



Gigantomastia as a Cause of Pulmonary Hypertension

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Abstract

Reduction mammoplasty is the gold standard treatment for gigantomastia. We report one female patient with juvenile gigantomastia associated with severe pulmonary hypertension where her pulmonary pressure decreased significantly after the surgery, improving her quality of life. A 22-year-old female patient with gigantomastia since 10 years old, tricuspid regurgitation, and pulmonary thromboembolism antecedent was admitted to the emergency department. Her oxygen saturation was 89%. Acute heart failure management was initiated. An echocardiogram reported left ventricle ejection fraction (LVEF) of 70% with severe right heart dilation, contractile dysfunction, and arterial pulmonary pressure (PASP) of 110 mm Hg. A multidisciplinary team considered gigantomastia could generate a restrictive pattern, so a Thorek reduction mammoplasty with Wise pattern was performed. Presurgical measurements were: sternal notch to nipple-areola complex, right 59 cm, left 56 cm. Three days after surgery, the patient could breathe without oxygen support. In the outpatient follow-up, patient referred reduction of her respiratory symptoms and marked improvement in her quality of life. Six months after surgery, a control echocardiogram showed a LVEF of 62% and PASP of 85 mm Hg. Pulmonary hypertension may be present in patients with gigantomastia. Reduction mammoplasty may be a feasible alternative to improve the cardiac signs and symptoms in patients with medical refractory management.

Keywords

- ▶ gigantomastia
- ▶ mammoplasty reduction
- ▶ pulmonary hypertension

Gigantomastia can generate different problems at a physic, psychologic, and social level.¹ It can be caused by glandular hypertrophy, excessive fatty tissue, or a combination of both.² Gigantomastia could cause neck, back, shoulders, or/and breast pain, as well as intertrigo or/and postural disorders.³ Some patients have referred breathing difficulties, possibly explained

by the restriction of chest wall compliance generated by excessive breast volume, which increases respiratory effort.^{4,5} Reduction mammoplasty is the gold standard surgical management to improving patient symptoms.^{6,7} The available surgical techniques are nipple-areola complex (NAC) irrigated by a vascular pedicle⁸ or free nipple grafting (Thorek).⁹

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There is no available literature that associates gigantomastia with pulmonary hypertension (PH), but there is a relationship between obesity as a proinflammatory factor and PH. We describe the reduction of pulmonary artery pressure in a woman with gigantomastia after reduction mammoplasty; she agreed and gave us informed consent to publish her photos and data from her medical history.

Case Report

A 22-year-old female patient with gigantomastia since 10 years old, tricuspid regurgitation, and pulmonary thromboembolism antecedent was admitted to the emergency department. At physical examination, she had minimal effort dyspnea, New York Heart Association (NYHA) IV scale, oliguria, orthopnea, and lower extremities edema. Her oxygen saturation (OS) was 89%. Acute heart failure management was initiated. A computed tomography angiography revealed a 43-mm pulmonary artery and right ventricular and atrium dilation. An echocardiogram reported left ventricle ejection fraction (LVEF) of 70% with severe right heart dilation, contractile dysfunction, and arterial pulmonary pressure (PASP) of 110 mm Hg. Anti-deoxyribonucleic acid was positive on 1:160 dilutions and C3 was 61 mcg/dL. Systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS) was suspected. Despite medical management, the patient's symptoms did not improve. A multidisci-

plinary team considered gigantomastia could generate a restrictive pattern, alveolar hypoventilation syndrome, and obstructive sleep apnea/hypopnea syndrome, so a Thorek reduction mammoplasty with Wise pattern was performed. Presurgical measurements were: sternal notch to NAC, right 59 cm, left 56 cm, NAC-NAC distances 37 cm, breast fold-NAC right 40 cm, left 34 cm (►Figs. 1 and 2).

