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COMMENTARY

Take the Road Less Traveled: Pulmonary Artery Banding to Rescue the Children's Failing Heart

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Comments

Pediatric dilated cardiomyopathy (DCM) is a leading cause of heart failure in children, presenting significant therapeutic challenges due to the limited efficacy of pharmacological treatments, the scarcity of donor hearts for transplantation, and the high costs associated with ventricular assist devices. Also, the economic burden of DCM medical management is a critical topic for world wide. In this context, the development of a safe, effective, and economically viable surgical intervention is of paramount importance. A recent study published in Congenital Heart Disease, titled "Cardiac Rehabilitation by Pulmonary Artery Banding after Induced Dilated Cardiomyopathy: A Pilot Study on a Rodent Model", represents a significant advancement in this field. This study evaluated the feasibility and therapeutic potential of pulmonary artery banding (PAB) in a drug-induced DCM rodent model, providing critical preclinical evidence to support its clinical translation [1].

The research team utilized doxorubicin (DOX) to induce DCM in a rat model, successfully replicating the systolic dysfunction characteristic of human DCM. Based on this model, they investigated the effects of PAB on left ventricular function, a novel approach in rodent studies. Despite a high mortality rate in the DOX-treated group (43.7%), echocardiographic analysis demonstrated significant improvements in left ventricular ejection fraction (EF) and fractional shortening (FS) following PAB. These findings are consistent with clinical observations that PAB can improve outcomes in infants with DCM, suggesting its potential as a therapeutic strategy for pediatric patients. To further illustrate the underlying mechanisms, the researchers used transmission electron microscopy (TEM) to examine ultrastructural changes in the myocardium. TEM revealed DOX-induced subcellular damage, including disorganized sarcomeres and vacuolization, providing morphological evidence of the drug's cardiotoxic effects. These findings not only enhance our understanding of DCM pathophysiology but also establish a methodological framework for future studies aimed at exploring the cellular and molecular mechanisms of PAB.

PAB, as a cost-effective and less invasive palliative surgical intervention, has demonstrated potential in clinical scenarios to reverse ventricular remodeling [2]. For example, Li et al. conducted a retrospective cohort study to evaluate the short-term efficacy of pulmonary artery banding (PAB) in 18 infants and young children with end-stage dilated cardiomyopathy (DCM) [3]. The results

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demonstrated that the surgical group exhibited a significantly higher left ventricular functional recovery rate compared to the nonsurgical group. Optimal degree of PAB for DCM hearts demand precision in hemodynamic targeting, individualized anatomic adjustments, and compatibility with underlying pathophysiology, since excessive pulmonary artery banding (PAB) raises right heart workload, leading to right heart failure. Back to this study, Padalino et al. posits that PAB facilitates the modulation of interventricular interactions by training the right ventricle, thereby improving left ventricular geometry and function. This finding is particularly promising for pediatric patients who are not candidates for heart transplantation or ventricular assist device support. Nonetheless, the study acknowledges certain limitations, such as the dose-dependent variability in DOX-induced DCM models [4] and the pathological distinctions from human idiopathic DCM, including right ventricular involvement [5]. Also, prioritizing stability and reproducibility—via Ang II or genetic models—would strengthen the translational relevance of PAB as a therapeutic strategy. Moreover, authors should assess the extent of PAB by measuring peak-Velocity data via echocardiography, which is beneficial in specifying the criteria for PAB. The elevated mortality rate in the animal model also constrained the sample size, potentially impacting the statistical robustness of some findings. The authors emphasize the necessity for model refinement, suggesting the exploration of genetic or pressure-overload-induced DCM models, and advocate for future research to summarize the molecular signaling pathways activated by PAB, which may inspire integrated pharmacologic and surgical therapeutic strategies.

Despite its preliminary nature, this study represents a significant contribution to the field of pediatric heart failure research. By validating the feasibility of PAB in improving cardiac function in an animal model, it addresses a critical gap in the literature and provides a robust foundation for future investigations. The establishment of a reliable rodent model for DCM, combined with the insights gained from this research, opens new avenues for exploring the biological mechanisms of PAB and its potential clinical applications.

In conclusion, this study provides compelling preclinical evidence supporting the potential of PAB as a therapeutic intervention for pediatric DCM. By addressing the limitations of current treatment options and offering a cost-effective alternative, it offers hope for improved outcomes in children with heart failure. Future research must achieve breakthroughs in optimizing model stability, expanding sample sizes, and deeply elucidating molecular mechanisms to accelerate the translation of PAB from bench to bedside. Overall, this work pioneers new research directions and methodologies in the surgical study of pediatric heart failure.

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