

CASE REPORT

Modified Thorek Mammoplasty for the Treatment of Idiopathic Gigantomastia

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Abstract

Idiopathic gigantomastia is a rare and severe condition characterized by excessive breast enlargement, which often causes significant physical and psychological distress. Although various surgical techniques have been described for the management of this condition, there is no universally accepted approach. This case report discusses the surgical management of a 29-year-old female patient with idiopathic gigantomastia using a modified Thorek reduction mammoplasty technique. The modified technique involved incorporating a superomedial pedicle to preserve breast volume. The superomedial dermoglandular flap was designed and adapted to the upper margin of the areola, providing autoaugmentation to restore breast volume and prevent post-operative ptosis. This modification allowed for the preservation of the breast's natural projection while achieving a satisfactory aesthetic result. The nipple-areola complex was grafted onto the pedicled flap, avoiding the risks of pedicle-based techniques due to the patient's existing breast anatomy and tissue characteristics. Following the surgery, the patient experienced no complications, and her post-operative appearance was significantly improved, with both physical symptoms and psychological distress alleviated. The total excised tissue weighed 3546 grams from the right breast and 4487 grams from the left, reflecting the extent of the hypertrophy. This report highlights the efficacy of the modified Thorek mammoplasty technique, particularly in cases of severe gigantomastia, and suggests that the incorporation of a superomedial pedicle can be an effective strategy for maintaining breast volume and ensuring an aesthetically pleasing outcome.

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Introduction

Although initially described by Palmuth in 1648, gigantomastia was formally defined in medical literature by Strombeck in 1964.^{1,2} Gigantomastia is a rare, benign condition characterized by excessive and rapid breast enlargement, which can result in significant physical discomfort and psychosocial distress. Despite the absence of a universally accepted definition, it is commonly described as the hypertrophy of breast tissue necessitating the excision of at least 1500 grams from a single breast.³⁻⁵ Some authors, however, define it as a condition where one breast exceeds 2500 grams in weight.⁶

Physiological breast enlargement is considered normal during puberty and pregnancy. The etiology of gigantomastia remains unclear, although gestation, hormonal dysregulation, medication use, and autoimmune diseases have been implicated in its pathogenesis.^{4,7,8} Idiopathic gigantomastia specifically refers to massive, bilateral or unilateral breast enlargement occurring in adolescent females, in the absence of any identifiable pathological cause.⁷ Gigantomastia can be classified into several subtypes, including juvenile, idiopathic, gestational, hormonal, and drug-induced forms.

Various surgical techniques have been described in the literature; however, there remains no standardized approach to the management of this condition.⁷ In the present report, we describe the surgical management of a case of idiopathic gigantomastia using a modified Thorek reduction mammoplasty technique incorporating a superomedial pedicle with free nipple-areola grafting.

Case

A 29-year-old G2P2 female patient presented to the plastic surgery clinic with complaints of progressive, massive, bilateral, and asymmetric breast enlargement. Her symptoms included difficulty finding appropriately fitting bras, breast, back, and lumbar pain, as well as occasional ulceration of the breast skin. Medical history revealed that breast enlargement had been present since puberty and had progressively worsened during both pregnancies. The patient had no known systemic disease, was not taking any medications or hormones, and had no family history of gigantomastia or breast cancer. She could not precisely recall the onset of hypertrophy but reported that her breast size had been noticeably larger than her peers' since approximately age 12–13. She had completed her last pregnancy about three years ago.

A mammogram performed at an external center two months prior had been reported as BIRADS-2.

Inspection revealed asymmetric, excessive hypertrophy of the breasts, skin thinning, minimal ulcerations, areas with peau d'orange appearance, significantly enlarged areolae, and dilated superficial veins. Indentation marks from bra straps were observed on both shoulders. Due to excessive weight, the inframammary fold had descended significantly below its anatomical position. On palpation, firm subcutaneous nodules were noted bilaterally, but there was no galactorrhea or axillary lymphadenopathy. The sternal notch–nipple distance was measured as 39 cm on the right and 46 cm on the left. At presentation, the patient weighed 59 kg and was 166 cm tall (BMI: 21,4 kg/m²). All preoperative laboratory results were within normal limits, and the β -HCG test was negative. Given the extensive tissue resection planned, two units of erythrocyte suspension were cross-matched preoperatively (initial hemoglobin: 13.8g/dL). A Thorek reduction mammoplasty under general anesthesia was planned. The patient was thoroughly informed that the nipple would be reconstructed as skin graft and that breastfeeding and lactation would not be possible in the event of a future pregnancy. Written informed consent was obtained regarding this matter. Preoperative clinical photographs are shown in Figure 1.