The patient was prepared for general anesthesia with oxygen flow at 2 L/min per cannula. The mammary and periareolar base were infiltrated with 500 mL of 0.9% saline solution plus adrenaline 0.5 mcg/mL. The oncologist surgeon performed the resection of glandular adipose tissue and excess skin. The extracted tissue weighed 3,815 g (right breast) and 2,021 g (left breast). Plastic surgeon performed mammary reconstruction. Subcutaneous cellular tissue was sutured with Vicryl 2.0, dermis with Vicryl 3.0, and the skin with Monocryl 3.0. The NACs were obtained as full-thickness skin grafts at 6 cm of the vertical breast pillar. The surgeon made the cotton bolster secured with sutures.

Three days after surgery, the patient could breathe without oxygen support. Eight days postoperatively, the patient was discharged with NYHA II without oxygen support. In the outpatient follow-up, patient referred reduction of her respiratory symptoms and marked improvement in her quality of life (►Figs. 3 and 4). Six months after surgery, a control echocardiogram showed a LVEF of 62% and PASP of 85 mm Hg.



Fig. 1 A case of gigantomastia which was reconstructed using Thorek reduction mammoplasty. Front view of the preoperative Wise pattern.



Fig. 2 A case of gigantomastia which was reconstructed using Thorek reduction mammoplasty. A preoperative photograph.



Fig. 3 A photograph at follow-up on postoperative 1 month, lateral view.



Fig. 4 A photograph at follow-up on postoperative 1 month, front view.

Discussion

The gigantomastia is a pathology with a high physical, psychological, and social impact. Consequences are diverse including postural disorders to marked impact in quality of life. We report a case of a decrease in pulmonary artery pressure in a patient with PH and severe gigantomastia after reduction mammoplasty. To our knowledge, this is the first case report in the world which describes changes in pulmonary pressure consequently to gigantomastia, which leads us to make two recommendations: First, cardiac pathologies should be ruled out in gigantomastia patients. Second, reduction mammoplasty may be a feasible alternative in patients with gigantomastia and heart failure symptoms and refractory medical management, postulating gigantomastia as partially responsible for the underlying cardiac condition.

Obesity is associated with low-grade systemic inflammation, insulin resistance, and oxidative stress that can exacerbate the vascular remodeling process in patients with PH.¹⁰ Excess adipose tissue in obese patients leads to a mechanical pulmonary restriction and a hypercirculatory state which, at a long term, cause right heart failure.¹¹ Some studies suggest a positive correlation between removed breast tissue and lung function improvement.¹² This phenomenon is possibly attributable to the relative restriction in chest wall compliance gigantomastia patients have which would explain the respiratory symptoms they refer. In our patient, although her body mass index was normal, her severe gigantomastia could

have had repercussions in mechanical (mechanical restriction up the chest) and chemical (proinflammatory state adipose tissue like obesity) dimension. Although SLE and APS were suspected and managed, the fast improvement of patient's symptoms after the surgery made us think gigantomastia could be partially responsible of her PH and right heart failure.

Improvement in OS, forced vital capacity, forced expiratory volume in the first minute, and chest wall compliance in patients with gigantomastia after reduction mammoplasty have been documented.^{12,13} Our patient improved very quickly. Her PASP reduced 23% compared with presurgical measure. She was able to breathe without respiratory support in just 3 days after surgery.

PH may be present in patients with gigantomastia. The cardiovascular condition of patients with gigantomastia should be evaluated. Reduction mammoplasty may be a feasible alternative to improve the cardiac signs and symptoms in patients with medical refractory management.

Authors Contributions

J.P.C. and A.M.R. conceived the study, designed the study, and obtained ethics approval. J.P.C. and L.R.S. were involved in all aspects of the paper generation and revised each draft and coordinated all coauthors' activities. L.R.S. collected all the data. J.P.C., L.R.S., and L.T.C. wrote the different versions. L.R.S. also prepared the figures. All authors provided critical input into all aspects of the

design and execution of the study and participated in all phases of the study.

Patient Consent

Informed consent was obtained from all individual participants included in the study.

Conflict of Interest

None declared.

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