Cardiology in the Young

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Pulmonary artery banding for dilated and depressed left ventricle: dilated cardiomyopathy versus left ventricular non-compaction cardiomyopathy

Andrey Semyashkin[1], Julia Nesteruk[2], Dimitra Giannikopouloui[3], Michael Scheid[3], Gleb Tarusinov[3], Aktam Tannous[3], Marcel Te Vrugt[4] and Lotfi Ben Mime[5]

[1]Department of Pediatric Cardiac Surgery, University Hospital Muenster, Muenster, Germany; [2]University Heart Center Freiburg Bad Krozingen, Freiburg, Germany; [3]Kaiser Wilhelm Krankenhaus, Pediatric Heart Center, Duisburg, Germany; [4]Department of Pediatric Hematology and Oncology, University Hospital Muenster, Muenster, Germany and [5]Pediatric Heart Surgery, Center for Congenital Heart Defects and Children's Heart Center, HDZ-NRW, University Hospital of the Ruhr-University Bochum Medical Faculty OWL University Bielefeld, Germany

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Corresponding author:
Andrey Semyashkin;
Email: semyashkin.doc@gmail.com

Abstract

Objectives: To retrospectively assess the suitability of pulmonary artery banding as a treatment strategy for dilated cardiomyopathy and left ventricular non-compaction cardiomyopathy with depressed left ventricular ejection fraction. *Methods:* The study was retrospective and included consecutive patients who met the inclusion criteria: diagnosed with dilated cardiomyopathy or left ventricular non-compaction cardiomyopathy and left ventricular ejection fraction less than 35%. Cardiac indices were documented, and clinical outcomes were followed for 5 years. *Results:* This study included 21 patients with depressed left ventricular ejection fraction due to dilated cardiomyopathy (n = 11) or left ventricular non-compaction cardiomyopathy (n = 10), treated either with anti-congestion medication alone or in combination with pulmonary artery banding. The groups treated with pulmonary artery banding showed significant improvement in left ventricular ejection fraction compared to controls (ANOVA, $p = 0.0002$), with no major adverse events. In the subgroup with left ventricular non-compaction, pulmonary artery banding led to significant improvement of the left ventricular ejection fraction ($p = 0.00002$) and significant reductions in the Z scores of left ventricular end-diastolic diameter ($p = 0.0002$) and of end-diastolic volume ($p = 0.004$). *Conclusions:* Pulmonary artery banding appears to be a viable strategy for improving heart function in patients with non-compaction and dilated cardiomyopathy and depressed left ventricular ejection fraction. While pulmonary artery banding demonstrated more pronounced benefits in the subgroup with non-compaction cardiomyopathy, significantly enhancing cardiac restoration indices throughout the follow-up period, warranting further investigation in larger studies.

Introduction

Dilated cardiomyopathy is the most common childhood cardiomyopathy and is associated with significant morbidity and mortality. The prognosis for paediatric dilated cardiomyopathy patients is generally poor, and current effective therapeutic options for dilated cardiomyopathy patients with progressive heart failure are limited to anti-congestion medication. Approximately 40% of paediatric dilated cardiomyopathy patients who progress to end-stage heart failure require a heart transplantation.[1] Left ventricular non-compaction cardiomyopathy is the third most common type of primary cardiomyopathy after dilated cardiomyopathy and hypertrophic cardiomyopathy.[2,3] It is a genetically heterogeneous disorder typically associated with reduced left ventricular ejection fraction and marked systolic dysfunction.[4] Over 80% of patients with non-compaction cardiomyopathy have depressed ventricular compliance due to hypertrabeculation and conduction abnormalities that can lead to malignant ventricular tachyarrhythmias and a high risk of sudden cardiac death.[5]

The presence of systolic dysfunction and/or arrhythmias was identified as the most significant risk factor for death or heart transplantation in paediatric patients with left ventricular non-compaction cardiomyopathy.[6] Patients diagnosed with left ventricular non-compaction cardiomyopathy within their first year of life were observed to have an increased independent risk of 25% for death or heart transplantation.[6,7]

