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ORIGINAL ARTICLE

Quantitative assessment and risk factors for chest wall deformity resulting from tissue expansion for breast reconstruction

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Abstract

Background Chest wall deformity after tissue expansion for breast reconstruction is less recognized than complications such as infection, hematoma, skin necrosis and capsular contracture. However, the condition should not be discounted because pain, rib fracture and dyspnea may occur in severe cases. The aim of this study is to evaluate the extent of chest wall deformity quantitatively using computed tomography (CT) and to identify risk factors for this condition after tissue expansion.
Methods The subjects were 34 patients who underwent unilateral two-stage reconstruction and were examined by multidetector-row CT before expander surgery and during maximal tissue expansion. Chest wall deformity was assessed quantitatively using the Chest Wall Deformity Index (CWDI), which was measured before expander surgery (pre-CWDI) and during maximal tissue expansion (post-CWDI). Post minus pre (post−pre) CWDI was used as the index of chest wall deformity in the assessment of risk factors.
Result Post-CWDI was significantly higher than pre-CWDI ($3.66 \pm 3.23\%$ vs. $0.03 \pm 2.74\%$, $P < 0.001$ by paired t test), showing that chest wall deformity occurred after maximum expansion. In a multiple linear regression model, capsular contracture emerged as a significant predictor of increased post−pre CWDI ($P = 0.003$). BMI was a significant predictor of decreased post−pre CWDI ($P = 0.003$), but this result may have been due to the measurement method.
Conclusions Our findings suggest that chest wall deformity is common after maximum tissue expansion for breast reconstruction. Awareness of the possibility of chest wall deformity during tissue expansion is important, particularly in cases with capsular contracture.

Keywords Chest wall deformity · Breast reconstruction · Computed tomography · Capsular contracture · Expander

Introduction

Two-stage breast reconstruction using a tissue expander is now a commonly performed method. Common complications of this procedure include infection, hematoma, skin necrosis and capsular contracture, but chest wall deformity after tissue expansion is less well recognized. This may be because this condition has only been examined in a few articles that are mostly old, and in which smooth-surfaced and round-shaped expanders were used [1–4]. In addition, most patients with chest wall deformity do not show apparent major symptom. However, this deformity should not be discounted because correlations with complaints of pain, and with rib fracture and dyspnea have been reported in severe cases [4–6].

Capsular contracture [1, 3], preoperative radiotherapy [6], osteoporosis [4, 7] and delayed reconstruction [2] have been reported as possible risk factors for chest wall deformity. However, most of these findings are from case reports and, thus, the factors leading to chest wall deformity are still unclear. The deformity has been evaluated using a 'semi-quantitative' scale of scoring and grading [1, 2, 5], but 'quantitative' measurements have not been used. Therefore, the aim of this study is to evaluate chest wall deformity after maximum tissue expansion by an anatomically shaped expander with a textured surface using a quantitative method based on computed tomography, and to examine risk factors for this condition.

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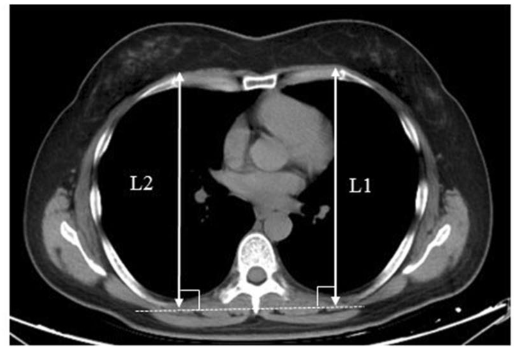
Patients and methods

The study had a nonrandomized, retrospective cohort design and, thus, was granted exemption from institutional Clinical Ethics Committee approval (no. 1297). Between August 2013 and November 2017, a total of 186 Japanese patients with breast cancer underwent two-stage reconstruction using an expander at the Department of Plastic and Reconstructive Surgery and Breast and Endocrine Surgery, Gunma University Hospital. Of these patients, 34 who underwent unilateral two-stage reconstruction and multidetector-row computed tomography (MDCT) scans before expander surgery and during maximal tissue expansion were included in the study. A total of 64, 128 or 320 MDCT scans (Aquilion 64 and ONE, Toshiba, Japan; VCT, GE Healthcare, Japan; Somatom Definition Flash, Siemens, Germany) were used. A textured and anatomical expander (Allergan) was used in all patients. All expanders were placed in a subpectoralis major muscle pocket. No acellular human dermis was used due to restrictions of the Japanese health insurance system.

