Pectoral Muscle Atrophy After Axillary Artery Cannulation for Aortic Arch Surgery

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To investigate postoperative pectoral atrophy in 141 patients undergoing aortic arch surgery involving bilateral axillary artery cannulations with side grafts. The depth from the skin to the axillary artery surrounding the thoracoacromial artery (zone 1), and the thicknesses of pectoralis major (zone 2) and pectoralis minor (zone 3) were measured by computed tomography before surgery, at 1 and 6 months after surgery, and at the most recent follow-up assessment (PostT2) (mean = 41 months, range 11–75 months). Based on the median value (47.4 mm) of zone 1, the preoperative pectoral thickness was categorized into 2 groups: pectoral thickness >47.4 mm (thick group) and ≤47.4 mm (thin group). Mean changes in the pectoral thickness from baseline were evaluated using the longitudinal mixed-effects model. Forty-three of 110 patients underwent total arch replacements and extra-anatomical bypasses for left subclavian artery anastomoses. In 3 patients, axillary artery grafts became infected. There was no obvious harm associated with muscle wasting. Mean changes from baseline in zones 1, 2, and 3 showed significant declines at PostT2 (-13.40 ± 9.73 mm [$P < 0.0001$], -7.00 ± 5.23 mm [$P < 0.0001$], and -7.23 ± 6.42 mm [$P < 0.0001$], respectively). In the thick group, the progression of pectoral atrophy in zones 1 and 3 was significantly more than that of the thin group ($P < 0.0001$ for both zones). Postoperative pectoral atrophy progressed rapidly. The preoperative pectoral size might be of no use in the prevention of pectoral atrophy. Further investigation to prevent the pectoral atrophy is needed.

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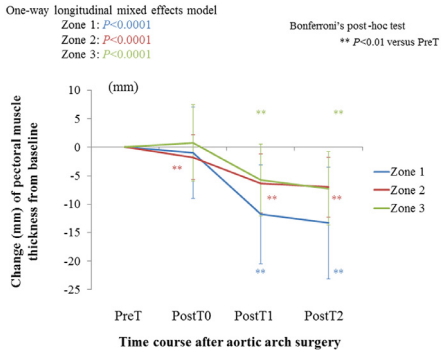
Keywords: Proximal aortic arch surgery, Axillary artery cannulation, Pectoral muscle atrophy, Side graft, LSCA bypass



Changes in pectoral muscle thickness from baseline in zones 1, 2, and 3.

Central Message

Pectoral muscle atrophy in patients undergoing proximal aortic arch surgery including cannulations to bilateral axillary arteries progresses rapidly after surgery.

Perspective Statement

Our data showed that pectoral muscle (PM) atrophy in patients undergoing proximal aortic arch surgery with cannulations to bilateral axillary arteries progressed rapidly after surgery and that preoperative PM thickness might be of no use in preventing the progression of muscle atrophy.

Abbreviations: AxA, axillary artery; CPB, cardiopulmonary bypass; CT, computed tomography; LSCA, left subclavian artery; PAAS, proximal aortic arch surgery; PM, pectoral muscle; PreT, preoperatively; PostT0, 1 month after surgery; PostT1, 6 months after surgery; PostT2, most recent follow-up assessment; TAR, total arch replacement

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INTRODUCTION

Cannulating the ascending aorta to achieve cardiopulmonary bypass (CPB) arterial inflow is a standard procedure. However, axillary artery (AxA) cannulation has become an alternative perfusion site, particularly for proximal aortic arch surgery (PAAS), complex cardiovascular surgery, and for patients with severe aortic atherosclerosis.[1–4] Cannulation-related complications are rare, and the most common complications are brachial plexus and AxA injuries. While brachial plexus injuries cause pectoral muscle (PM) atrophy,[5,6] PM atrophy occasionally occurs after cardiovascular surgery involving AxA cannulation in patients without brachial plexus palsies. PM atrophy may be caused by intraoperative direct injury of the PM and the denervation or damage of the pectoral

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nerves of the brachial plexus, which innervate the pectoralis major or minor, during dissection of the AxA.[7] The side graft used for AxA cannulation is usually oversewn as close to the AxA as possible.[1,4] With progressive PM atrophy occurring after surgery, the remnant of side graft could be a potential site for wound infection. To date, few studies have been undertaken to clarify PM atrophy in patients who undergo cardiovascular surgery that involves AxA cannulation. At our institute, our standard perfusion approach comprises bilateral AxA cannulation via side grafts for PAAS. The aim of this study was to evaluate the degree of PM atrophy after PAAS with bilateral AxA cannulations based on computed tomography (CT) findings.

