Available online at www.sciencedirect.com

ScienceDirect

www.onlinepcd.com

Pulmonary Hypertension in Aortic Stenosis and Mitral Regurgitation: Rest and Exercise Echocardiography Significance

Christophe Martinez[a], *Anne Bernard*[a,b], *Raluca Dulgheru*[a], *Pierluigi Incarnato*[a], *Cécile Oury*[a], *Patrizio Lancellotti*[a,c,*]

[a]*University of Liège Hospital, GIGA Cardiovascular Sciences, Departments of Cardiology, Heart Valve Clinic, CHU Sart Tilman, Liège, Belgium*
[b]*University of Tours Hospital, Cardiology Department, Tours, France and University François Rabelais, Tours, France*
[c]*Gruppo Villa Maria Care and Research, Anthea Hospital, Bari, Italy*

ARTICLE INFO

Keywords:
Aortic stenosis
Echocardiography
Exercise
Mitral regurgitation, pulmonary hypertension
Outcome

ABSTRACT

Valvular heart disease is a common cause of increased mean pulmonary artery pressure (PAP). Aortic stenosis and mitral regurgitation are frequently accompanied by pulmonary hypertension (PH), especially when they are severe and symptomatic. In asymptomatic patients, PH is rare, though the exact prevalence is unknown and mainly stems from the severity of the valvular heart disease and the presence of diastolic dysfunction. Exercise echocardiography has recently gained interest in depicting PH. In these asymptomatic patients, exercise PH is observed in about >40%. Either PH at rest (systolic PAP >50 mmHg) or during exercise (systolic PAP >60 mmHg) is a powerful determinant of outcome and is independently associated with reduced survival, regardless of the severity of the underlying valvular pathology.

© 2016 Elsevier Inc. All rights reserved.

Current guidelines provide a classification system that categorizes pulmonary hypertension (PH) into five groups with specific pathogeneses and clinical characteristics (Table 1).[1] Group 2, according to the World Symposium classification, includes PH caused by left heart diseases such as valvular heart disease (VHD) or myocardial disease. VHD is a frequent cause of PH.[2] Initially, PH usually results from the hemodynamic effects of VHD. With the increasing severity and chronicity of PH, progressive right ventricular (RV) dysfunction and RV failure can develop, compromising the patient outcome.[3] Because pulmonary artery pressures (PAP) are dynamic, exercise-induced PH can identify patients with VHD at higher risk of further clinical worsening.[4] Therefore, the assessment of PH at rest and during exercise is of major interest for individual risk stratification and therapeutic management of VHD.[5]

Definitions

PH is defined by a resting mean PAP ≥25 mmHg regardless of the etiology.[1] The diagnostic criteria of PH also include an abnormal increase in pulmonary capillary artery pressure (PAWP) or in left ventricular end-diastolic pressure (>15 mmHg). Post capil-

Statement of Conflict of Interest: see page 68.

* Address reprint requests to Prof. Patrizio Lancellotti, MD, PhD, Domaine Universitaire du Sart Tilman, Batiment B35, Department of Cardiology, University Hospital, Université de Liège, CHU du Sart Tilman, 4000 Liège, Belgium.
E-mail address: plancellotti@chu.ulg.ac.be (P. Lancellotti).

http://dx.doi.org/10.1016/j.pcad.2016.06.004
0033-0620/© 2016 Elsevier Inc. All rights reserved.

