0140

Prevalence of pulmonary hypertension in patients undergoing hemodialysis
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Introduction: Pulmonary hypertension (PH) is one of the most important accompanying comorbidities with hemodialysis in patients with end-stage renal disease. The prevalence of hemodialysis-induced PH is still a subject of debate. The goal of the present work was to determine the prevalence of PH in patients undergoing hemodialysis.

Materials and Methods: This study was carried out on 111 patients undergoing hemodialysis for at least 6 months. Pulmonary artery pressure (PAP) was measured using echocardiography, and a value equal to or higher than 35 mmHg was considered PH. The relationship of a high PAP with demographic and clinical characteristics of the patients was assessed.

Results: A total of 111 patients were included in the study. The mean age was 44 ± 14 years. The mean duration of hemodialysis was 146 ± 80 months. The most common cause of end-stage renal disease was glomerulonephritis. The mean ejection fraction and PAP were 62.5 ± 10.5% (range, 29% to 81%) and 26.6 ± 12 mmHg (range, 11 mmHg to 100 mmHg), respectively. Overall, 16% of the patients had PH. These patients were more likely to have low ejection fractions, valvular calcifications and valvulopathy. They were also older than other patients.

Conclusion: Our findings show that PH is associated with duration age, ejection fraction, and valvular calcifications. Due to the high prevalence of PH among hemodialysis patients, it is necessary to screen this disorder and minimize its effects.

0337

Echocardiographic findings in passive and reactive postcapillary pulmonary hypertension
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Background: Left heart diseases are the most common cause of pulmonary hypertension (PH). The reactive form of PH confers a worse prognosis than the passive one. Therapeutic intervention are going on investigating vasodilatation drugs.

Aims: The aim of this study was to seek for echocardiographic differences between passive and reactive postcapillary pulmonary hypertension in patients with heart failure and preserved ejection fraction.

Methods: This study enrolled 58 patients who were assigned both to pulmonary artery catheterization and standard echocardiographic examination. Patients were classified into 2 groups: reactive PH (mean pulmonary artery pressure >25 mmHg, pulmonary capillary wedge pressure >15 mmHg, and transpulmonary gradient >12 mmHg and passive PH (mean pulmonary artery pressure >25 mmHg, pulmonary capillary wedge pressure >15 mmHg, and transpulmonary gradient <12 mmHg).

Results: Despite similar wedge pressure (p = 0.67), patients with reactive PH showed significantly higher systolic pulmonary artery pressure (p < 0.05) and pulmonary arterial resistance (p < 0.05). There was a trend of correlation with a better contractility measured by the lateral tricuspid annulus motion in tissue Doppler imaging (10.2±2.1 vs. 8.3±1.9 m/s, p = 0.094) and lower values on the right ventricle long axis (73.8±12.4 vs. 80.1±23.5 mm, p = 0.05) in patients with reactive PH compared with passive PH.

Conclusion: Despite higher pressure and resistance, patients with reactive PH showed better right ventricular function and less remodeling.

0123

Nebivolol for improving endothelial dysfunction, pulmonary vascular remodeling, and right heart function in pulmonary hypertension
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Background: Endothelial cell (EC) dysfunction plays a central role in the pathogenesis of pulmonary arterial hypertension (PAH), promoting vasoconstriction, smooth muscle proliferation and inflammation.

Objectives: This study sought to test the hypothesis that nebivolol, a β1 antagonist and β2-agonist, may improve PAH and reverse the PAH-related phenotype of pulmonary ECs (P-EC).

Methods: We compared the effects of nebivolol, with metoprolol, a first generation β-blocker on human cultured PAH and control P-EC proliferation, vasoactive and proinflammatory factor production and crosstalk with PA smooth cells (PASMC). We assessed the effects of both β-blockers in precontracted PA rings. We also compared the effects of both β-blockers in experimentalPAH.

Results: PAH P-EC overexpressed the proinflammatory mediators IL-6 and MCP-1, the growth factor FGF2, and the potent vasoconstrictive agent endothelin-1 as compared to control cells. This pathological phenotype was corrected by nebivolol but not metoprolol in a dose-dependent fashion. We confirmed that PAH P-EC proliferate more than control cells, and stimulate more PASMC mitosis, a growth abnormality which was normalized by nebivolol but not by metoprolol. Nebivolol but not metoprolol induced an endothelial- and nitric oxide-dependent relaxation of PA. Nebivolol was more potent than metoprolol in improving, cardiac function, pulmonary vascular remodeling and inflammation of rats with monocrotaline-induced pulmonary hypertension.

Conclusions: Nebivolol could be a promising option for the management of PAH, improving endothelial dysfunction, pulmonary vascular remodeling, and right heart function. Until clinical studies are undertaken, however, routine use of β-blockers in PAH cannot be recommended.