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Research Article

The Akor® Extracardiac Mesh Implantation in Dilated Cardiomyopathy Management

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Abstract

Objective: The aim of the study was to assess the long-term effects of the AKOR® extracardiac mesh implantation alone or in combination with mitral valve replacement (MVR) on left ventricular (LV) structure and function in patients with idiopathic dilated cardiomyopathy (IDCM) and heart failure, evaluate the safety and efficacy of this procedure.

Background: Dilated cardiomyopathy progression depends on ventricular remodeling. Changes in myocardial stroma based on collagen type I/type III ratio, affect cardiac compliance and lead to further LV dilation. The originally designed "AKOR®" extracardiac mesh was implemented in the trial. It was different from the ACORN CorCap CSD, that was used in previous trials. The trial was aimed to check, whether its implantation (alone or in combination with MVR) may compensate negative changes in myocardial stroma and prevent remodeling development.

Methods: A total of 36 patients with IDCM, severe LV dysfunction and advanced heart failure (NYHA class III-IV) were enrolled in the trial. All patients underwent extracardiac mesh of original design implantation either alone (Group I; n = 16) or in combination with MVR (Group II; n = 20). Long-term results were followed up to 4 years postop. Echocardiograms were obtained annually until the last patient was followed for 4 years. Standard measurements of LV volumes and dimensions, ejection fraction were made. The NYHA functional class, 6-min walking distance, working capacity were evaluated at annual checkups.

Results: The total LV EDD preoperatively was 7,2 ± 0,8 cm; LV EDV 258,5 ± 76,3 ml; LV ejection fraction 24,7 ± 6,7%. There were no cases of intraoperative mortality in both groups. The total 4 years' actuarial survival in total group was 79,6 ± 7,4% ($p < 0,05$). The 4 years' actuarial survival in Group I (AKOR mesh alone) was 92,7 ± 7,0% ($p < 0.05$). The 4 years' actuarial survival in Group II (AKOR mesh +MVR) was 70,0 ± 2,5% ($p < 0.05$). The LV EDD diameter decreased in total group by 13% (from 7,2 ± 0,8 cm to 6,1 ± 0,5 cm; $p < 0.05$). The LV EDV decreased by 38% (from 258,5 ± 76,3 ml to 160,8 ± 19,7ml; $p < 0.05$). There were no cases of LV EDD or LV EDV increase after AKOR mesh implantation. LV EF increased from 24,7 ± 6,7% to 38,5 ± 5,6%. The number of annual hospitalizations decreased 3 times. NYHA mean class changed from 3,3 ± 0,7 to 2,0 ± 0,5 ($p < 0,05$). 19 (52,7%) patients restored working capacity. In 7 (19,4%) patients the AKOR® mesh implantation stimulated the trend for reverse LV remodeling and NYHA class improvement.

Conclusions: The AKOR® extracardiac mesh implantation (alone or in combination with MVR) stopped remodeling progression in IDCM patients, improved cardiac structure and functional status. When implemented in the adaptive stage of LV remodeling it may stimulate reverse heart remodeling. No signs of cardiac constriction were noted. Randomized controlled trials are needed to further evaluate the efficacy of this medical technology.

Keywords: Dilated Cardiomyopathy; Extracardiac Mesh

Methods

The study was prospective, nonrandomized clinical trial. The trial protocol was approved by the local ethics committee.

All patients provided written consent before enrollment.

Enrollment criteria: idiopathic dilated cardiomyopathy: left ventricular end diastolic dimension (LV EDD) ≥ 6,0 cm; left ventricular end diastolic volume (LV EDV) > 200,0 ml; left ventricular ejection fraction (LV EF) ≤ 30%; NYHA class III-IV.

Exclusion criteria

Coronary artery disease (CAD); tricuspid valve regurgitation; suspected myocarditis.

Extracardiac mesh was implanted to all the patients enrolled to the trial. Mitral valve replacement (MVR) was performed in patients with mitral regurgitation > 2 grade.

All of the patients were treated with five-component medical therapy (diuretics, beta-blockers, ACE inhibitors, aldosterone antagonists, small doses of glycosides).

Preoperatively, after reaching the optimal clinical status, multispiral computed tomography of the heart was performed.

The mesh was manufactured for each patient individually, accordingly to its heart dimensions in diastole.

The plaster cast (moulage) of the heart was manufactured by 3D printing, accordingly the diastolic dimensions.