Figure 1. Preoperative appearance of the patient.

Due to the significantly low breast footprint, a superomedial dermoglandular flap resembling a pedicle was planned to preserve breast volume, and the nipple-areola complex was to be grafted onto this flap. The superior border of the areola was marked 19 cm from the sternal notch. A Wise-pattern skin excision design with 6,5 cm lateral limbs was drawn. The planned superomedial dermoglandular flap and surgical markings are shown in Figure 2.

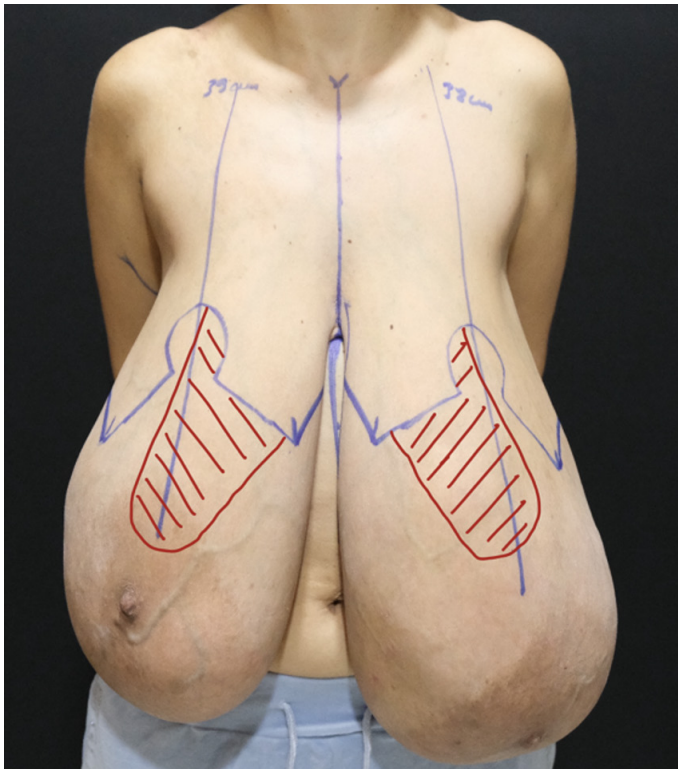


Figure 2. Surgical planning and the superomedial dermoglandular flaps marked for de-epithelialization to restore breast volume.

In the first stage of the operation, the areola was excised as a full-thickness skin graft (FTSG), followed by de-epithelialization of the superomedial tissue. The de-epithelialized flaps were then dissected from the medial, inferior, and lateral breast tissues through incisions extending down to the pectoral fascia. Subsequently, all breast tissue—except for the superomedial dermoglandular flap—was excised at the level of the pectoral fascia in accordance with the preoperative markings. The superomedial dermoglandular flap was then adapted to the site designated for the neo-areola. Thereafter, the medial and lateral breast pillars were approximated and sutured, and the areola graft was inset into its new position. The same procedure was performed bilaterally, resulting in the excision of 3546 grams of breast tissue from the right breast and 4487 grams from the left. After placement of tie-over dressings, one active closed-suction drain was inserted into each breast, and the procedure was completed. The intraoperative view of the patient is presented in Figure 3.

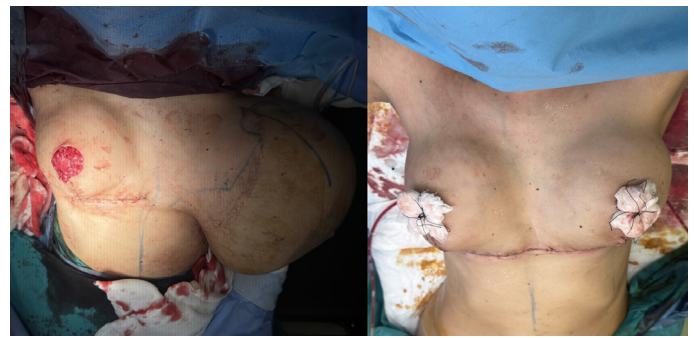


Figure 3. Early and late intraoperative views of the patient.

In the postoperative period, the patient was hospitalized for two days and monitored under intravenous antibiotic therapy and analgesia. As the patient did not exhibit any signs of pallor or tachycardia and her follow-up hemoglobin level was 10.1 g/dL, no blood transfusion was deemed necessary. The patient was discharged on postoperative day 2 with her drains in place.

At the first postoperative follow-up visit on day 7, the drains were removed, and the tie-over dressings were opened. The nipple grafts were observed to have begun to take well, and no wound healing complications were noted. The patient's appearance on postoperative day 7 is shown in Figure 4.



Figure 4. Postoperative appearance of the patient at 1st week.