The chest wall size in a CT image changes slightly depending on the timing of stopping of breathing while recording the CT scan. Therefore, a quantitative index of deformity should be defined relative to the unaffected side preoperatively and during maximum tissue expansion. Therefore, we evaluated chest wall deformity using a quantitative measurement that we refer to as the Chest Wall Deformity Index (CWDI) (Fig. 1). The distance of a perpendicular line drawn from the boundary of the fourth costal bone and cartilage to a line connecting the most posterior point of the chest wall on the right and left was measured on the unaffected (L1) and affected (L2) sides. Pre-CWDI (measured preoperatively) was then defined as (L1 − L2)/L1 × 100 (Fig. 1a), and post-CWDI measured during maximum tissue expansion was defined as (L1′ − L2′)/L1′ × 100 (Fig. 1b). To correct for natural asymmetry of the chest, post−pre CWDI (post-CWDI minus pre-CWDI) was used as the final index of chest wall deformity for evaluation of risk factors (Fig. 1b). CWDI was measured again in patients who underwent further CT examinations at more than six months after the second surgery.

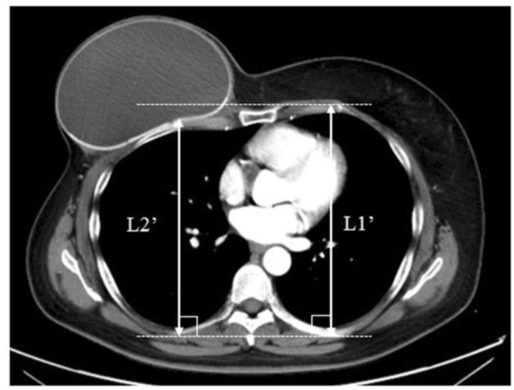
Data were collected for age, body mass index (BMI), size of expander, saline solution volume injected into the expander (volume of initial injection and total volume injected), ratio of total saline solution volume injected to the expander size, immediate (vs. delayed) expander insertion, radical mastectomy involving the skin and nipple–areolar complex (vs. nipple-sparing (NSM) or skin-sparing (SSM) mastectomy), indwelling duration of expander and postoperative complications (with or without

(a)



$$\text{Pre-CWDI (\%) (Chest Wall Deformity Index)} = \frac{\text{L1(mm)} - \text{L2(mm)}}{\text{L1(mm)}} \times 100$$

(b)



$$\text{Post-CWDI (\%) (Chest Wall Deformity Index)} = \frac{\text{L1'(mm)} - \text{L2'(mm)}}{\text{L1'(mm)}} \times 100$$

$$\text{Post–Pre CWDI} = \text{Post-CWDI} - \text{Pre-CWDI}$$

Fig. 1 Chest Wall Deformity Index (CWDI) obtained from a CT image. **a** The distance of the perpendicular line drawn from the boundary of the fourth costal bone and cartilage to the line connecting the most posterior point of the chest wall on the right and left was measured on the unaffected (L1) and affected (L2) sides. Pre-CWDI was defined as (L1 − L2)/L1 × 100 in MDCT before expander surgery. **b** Post-CWDI was similarly defined as L1′ − L2′/L1′ × 100 at maximum tissue expansion. To correct for the natural asymmetry of the chest, post−pre CWDI (post-CWDI minus pre-CWDI) was used as the index of chest wall deformity

infection, seroma, and capsular contracture). Capsular contracture was defined as early capsular contracture with Baker capsules of class II or greater before completion of tissue expansion. No patients underwent preoperative radiotherapy or had postoperative hematoma, and these factors were excluded for the cohort.