The mesh was manufactured of the stripes, being done of the cutted Gelweave vascular graft. The Sulzer Vascutec Gelweave Vascular Graft is made of a woven polyester material that has been impregnated with an absorbable mammalian gelatin. The gelatin coating of stripes provided less damage to the epicardium.

Each stripe was 10 mm wide. The stripes were connected with each other under the right angle. The mesh covered all the surface of the ventricles and atria (partially).

Due to the "ribbing" of stripes, the mesh was not rigid, but instead had a certain flexibility that appeared to be important in functional activity of the device. The stripes were sutured to a Gelweave patch, positioned at the heart's apex.

The mesh was exposed to gas sterilization after the manufacture.

Figure 1: The AKOR® extracardiac mesh design.

The mesh implantation and mitral valve replacement were performed at the open-heart surgery, cardiopulmonary bypass.

The mesh was implanted intraoperatively under control of central venous pressure and cardiac output. It was fixed to the pericardium at the level of its bottom line.

Figure 2: The AKOR® mesh implantation.

The MedEng Ltd mechanical heart valves were implanted in case of mitral regurgitation > 2 grade.

Impella 5,0 microaxial pump was implemented in post cardiopulmonary bypass to prevent acute heart failure development.

The study was a 4 years trial. All the patients were assessed intraoperatively, at 30 days postop and then followed annually up to the 4th year postop. General clinical assessment, including physical examination, ECG, X-Ray, echocardiograhy and 6-minutes walking tests were performed.

Cox proportional-hazards modeling was used for efficacy and safety analyses. A P value of 0.05 or less was considered to indicate statistical significance.

Results

- 36 patients were enrolled into the trial, being operated on from March 1st 2017 to March 1st 2020.
- Long-term results were followed up to 4 years post operation.
- There were 34 (94,4%) males and 2 (5,6%) females of 40,8 ± 10,5 years of age.
- All the patients were divided into 2 groups. Group I: mesh implantation alone (n = 16), group II mesh implantation + MVR (n = 20).
- Impella 5.0 microaxial pump was implemented in 12 patients in post cardiopulmonary bypass period and prevented postcardiotomy syndrome development.

Preoperative status

- **Total group:** Group I and Group II; (n = 36): LV EDD 7,2 ± 0,8 cm; LV EDV 268,5 ± 50,3 ml; LV EF 24,7 ± 6,7%; NYHA class 3,3 ± 0,5.
- **Group I (n = 16):** LV EDD was 7,1 ± 0,9 cm; LV EF 24,8 ± 5,6%; mean NYHA class 3,2 ± 0,3; none of the group was employable; average number of annual hospitalizations was 4 ± 1.
- **Group II (n = 20):** LV EDD was 7,4 ± 0,9 cm; LV EF 25,8 ± 7,4%; NYHA class 3,3 ± 0,6; none of the group II was employable. Average number of annual hospitalizations was 4 ± 2.

	Pre op	30 days	1 year	2 years	3 years	4 years
LVEDD (cm)	7,2 ± 0,8	6,9 ± 0,1	6,5 ± 0,5	6,4 ± 0,4	6,1 ± 0,5	6,1 ± 0,5
LVEDV (ml)	268,5 ± 50,3	211,3 ± 21,7	186,7 ± 14,4	172,8 ± 18,3	160,8 ± 19,7	160,8 ± 19,7
LV EF (%)	24,7 ± 6,7	32,5 ± 3,3	36,6 ± 4,2	34,6 ± 5,6	36,4 ± 4,8	38,5 ± 5,6
Mitral regurgitation (grade)	2,5 ± 0,8	1,0 ± 0,3	1,0 ± 0,3	1,0 ± 0,3	1,0 ± 0,3	1,0 ± 0,3
6 min walking test (m)	220,0 ± 40,0	-	350,0 ± 20,0	360,0 ± 10,0	380,0 ± 25,0	390,0 ± 35,0
NYHA class	3,3 ± 0,7	-	2,0 ± 0,5	1,8 ± 0,6	1,8 ± 0,6	2,0 ± 0,5

Table 1: Total group (AKOR® mesh alone + mesh/MVR) (n = 36).

Figure 3: Actuarial 4 years survival in total group (AKOR® mesh alone and AKOR® mesh + MVR implantation).

The 4 years actuarial survival is 79,6 ± 6,8%. The progression of LV remodeling was stopped: no cases of LV EDD, LV EDV increase were noted. There were no cases of LV EF decrease or mitral valve regurgitation increase.

