Turk Gogus Kalp Dama 2015;23(1):19-25
doi: 10.5606/tgkdc.dergisi.2015.9581

Original Article / Özgün Makale

The effect of coronary artery bypass grafting on aortic functions

Koroner arter baypas greftlemenin aort fonksiyonları üzerine etkisi

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ABSTRACT

Background: This study aims to investigate the effect of coronary artery bypass grafting (CABG) on aortic functions.

Methods: Thirty-two consecutive patients (mean age 61.6±9.4 years) with multi-vessel coronary artery disease (CAD) who underwent CABG surgery with cardiopulmonary bypass by the same surgical team were included in this study. The severity of the disease was calculated by the Gensini score index. Aortic function indices such as aortic cross-sectional compliance, aortic distensibility, and aortic stiffness index were calculated by echocardiography preoperatively, and at third and six months postoperatively.

Results: Aortic functions of patients with severe or extensive CAD with high Gensini score (mean 98.3±38.1) improved at postoperative period. Aortic cross-sectional compliance increased from preoperative 2.62±1.5 cm^2/mmHg to 3.52±1.6 cm^2/mmHg at three months (p=0.21), and to 3.79±1.5 cm^2/mmHg at six months (p<0.01) postoperatively. Increase in aortic distensibility was measured as 3.5±2.3 cm^2/dyne $x10^{-6}$, 4.6±2.5 cm^2/dyne $x10^{-6}$ (p=0.33), and 4.8±2.1 cm^2/dyne $x10^{-6}$ (p<0.01) preoperatively, and at three months and six months postoperatively, respectively. Aortic stiffness index decreased from 3.2±0.7 to 2.9±0.6 (p=0.20), and to 2.8±0.4 (p<0.01) preoperatively, at three months and at six months postoperatively, respectively.

Conclusion: Proper revascularization via on-pump CABG with aortic cross-clamp results in a significant improvement in aortic functions.

Keywords: Aortic stiffness; coronary artery disease; coronary bypass surgery.

ÖZ

Amaç: Bu çalışmada koroner arter baypas greftleme (KABG)'nin aortik fonksiyonlar üzerindeki etkisi incelendi.

Çalışma planı: Aynı cerrahi ekip tarafından kardiyopulmoner baypas ile KABG ameliyatı yapılan çokdamar koroner arter hastalığı (KAH) olan ardışık 32 hasta (ort. yaş 61.6±9.4 yıl) çalışmaya alındı. Hastalığın şiddeti Gensini skor indeksi ile hesaplandı. Aortik kesit uyumu, aortik esneklik ve aortik sertleşme gibi aortik fonksiyon indeksleri ekokardiyografi ile ameliyat öncesi dönemde ve ameliyat sonrası üçüncü ve altıncı aylarda hesaplandı.

Bulgular: Aortik fonksiyonlar yüksek Gensini skoru (ortalama 98.3±38.1) ile ciddi veya yaygın KAH'si olan hastalarda ameliyat sonrası dönemde iyileşti. Aortik kesit uyumu ameliyat öncesi 2.62±1.5 cm^2/mmHg'den ameliyat sonrası üçüncü ayda 3.52±1.6 cm^2/mmH'ye (p=0.21) ve altıncı ayda 3.79±1.5 cm^2/mmHg'ye (p<0.01) yükseldi. Aortik esneklikte artış ameliyat öncesinde, ameliyat sonrası üçüncü ay ve altıncı ayda sırasıyla 3.5±2.3 cm^2/dyne $x10^{-6}$, 4.6±2.5 cm^2/dyne $x10^{-6}$ (p=0.33) ve 4.8±2.1 cm^2/dyne $x10^{-6}$ (p<0.01) olarak ölçüldü. Aortik sertlik indeksi ameliyat öncesi, ameliyat sonrası üçüncü ay ve altıncı aydaki dönemde sırasıyla 3.2±0.7'den 2.9±0.6'ya (p=0.20) ve 2.8±0.4'e (p<0.01) azaldı.

Sonuç: Aortik kross klemp ile açık pompa KABG yoluyla düzgün revaskülarizasyon aortik fonksiyonlarda anlamlı iyileşme sağlar.

Anahtar sözcükler: Aortik sertlik; koroner arter hastalığı; koroner baypas cerrahisi.