Univariate analysis was used to examine the correlation of each variable with post−pre CWDI. Comparisons of categorical variables were performed by Mann–Whitney *U* test or Student's t test, depending on whether the data were normally distributed. Continuous variables were analyzed by Spearman rank correlation test. Variables with $P<0.2$ in univariate analysis were included in multivariate analysis using a stepwise multiple linear regression model to identify factors associated with post−pre CWDI. All data were analyzed using SPSS ver. 25 (Chicago, IL, USA). $P<0.05$ was considered to be statistically significant.

Results

The 34 patients who underwent two-stage reconstruction and MDCT scans before expander surgery and during maximal tissue expansion had a mean age of 48.7 (range 36–68) years and a mean BMI of 25.0 (9.1–34.7) kg/m^2. The average tissue expander size was 382.4 (250–700) mL, the average initial tissue expander fill volume was 126.5 (80–250) mL, and the ratio of the total saline solution volume injected to the expander size was 117.8 (64–160) %. Of the 34 patients, 28 underwent immediate expander surgery and 6 had delayed surgery. The mean indwelling duration of expander was 286.8 ± 99.3 (range 85–577) days. The mean interval from mastectomy to delayed expander surgery of all six patients was 35.8 ± 41.0 (range 11–118) months. 19 underwent radical mastectomy involving the skin and nipple–areolar complex, while 15 received NSM or SSM. Postoperative infection, seroma, and capsular contracture occurred in 2 (5.9%), 4 (11.8%), and 5 (14.7%) cases, respectively.

The mean post-CWDI was significantly higher than the mean pre-CWDI (3.66 ± 3.23% (range −2.7 to 8.8%) vs. 0.03 ± 2.74% (range −8.2 to 4.4%), $P<0.001$ by paired t test; Fig. 2). This result clearly shows that chest wall deformity occurred after maximum expansion, but no rib fracture or dyspnea occurred in any patients. The post−pre CWDI was 3.63 ± 2.86% (range −0.97 to 11.90). In univariate analysis, capsular contracture was a significant factor related to post−pre CWDI (Tables 1, 2). Multiple linear regression showed that capsular contracture was a significant predictor of an increased post−pre CWDI ($P=0.003$), and BMI ($P=0.027$) was a significant predictor of a decreased post−pre CWDI (Table 3).

Further CT examinations and measurements of CWDI at more than six months after the second surgery (mean 19.0 ± 7.7 months: range 6–34 months) were performed in 14 of the 34 patients. The mean CWDI at this time was 1.58 ± 3.16% (range −7.4 to 6.7%).

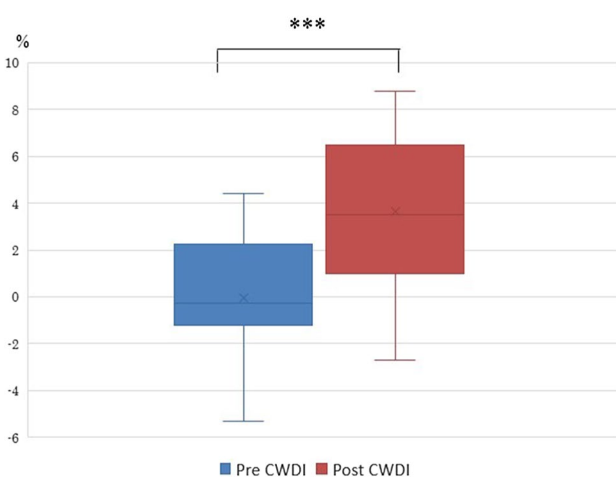


Fig. 2 Comparison between pre- and post-CWDI (%). Post-CWDI was significantly higher than pre-CWDI ($P<0.001$ by paired t test)

Discussion

Chest wall deformity during maximal tissue expansion is not commonly recognized, despite a rate of occurrence of 53.0–77.8% [1, 2, 5]. This may partly be because most patients with this deformity do not complain of major symptoms. However, Cherbino et al. found that all patients with chest wall deformity complained of pain after the expansions, whereas pain was not present in those without chest wall deformity [5]. Also, a year after the implant, chest wall deformity had not improved in 72.1% of cases, and rib fracture and dyspnea occurred in severe cases [4, 5]. These findings suggest that it is important to examine the extent of chest wall deformity after maximum tissue expansion and to identify the risk factors for this deformity.