LV EDD decreased from 7,2 ± 0,8 cm 6,1 ± 0,5 cm ($p < 0.05$). LV EDV decreased from 268,5 ± 50,3 cm to 160,8 ± 19,7 ml ($p < 0.05$).

LV EF increased from 24,7% ± 6,7% to 38,5% ± 5,6% ($p < 0,05$).

Mean mitral regurgitation decreased from 2,5 ± 0,8 to 1,0 ± 0,3 grade ($p < 0,05$).

6 min walking distance increased from 220,0 ± 40,0 m to 390,0 ± 35,0 m ($p < 0,05$).

Mean NYHA functional class changed from 3,3 ± 0,7 to 2,0 ± 0,5 ($p < 0,05$).

There were no cases of intraoperative mortality. 2 patients of Group II died within 30 days postoperatively. 1 patient of Group I and 2 patients of Group II died within the period from the 1st and 2d year after the surgery.

Group I. The AKOR® mesh implantation alone (n = 16).

	Preop	30 days	1 year	2 years	3 years	4 years
LV EDD (cm)	7,1 ± 0,9	6,1 ± 0,8	6,1 ± 0,6	6,1 ± 0,1	6,2 ± 0,1	6,1 ± 0,3
LV EDV (ml)	277,7 ± 64,5	211,5 ± 44,0	220,3 ± 44	210,3 ± 21,5	180,5 ± 20,5	160,0 ± 20,5
LV EF (%)	24,8 ± 5,6	40,2 ± 0,3	42,4 ± 0,2	41,4 ± 0,4	40,2 ± 0,4	40,3 ± 0,6
Mitral regurgitation	1,5 ± 0,5	1,0 ± 0,5	1,0 ± 0,5	1,0 ± 0,5	1,0 ± 0,5	1,0 ± 0,5
6 min walking test (m)	220,0 ± 20,0	-	360,0 ± 20,0	390,0 ± 10,0	400,0 ± 15,0	400,0 ± 20,0
NYHA class	3,0 ± 0,3	-	2,0 ± 0,5	1,8 ± 0,4	1,8 ± 0,3	1,8 ± 0,2

Table 2: AKOR® mesh implantation alone (n = 16).

Figure 4: Actuarial 4-years survival in AKOR® mesh implantation alone.

The 4 years actuarial survival in Group I is 92,7 ± 6,8% ($p < 0.05$).

LV EDD decreased from 7,1 ± 0,9 cm 6,1 ± 0,3 cm ($p < 0.05$). LV EDV decreased from 277,7 ± 64,5 cm to 160,0 ± 20,5 ml ($p < 0.05$).

LV EF increased from 24,8% ± 5,6% to 40,3% ± 0,6% ($p < 0,05$).

Mean mitral regurgitation decreased from 1,5 ± 0,5 to 1,0 ± 0,5 grade ($p < 0,05$).

6 min walking distance increased from 220,0 ± 20,0 m to 400,0 ± 20,0 m ($p < 0,05$).

Mean NYHA functional class changed from 3,3 ± 0,3 to 1,8 ± 0,2 ($p < 0,05$).

1 patient of Group I died during the 4 years period (between the 1st and 2d year post op).

Group II: AKOR® mesh implantation +MVR (n = 20).

	Preop	30 days	1 years	2 years	3 years	4 years
LV EDD (cm)	7,4 ± 0,9	6,9 ± 0,1	6,9 ± 0,1	6,7 ± 0,5	6,5 ± 0,2	6,5 ± 0,2
LV EDV (ml)	280,5 ± 8,0	210,6 ± 6,2	215,4 ± 3,3	190,5 ± 2,5	180,7 ± 4,2	180,2 ± 4,0
LV EF (%)	25,8 ± 7,4	38,5 ± 0,5	35,5 ± 1,7	38,2 ± 0,6	39,4 ± 4,5	36,8 ± 4,4
Mitral regurgitation (grade)	2,6 ± 0,8	-	-	-	-	-
6 min walking test (m)	170,2 ± 10,1	-	360,0 ± 20,2	350,0 ± 10,2	350,2 ± 15,6	330,5 ± 20,5
NYHA class	3,2 ± 0,6	-	3,0 ± 0,1	2,5 ± 0,5	2,2 ± 0,5	2,2 ± 0,6

Table 3: Group II: extracardiac mesh + MVR (n = 20).

Figure 5: 4-years actuarial survival in Group II: AKOR® mesh + MVR (n = 20).