METHODS

Study Population

This study was approved by our institutional review board, and the need for individual patient consent was waived. Between January 2011 and December 2016, 225 consecutive patients underwent PAAS with selective antegrade cerebral perfusion and moderate hypothermic circulatory arrest at our institute. In principle, the bilateral AxAs and a unilateral femoral artery (multiple perfusion sites) were utilized for the CPB arterial return. Patients who underwent PAAS with selective antegrade cerebral perfusion and moderate hypothermic circulatory arrest involving a unilateral AxA or femoral artery for systemic arterial perfusion ($n = 10$), died ($n = 14$), or were lost to follow-up ($n = 60$) were excluded from the study. Consequently, 141 consecutive patients were retrospectively enrolled to participate in this study. Data for the PM thickness of both sides (left and right) in 141 consecutive patients were used to perform statistical analysis in our study.

Operative Procedures

The standard operative procedure for PAAS at our institute has been described.[8] Briefly, 8-mm Hemashield (Boston Scientific Corp., Natick, MA) or 8-mm Triplex prosthetic grafts (Termo, Tokyo, Japan) were anastomosed to the bilateral AxAs and unilateral femoral artery to achieve systemic arterial perfusion before sternotomy. To cannulate the AxAs, 5−7-cm bilateral infraclavicular incisions were made at 1.5 cm below the middle and lateral parts of the clavicle that extended into the deltopectoral groove. The pectoralis major was split in the direction of the muscle fibers, and the clavipectoral fascia was cut. The pectoralis minor was then exposed, and it was retracted laterally or divided. A sharp dissection gently mobilized the AxA without touching the medial and lateral plexus cords that embrace the artery; this was controlled with loops of silicon elastomer tape. An 8-mm Hemashield graft was anastomosed end-to-side to the AxA (Video 1).

The order in which the proximal or distal end in PAAS was anastomosed was at the surgeon's discretion. We prefer undertaking the proximal anastomosis first.[8] The distal anastomosis was performed during selective antegrade cerebral perfusion and moderate hypothermic circulatory arrest. Reperfusion to the lower body from the femoral artery began after completing the open distal anastomosis. In patients subjected to total or partial arch replacements, the reconstruction of the arch vessels was performed during the systemic rewarming period after systemic reperfusion. In patients with severely diseased left subclavian arteries (LSCAs) or requiring hybrid approaches, including the frozen elephant trunk or the standard elephant trunk with a second-stage thoracic endovascular aortic repair for complex lesions, or in patients with thoracic aneurysms who were undergoing total arch replacements (TARs) using 4-branched prosthetic grafts, the LSCA was reconstructed in an extramediastinal fashion at the left infra subclavian space, and the graft branch passed through the second intercostal space (ie, LSCA bypass).

After completing rewarming, the patient was weaned off the pump. The prosthetic grafts used to cannulate the AxAs were clamped as close to the AxAs as possible, and they were then divided as far as the clamp, and ligated or oversewn.

The closure of the subclavicular incision was performed exclusively by closing the fascia of the pectoralis major. When there was excessive bleeding, the 10F Silastic drain (Ethicon, Inc, Somerville, NJ) was placed in the subclavicular wound space. The drain was connected to a J-VAC suction reservoir with a 100 mL capacity.

Computed Tomography Imaging and Data Analyses

In addition to the CT scans carried out before surgery, the standard follow-up assessments included CT scans at approximately 1 month and 6 months postoperatively, followed by imaging every 6 months for the first 2−3 years, and annually thereafter. During CT imaging, the depth from the skin to the AxA (zone 1), the thickness of the pectoralis major (zone 2), and the thickness of the pectoralis minor and the fat tissue (zone 3) were measured bilaterally at the site of the thoracoacromial artery (Fig. 1). The variables that were measured preoperatively (PreT), at approximately 1 month after surgery (PostT0), at approximately 6 months after surgery (PostT1), and at the most recent follow-up CT assessment (PostT2: mean follow-up 41 ± 21 months after surgery; range 11−75 months) were analyzed statistically.

Definition of Clinical Parameters

Partial arch replacement was defined as anastomosis of the brachiocephalic artery, or both the brachiocephalic and left common carotid artery, using the thoracic aortic surgical procedure. TAR was defined as the anastomosis of supra-aortic vessels (brachiocephalic artery, left common carotid artery, and LSCA) using the thoracic surgical procedure.