Children with left ventricular non-compaction cardiomyopathy are more likely to experience death or require transplantation if they exhibit a reduced left ventricular fractional shortening z-score. In contrast, those whose left ventricular systolic function remains intact tend to have

more favourable outcomes compared to children with dilated cardiomyopathy or restrictive cardiomyopathy.[1]

The mortality rate among patients with progressive heart failure remains high.[8] Due to a lack of prospective studies conducted on large patient cohorts, the long-term effectiveness of medical treatments for patients with left ventricular non-compaction cardiomyopathy remains uncertain.[9] Management of patients with progressive heart failure is still largely guided by clinical symptoms, with no significant effect on life expectancy or prevention of major adverse events.[9]

The primary goal of treatment in patients with cardiomyopathy is to reverse myocardial remodelling and improve contractile function.[10] Pulmonary artery banding is considered one of the few treatment options that directly affects the hemodynamic aspects of heart failure, aiming to improve left ventricular geometry and enhance systolic cardiac function.[11,12,13] Several authors have documented the benefits of pulmonary artery banding in patients with dilated cardiomyopathy.[14,15] However, the specific impact of pulmonary artery banding on patients with left ventricular non-compaction cardiomyopathy remains uncertain and warrants further investigation.[16]

The authors of this study aim to retrospectively evaluate and compare the impact of pulmonary artery banding on the dynamics of left ventricular geometry and myocardial contractility in patients with cardiomyopathy and reduced left ventricular ejection fraction. Additionally, the study seeks to determine any differences in response between subgroups of patients with left ventricular non-compaction cardiomyopathy and dilated cardiomyopathy.

Materials and methods

The study initially included a cohort of 33 patients. This retrospective analysis was conducted at the Heart Center Duisburg, Germany, over a 5-year period beginning on April 1, 2016. Consecutive patients were enrolled based on specific inclusion criteria: a confirmed diagnosis of either dilated cardiomyopathy or left ventricular non-compation cardiomyopathy and a left ventricular ejection fraction of less than 35%. Additional exclusion criteria included severe right ventricular dysfunction, pulmonary vascular resistance >6 Wood units and incomplete follow-up data. Throughout the study period, all patients underwent functional echocardiography imaging and were followed clinically for 5 years. The follow-up schedule began with baseline assessments at admission and the initiation of anti-congestion medication (BL 0). Subsequent evaluations occurred at key time points: BL 1 (pulmonary artery banding application), FU 1 (1 month post-application), FU 2 (2 months post-application), FU 3 (3 months post-application), FU 4 (1 year post-application) and FU 5 (5 years post-application). Control groups receiving anti-congestion medication were monitored accordingly. A comprehensive evaluation of treatment strategies included assessing cardiac restoration indices such as left ventricular ejection fraction, left ventricular end-diastolic diameter, left ventricular end-diastolic volume and right ventricular end-diastolic diameter, along with documentation of clinical progression.

Statistical analysis was performed using the SPSS version 23 software package (IBM SPSS Statistics 23) and Rstudio, Version 4.2.3. A p-value less than or equal to 0.05 was considered statistically significant. Continuous variables were presented as mean ± SD or median with range. The missing values were reconstructed by median imputation. It was 4.3% missing values. Student's independent *t*-test or Mann–Whitney test were chosen for comparison of continuous variables with normal or not normal distribution (Kolmogorov–Smirnov test was used), accordingly. Standard error and 95%CI – confidence interval are presented for all calculations.

All categorical variables were compared using Fisher's exact test to determine the significance of the relationships between the variables.

To evaluate the main effects of pulmonary artery banding, time, and their interaction in a cohort of patients with cardiomyopathy, we employed a two-way repeated measures analysis of variance (ANOVA). This statistical approach allowed us to assess both the independent effects of treatment and time, as well as their combined impact.