The 4 years actuarial survival in Group II is 70,0 ± 6,8% ($p < 0.05$).

LV EDD decreased from 7,4 ± 0,9 cm 6,5 ± 0,2 cm ($p < 0.05$).

LV EDV decreased from 280,5 ± 8,05 cm to 180,2 ± 4,0 ml ($p < 0.05$).

LV EF increased from 25,8% ± 7,4% to 36,8% ± 4,4 ($p < 0,05$).

Mean mitral regurgitation before surgery was 2,6 ± 0,8 grade ($p < 0,05$).

6 min walking distance increased from 170,2 ± 10,1 m to 330,5 ± 20,5 m ($p < 0,05$).

Mean NYHA functional class changed from 3,2 ± 0,6 to 2,2 ± 0,6 ($p < 0,05$).

2 patient of Group II died during 30 days post op; 3 patients died between the 1st and 2d year post op.

In 7 (19,4%) of 36 patients the trend for regression of IDCM and reverse LV remodeling were noted; in 4 (11%) patients the clinical status improved significantly; in 14 (38,8%) patients the clinical status remained stable.

All in all, the positive effects of extracardiac mesh implantation was achieved in 69,3% of IDCM patients.

	LVEDD preop (cm)	Relative LV walls thickness index	BP syst (mm Hg)	NYHA preop	LVEDD post op (cm)	NYHA post op
Patient 1	6,4	0,31	120	III	5,0	I
Patient 2	6,6	0,37	120	III	5,1	I
Patient 3	6,8	0,43	120	III	5,8	I
Patient 4	6,3	0,32	100	III	5,4	I
Patient 5	7,7	0,30	125	III	5,9	II
Patient 6	6,5	0,32	100	III	5,5	I
Patient 7	6,8	0,32	100	III	5,4	I

Table 4: The subgroup of patients, where LV reverse remodeling was noted.

Relative LV walls thickness index = (LV back wall thickness + Interventricular septum thickness)/LV EDD.

Discussion

Idiopathic dilated cardiomyopathy (IDCM) is a primary myocardial disease of unknown cause [1].

Dilative cardiomyopathy is one of the most common causes of heart failure [2]. It is characterized by ventricular dilatation and systolic dysfunction in the abnormal loading conditions.

It is considered one of the leading causes of heart failure with reduced ejection fraction worldwide. In IDCM heart failure with reduced ejection fraction represents the most frequent cause of death, despite improvements in treatment and is one of the leading indications for heart transplantation [3].

Despite considerable improvements in the medical treatment of heart failure, the "gold standard" for the treatment of these patients

remains heart transplantation. Nevertheless, in consideration of the shortage of donor organs, this procedure can be offered only to a small percentage of patients who could benefit from a new heart. A number of innovative approaches are being investigated in terms of improved survival and quality of life in patients refractory to medical therapy [4].

The pathological end point of dilative cardiomyopathy is cardiac remodeling, characterized my myocyte hypertrophy and chamber dilatation, leading to a more spherical shape of left ventricle and to a reduced pump function [4].

The cardiac stroma plays essential role in cardiac remodeling [5].

The structure of the cardiac stroma depends on collagen. The bulk of the network of structural proteins that make up the extracellular matrix of the heart is composed of collagen type I and type III, which provide structural support for the muscle cells and are crucial for cardiac function. The prognosis and progression of a disease or diseased state may be significantly impacted by the upregulation or downregulation of the collagen types, particularly Col I and Col III. For example, increasing Col I protein levels may impose increasing myocardial stiffness, impairing the diastolic and systolic function of the myocardium. Collagen I is a stiff fibrillar protein that gives tensile strength, whereas Col III produces an elastic network that stores kinetic energy as an elastic rebound. These two collagen proteins have distinct physical properties in nature. Therefore, the control of Col I and Col III as well as the potential relevance of the Col I/Col III ratio in IDCM progression [6].

Heart failure is characterized by major changes in cardiac structure and eventual cardiac dilatation leading to progressive decrease in myocardial function [7].

Progression of heart failure suggests that medications alone may not shield the decline in myocardial structure and function, hence, the search for innovative strategies continued. Surgical approaches which unload the ventricle have been greatly considered [8].

A surgical approach evolved through the introduction of the Acorn CorCap cardiac support device (CSD; Acorn Cardiovascular, Inc., St Paul, Minn.), a mesh-like woven polyester jacket that is surgically placed around in the heart circumferentially tailored to provide assistance during diastole. It is designed to decrease-wall stress-and myocyte overstretching and unloads the heart, thus ventricular remodeling (stretching of the myocardium) may be arrested and even be annulled [8].