Statistical Analyses

Data were analyzed using IBM SPSS software, version 24.0 for Windows (IBM Corporation, Armonk, NY). Summary results for numeric variables were presented as mean ± standard deviation (SD). Data for mean changes from the baseline

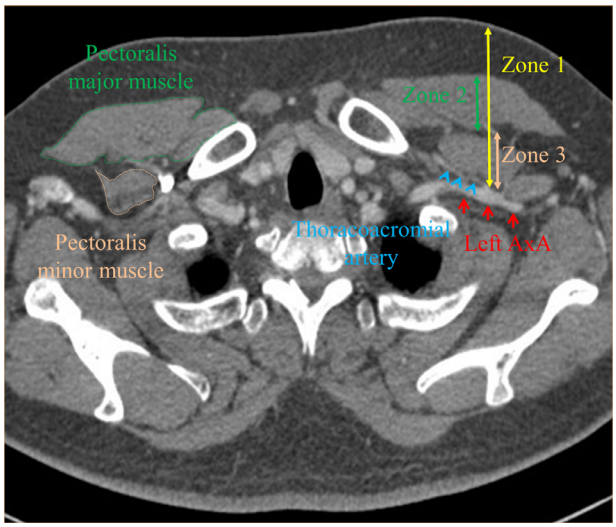


Figure 1. Both sides of shoulder were measured at 3 sites, namely, zone 1, which represented the depth from the skin to the axillary artery (AxA), zone 2, which represented the pectoralis major thickness, and zone 3, which represented the thickness of the pectoralis minor and the fat tissue, around the thoracoacromial artery. The areas enclosed by the green or peach dotted lines show the pectoralis major and pectoralis minor, respectively, and the blue and red arrows indicate the thoracoacromial artery and AxA, respectively. AxA, axillary artery. (Color version of figure is available online at http://www.semthorcardiovascsurg.com.)

in the PM thickness of zones 1, 2, and 3 were used to perform statistical analysis. Preoperative PM thickness in zone 1 was 46.44 ± 10.01 mm (median: 47.4 mm, interquartile range [IQR]: 40.3–52.7 mm). Based on the median value of zone 1, patients were classified into 2 groups: preoperative PM thickness >47.4 mm (thick group, $n = 139$) and ≤47.4 mm (thin group, $n = 143$). Differences were analyzed using one-way or two-way longitudinal mixed-effects model analysis of variance, followed by a post hoc Bonferroni's test. A two-way mixed-effects model with repeated measures was appropriate to evaluate the association between the mean change in PM thickness between a preoperative value at PreT (baseline) and each group of the patients (A: right side [$n = 141$] vs left side [$n = 141$], B: with [$n = 43$] vs without [$n = 98$] a LSCA bypass, and C: the thick group vs the thin group). The mixed-effects model analysis of B was performed using sample data of left PM thickness. A P value <0.05 was considered statistically significant.

RESULTS

Table 1 presents the patients' characteristics and surgical data. A total of 24 patients (17%) underwent emergent or urgent PAAS. The surgical procedures comprised TARs in 110 (78%), partial arch replacements in 13 (9%), and ascending aortic replacements, including hemiarch replacements, in 18 patients (13%). Forty-three patients (39.1%) underwent TARs including a LSCA bypass. There was no operative or

Table 1. Patients' Characteristics and Surgical Data

Variable	Overall
Age, y	70 ± 11
Octogenarian	28 (20)
Male	90 (64)
Emergent or urgent surgery	24 (17)
Chronic renal dysfunction (serum creatinine ≥2.0 mg/dL)	6 (4)
COPD ($FEV_{1.0}$% < 60% and/or drug use of prednisolone)	29 (21)
Previous cerebrovascular accident	25 (18)
Preoperative EF, %	66.6 ± 6.8
Atherosclerotic aneurysm	79 (56)
Shaggy aorta	21 (15)
Acute aortic dissection	23 (16)
Previous cardiac surgery via median sternotomy	5 (4)
Type of operative procedure	
Total arch replacement	110 (78)
Partial arch replacement	13 (9)
Ascending aortic replacement	18 (13)
Concomitant procedure	68 (34)
Aortic root repair	3 (2)
CABG	25 (18)
Frozen elephant trunk technique	12 (9)
Extra-anatomical bypass	43 (30)
CPB time, min	278 ± 52
Coronary ischemic time, min	87 ± 41
HCA time, min	69 ± 26
SACP time, min	148 ± 52
Lowest rectal temperature, °C	26.5 ± 4.9
Operation time, min	617 ± 106

CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; CPB, cardiopulmonary bypass; $FEV_{1.0}$%, forced expiratory volume in 1 s percent; HCA, hypothermic circulatory arrest; SACP, selective antegrade cerebral perfusion.
The data presented are the means and standard deviations or the numbers (percentages).

in-hospital mortality. The overall incidence of cerebral stroke and paraplegia or paraparesis was 7.8% ($n = 11$, 7 [6.0%] in elective procedures and 3 [12.5%] in emergent or urgent procedures) and 2.1% ($n = 3$, 2 [1.7%] and 1 [4.2%]), respectively. No mediastinitis was observed after surgery. Three patients had remnant axillary graft infections (2 [1.7%] and 1 [4.2%]). Moreover, a patient undergoing an elective TAR had bilateral pseudoaneurysms around the remnant axillary grafts (Table 2 and Fig. 2). In case 3, the LSCA bypass in the TAR was ligated and resected at a position that was far from infected axillary graft.

None of the patients experienced arm ischemia, AxA injuries, or aortic dissections. Four patients (2.6%) had postoperative brachial plexus injuries that included 1 case with a mild right-hand weakness and 3 cases with left-hand weaknesses. The symptoms in all of these patients were alleviated before discharge.

Table 2. Complications in the Axillary Artery Area

Case	Age/Sex	Primary Surgery	Cause of Complication	Site of Complication	Extra-anatomical Bypass	Interval Between Primary Surgery and Treatment (mo)	Treatment of AxA
1	79/M	TAR	I	R	N	4	Re, PA
2	88/F	TAR	I	L	N	32	Re, PA
3	80/M	TAR	I	L	Y	13	Re, Direct closure
4	77/F	TAR	P	Both	N	32	Re, PA

AxA, axillary artery; F, female; I, infection; L, left; M, male; N, none; P, pseudoaneurysm; PA, patch angioplasty; R, right; Re, remove; TAR, total arch replacement; Y, yes.

Patch angioplasty was performed by using the saphenous vein. In case 3, the prosthetic graft of the extra-anatomical bypass for the left subclavian artery anastomosis was ligated and resected to a position far from the infected axillary graft. The prosthetic graft anastomosed to the left subclavian artery was removed, and then the left subclavian artery was repaired by direct suturing.

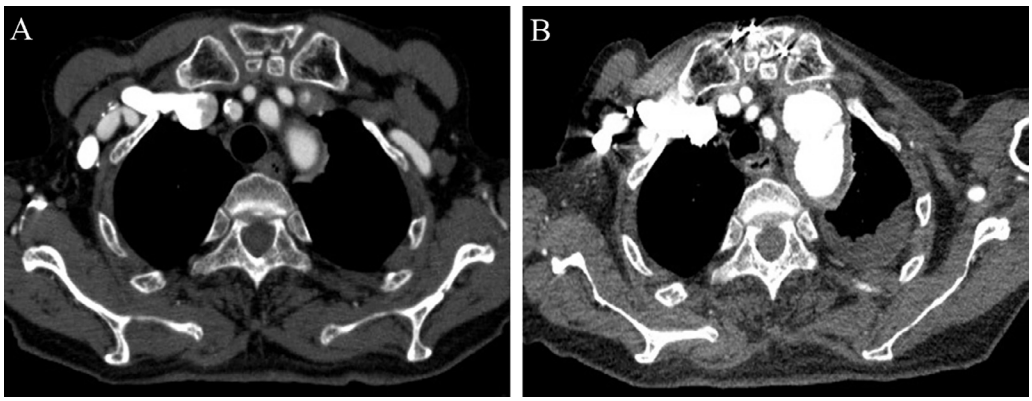


Figure 2. Computed tomographic images from case 2 (A) before and (B) 32 months after surgery. Remarkable pectoral muscle atrophy that had occurred on both sides is evident in panel B. This patient underwent a total arch replacement for an ascending and arch aneurysm that was 70-mm wide at its maximum diameter. At 32 months after surgery, the remnant graft of the left axillary artery was removed, because the prosthetic graft was infected.

Mean Changes From the Baseline in Preoperative Muscle Thickness of Zones 1, 2, and 3

The preoperative PM thicknesses in zones 1, 2, and 3 were 46.44 ± 10.01 mm (median: 47.4 mm, IQR: 40.3−52.7 mm), 16.88 ± 5.19 mm (median: 16.1 mm, IQR: 13.4−20.5 mm), and 19.61 ± 5.70 mm (median: 19.9 mm, IQR: 15.7−23.4 mm), respectively.