Further subgroup analysis was conducted to assess potential differences between patients with left ventricular non-compaction and those with dilated cardiomyopathy regarding their response to pulmonary artery banding. Given the small sample sizes within each subgroup, non-parametric tests were employed to analyse the data. For each subgroup, a Friedman test was used to assess differences across more than three dependent categories. Additionally, Pair's Exact Test was employed as a post hoc analysis to further investigate pairwise differences between specific time points. To control for the risk of Type I error due to multiple comparisons, a Bonferroni correction was applied, ensuring robust statistical validity. Missing data were addressed using median imputation, a method chosen to minimise bias while preserving the overall structure and integrity of the dataset.

Results

After applying additional selection criteria and assessing patient eligibility, the final cohort consisted of 21 patients. The baseline characteristics of the 21 patients are summarized in Table1. All patients received anti-congestion medication upon admission to the hospital. Following stabilisation, 11 patients continued to receive anti-congestion medication. However, for the remaining 10 patients, anti-congestion medication failed to improve their condition, leading to the administration of pulmonary artery banding supplementation one month later. Consequently, the study generated two distinct groups: PAB-LVNC (n = 5) and PAB-DCM (n = 5), who received pulmonary artery banding in addition to anti-congestion medication, along with two corresponding control groups: ACM-LVNC (n = 5) and ACM-DCM (n = 6), who received anti-congestion medication only.

The baseline characteristics of patients in the study group and the control group are summarised in the table. The median age at the initiation of therapy, which included either pulmonary artery banding with anti-congestion medication or anti-congestion medication alone in control groups, was 4.4 years for patients diagnosed with dilated cardiomyopathy and 1.4 years for patients diagnosed with left ventricular non-compaction cardiomyopathy. Among the patients with dilated cardiomyopathy, 10 (91%) were male, while among those with left ventricular non-compaction cardiomyopathy, 4 (40%) were male.

Both groups appeared to benefit from the application of pulmonary artery banding. Three primary outcomes were assessed: death or heart transplantation, sudden cardiac death or malignant ventricular arrhythmia and implantation of a left ventricular assist device. No major adverse events occurred during the follow-up period in the group treated with pulmonary artery banding. In contrast, two major events (one heart transplantation and one

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Table 1. Baseline characteristics of 21 patients in treated and control groups

	PAB DCM (N = 5) mean, SD; median, min-max	ACM DCM (N = 6) mean, SD; median, min-max	p-value	PAB LVNC (N = 5) mean, SD; median, min-max	ACM LVNC (N = 5) mean, SD; median, min-max	p-value
Sex/male (%)	5 (100)	5 (83)	0.999[c]	2 (40)	2 (40)	0.999[c]
Age (days)	1563.7 ± 1713.04	1020.1 ± 2017.13	0.661[a]	61.8 ± 37.31	43.4 ± 73.66	0.632[a]
Weight (g)	14,500.0 ± 11,824.13	11,566.7 ± 11,224.5	0.683[a]	5024.0 ± 1188.12	3838.0 ± 1178.91	0.152[a]
Height (cm)	92.0 ± 37.38	75.5 ± 33.43	0.459[a]	59.2 ± 6.06	51.4 ± 6.99	0.096[a]
pro BNP (pg/mL)	7817(2766–38447)	27,654 (4214–70000)	0.327[b]	13,512 (6528–18678)	15,784 (2363–70000)	0.999[b]
LVEF (%)	21.2 ± 6.57	27.5 ± 6.09	0.134[a]	18.9 ± 6.56	26.0 ± 13.42	0.319[a]
FS (%)	11.2 ± 1.78	14.8 ± 3.4	0.401[a]	15.0 ± 3.61	17.8 ± 8.14	0.502[a]
Z-score LVEDV (mL)	3.4 ± 2.94	2.8 ± 1.01	0.699[a]	3.2 ± 0.02	4.9 ± 2.27	0.166[a]
Z-score LVED mm	5.6 ± 3.95	6.1 ± 2.84	0.812[a]	5.9 ± 1.00	6.0 ± 0.96	0.900[a]
Z-score RVED mm	3.4 ± 4.13	0.9 ± 2.67	0.181[a]	0.8 ± 1.05	0.6 ± 1.18	0.479[a]
TAPSE mm	10.8 ± 2.87	10.3 ± 5.19	0.872[a]	10.6 ± 2.70	10.0 ± 6.52	0.854[a]
MV Regurgitation (grad)	1 (1–2)	2 (0–3)	0.409[b]	1 (1–2)	1 (1–3)	0.700[b]
β-blocker n (%)	5 (100)	6 (100)	0.999	4 (80)	4 (80)	0.999[c]
ACE inh. n (%)	5 (100)	6 (100)	0.999	3 (60)	4 (80)	0.999[c]
Esedrix n (%)	2 (40)	0 (0)	0.182[c]	2 (40)	3 (60)	0.999[c]
Aldosteron Antag. n (%)	3 (60)	0 (0)	0.061[c]	2 (40)	3 (60)	0.999[c]

