vivid 7 system after obtaining informed consent from all the participants.
The mean age of the healthy group was 46 years, while the mean age of the patients was 59 years (P = 0.001). The mean systolic PAP was 25.13 ± 2.79 in the healthy group and 61.4 ±14.41 in the patient group (P = 0.001). Three consecutive cardiac cycles were recorded during expiration with 2D color-coded Doppler myocardial imaging at the level of the aortic valve. A region of interest of 6 × 4 mm was placed in the proximal, mid, and distal parts of the medial and lateral walls of the pulmonary artery and regional velocity, strain, and strain rates were estimated.

RESULTS

In the healthy group, strain was positive in most segments—except in some proximal parts of the pulmonary artery in the medial and lateral walls that had negative strain. The pattern was different in the patients with pulmonary hypertension inasmuch as although strain was positive in most segments, not only some proximal segments but also some parts of the mid or distal segments had negative strain.
Velocity and strain rate waves had 3 defined peaks: 1 positive in systole and 2 negative in diastole in both healthy and pulmonary hypertension groups—except in parts with negative strain that had negative strain rates as well (Fig. 1).
The analysis of systolic velocity, strain, and systolic strain rate showed no significant differences between the 2 opposite walls in the medial and lateral portions in the normal and pulmonary hypertension groups (Table 1). These findings suggested that the pulmonary artery had synchronous contractions, which were not affected by the presence of pulmonary hypertension.

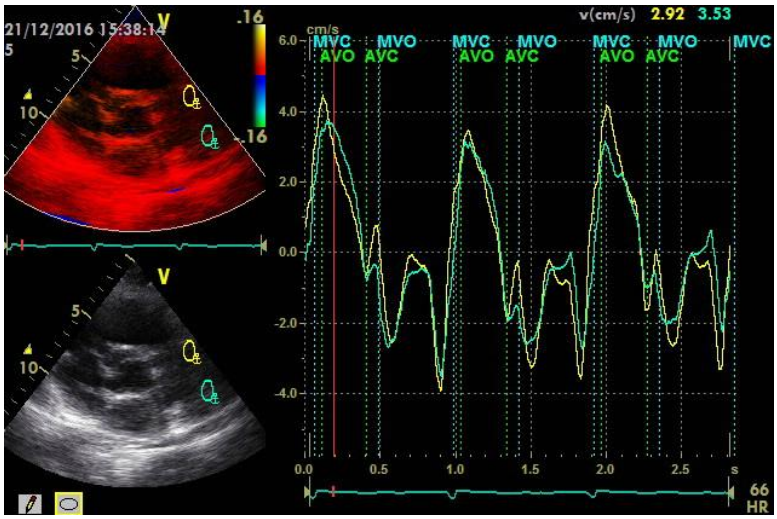

Figure 1 a. Velocity waves in the base and middle parts of the lateral wall of the pulmonary artery.

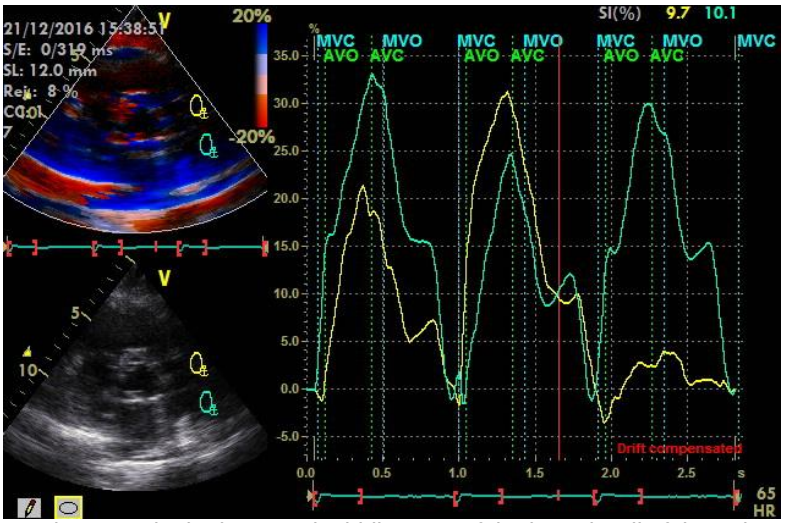

Figure 1 b. Strain waves in the base and middle parts of the lateral wall of the pulmonary artery.