Central Picture and Supplementary Table E1 show mean changes from the baseline of PM muscle in zones 1, 2, and 3, respectively. In each zone, there were constant reductions in PM thickness relative to the baseline values ($n = 282$; 141 on the right side plus 141 on the left side; $P < 0.0001$ for all zones).

Comparison of Mean Changes From the Baseline in Pectoral Muscle Thickness Between A: the Right and Left Sides, B: in Patients With and Without a LSCA Bypass, and C: the Thick Group and the Thin Group

Tables 3−5, Supplementary Figures E1−E3 show mean changes over time from the baseline in PM thickness in A: the left and the right sides, B: in patients with and without a LSCA bypass, and C: in patients of the thick and the thin group, in zones 1, 2, and 3, respectively. Moreover, the estimates of mean change from the baseline in zones 1, 2, and 3 are shown in Supplementary Tables E2−E4, respectively.

Zone 1

A: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). In addition, mean change over time from the baseline in the right PM thickness was significantly less than that of in the left ($P < 0.0001$). Moreover, the decrease over time of right PM thickness was significantly more than that of the left ($P = 0.038$).

B: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). There was no difference in mean change from the baseline in PM thickness between both groups ($P = 0.058$). Moreover, the interaction between group and time was not statistically significant ($P = 0.079$).

C: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). In addition, the mean change over time from the baseline in PM thickness in the thick group was

Table 3. Longitudinal Data in Pectoral Muscle Thickness of Zone 1

	PreT	PostT0	PostT1	PostT2	*P* (Time)	*P* (Group)	*P* (Interaction)
(A) Right vs left							
Right side					<0.0001	<0.0001	0.038
Mean (SD), mm	0	−2.54 (7.74)	−12.62 (9.05)	−14.12 (9.81)			
Median (IQR), mm	0	−2.2 (−8.5, 3.1)	−12.7 (−18.0, −6.7)	−15.0 (−19.7, −7.2)			
Left side							
Mean (SD), mm	0	0.52 (8.01)	−10.95 (8.33)	−12.68 (9.65)			
Median (IQR), mm	0	0.9 (−4.0, 5.0)	−11.4 (−16.3, −6.5)	−11.3 (−18.7, −6.4)			
(B) With vs without a LSCA bypass							
With					<0.0001	0.058	0.079
Mean (SD), mm	0	1.07 (7.85)	−8.55 (8.26)	−10.55 (9.48)			
Median (IQR), mm	0	1.4 (−3.8, 5.9)	−8.5 (−14.5, −2.8)	−10.2 (−15.7, −6.2)			
Without							
Mean (SD), mm	0	−1.39 (7.99)	−12.37 (8.69)	−13.92 (9.71)			
Median (IQR), mm	0	−1.6 (−6.5, 4.1)	−12.3 (−17.5, −6.8)	−13.7 (−19.2, −7.1)			
(C) Thick group vs thin group							
Thick					<0.0001	<0.0001	<0.0001
Mean (SD), mm	0	−3.29 (8.06)	−14.70 (8.95)	−16.48 (10.10)			
Median (IQR), mm	0	−3.0 (−8.5, 1.6)	−14.0 (−20.7, −8.9)	−16.0 (−22.0, −10.1)			
Thin							
Mean (SD), mm	0	1.20 (7.33)	−8.95 (7.49)	−10.41 (8.38)			
Median (IQR), mm	0	1.4 (−3.4, 5.6)	−8.9 (−13.9, −4.5)	−10.2 (−16.3, −4.2)			

IQR, interquartile range; LSVC, left subclavian artery; PM, pectoral muscle; PreT, preoperatively; PostT0, 1 month after surgery; PostT1, 6 months after surgery; PostT2, the most recent follow-up assessment; SD, standard deviation.
P values were tabulated for time effect, group effect, and interaction by two-way longitudinal mixed-effects model analysis of variance, followed by a post hoc Bonferroni's test.