Table 1 Univariate analysis of the relationships of categorical variables with post−pre CWDI

Variables	Cases (%)	Post−pre CWDI (mean ± SD)	P
Expander surgery			
Immediate	28 (82.4%)	3.45 ± 2.66	0.651[b]
Delayed	6 (17.6%)	4.47 ± 3.87	
Resection method			
Radical mastectomy	19 (55.9%)	4.07 ± 2.34	0.567[b]
NSM/SSM	15 (44.1%)	3.07 ± 2.34	
Complication			
With infection	2 (5.9%)	7.33 ± 6.46	0.242[b]
Without infection	32 (94.1%)	3.40 ± 2.54	
With seroma	4 (11.8%)	4.51 ± 5.01	0.525[a]
Without seroma	30 (88.2%)	3.52 ± 2.57	
With capsular contracture	5 (14.7%)	6.50 ± 3.89	0.013*[a]
Without capsular contracture	29 (85.3%)	3.14 ± 2.40	

*$P<0.05$

[a]Student's t test

[b]Mann–Whitney U test

Table 2 Correlations between continuous quantitative variables and post−pre CWDI

Variables	*r*	*P*
Age (years)	0.052	0.768
BMI (kg/m^2)	− 0.257	0.142
Size of expander (mL)	− 0.300	0.085
Initial saline solution volume injected into the expander (mL)	− 0.150	0.396
Ratio of saline solution volume injected to size of expander (%)	0.248	0.157
Indwelling duration of expander (days)	0.274	0.117

r Spearman rank correlation coefficient

Table 3 Significant predictors of chest wall deformity in a logistic multiple linear regression model

Variables	*B*	SE *B*	*β*
Capsular contracture	3.86	1.26	0.48**
BMI (kg/m^2)	− 0.25	0.11	− 0.35*
Size of expander (mL)	n.s	n.s	n.s
Ratio of saline solution volume injected to size of expander (%)	n.s	n.s	n.s
Indwelling duration of expander (days)	n.s	n.s	n.s
R^2	0.30		
F	5.36*		

B partial regression coefficient, *SE B* standard error of partial regression coefficient, *β* standardized partial regression coefficient, R^2 coefficient of determination, *n.s.* not significant

*$P<0.05$, **$P<0.01$

We often perform a two-stage approach in flap reconstruction, with transplant of a de-epithelialized flap following insertion of a tissue expander. This avoids the patchwork-like scar around the skin paddle of the flap [8]. All patients in this study received two-stage abdominal flap reconstruction, and all underwent chest and abdominal enhanced CT for assessment of recipient and donor vessels just before the flap insertion surgery. This allowed a retrospective evaluation of chest wall deformity at maximum tissue.

The distance of a perpendicular line drawn from the boundary of the fourth costal bone and cartilage to a line connecting the most posterior point of the chest wall was measured. Post−pre CWDI at the 6th costal bone was significantly smaller than post−pre CWDI at the 4th costal bone (1.61 ± 3.09% vs. 3.63 ± 2.86%, $P=0.008$ by paired *t* test). There was almost no change in the 8th costal bone. Therefore, we used changes in the chest wall at the 4th costal bone to indicate the degree of chest wall deformity. However, more accurate evaluation of the deformity might have been achieved using three dimensions including the transverse diameter, such as analysis with 3D-CT, rather than the single dimension. This is a limitation of the study.

Our quantitative results clearly showed that chest wall deformity occurred after maximal tissue expansion, and that capsular contracture was a significant predictor of increased chest wall deformity. This result is in contrast with the finding of Sinow et al. [1] that capsular contracture is a significant predictor of lower chest wall deformity. The capsule is present around the entire circumference of the expander, and therefore resists distortion in all dimensions, including the thoracic skeleton, and buffers' deformation. Conversely, a few case reports have shown that thoracic deformity might result from tight periprosthetic capsules, as in our results. Dickson and Sharpe attributed chest wall deformity in two patients to tight periprosthetic capsules [3]. In five patients with capsular contracture in our study, the mean ratio of the total saline solution volume injected to the expander size was no less than 144.0% to accomplish the required skin expansion, whereas the mean ratio in all 34 cases was 117.8%.