ACM = Anti-congestion medication; DCM = Dilated cardiomyopathy; LVEF = Left ventricle ejection fraction; LVEDD = Left ventricle end-diastolic diameter; LVEDV = Left ventricle end-diastolic volume; LVNC = Left ventricular noncompaction cardiomyopathy; MV = Mitral valve; PAB = Pulmonary artery banding; RVEDD = Right ventricle end-diastolic diameter; TAPSE = Tricuspid annular plane systolic excursion; ACE inh.= Angiotensin-converting enzyme inhibitorpro; BNP = Pro-brain natriuretic peptide.

death) were observed in the subgroup of patients with dilated cardiomyopathy treated with anti-congestion medication alone.

In the cohort of patients with cardiomyopathy, a two-way repeated measures analysis of variance was used to evaluate the interaction between treatment and time. This analysis revealed a significant interaction effect on left ventricular ejection fraction ($F(6,80) = 5.04$, $p = 0.0002$), indicating that left ventricular ejection fraction differed significantly between the treatment groups and changed over time in the group receiving pulmonary artery banding. Notably, treatment with pulmonary artery banding showed a statistically significant beneficial effect, with a marked improvement in left ventricular ejection fraction over the study period. Left ventricular ejection fraction exhibited a significant and sustained increase over 5 years ($p = 0.001$ at the fourth follow-up and $p = 0.001$ at the fifth follow-up) (Supplementary Figure S1).

In the control group, which included all cardiomyopathy patients treated with anti-congestion medication alone, no significant improvement in left ventricular ejection fraction was observed ($p = 0.125$ at the fourth follow-up and $p = 0.156$ at the fifth follow-up) (Supplementary Figure S1).

Further subgroup analysis of the patients with left ventricular non-compaction cardiomyopathy treated with pulmonary artery banding revealed significant differences in left ventricular ejection fraction across the six time points, as determined by the Friedman test ($CHI^2[6] = 29.1$, $p < 0.00002$). Pair's exact test showed significant differences between baseline measurements taken at the time of pulmonary artery banding and the fourth follow-up ($p = 0.001$), as well as between baseline and the fifth follow-up ($p = 0.0001$).

Similarly, significant differences were observed in the Z scores for left ventricular end-diastolic diameter across the six time points ($CHI^2[6] = 26.0$, $p = 0.0002$). Pair's exact test indicated significant differences between baseline and the third follow-up ($p = 0.0135$), the fourth follow-up ($p = 0.0001$) and the fifth follow-up ($p = 0.006$).

For the Z scores of left ventricular end-diastolic volume, the Friedman test also revealed significant differences across the six time points ($CHI^2[6] = 19.3$, $p = 0.004$). Significant differences were observed in the Pair's exact test between baseline and the fourth follow-up ($p = 0.0003$) and the fifth follow-up ($p = 0.027$).