Table 4. Longitudinal Data in Pectoral Muscle Thickness of Zone 2

	PreT	PostT0	PostT1	PostT2	*P* (Time)	*P* (Group)	*P* (Interaction)
(A) Right vs left							
Right side					<0.0001	0.371	0.483
Mean (SD), mm	0	−2.12 (3.94)	−6.34 (5.17)	−7.07 (5.40)			
Median (IQR), mm	0	−1.6 (−4.5, 0.2)	−5.4 (−9.0, −2.9)	−6.4 (−10.2, −3.7)			
Left side							
Mean (SD), mm	0	−1.37 (3.94)	−6.39 (5.19)	−7.02 (5.00)			
Median (IQR), mm	0	−1.4 (−3.8, 1.2)	−6.0 (−10.1, −3.2)	−7.1 (−10.1, −3.9)			
(B) With vs without a LSCA bypass							
With					<0.0001	0.437	0.384
Mean (SD), mm	0	−0.62 (4.34)	−5.85 (5.53)	−7.14 (4.95)			
Median (IQR), mm	0	0.3 (−4.2, 2.8)	−5.2 (−9.1, −2.7)	−7.1 (−11.0, −3.8)			
Without							
Mean (SD), mm	0	−1.95 (3.85)	−6.46 (5.11)	−7.03 (5.25)			
Median (IQR), mm	0	−1.6 (−4.0, 0.4)	−5.8 (−9.3, −3.2)	−6.7 (−10.1, −3.7)			
(C) Thick group vs thin group							
Thick					<0.0001	0.073	0.070
Mean (SD), mm	0	−1.90 (4.01)	−6.90 (5.52)	−7.68 (5.42)			
Median (IQR), mm	0	−1.7 (−4.6, 0.7)	−5.9 (−10.5, −3.3)	−7.1 (−11.5, −4.0)			
Thin							
Mean (SD), mm	0	−1.60 (3.89)	−5.85 (4.78)	−6.43 (4.91)			
Median (IQR), mm	0	−1.2 (−3.7, 0.8)	−5.2 (−8.4, −2.8)	−6.1 (−9.6, −3.6)			

IQR, interquartile range; LSVC, left subclavian artery; PM, pectoral muscle; PreT, preoperatively; PostT0, 1 month after surgery; PostT1, 6 months after surgery; PostT2, the most recent follow-up assessment; SD, standard deviation.
P values were tabulated for time effect, group effect, and interaction by two-way longitudinal mixed-effects model analysis of variance, followed by a post hoc Bonferroni's test.

Table 5. Longitudinal Data in Pectoral Muscle Thickness of Zone 3

	PreT	PostT0	PostT1	PostT2	*P* (Time)	*P* (Group)	*P* (Interaction)
(A) Right vs left							
Right side					<0.0001	<0.0001	0.028
Mean (SD), mm	0	−0.49 (6.11)	−6.54 (6.33)	−7.98 (6.27)			
Median (IQR), mm	0	−0.3 (−5.0, 3.5)	−6.0 (−10.6, −2.2)	−8.1 (−12.3, −3.8)			
Left side							
Mean (SD), mm	0	2.00 (7.05)	−4.97 (6.21)	−6.57 (6.44)			
Median (IQR), mm	0	1.5 (−2.2, 4.9)	−4.6 (−9.6, −1.0)	−6.1 (−11.1, −2.3)			
(B) With vs without a LSCA bypass							
With					<0.0001	0.027	0.002
Mean (SD), mm	0	1.94 (6.99)	−2.87 (6.44)	−4.18 (6.58)			
Median (IQR), mm	0	1.1 (−3.1, 4.5)	−2.0 (−6.9, 1.6)	−3.5 (−9.0, −0.6)			
Without							
Mean (SD), mm	0	0.54 (6.64)	−6.27 (6.16)	−7.83 (6.20)			
Median (IQR), mm	0	0.4 (−3.3, 4.3)	−5.8 (−10.5, −2.1)	−8.0 (−12.3, −3.8)			
(C) Thick group vs thin group							
Thick					<0.0001	<0.0001	<0.0001
Mean (SD), mm	0	−1.00 (6.48)	−7.91 (6.12)	−9.47 (6.29)			
Median (IQR), mm	0	−1.5 (−5.3, 3.0)	−7.9 (−12.9, −4.0)	−9.8 (−13.5, −5.8)			
Thin							
Mean (SD), mm	0	2.46 (6.50)	−3.66 (5.78)	−5.14 (5.76)			
Median (IQR), mm	0	2.4 (−1.3, 5.8)	−3.6 (−7.0, 0.2)	−4.9 (−9.1, −0.8)			

IQR, interquartile range; LSVC, left subclavian artery; PM, pectoral muscle; PreT, preoperatively; PostT0, 1 month after surgery; PostT1, 6 months after surgery; PostT2, the most recent follow-up assessment; SD, standard deviation.
P values were tabulated for time effect, group effect, and interaction by two-way longitudinal mixed-effects model analysis of variance, followed by a post hoc Bonferroni's test.

significantly less than that of in the thin group ($P < 0.0001$). The progression of PM atrophy in the thick group was significantly more than that of in the thin group ($P < 0.0001$).