As stated by Sinow et al. [1], capsular contracture itself, without overinflation, might be negatively correlated with deformity, as a buffer to deformation (Fig. 3a). In a case with overinflation, but without capsular contracture, the expander relatively smoothly expands in all directions and the pressure on the chest wall is relatively low, resulting in only moderate deformity (Fig. 3b). However, in a case with capsular contracture and overinflation, the expander places very strong pressure in all directions, including the chest wall, to accomplish the required skin expansion against capsular contracture, and this is more likely to have a positive correlation with deformity (Fig. 3c).

BMI has not previously been found to be a relevant factor for chest wall deformity after expander reconstruction. In our study, BMI was a predictor of lower chest wall deformity. However, this result might be due to our evaluation method, since a larger BMI tends to be related to a larger thickness of the chest wall (L1′ is larger). In patients with thick and flat chest walls, but with the same chest wall deformity (L2′–L1′ is the same), post-CWDI (L2′ − L1′/L1′ × 100) would be lower in the patient with the thick chest wall. This may explain why BMI was found to be a negative predictor of chest wall deformity in this study.

Moor et al. suggested that delayed expander surgery may be a significant risk factor for chest wall deformity [2], based on the presence of fresh tissues in immediate expansion after mastectomy, generating less scar tissue compared to a delayed procedure. However, in our

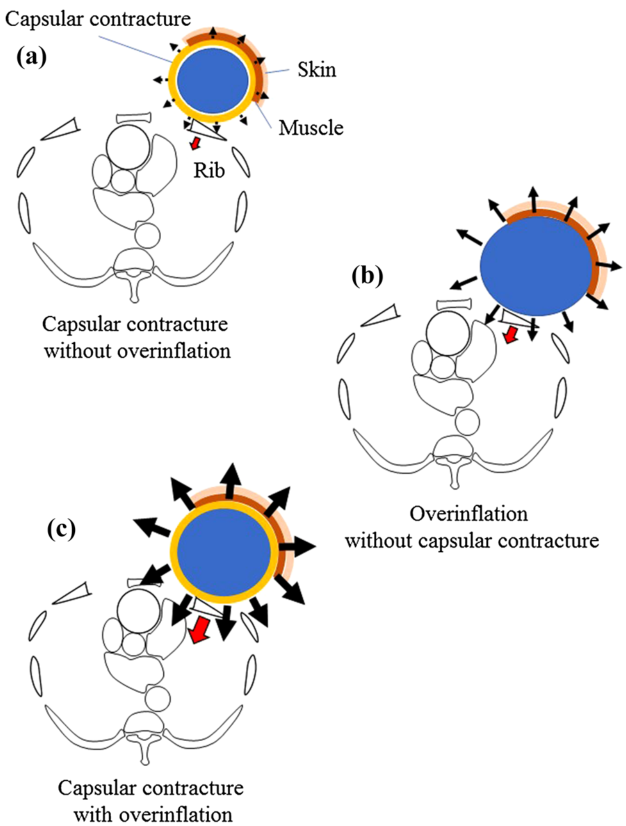


Fig. 3 Effects of capsular contracture and overinflation on chest wall deformity. **a** Capsular contracture alone may be negatively correlated with deformity to buffer deformation, as suggested by Sinow et al. [1]. **b** In a case with overinflation only, the expander smoothly expands in all directions and the pressure on the chest wall is relatively small, resulting in minor deformity only. **c** In a case of capsular contracture with overinflation, the expander exerts very strong pressure in all directions, including on the chest wall, to accomplish the required skin expansion against capsular contracture, and this might cause severe deformity

patients, delayed expansion was not a significant predictor, although chest wall deformity was slightly higher after delayed surgery compared to immediate surgery (post–pre CWDI: 4.47 ± 3.87 vs. 3.45 ± 2.66). These results suggest that a delayed procedure does not greatly influence chest wall deformity, provided that adequate incision or resection of the capsule is carried out in expander insertion. The mean interval from mastectomy to delayed expander surgery in all six patients was 35.8 ± 41.0 months (range 11–118 months). There was no significant correlation between the intervals and pre − post CWDI in delayed cases (Spearman rank correlation test: $P = 0.903$, $r = 0.06$). In all patients except one, the interval from mastectomy to expander surgery was more than 1 year, during which time the scar may have matured and softened. This might be another reason why delayed expansion was not a significant predictor of chest wall deformity.