The pathological examination of the patient's breast tissue reported "Widespread pseudoangiomatous stromal hyperplasia, fibroadenomas, and fibrocystic changes in both breasts." The patient was advised to undergo follow-up with a new mammogram after 6 months. The histopathological appearance of the patient's breast tissue is shown in Figure 5 and the postoperative appearance of the patient at 6th month is shown in Figure 6. No postoperative complications were observed and the peroperative period was uneventful.

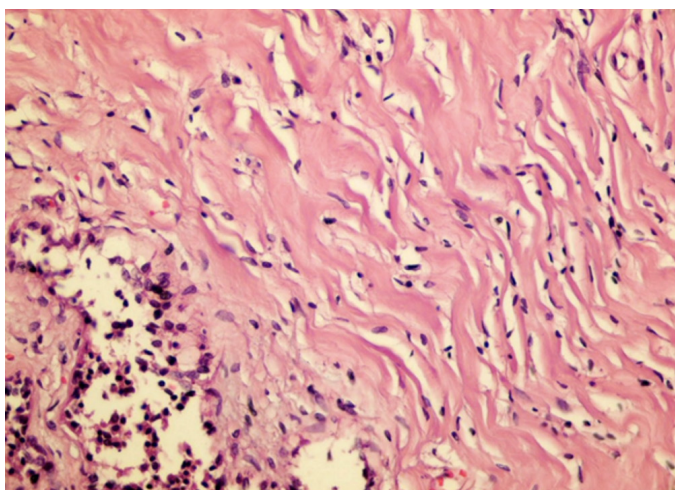


Figure 5: Histopathological appearance of the patient's breast tissue.



Figure 6: The postoperative appearance of the patient at 6th month.

Discussion

Gigantomastia is a rare condition, predominantly observed in individuals of the white race.⁵ The white-to-black ratio has been reported as 9:4.⁵ Typically, rapid and sudden breast enlargement occurs unilaterally or bilaterally, in a symmetric or asymmetric manner. There are suggestions that the etiology may involve an increase in the number of prolactin, estrogen, or progesterone receptors in the target organ, or an excessive increase in the sensitivity of these receptors to the hormones at the receptor level.^{5,8} The patient's Caucasian background and the bilateral nature of the gigantomastia support the literature.

Tension in the breast skin, tenderness, peau d'orange appearance, and enlargement of the superficial breast veins leading to a varicose appearance may occur.⁵ Ulceration and skin loss may be present in the breasts. Literature reports deaths due to infections caused by ulcers and bleeding in breast vessels.

The main symptoms of gigantomastia include breast pain, hygiene difficulties, intertriginous lesions in the inframammary folds, neck, back, and lumbar pain, shoulder impressions and indentations from bra straps, orthopnea, skin necrosis, kyphosis, and lumbar lordosis.^{9,10,11} In addition, psychological disturbances such as social isolation, peer bullying, and depression or anxiety leading to suicide may occur. The patient discussed in this case presented to us due to the presence of similar physical complaints.

Normal breast development occurs over a period of 3-5 years and involves all tissues that constitute the breast.⁷ The breast is composed of three main tissues: fat, stroma, and gland. These tissue types are organized around the alveoli and lobules within the breast. Three dominant hormones regulate breast physiology.² These hormones are prolactin, estrogen, and progesterone. The ductal growth of the breast is influenced by anterior pituitary hormones such as luteinizing hormone, growth hormone, and adrenocorticotrophic hormone, as well as estrogen, which is the primary stimulator of the breast. Lobuloalveolar growth, on the other hand, is influenced by progesterone and prolactin. Corticosteroids and prolactin are hormones that independently affect breast development.⁷ Normal breast development begins with the formation of the mammary ridge from the ectoderm on the 20th day of the embryonic period and is completed towards the end of the fetal period.⁷ Looking at the embryology of the breast, it is observed that both ectoderm and mesoderm are responsible for the formation of the breast; the breast tissue originates from the ectoderm, while the skin derives from the mesoderm.^{9,10} The nipple and areola arise from the mammary pit and the inward folding of the epidermis, surrounding connective tissue, and mesenchyme.⁷

The primary cause of excessive breast enlargement during or after puberty, without any underlying pathology, remains unknown. Idiopathic gigantomastia is not associated with hormonal disorders.¹¹ Additionally, chromosomal analysis studies of patients with gigantomastia have shown a normal 46 XX karyotype.⁷ Pregnancy-related gravid hypertrophy is usually observed between the ages of 20 and 30, distinguishing it from idiopathic gigantomastia based on the patient's age and pregnancy history. Pseudo-gigantomastia is associated with excessive fat accumulation in the breasts due to obesity.¹²