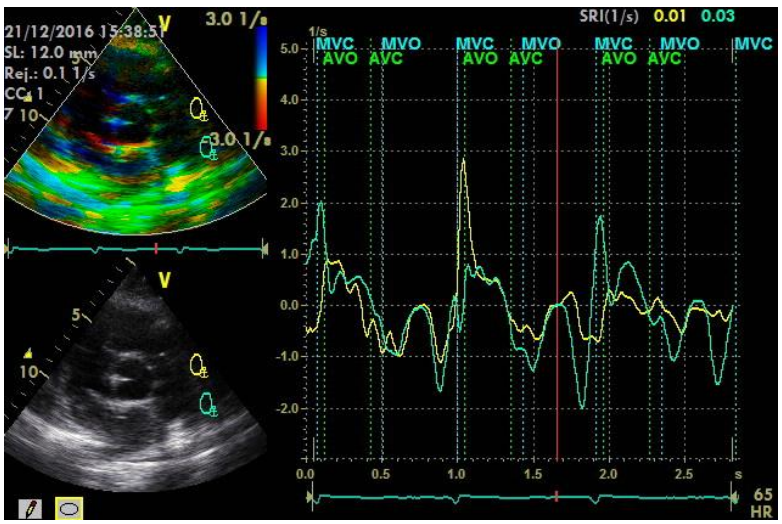

Figure 1 c. Strain rate waves in the base and middle parts of the lateral wall of the pulmonary artery.

The analysis of age-dependent pulmonary artery characteristics showed an increased age-dependent velocity in the normal group but not in the pulmonary hypertension group. There was no significant change in the other deformation indices (strain and strain rate) by increasing age in both healthy and pulmonary hypertension groups.

Table 1. Velocity, strain, and strain rate in the healthy and pulmonary hypertension groups

	Healthy			Pulmonary Hypertension		
Velocity (cm/s)	BM=4.5-9.18 mean=6.57 BL=0.65-12.5 mean=3.66 *P* value=0.488	MM=2.7-9.47 mean=6.07 ML=0.68-11.3 mean =5.41 *P* value =0.548	DM=3.30-7.30 mean=5.35 DL=0.49-9.80 mean =2.65 *P* value =0.11	BM=2.0-7.9 mean=4.69 BL=1.2-6.9 mean=4.22 *P* value =0.242	MM=1.3-8.5 mean=3.97 ML=0.46-9.6 mean=4.46 *P* value =0.481	DM=1.60-5.90 mean=3.56 DL=0.9-6.8 mean =2.76 *P* value =0.936
Strain (%)	BM=-34.6-61.7 mean=10.39 BL=-23.15-21.7 mean=5.51 *P* value =0.27	MM=4.20-39.00 mean=14.57 ML=1.1-47.00 mean=18.66 *P* value =0.593	DM=4.2-43.7 mean=14.57 DL=1.54-104.0 mean=19.1 *P* value =0.265	BM=-11.7-23.5 mean=9.06 BL=-19.8-19.7 mean=4.68 *P* value =0.527	MM=-12.1-13.3 mean=3.97 ML=-21.8-30 mean=6.06 *P* value =0.593	DM=-2.4-19.8 mean=7.12 DL=-23.6-17.3 mean=3.39 *P* value =0.637
Strain rate (1/s)	BM=-4.4-4.4 mean=0.79 BL=-4.6-7.3 mean=0.84 *P* value =0.21	MM=0.87-3.10 mean=1.85 ML=0.5-3.84 mean=1.69 *P* value =0.62	DM=0.48-3.10 mean=1.45 DL=0.51-3.2 mean=1.61 *P* value =0.24	BM=-1.79-2.7 mean=1.05 BL=-1.42-1.76 mean=0.55 *P* value =0.33	MM=-2.2-1.70 mean=0.38 ML=-1.68-2.90 mean=0.59 *P* value =0.177	DM=-0.2-1.30 mean=0.7 DL=-1.90-1.80 mean=0.33 *P* value =0.436