In the control subgroup of patients with left ventricular non-compaction cardiomyopathy treated with anti-congestion medication alone, no significant improvement in left ventricular ejection fraction was observed at the fourth follow-up ($p = 1.00$). Similarly, no significant changes were noted in the reversal of left ventricular remodelling, as measured by Z scores for left ventricular end-diastolic volume (baseline versus fourth follow-up, $p = 1.00$) or left ventricular end-diastolic diameter (baseline versus fourth follow-up, $p = 1.00$) (Supplementary Figure S2).

Further subgroup analysis of patients with dilated cardiomyopathy treated with pulmonary artery banding revealed significant differences in left ventricular ejection fraction across the six time points ($CHI^2[6] = 14.2$, $p < 0.03$). Pair's exact test identified significant differences between baseline and the fourth follow-up ($p = 0.04$) and between baseline and the fifth follow-up ($p = 0.017$). No significant differences were observed for the Z scores of left ventricular end-diastolic volume or left ventricular end-diastolic diameter.

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In the control subgroup of patients with dilated cardiomyopathy treated with anti-congestion medication alone, no significant improvement in left ventricular ejection fraction or reversal of left ventricular remodelling was observed over the same period.

Specific outcomes of the 12 excluded patients

Patients with left ventricular ejection fraction above 35% (n = 6) had relatively preserved left ventricular function compared to the study cohort. They were managed effectively with medical therapy alone and were not eligible for pulmonary artery banding. During follow-up, they experienced mild to moderate symptoms but no major clinical deterioration. Patients with severe right ventricular dysfunction (n = 3) were excluded because their compromised right ventricle could not sustain adequate ventricular–ventricular interaction following pulmonary artery banding. Outcomes for this group were poor, with two patients requiring mechanical circulatory support (ventricular assist devices) and one undergoing heart transplantation. Patients with pulmonary vascular resistance exceeding 6 Wood units (n = 2) were excluded, as this level is considered a contraindication for pulmonary artery banding. Despite receiving treatment with pulmonary vasodilators, both patients exhibited progressive right-sided heart failure. One patient ultimately required palliative care, while the other was listed for heart transplantation. One patient (n = 1) was excluded due to incomplete follow-up data, making it impossible to reliably assess their long-term clinical course or outcomes.

Discussion

Patients treated exclusively with anti-congestive therapy demonstrated initial stabilisation but no significant improvement in cardiac function was observed throughout the duration of the study. In contrast, patients who underwent pulmonary artery banding experienced notable clinical benefits, with significant improvements in echocardiographic measures. While the analysis of variance did not show statistically significant changes in the Z-scores of left ventricular end-diastolic volume and left ventricular end-diastolic diameter, graphical representations suggested a potential trend toward left ventricular remodelling. Although these trends did not reach statistical significance, the visual data hint at a remodelling process that warrants further investigation in future studies involving larger patient cohorts (Supplementary Figure S1).

Patients with left ventricular non-compaction cardiomyopathy and dilated cardiomyopathy demonstrated differing responses to pulmonary artery banding. In the left ventricular non-compaction cardiomyopathy group, significant improvements in left ventricular ejection fraction were observed early after the initiation of pulmonary artery banding and were sustained over the 5-year follow-up period. Additionally, evidence of left ventricular remodelling reversal was noted, with consistent and significant reductions in the Z scores of left ventricular end-diastolic diameter and volume over time.

In contrast, patients with dilated cardiomyopathy exhibited improvements in left ventricular ejection fraction following pulmonary artery banding, but changes in left ventricular end-diastolic diameter and volume were not significant.

These differing responses may reflect variations in the degree and nature of myocardial remodelling, driven by the distinct pathophysiological mechanisms underlying left ventricular non-compaction cardiomyopathy and dilated cardiomyopathy.

