The Impact of the New Hemodynamic Definition on the Prevalence of Pre-Capillary Pulmonary Hypertension

Yeni Hemodinamik Tanımlamanın Pre-Kapiller Pulmoner Hipertansiyon Prevalansına Etkisi

The underlying mechanism of pulmonary hypertension (PH) is increased right ventricle (RV) afterload due to pulmonary vascular negative remodeling and vasculopathy and it is characterized with increased mortality risk due to RV failure if not diagnosed and treated early.1 Five PH groups with similar pathophysiology, clinical presentation, and treatment strategy are diagnosed by the World Heart Organization (WHO).2 In 2015 European Society of Cardiology (ESC)/European Respiratory Society (ERS) Guidelines for the diagnosis and treatment of PH, pre-capillary PH has been defined as mPAP ≥ 25 mmHg, PCWP ≤ 15 mmHg and PVR >3 WU and described group 1,3,4, and 5 of PH patients.3 In this guideline while patients with mPAP ≥ 25 mmHg, PCWP > 15 mmHg, and PVR <3WU described as isolated post-capillary PH (Ipc–PH), if the patient had PVR higher than 3 WU with similar mPAP and PCWP the hemodynamic definition has been made as combined pre- and post-capillary PH (Cpc–PH).3

Data accumulated from healthy individuals showed us that a normal mPAP at rest is 14.0 ± 3.3 mmHg. The upper limit of normal PVR in healthy volunteers and the lowest prognostically relevant threshold of PVR is ~ 2WU. Hence, the definition of pre-capillary PH is updated as mPAP > 20 mmHg, PCWP ≤ 15 mmHg, and PVR >2WU in 2022 ESC/ERS PH guideline.4 Furthermore, for the first time in this new guideline, patients with mPAP > 20 mmHg but PVR < 2 WU are defined as undefined PH.4 After this new hemodynamic definition, we re-evaluated our right heart catheterization (RHC) procedures that have been performed between 2017 and 2023. The clinical indications for RHC were suspicion of congenital heart disease associated pulmonary arterial hypertension (APAH-CHD) in 32.5% of patients, idiopathic PAH in 43.9% of patients, PH associated with left heart disease (APAH-LHD) in 17.9% of patients, and chronic thromboembolic pulmonary hypertension in 5.7% (Figure 1). As the results of the evaluation 123 incident cases, we realized almost 10% increase in the prevalence of pre-capillary PH patient population (Table 1).

Although the number of patients diagnosed with pre-capillary PH is increasing after releasing of new ESC guideline, PAH–specific drugs have not yet been tested and approved for efficacy and safety in these patient groups. As the old definition (mPAP ≥ 25 mmHg, PVR > 3WU, and PCWP < 15 mmHg) was used in randomized controlled trials that PAH–specific treatments were approved, we should only treat PAH patients who meet this hemodynamic criteria. We must keep this fact in mind. If the PAH specific drugs are approved in these patients in the future, we will be able to protect RV and improve life expectancy with early diagnosis and rapid onset of initial combination therapy.

Furthermore, we should keep in mind that clinical suspicion is initiated the PH diagnostic algorithm. In patients with symptoms, risk factors, and clinical signs suggesting PH, evaluating the probability of PH with echocardiography is the main strategy of the
diagnostic algorithm. The threshold of tricuspid regurgitation velocity for low, intermediate, and high PH probability remained unchanged (<2.8 m/s, 2.9–3.4 m/s, >3.4 m/s, respectively).

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**References**