No rib fracture or apparent dyspnea occurred in any patient in the study. Previous studies suggest that rib fracture or dyspnea occurs with severe deformity [1]. However, a high degree of deformity does not necessarily result in rib fracture or dyspnea [5], and the degree of chest wall deformity that could cause rib fractures or dyspnea is still unclear. In several case reports, preoperative radiotherapy, osteoporosis and steroid intake have been suggested as predictors of chest wall deformity and rib fracture or dyspnea [4, 6, 7], but without significance. None of our patients underwent radiotherapy to the chest wall or mammary region, and none had osteoporosis or steroid intake. Therefore, further studies are needed to evaluate the influence of these factors on chest wall deformity, rib fracture and dyspnea.

At more than 6 months after flap reconstructions, further CT was performed in 14 of the 34 patients and the mean CWDI was 1.58 ± 3.16%. Therefore, chest wall deformity somewhat improved after flap insertion because the mean CWDI during maximal tissue expansion was 3.66 ± 3.23%. However, CWDI at more than 6 months after flap reconstruction was still significantly higher than the preoperative CWDI of 0.03 ± 2.74% ($P = 0.026$ by paired t test). Cherbino et al. found that chest wall deformity had not improved in 72.1% of cases at 1 year after implant insertion. Therefore, deformity may improve in some cases, but in others a greater or lesser degree of deformity may remain after implant or flap insertions. Further studies are needed to evaluate whether the deformity would improve or not, more years after the implant or flap insertions.

In conclusion, we evaluated chest wall deformity quantitatively using CT after maximum tissue expansion for breast reconstruction with an anatomically shaped expander with a textured surface, and examined the related risk factors. Our findings suggest that chest wall deformity is common after this procedure. Therefore, awareness of possible chest wall deformity during tissue expansion is important, particularly in cases with capsular contracture.

Compliance with ethical standards

Conflict of interest The authors have declared that no conflict of interest exists. Funding was from institutional sources only.

References

1. Sinow JD, Halvorsen RA Jr, Matts JP, Schubert W, Letourneau JG, Cunningham BL. Chest-wall deformity after tissue expansion for breast reconstruction. Plast Reconstr Surg. 1991;88:998–1004.
2. Moor EV, Wexler MR, Bar-Ziv Y, Weinberg A, Chaouat M, Ad-El D, et al. Chest wall deformity following maximal tissue expansion for breast reconstruction. Ann Plast Surg. 1996;36:129–32.

3. Dickson MG, Sharpe DT. The complications of tissue expansion in breast reconstruction: a review of 75 cases. Br J Plast Surg. 1987;40:629–35.
4. McKinney P, Edelson R, Terrasse A, Zukowski M. Chest-wall deformity following soft-tissue expansion for breast reconstruction. Plast Reconstr Surg. 1987;80:442–4.
5. Cherubino M, Scamoni S, Maggiulli F, Floridi C, Mangini M, Di Giovanna D, et al. Breast reconstruction by tissue expansion: what is the integrity of the chest wall? J Plast Reconstr Aesthet Surg. 2016;69:e48–54.
6. de Wildt RP, Tuinder S, van der Hulst RR. Substantial chest-wall deformity following tissue expansion after radiotherapy. Eur J Plast Surg. 2009;32:337–40.
7. Sariguney Y, Ayhan S, Eryilmaz T. Chest wall deformity after tissue expansion. Scand J Plast Reconstr Surg Hand Surg. 2008;42:108–9.
8. Kajikawa A, Ueda K, Tateshita T, Katsuragi Y. Breast reconstruction using tissue expander and TRAM flap with vascular enhancement procedures. J Plast Reconstr Aesthet Surg. 2009;62:1148–53.