Available online at
www.tgkdc.dergisi.org
doi: 10.5606/tgkdc.dergisi.2015.9581
QR (Quick Response) Code

Received: November 25, 2013 *Accepted:* June 10, 2014

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Aortic distensibility is the measurement of aortic elasticity and reflects the expandability and stiffness of the aorta. When the aorta loses its elasticity, aortic expandability decreases and aortic function is compromised. In addition, aortic elastic properties determine the left ventricular (LV) function and coronary blood flow in coronary artery disease (CAD). Furthermore, aortic stiffness is an independent predictor of cardiovascular risk, all-cause, and cardiovascular mortality.[1] Recent studies have revealed that age, smoking, diabetes mellitus (DM), familial hypercholesterolemia, hypertension, end-stage renal disease (ESRD), metabolic syndrome, hypothyroidism, and CAD compromise aortic elastic properties, thus inducing functional and histological changes in the aortic wall.[2-4] In patients with CAD, the aortic elastic properties reflect the extent and severity of the disease.[5]

Particularly when extensive impairment is present because of multi-vessel or left main CAD, coronary artery bypass grafting (CABG) surgery is the most reasonable treatment approach. However, myocardial and endothelial damage is still a widely debated problem during the ischemia-reperfusion (IR) sequence in heart surgery. Debates have also arisen regarding aortic cross-clamping, the implantation of the conduits (since it causes direct trauma to the aortic wall), and inflammation.[6-9] Moreover, CABG surgery may compromise aortic functions, which is thought to reflect the extent and severity of the disease.

Previous studies have revealed that the extent and severity of CAD is proportional to aortic function, but the effect of CABG on aortic functions in patients with CAD has yet to be investigated. Therefore, the aim of this study was to investigate this issue.

PATIENTS AND METHODS

Thirty-two consecutive patients with stable CAD and multi-vessel involvement who underwent elective CABG by the same team were included in this study. Patients were excluded who had moderate or severe valve disease, one-vessel CAD, a rhythm other than sinus, aortic aneurysms, congenital heart diseases or cardiomyopathies, severe hepatic or renal impairment, thyroid disease, or severe anemia. In addition, those with porcelain aorta or connective tissue disease were also excluded since these can cause structural changes in the aortic wall.

We obtained both verbal and written informed consent from all of the patients before they were enrolled in the study, and the Institutional Review Board approved the study protocol. The scores for each patient before CABG were calculated using the 2011 European System for Cardiac Operative Risk Evaluation (EuroSCORE) II.

Blood samples were collected from the patients after a fasting period of 12 hours, and the glucose, urea, creatinine, total cholesterol, triglyceride, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol levels were analyzed.

Subsequent to performing a median sternotomy, standard cardiopulmonary bypass (CPB) with moderate systemic hypothermia and topical cooling was used for all patients. Ascending aortic cannulation and two-stage venous cannulation of the right atrium were performed for the CPB, with a cardioplegia delivery cannula being inserted into the ascending aorta. Myocardial protection was achieved by an initial antegrade infusion of a cold crystalloid cardioplegic potassium solution after the application of the aortic cross-clamp, and this was continued along with intermittent antegrade cold blood cardioplegia at the completion of each distal anastomosis via the cardioplegia delivery cannula from the aortic root. All proximal and distal anastomoses were performed during a single aortic cross-clamp period, and the left internal mammary artery (LITA) was the preferred site for arterial grafting in the patients with additional saphenous vein (SV) grafts. Additionally, all of the study participants received warm induction just before the aortic cross-clamp was removed.

Each patient underwent standard transthoracic two-dimensional (2D) and Doppler echocardiography in three sessions: preoperatively within three days of the surgery and postoperatively at the third and six months. After 15 minutes of rest, the echocardiographic measurements were performed via a standard technique using the Vivid 7 ultrasound system with a 2.5-MHz probe (GE Vingmed Ultrasound AS, Horten, Norway) in the left lateral position. All of the echocardiographic measurements were done in three consecutive cycles, and their average was then calculated, with the M-mode being recorded at 100 mm/s. Based on the recommendations for quantitation of the left ventricle by two- dimensional echocardiography by the American Society of Echocardiography Committee on Standards, Subcommittee on Quantitaion of Two-dimensional Echocardiograms, the M-mode measurements of the LV diastolic and systolic diameters and the left atrium systolic diameter were obtained from the image of the parasternal long axis,[10] and the LV ejection fraction (LVEF) was calculated using the modified Simpson's rule. The Doppler echocardiographic recording

