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CILP1 as a biomarker for right ventricular maladaptation in pulmonary hypertension

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CILP1 is a novel biomarker of RV and LV pathological remodelling that is associated with RV maladaptation and ventriculoarterial uncoupling in patients with pulmonary hypertension
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ABSTRACT The aim of our study was to analyse the protein expression of cartilage intermediate layer protein (CILP)1 in a mouse model of right ventricular (RV) pressure overload and to evaluate CILP1 as a biomarker of cardiac remodelling and maladaptive RV function in patients with pulmonary hypertension (PH).

Pulmonary artery banding was performed in 14 mice; another nine mice underwent sham surgery. CILP1 protein expression was analysed in all hearts using Western blotting and immunostaining. CILP1 serum concentrations were measured in 161 patients (97 with adaptive and maladaptive RV pressure overload caused by PH; 25 with left ventricular (LV) hypertrophy; 20 with dilative cardiomyopathy (DCM); 19 controls without LV or RV abnormalities).

In mice, the amount of RV CILP1 was markedly higher after banding than after sham. Control patients had lower CILP1 serum levels than all other groups ($p < 0.001$). CILP1 concentrations were higher in PH patients with maladaptive RV function than those with adaptive RV function ($p < 0.001$), LV pressure overload ($p < 0.001$) and DCM ($p = 0.003$). CILP1 showed good predictive power for maladaptive RV in receiver operating characteristic analysis (area under the curve (AUC) 0.79). There was no significant difference between the AUCs of CILP1 and N-terminal pro-brain natriuretic peptide (NT-proBNP) (AUC 0.82). High CILP1 (cut-off value for maladaptive RV of $\geq 4373 \text{ pg mL}^{-1}$) was associated with lower tricuspid annular plane excursion/pulmonary artery systolic pressure ratios ($p < 0.001$) and higher NT-proBNP levels ($p < 0.001$).

CILP1 is a novel biomarker of RV and LV pathological remodelling that is associated with RV maladaptation and ventriculoarterial uncoupling in patients with PH.