The helical architecture of the interventricular septum, as confirmed by diffusion tensor MRI, establishes its role as a unified structure rather than a left ventricular structure, facilitating interaction between the left and right ventricles.[17,18] When considering pulmonary artery banding intervention, it imposes left ventricular conditions on the right ventricle to the extent that the latter is still able to cope with the imposed high pressure. In cases of hypertension affecting the right ventricle, its pressure–volume relationships begin to resemble those of a healthy left ventricle. Consequently, the hypertensive right ventricle becomes the driving force of global cardiac function. Due to the significance of ventricular interdependence, pulmonary artery banding could pose an excessive burden on a compromised right ventricle or fail to facilitate positive ventricular–ventricular interaction in cases of severely reduced left ventricular ejection fraction.[20,21] In the presence of right ventricular scarring or replacement with a non-contractile patch, the interventricular septum can still maintain circulatory stability as long as the right ventricle is not dilated.[22] The compressive forces of the basal loop of the right ventricle's free wall alone are sufficient to cause normal right ventricle ejection when pulmonary vascular resistance is low, but cannot efficiently maintain cardiac output when pulmonary vascular resistance is increased without concomitant septal twisting.[23,24] Within physiological limits, an increase in right ventricle preload can enhance myocardial contraction via the Frank–Starling mechanism.[25]

In our study, patients with dilated and left ventricular non-compaction cardiomyopathy exhibited baseline left ventricular ejection fraction values around 20%. Both groups benefited from pulmonary artery banding, but the question remains whether a baseline ejection fraction of 20% represents a definitive threshold for its success. At this stage of cardiac dysfunction with an initial left ventricular ejection fraction below 20%, the right ventricular ejection fraction is often already reduced, impairing its ability to sustain effective ventricular–ventricular interaction.[26,27] This supports the notion that preserved right ventricular septal function is critical for the success of pulmonary artery banding. Therefore, pulmonary artery banding should likely be considered primarily in conditions with preserved right ventricular ejection fraction, which is typically the case in left ventricular non-compaction cardiomyopathy, as the right ventricle is usually less affected.

Recent studies using novel cardiac magnetic resonance data analysis revealed that in left ventricular non-compaction cardiomyopathy patients with reduced left ventricular ejection fraction, global circumferential strain and global longitudinal strain are significantly impaired in midventricular and apical segments, but preserved at the base.[28,29] The predominant localisation of non-compaction in midventricular and apical segments may explain the benefit of pulmonary artery banding-induced enhanced ventricular–ventricular interaction through common fibres predominantly localised in these same segments.[28,30]

Given the biventricular interaction, the results suggest that pulmonary artery banding in patients with left ventricular non-compaction cardiomyopathy may restore the elliptical configuration of the heart and improve the hemodynamic function of the left ventricle. The coupled left ventricle is supported by the right ventricle when the common myofibrils are preserved in structure and function.[21] This appears to be the case in left ventricular non-compaction cardiomyopathy with chronic progressive heart failure but not in dilated cardiomyopathy with reduced left ventricular ejection fraction.

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Limitations

The limitations of our study include its retrospective design, small sample size due to the rarity of left ventricular non-compaction cardiomyopathy and the limited number of eligible patients, and a relatively short follow-up period of 5 years, which may not fully reflect long-term outcomes. While a multi-center study would provide a larger cohort, greater statistical power, and broader insights across diverse populations, our study is the first to compare outcomes of pulmonary artery banding in left ventricular non-compaction and dilated cardiomyopathy. It offers valuable preliminary findings, demonstrating significant improvements in cardiac function and highlighting the feasibility of this intervention. These results, along with our subgroup analyses, provide a strong foundation for future research.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S1047951125000460.

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Competing interests. None

Ethical standards. The study received approval from the institutional ethics committee (Nr.69/2024, 08.04.2024), and written informed consent was obtained from the parents of all participating patients. The authors assert that all referenced work contributing to this review complies with the ethical standards of biomedical or medicolegal investigation.

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