BM, Basal of the medial wall; MM, Mid of the medial wall; DM, Distal of the medial wall; BL, Basal of the lateral wall; ML, Mid of the lateral wall

The comparisons of the deformation indices revealed reduced peak systolic velocity, strain, and strain rate in the mid and distal parts of the pulmonary hypertension group compared to the control group. The reduced velocity was mainly in the medial wall of the pulmonary artery. The proximal part of the medial and lateral walls had no difference regarding strain and strain rate (Table 2).

Table 2. Comparisons of velocity, strain, and strain rate between the normal and pulmonary hypertension groups (*P* value) at different parts of the medial and lateral vessel walls

	Velocity (*P* value)	Strain (*P* value)	Strain Rate (*P* value)
BM	0.003	0.846	0.714
MM	0.006	0.0001	0.001
DM	0.001	0.025	0.004
BL	0.32	0.075	0.694
LM	0.353	0.014	0.01
DL	0.003	0.038	0.002

BM, Basal of the medial wall; MM, Mid of the medial wall; DM, Distal of the medial wall; BL, Basal of the lateral wall; ML, Mid of the lateral wall; DL, Distal of the lateral wall

DISCUSSION

Our study demonstrated that the novel noninvasive indices of the pulmonary artery obtained by echocardiography were significantly affected by pulmonary hypertension.

Changes in the pulmonary vascular function in individuals without overt cardiovascular diseases and normal pulmonary pressures novel noninvasive indices remain undetected. So far, only a single study has reported synchronous movements of the pulmonary artery in reverse of the heart movements: when the right ventricle is contracting, the pulmonary artery is longitudinally expanding and vice versa.[3] Our study confirmed those findings in a normal population. Strain was positive in systole and showed arterial dilation by the right ventricular contraction. Velocity and strain rate had 1 positive systolic and 2

negative diastolic curves. These patterns were maintained in the pulmonary hypertension group, although their values of strain, strain rate, and velocity were reduced compared to those in the healthy group. These reduced pulmonary artery velocity and strain have been previously confirmed in only small studies by phase-contrast MRI. [4-5-6-2] There is no study in the existing literature on the measurement of these parameters by echocardiography and our study is the first investigation of its kind to evaluate and compare theses parameters between patients with pulmonary hypertension and individuals free of overt cardiovascular diseases.

An increased accumulation of elastin and collagen in the pulmonary artery during hypoxemia has been previously described in an animal model. [7-8] Changes in the amount of collagen and elastin can affect the behavior of the vessel wall insofar as elastin is load-bearing at low stresses and collagen at physiologic and mostly at high stresses. [9] The pulmonic function in pulmonary hypertension may also be attributed to the accelerated breakdown of elastin in the vessel wall, leading to the stiffening of the wall as elastin is replaced with stiffer collagen—similar to the effects of long-standing hypertension on the aorta. [10] A higher pressure in the aortic artery leads to greater smooth muscle cell production and elastin synthesis, and the structure of the aortic or pulmonic artery may be incomparable. [11] Previous research has proved histopathologic changes in patients with chronic severe pulmonary arterial hypertension due to congenital heart disease. These changes may happen in other forms of pulmonary hypertension such as severe atherosclerotic changes of the pulmonary artery wall and fibrosis and cystic-like formation of the vessel wall. [12]

The major limitations of the current study are its small sample size and the inclusion of patients with only pulmonary venous hypertension. Whether these results hold true in other types of pulmonary hypertension requires further research.

In conclusion, a variety of pulmonary artery deformation indices such as velocity, strain, and strain rate were reduced in our patients with pulmonary hypertension. We believe that these parameters may confer important clinical use in this group of patients.

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