allowed for the analysis of the diastolic mitral flow velocities of the E wave (m/s), A wave (m/s), and E/A ratio. The pulsed- wave Doppler tissue (PWDT) sample volume was also placed on the mitral annulus at the lateral LV wall in the apical four-chamber view, and the systolic myocardial velocity, peak velocity of the early diastolic wave (Em), peak velocity of the late diastolic wave (Am), and Em/Am ratio were then measured. The ratio of the early diastolic transmitral inflow velocity to the annular tissue velocity (E/E′) was also measured, and this was used as an index of LV diastolic function. By placing the M-mode stick 3 cm above the aortic valve, traces were obtained, and these were used to calculate the aortic systolic and diastolic diameters.[11] While the systolic diameter was measured from the peak of the aortic trace, the diastolic diameter was obtained from the R peak of the electrocardiogram. The following formulas were used to calculate the aortic cross-sectional compliance (CSC), aortic distensibility (AD), and aortic stiffness index (ASI):[12]

Aortic CSC (cm^2/mmHg)= (AoS-AoD) x AoD/2PP

AD (cm^2/dyne $x10^{-6}$)= 2xAoS-AoD)/AoDx PP.

ASI= ln (SBP/DBP) x AoD/(AoS-AoD).

Furthermore, the pulse pressure (PP) of the left brachial artery was obtained simultaneously via cuff sphygmomanometer and calculated as systolic blood pressure (SBP) minus diastolic blood pressure (DBP) using the fifth Korotkoff sound for DBP.

All of the echocardiographic measurements were made by the same cardiologist. In addition, the intraobserver variability when calculating the aortic root distensibility at our laboratory was 3.4%.

The angiographic evaluations were done by two experienced cardiologists who were blinded to the study, and any discrepancies were solved by consensus. The extent and severity of CAD was assessed using the Gensini scoring system,[13] and these scores were calculated by grading the narrowing of the lumen of the coronary artery as follows: scores of one for 1-30% occlusion, two for 31-50%, four for 51-74%, eight for 75-90%, and 16 for 91-99%. Complete occlusion was given a Gensini score of 32. Next, this primary score was multiplied by a factor that took into account the importance of the position of the lesion in the coronary arterial tree, with it being multiplied by five for the left main coronary, 2.5 for the proximal left anterior descending coronary artery (LAD) or proximal left circumflex artery, 1.5 for the mid-region of the LAD, one for the distal LAD, the mid-distal region of the left circumflex artery, the right coronary artery (RCA), the first diagonal branch, or the obtuse marginal branch, and 0.5 for the second diagonal branch or the posterolateral branch. The final Gensini score was expressed as the sum of these scores.

Statistical analysis

The IBM SPPS Statistics for Windows version 20.0 software package (IBM Corp. Armonk, NY, USA) was used for all statistical analyses, and all of the data was expressed as mean ± standard deviation (SD). Categorical variables were compared via the Fisher's exact test. Normally distributed variables were compared across groups utilizing Student's t-test, whereas variables which were not normally distributed were compared using the Mann-Whitney U test. In addition, the paired sample t-test was used to compare the preoperative aortic root indices to the postoperative values, and Pearson's correlation coefficient was used to evaluate the relationship between the variables. A p value of <0.05 was considered to be statistically significant.

RESULTS

The clinical characteristics of the patients, including age, gender, the risk factors for CAD, the EuroSCORE II and Gensini scores, and the echocardiographic measurements are shown in Table 1.

Our study involved patients with severe and extensive multi-vessel CAD with high Gensini scores (mean: 98.3±38.1) as well as those with a moderate operative risk based on the EuroSCORE II scores (mean: 3.1±1.8). There was no in-hospital mortality, and no major cardiovascular events occurred. All of the patients underwent on-pump CABG with a mean of 3.81±0.78 grafts per patient, and the LITA was used in all patients with additional SV grafts. The mean cross-clamp time was 53±21.4 minutes, but there were no significant correlation between this cross-clamp time and the aortic CSC, AD and ASI at the postoperative third and sixth months.