Initial studies [9] in 48 patients supported with the device between April 1999 and April 2001 revealed a significant decrease in LV end-diastolic dimension (LVEDD), as early as 3 months post-implantation and further diminution at 6 months. The decrease in LVEDD was sustained during a 3-year follow-up, hence regarded as a remarkable support. The ejection fraction (EF) significantly increased at 3 months reaching an enhanced improvement at 6 months, and sustained within 3 years post-surgery. No manifestation of constriction was evident hemodynamically.

These initial studies demonstrated that the device implantation was safe with no additional surgical adverse events.

Oz., *et al*. [10] implanted the device with international colleagues worldwide, as part of a trial, in more than 130 patients with DCMP with or without simultaneous cardiac procedures. There was a 4.6% mean intraoperative reduction in LVEDD and there were neither device-related intraoperative complications and adverse events nor evident constrictive physiology with maintenance of coronary artery flow reserve, decrease of ventricular size and improvement in ejection fraction and NYHA functional class during a 2-year follow-up. Survival rates were 73% and 68% at 12 and 24 months, respectively. The group involved in the said trial confirmed the safety of the Acorn CorCap CSD device in patients with DCMP. Further randomized clinical trials have been performed then in Europe, Australia, and North America and enrolled 300 patients with Acorn mesh implantation alone and Acorn plus mitral valve repair/replacement. At 5 years the total mortality was 30%. These studies then concluded that the Acorn CorCap CSD was a promising surgical therapy for DCMP [11,12].

The most commonly cited concern about the Acorn CorCap CSD implantation was then the late development of pericardial constriction. However, in a report by Mann., *et al*. [13] during the 5-year follow-up, there have been no cases of pericardial constriction detected during the 5-year follow-up.

Nonetheless, the long-term effects of the Acorn CorCap CSD on my ofiber stress and ventricular function remained largely

unknown and unreported. Further device trials have been disapproved by the FDA in 2008 based on the following issues: (I) NYHA class results are not clinically significant [14], (II) no survival difference between treatment and control groups [14], (III) high perioperative mortality in no-MVR group [14], (IV) severe adhesions and fibrosis [14].

Hence, with the formidable concerns over the long-term pericardial constriction from the excessive adhesions and which could exacerbate heart failure [14], the device was completely withdrawn from the market.

Clinical results demonstrated the need to optimize its design and Acorn fabric orientation to get the maximal myofiber stress reduction [15].

Electromechanical cardioplasty using a wrapped elasto-conductive epicardial mesh were tested in animal experiments [16].

The implantable soft robotic sleeve mimicked the form and function of the native heart, with a stiffness value of the same order of magnitude as that of the heart tissue. It supported failing heart in porcine model of acute heart failure and may have the potential to act as a bridge to transplant for patients with heart failure [17].

Previous soft robotic ventricular assist devices have generally targeted biventricular heart failure and have not engaged the interventricular septum that plays a critical role in blood ejection from the ventricle. We propose implantable soft robotic devices to augment cardiac function in isolated left or right heart failure by applying rhythmic loading to either ventricle. Our devices anchor to the interventricular septum and apply forces to the free wall of the ventricle to cause approximation of the septum and free wall in systole and assist with recoil in diastole. Physiological sensing of the native hemodynamics enables organ-in-the-loop control of these robotic implants for fully autonomous augmentation of heart function [18].

In our research we modified the mesh design. The AKOR® mesh differs from the ACORN CSD: it is more elastic, it covers both ventricles, orientation of strips provide.

Choosing the proper timing for mesh implantation is of a special importance, as well as the proper patient selection. The predictors of reversed remodeling after AKOR extracardiac mesh implantation were: stage of adaptive LV remodeling; relative LV walls thickness index > 0,3; BP syst > 100 mm Hg.

We guess that these minor details led to rather positive clinical results and give a certain hope for the implementation of the AKOR extracardiac mesh in IDCM management.

Conclusions

The AKOR® extracardiac mesh implantation prevents the progression of LV remodeling in IDCM patients, improves functional status and quality of life. It is mostly efficient if implemented at the stage of adaptive LV remodeling when it may stimulate the reverse LV remodeling. The randomized clinical trials are necessary to further evaluate the clinical efficiency of this ®method.

Conflict of Interests Statement

None declared.

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