Abbreviations and Acronyms

AcT = pulmonary acceleration time

DPD = diastolic pressure difference

LV = left ventricular

LA = left atrial

NYHA = New York Heart Association

PAP = pulmonary artery pressure

PAWP = pulmonary capillary wedge pressure

PH = pulmonary hypertension

PVR = pulmonary vascular resistance

RV = right ventricular

sPAP = systolic pulmonary artery pressure

TPG = transpulmonary pressure gradient

VHD = valvular heart disease

lary PH or pulmonary venous hypertension resulting in passive increase in systolic PAP (sPAP), is observed when the transpulmonary pressure gradient (TPG = meanPAP–PAWP) is <7–10 mmHg.[6] In advanced stage of the diseases, a mixed mechanism with both pre-capillary and post-capillary PH can be observed (TPG >12 mmHg). This form of PH has been considered as "out of proportion" to the left ventricular (LV) filling pressure and is characterized by both pulmonary arterial vasoconstriction, which is reversible, and by fixed pulmonary vasculature remodeling.[6] However, TPG is influenced by all determinants of mean PAP which includes flow, resistance, and LV filling pressure thus may not serve as the best parameter to define the presence of pulmonary vascular disease. Diastolic pressure gradient (DPG), defined as diastolic PAP– mean PCWP, seems to be a better approach and is now used to characterize the presence of PVD. Isolated post-capillary PH (Ipc) is observed when PCWP is >15 mmHg and DPG is <7 mmHg. Combined post-capillary PH and pre-capillary PH (Cpc–PH) is defined as a PAWP >15 mmHg and a DPG ≥7 mmHg.[1]

Table 1 – The 2015 updated clinical classification of pulmonary hypertension.

Class	Denomination
1	Pulmonary arterial hypertension
1'	Pulmonary veno-occlusive disease and/or pulmonary capillary haemangiomatosis
2	Pulmonary hypertension due to left heart disease
2.1	Systolic dysfunction
2.2	Diastolic dysfunction
2.3	Valvular heart disease
3	Pulmonary hypertension due to lung diseases and/or hypoxia
4	Chronic thromboembolic pulmonary hypertension
5	Pulmonary hypertension with unclear and/or multifactorial mechanisms

Adapted from reference.[1]

Pathophysiology of Group 2 PH

LV pressure or volume overload due to aortic or mitral valve disease causes a progressive increase in left atrial (LA) pressure, which is passively transmitted backward to the pulmonary venous system with an increase in PV pressure.[3] PV congestion may trigger pulmonary vasculature vasoconstriction, which together with decreased nitric oxide availability, dysregulation in prostacyclin (PGI2) metabolism, increased endothelin-1 expression (potent vasoconstrictor), desensitization to natriuretic peptide induced vasodilatation and vascular remodeling, have a negative impact on alveolar-capillary units leading to further increase in PAP in excessive degree when considered in regards to PAWP.[7–10] With time, irreversible remodeling of alveolar-capillary membrane with excessive deposition of type IV collagen and structural changes in pulmonary veins and arteries (abnormalities of elastic fibers and hypertrophy and fibrosis of medial and intimal tunic) can be seen.[11] These structural damages may induce functional injuries, decrease lung diffusion capacity, and impair gas exchange. Hemodynamically, the chronic increase in PAP primarily promotes RV hypertrophy followed later on by tricuspid annulus dilatation, increase in tricuspid regurgitation severity, and RV dysfunction and failure. In end-stage, PAP can decrease despite the increase in PVR due to the fall in RV stroke volume related to advanced RV failure.[12]

To summarize, PH secondary to VHD first results from an upstream transmission of increased LV filling pressure and LA pressure to pulmonary vessels. With chronicity resulting in worsening VHD severity and increase in PAP, remodeling of pulmonary vessels and irreversible PH occur with similar pathophysiologic consequences seen in pulmonary arterial hypertension (Fig 1). Again there is no evidence that left-sided PH determines similar arterial vessel remodeling compared to PAH.

Diagnostic work-up

Clinical signs of PH

During the early stages, patients with PH usually remain asymptomatic for a prolonged period of time.[1] The most frequent initial symptoms are exercise-related dyspnea, which may be accompanied by fatigue and dizziness. Signs of RV failure, such as peripheral edema and ascites, syncope and decompensated RV failure are seen at a more advanced stage of the disease. In clinical practice, the reported symptoms are mainly driven by the left-sided VHD.[13,14]

Clinical tests often reveal findings suggestive of left-sided VHD PH: presence of VHD on physical exam; LV/LA hypertrophy on ECG; pulmonary vascular congestion, pleural effusion or pulmonary edema on chest X-ray or computed tomography. Nonetheless, the signs and symptoms of PH are nonspecific and may be subtle, explaining why the diagnosis often only occurs late in the course of the disease. Orthopnea and paroxysmal nocturnal dyspnea may occur and are more specific to left heart disease-related PH.

Download English Version:

https://daneshyari.com/en/article/5619664

Download Persian Version:

https://daneshyari.com/article/5619664

Daneshyari.com