Zone 2

Both groups of A, B, and C had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$ for all groups). Meanwhile, there were no significant differences in mean changes from the baseline between both groups in A, B, and C ($P = 0.371$, $P = 0.437$, and $P = 0.073$, respectively). Moreover, no differences in interactions between groups and time in A, B, and C were found ($P = 0.483$, $P = 0.384$, and $P = 0.07$, respectively).

Zone 3

A: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). In addition, the mean change over time from the baseline in right PM thickness was significantly less than that of in the left ($P < 0.0001$). The decrease over time of the right PM thickness was significantly more than that of the left ($P = 0.028$).

B: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). In addition, the mean change over time from the baseline in PM thickness in patients with a LSCA bypass was significantly more than that of patients without ($P = 0.027$). Moreover, the progress over time of PM atrophy in patients with a LSCA bypass was significantly less than those without ($P = 0.002$).

C: Both groups had a significant decline in mean change over time from the baseline in PM thickness ($P < 0.0001$). In addition, the mean change over time from the baseline in PM thickness in the thick group was significantly less than that of in the thin group ($P < 0.0001$). The progression of PM atrophy in the thick group was significantly more than that of in the thin group ($P < 0.0001$).

DISCUSSION

The key findings from this study were that (1) PM atrophy progressed rapidly after surgery and (2) preoperative PM size might be of no use in preventing the progress of muscle atrophy. Of note, a total of 4 patients (2.8%) experienced complications as a direct result of AxA cannulation, which included graft infections and pseudoaneurysms.

Although the femoral artery is the most common inflow site for CPBs in cases subjected to complex cardiovascular surgery, this procedure is associated with a significant risk of atherothrombotic embolization that is caused by retrograde perfusion from the CPB.[9,10] Using the AxA as an alternative option for arterial inflow during complex cardiovascular surgery was first

popularized by Sabik et al.[1] AxA cannulation has several advantages over femoral artery and aortic cannulations in terms of avoiding sandblasting effects during CPB,[1,2,4,9,10] the absence of atheromas,[1,2,4,10] and preventing malperfusion during surgery to repair acute type A dissections.[3,4] We believe that multi-inflow sites, including the AxA, reduce the velocity compared with a single inflow site and the risk of atherosclerotic debris detachment.[11,12] Specifically, AxA perfusion has positive effects on the maintenance of uniform circulation in both hemispheres in cases with an incomplete circle of Willis, and acts to avoid spinal cord ischemia, and prevent arterial complications associated with insufficient unilateral perfusion in patients with small physiques.[13] Therefore, utilizing bilateral AxAs and a unilateral femoral artery is our standard perfusion approach for PAAS.

AxA cannulation-related complications occur at frequencies of between 2% and 6.5%.[1–4] Sabik et al showed that AxA cannulation using a side graft significantly reduces the frequencies of brachial plexus injuries, AxA injuries, arm ischemia, and aortic dissection.[1] Since the AxA is often fragile, decannulation of the AxA using a side graft is performed by transecting it, and oversewing or ligating its stump. Surprisingly, PM atrophy progressed rapidly after surgery in the present study. We believe that with the progression of PM atrophy, the remnant of the side graft may represent a potential site for wound infection. Therefore, the side graft used for AxA cannulation should be removed after CPB termination.

During TAR, the LSCA anastomosis and the distal anastomosis of the descending aorta can be difficult to perform, because they are located deeply within the surgical field. Therefore, ligating the LSCA, bypassing the LSCA with a side arm from the 4-branched prosthetic graft, that is, an extra-anatomical bypass, and performing a more proximal distal anastomosis combined with a stented or nonstented elephant trunk implantation is an alternative and reliable technique for completing TARs.[14,15] Meanwhile, Cui et al showed that ligation of the LSCA in TAR procedures is an effective approach for selected patients with insufficient LSCA exposure.[16] However, following LSCA ligation, it has previously been reported that the incidence of left-arm ischemia was approximately 36%.[17] With the progression of PM atrophy after surgery, a LSCA bypass could be a potential site of wound infection after surgery, as is the remnant graft used for the AxA cannulation. Once the subclavicular wound infection extends to a LSCA bypass graft, it is difficult to cure the graft infection. Therefore, in cases using a LSCA bypass in TAR, it is suggested that the bypass graft be placed in the left intrathoracic cavity as far as possible to avoid exposure of the graft on the cutaneous surface.