Severe anemia was diagnosed when the hemoglobin (Hb) concentrations were below 8.0 g/dL, and, as previously mentioned, these patients were excluded from the study. In our study group, the mean Hb was 13.63±1.75 g/dL preoperatively and the perioperative transfusion rates were 2.1±1.66 units of whole blood and 1.26±0.58 units of red blood cells (RBCs) per patient. The postoperative mean Hb values for the third month were 12.58±1.47 g/dL, and there was no statistically significant difference in the pre- and postoperative mean Hb values (p=0.8).

Table 1. Clinical and echocardiographic characteristics of the study population

Characteristics	n	%	Mean±SD
Patients (n)	32		
Gender			
Male	30		
Female	2		
Age (years)			61.6±9.4
Dyslipidemia	13	40	
Hypertension	13	40	
Smoking	14	43	
Diabetes mellitus	12	37	
Family history of premature coronary artery disease	4	12	
Total cholesterol (mg/dL)			182.1±4
Low-density lipoprotein (mg/dL)			106.2±3
High-density lipoprotein (mg/dL)			39.7±8
Triglycerides (mg/dL)			182.2±102
Hemoglobin (g/dL)			13.6± 1.8
Number with transfused whole blood			2.1±1.7
Number with transfused red blood cells			1.3±0.6
Mean EuroSCORE II score			3.1±1.8
Mean Gensini score			98.3±38.1
Left ventricular end-diastolic diameter (mm)			5.0±0.4
Left ventricular end-systolic diameter (mm)			3.3±0.6
Left atrial diameter (mm)			4.1±0.4
Interventricular septal thickness (mm)			1.1±0.8
Posterior wall thickness (mm)			1.1±0.9

SD: Standard deviation; EuroSCORE II: European System for Cardiac Operative Risk Evaluation II.

The pre- and postoperative clinical, echocardiographic characteristics, and aortic function indices of the patients are shown in Table 2. The preoperative and postoperative three and six-month systolic arterial pressures (SAPs) were 122.8±13.8, 121.1±15.1, and 118.8±11.4 mmHg, respectively, and the diastolic pressures were 77.9±4.8, 76.4±7.7, and 77.1±7.2 mmHg. None of these values were statistically significant. In addition, there were no differences in the PP values at the same times (44.8±11.6; 44.6±9.4 and 41.8±7.7 mmHg, respectively), and these also did not reach statistical significance. Furthermore, the preoperative and three and six-month postoperative systolic and diastolic diameters of the aorta were 3.41±0.38 and 3.18±0.41, 3.45±0.30 and 3.16±0.31, 3.50±0.28 and 3.19±0.27 mm, respectively, and these differences were also not statistically significant. Additionally, the pre- and postoperative three and six-month pulsatile changes in the aortic diameter were 0.23±0.14, 0.27±0.12, and 0.31±0.12 mm, respectively ($p=0.13$ and $p=0.01$), and the changes in the sixth month were statistically significantly higher.

The aortic CSC increased from 2.62±1.5 preoperatively to 3.52±1.6 at three months ($p=0.21$) and 3.79±1.5 cm^2/mmHg at six months ($p<0.01$) postoperatively. There was also an increase over the same time periods in the AD from 3.5±2.3 to 4.6±2.5 ($p=0.33$) and 4.8±2.1 cm^2/dyne $x10^{-6}$ ($p<0.01$) and a decrease in the ASI from 3.2±0.7 to 2.9±0.6 ($p=0.20$) and 2.8±0.4 ($p<0.01$), respectively. Furthermore, improvement in aortic function was seen, and this reached statistical significance at the sixth postoperative month.

