HEMODYNAMIC RESPONSE OF SILDENAFIL DEPENDS ON LEFT VENTRICULAR CHAMBER STIFFNESS IN PATIENTS WITH DECOMPENSATED CONGESTIVE HEART FAILURE

Poster Contributions
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Background: Previous studies reported that sildenafil improves clinical status, left ventricular (LV) function and exercise capacity in patients with heart failure. However, it is not yet clear whether there are types of hemodynamic conditions in which sildenafil exerts beneficial effects. Therefore, we assessed acute hemodynamic effects of sildenafil in patients with decompensated congestive heart failure using echocardiography. We showed for the first time that there is a risk of sildenafil administration, and also developed a useful parameter which may be used to predict hemodynamic response to sildenafil in patients with decompensated congestive heart failure.

Methods: We performed echocardiography before and an hour after a single oral administration of sildenafil (20 mg) in 20 patients who were admitted to our hospital due to decompensated congestive heart failure.

Results: Tricuspid regurgitation peak gradient (TR-PG) and the ratio of peak velocity of early LV filling to early diastolic myocardial velocity (E/E') which is an indicator of LV filling pressure was elevated at baseline. These results suggest presence of decompensated congestive heart failure. Sildenafil decreased pulmonary vascular resistance by 16% (p<0.05) and TR-PG by 10 % (p<0.05), and increased LV stroke volume index by 12% (p<0.05). The increase in E/E' following administration of sildenafil were positively correlated with the deceleration time of early filling wave (DcT), an indicator of LV chamber stiffness, at baseline. We divided the patients into two groups: group A (n = 8), baseline DcT of 200 msec or longer, and group B (n = 12), baseline DcT of shorter than 200 msec. Mean E/E' increased by 19 % in group B, whereas it decreased by 12 % in group A.

Conclusions: Sildenafil significantly improves pulmonary circulation and cardiac output in patients with decompensated congestive heart failure. However, we have to be cautious to further elevation of LV filling pressure particularly in patients with shortened DcT or high LV chamber stiffness at baseline.