Several possible explanations could account for the progression of PM atrophy over time in patients undergoing AxA cannulation for PAAS. The more likely mechanism contributing to PM atrophy could involve direct muscle injuries caused by incisions, muscle ischemia associated with the division of arterial branches or surgical wound suturing, and muscle denervation caused by injuries to the brachial plexus or pectoral nerve branched from the brachial plexus.[5,6] Hoffman et al showed that the transection of both the medial and the lateral pectoral nerve leads to severe atrophy of the muscle after a modified radical mastectomy based on the anatomy of the pectoral nerve supply to the pectoralis major and minor.[7] The pectoralis major is innervated by both the medial and lateral pectoral nerves. The distal branches of the medial pectoral nerve pass through the pectoralis minor to innervate the lower portion of the pectoralis major. The lateral pectoral nerve supplies the upper portion of the pectoralis major.[7] The division of the pectoralis minor may lead to more severe muscle atrophy than its retraction. Moreover, Borisov et al showed that the denervation of skeletal muscle results in the progressive impairment of its functional properties atrophy of muscle fibers, and a rapid loss of up to 70–85% of its tissue mass during the first several months following denervation.[18] The trend in PM atrophy over time in the present study is congruent with that reported by Borisov et al.[18] Understanding the anatomy of fine nerves supplying the pectoralis major and minor may contribute to the prevention of muscle atrophy. In addition, a subclavian wound closure that involves closely suturing the PM to eliminate the dead space is likely to worsen muscle ischemia by reducing the blood supply. We closed the fascia only of the pectoralis major to prevent muscle ischemia caused by the aforementioned suturing method. Limited use of the electrical cauterizer must also be considered. A less invasive management of PM is recommended.

In the substudy evaluating confounding factors (right vs left, with vs without a LSCA bypass, and preoperative PM size), interactions between the mean change in PM thickness from the baseline and each confounding factors in zone 2 were not statistically significant. On the other hand, those in zone 3 were statistically significant. The management of the pectoralis major was performed with the same consistent approach in our institute. On the other hand, the management of the pectoralis minor was performed based on the surgeons' choice. Dividing the pectoralis minor for AxA cannulation may impact the progression of PM atrophy after surgery.

LIMITATIONS

There are several limitations to the present study. First, this was a retrospective observational study from a single center that involved a small number of patients. Second, the difference in PM atrophy after surgery observed in patients may reflect operative differences including the LSCA bypass, management of the pectoralis minor (muscle division vs retraction), muscle incorporation in wound closure, and the disturbance of the arterial supply or pectoral nerve of the pectoral muscle. In particular, management of the pectoralis minor by using muscle division would have had a profound impact on muscle atrophy after surgery. Unfortunately, the details on the management of pectoralis minor could not be identified. Therefore, the causes underlying PM atrophy remain unclear due to different compounding factors in patients undergoing PAAS including bilateral AxAs cannulation. Further studies are needed to

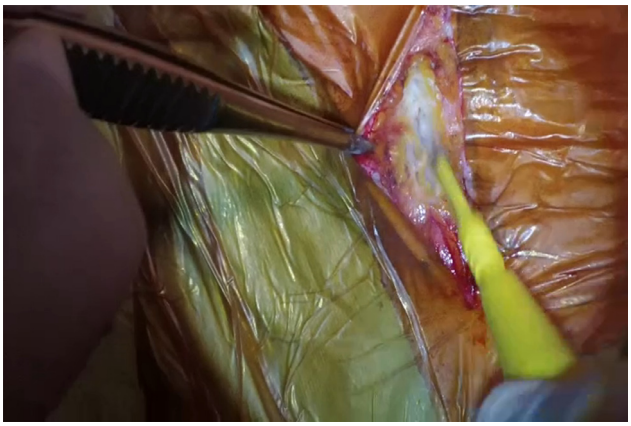
clarify the causes of postoperative PM atrophy. Finally, the follow-up period after surgery was variable among study patients.

CONCLUSIONS

PM atrophy progressed rapidly after surgery. Moreover, preoperative PM size might be of no use in preventing the progress of muscle atrophy. With the worsening of the PM atrophy, the remnant of side graft used for AxA cannulation or a LSCA bypass graft could be a potential site of wound infection. Further investigations on how to prevent PM atrophy after surgery are warranted.

SUPPLEMENTARY MATERIAL

The following is the supplementary data to this article:



Video 1. Right axillary artery cannulation using a prosthetic graft.

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