There was also a statistically significant correlation between the preoperative CSC, AD, and ASI and the Gensini scores ($p<0.001$, $p<0.001$, and $p<0.001$, respectively), and the Gensini scores were positively correlated with smoking ($p=0.014$; $r=0.43$). However, we did not find any significant correlations between the Gensini scores and the CSC, AD and ASI changes between the patients' pre- and postoperative values. The perioperative change in the ASI was positively correlated with hyperlipidemia ($p=0.027$; $r=0.39$), but we found no correlations between age, hypertension, DM, smoking, family history, the LVEF, the LV end-diastolic diameters, the LV end-systolic diameters, the left atrial diameters and the perioperative pre- and postoperative changes in the CSC, AD, and

Table 2. Preoperative and postoperative clinical and echocardiographic parameters of the study population

	Preoperative			Postoperative third month			Postoperative sixth month				
	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	*p* (0-3rd month)	*p* (0-6th month)
SBP (mmHg)			122.8±13.8			121.1±15.1			118.8±11.4	NS	NS
DBP (mmHg)			77.9±4.8			76.4±7.7			77.1±7.2	NS	NS
Pulse pressure (mmHg)			44.8±11.6			44.6±9.4			41.8±7.7	NS	NS
Ejection fraction (%)			48.5±10.4			50±8.1			51.3±7.5	NS	<0.01
E/Em			8.7±2.6			8.1±3.2			7.1±4.7	0.01	<0.01
AoSD (mm)			3.4±0.4			3.5±0.3			3.5±0.3	NS	NS
AoDD (mm)			3.2±0.4			3.2±0.3			3.2±0.3	NS	NS
Pulsatile change in aortic diameter (mm)			0.2±0.1			0.3±0.1			0.3±0.1	NS	0.01
AD (cm^2/dyne $x10^{-6}$)			3.6±2.3			4.6±2.5			4.8±2.1	NS	<0.01
ASI			3.3±0.7			2.9±0.6			2.9±0.4	NS	<0.01
CSC (cm^2/mmHg)			2.6±1.5			3.5±1.6			3.8±1.5	NS	<0.01
Beta blocker	11	34		13	40		12	39		NS	NS
ACE inhibitor/ARB	22	68		25	78		25	78		NS	NS
Ca canal blocker	0	0		2	2		3	3		NS	NS
Acetylsalicylic acid	24	75		31	96		31	96		NS	NS
Statin	23	71		30	93		30	93		NS	NS

SD: Standard deviation; NS: Not significant; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; AoSD: Aortic systolic diameter; AoDD: Aortic diastolic diameter; AD: Aortic distensibility; ASI: Aortic stiffness index; CSC: Cross-sectional compliance; ACE: Angiotensin-converting enzyme; ARB: Angiotensin II receptor blocker.

ASI. Furthermore, the pre- and three and six-month postoperative EFs of the patients were 48.5±10.4%; 50±8.1%, and 51.3±7.5, respectively, and the recovery in the systolic functions of the patients reached statistical significance at the postoperative sixth month (p<0.01). Moreover, the differences in the E/Em ratio were statistically significant (p<0.01) and showed improvement in the diastolic functions.

DISCUSSION

We aimed to demonstrate the effect of revascularization on aortic function, and to the best of our knowledge, this is the first prospective study that has analyzed this via on-pump surgery.

Recent studies have shown acute early deterioration in the aortic functions in patients with aortic valve replacement and aortic root replacement. For example, Barbetseas et al.[12] studied aortic functions after valve replacement in patients with aortic stenosis and showed that early deterioration improved at the sixth month postoperatively. Nemes et al.[14] analyzed the ASI in patients who underwent aortic full root replacement and found non-significant transient deterioration immediately after surgery followed by significant progressive improvement in the AD at the sixth postoperative month. In addition, AD has been studied in patients who underwent different operative techniques, such as the Ross procedure and total aortic root replacement with homografts and xenografts, and the results showed nearly normal AD at the one-year follow-up.[15,16] In order to avoid the acute postoperative effects of aortic wall trauma, inflammation, and anesthetic drugs, we evaluated our patients at the postoperative third and sixth months. Therefore, since no procedure was performed on the aortic root and all of the patients were revascularized to relieve their ischemia, an improvement in aortic functions was seen in our study group.

The relationship between aortic functions and CAD has been previously evaluated. Yildiz et al.[17] showed a direct correlation between the severity of CAD and impaired aortic elasticity and also stated that the impaired aortic elasticity might be an indicator of the presence and severity of CAD. In our study we similarly showed that the AD was negatively correlated and that the ASI was positively correlated with the Gensini scores of our patients. Furthermore, our findings support the fact that aortic root functions can be a predictor of the severity of atherosclerosis, which is evaluated using the Gensini score index.

Tanriverdi et al.[18] studied aortic function impairment on coronary slow flow patients. They compared these patients with those with normal coronary blood flow and concluded that impaired aortic elasticity in the patients with coronary slow flow might also be responsible for their impaired LV diastolic parameters.

Oxidative stress (OS) and endothelial damage are major events that can occur during CABG surgery, and these can be related to CAD severity.[19] Clinical conditions that enhance OS and inflammation act to accelerate the degree of arterial aging and stiffness. Moreover, additional extracorporeal circulation, direct trauma to the aortic wall during cross-clamping, and aortotomies during the operation can lead to injury and inflammation that can further increase aortic stiffness.[20,21] However, in previous studies,[14-16] these effects resulted in a transient deterioration in the recovery of aortic functions in patients with aortic stenosis and aortic aneurysms during patient follow-ups. In our study, we showed a trend toward recovery in aortic functions after revascularization at the third postoperative month, and this reached statistical significance at the sixth postoperative month.

Ozdemir et al.[22] failed to show a relationship between CABG patency and aortic functions in their study composed of 53 patients. They stated that their retrospective study was done on a considerably small number of patients and that prospective studies with a larger sample size might provide a better understanding of this issue. Cay et al.[23] studied 240 patients and showed that the aortic pulse and fractional pulse pressures were significantly and independently higher in patients with occluded SV grafts than for those with patent SV grafts.

Decreased compliance of the central arteries can alter arterial pressure and flow dynamics. When this happens, cardiac performance and coronary perfusion are affected.[2] Some studies have recently found that some lifestyle changes and medical therapies improved aortic functions.[24,25] However, none of these studies investigated the effect of revascularization on aortic functions. Giannattasio et al.[26] studied arterial functions after revascularization by CABG or stenting and showed that revascularization had no effect, and it made no difference whether it was assessed as arterial stiffness or as flow-mediated vasodilatation. These findings seem to contraindicate our findings, but because they used a different technique, it is not possible to adequately compare our results with theirs. As far as we know, our study is the first to show improvement in aortic functions after revascularization. Van Bussel et al.[9] demonstrated that aortic functions deteriorate in patients with CAD and that impaired aortic distensibility may predict severe coronary atherosclerosis. In our study population, the Gensini scores were significantly correlated with the preoperative aortic CSC, AD, and ASI. Dagdelen et al.[25] found that deterioration in aortic functions was related to the common atherosclerotic process and the reduced vasa vasorum flow of the ascending aorta that is supplied by narrowed coronary arteries. Furthermore, according to Ohtsuka et al.,[27] chronic decreases in AD can cause a deterioration in coronary perfusion. In these cases, after revascularization, the perfusion of the ascending aorta increases, which gets rid of the vicious cycle. In turn, this can lead to an improvement in aortic functions. In our study, the revascularization via CABG of the patients with high Gensini scores resulted in improved aortic functions, and this could have been related to dynamic and/or constitutional changes. We used the same interventions during surgery for all of our patients which might have caused constitutional changes in the aortic wall.

Revascularization via CABG does not treat atherosclerosis, but we did see increased myocardial and aortic wall perfusion in our patients. This then led to a recovery in the systolic and diastolic functions of the myocardium. The increased contractility and improved diastolic functions in the LV also led to changes in pressure and volume in the ascending aorta, and the improvement in the CSC, AD, and ASI represent these dynamic changes. Hence, we believe that the alterations in the aortic functions were secondary to the improvement in the LV systolic and diastolic functions.

All of our study participants were in clinical follow-up for stable CAD preoperatively, and the medical treatment of our patients, which was based on current guidelines, was maximized before CABG. In addition, the pharmacological treatment of our patients remained substantially unchanged over the six-month follow-up period.

Our study had a few limitations, one of which was that it only consisted of 32 patients. Moreover, we had no control group of patients who were operated on without the use of a cross-clamp. In spite of these drawbacks, we still believe that our findings were relevant.

In conclusion, our results indicate that in patients with multi-vessel CAD, CABG does not deteriorate aortic functions. In fact, we noted improvement in the aortic functions of the patients in our study. However, further studies on a larger patient group are needed to confirm our findings.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

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