In this case, given the patient's age, the onset of breast enlargement during adolescence, the absen-

ce of a history of hormone-containing or non-hormone-containing medications such as birth control pills, and no clinical or history findings suggestive of autoimmune diseases like SLE or Graves' disease that could cause gigantomastia led to the conclusion that the patient's condition is idiopathic since by definition, juvenile gigantomastia is characterized by rapid and excessive breast enlargement that occurs during puberty, typically between the ages of 11 and 17, in the absence of any identifiable underlying pathology. Although the patient states that breast enlargement began in adolescence, the hypertrophy worsened significantly during her pregnancies and persisted into adulthood. Thus, the clinical picture does not fulfill the accepted criteria for juvenile gigantomastia.

The diagnosis of idiopathic gigantomastia is made through the exclusion of all other causes.² Drug-induced gigantomastia, caused by D-penicillamine, neotetazone, cyclosporine, or protease inhibitors, can be identified by reviewing the medications the patient is taking.⁷ D-penicillamine, a chelator that breaks down large protein molecules into smaller molecules and simultaneously increases free estrogen, is one of the riskiest drugs in this regard.⁵ Additionally, a physical examination should be conducted to rule out breast enlargement due to trauma-induced panniculitis or palpable masses. At this stage, a pregnancy test must also be performed. To exclude pseudoprecocious puberty, granulosa cell tumors of the ovary, ovarian follicular cysts, and hormonal abnormalities, serum levels of estrogen, progesterone, prolactin, and gonadotropins should be assessed. For adrenal dysfunction, urine 17-ketohydroxysteroid levels should be checked.⁷ To exclude pituitary growth disorders, a direct head X-ray or cranial magnetic resonance imaging (MRI) can be used, and any possible enlargement of the sella turcica can be observed.⁷

Pathological examination of gigantomastia excision materials typically reveals some common features. These include ductal and alveolar proliferation, severe hypertrophy and fibrosis of periductal and periacinar stromal tissues, an increased number of lactiferous ducts, their enlargement, and lining by at least two layers of cubic, inactive cells. Usually, lymphocytic infiltration accompanies hyaline connective tissue in the materials.^{5,8} In some areas of the breast, necrotic degeneration due to secondary decreased blood supply may result in calcifications.

Another problem related to gigantomastia is its occurrence during the first trimester of pregnancy.

Although rare, with an incidence of 1:100,000, this condition can be life-threatening and may require emergency bilateral mastectomy. Deaths due to gravid gigantomastia have been reported in the literature. Infection, ulceration, and hemorrhage are absolute indications for surgery.⁸ Therefore, early surgery is recommended before complications such as skin loss, sepsis, or sudden massive bleeding.

Throughout history, various surgical techniques have been applied for gigantomastia, including breast reduction, skin-preserving or total mastectomy with breast-nipple reconstruction, hormonal therapy, or combinations of these approaches.¹³ However, there is still no consensus regarding the optimal treatment.¹⁴ A review of the literature reveals a case where a total of 38 kg of breast tissue was excised through mastectomy, followed by late-stage breast reconstruction with implants.³ Breast reduction with or without hormonal therapy is generally considered the first-line treatment for gigantomastia. Free nipple grafting is also frequently used, depending on the size of the breasts. However, breast reduction surgery does not provide a definitive cure, and recurrence can occur, with advanced pregnancies being a primary cause of recurrence. On the other hand, mastectomy and breast reconstruction are associated with several drawbacks, such as less natural aesthetic results, issues related to breast implants, insufficient lactation, and potential psychological side effects.

Although hormonal manipulation of the breast with tamoxifen, norethindrone, testosterone, progesterone, and stilbestrol has been attempted in treatment, success has not been achieved. Hydrocortisone and prednisone have been beneficial in controlling the inflammatory response but have not reduced breast growth.² Diuretics such as hydrochlorothiazide and furosemide have also been tried in the literature, but no benefit was achieved.² Bromocriptine has reduced prolactin levels in some cases, but its benefit is limited.^{2,8} For all these reasons, the appropriate treatment for gigantomastia remains a subject of debate.

The optimal surgical technique for reduction mammoplasty in the surgical treatment of gigantomastia is still debated. The main reason for this is that each technique has different complications, and none has a clear superiority over the others.¹⁴ The use of the inferior pedicle, considered safer for NAC transposition, has a significant disadvantage over time: the risk of bottoming out. This occurs because the excision is made from the upper part of the breast, and the lower

pole of the breast gradually sags due to gravity.¹⁴ The majority of excisions in gigantomastia are performed from the lower pole, which provides more aesthetic advantages in the long term. Vertical scar techniques have gained significant popularity in recent years, but they can cause the scar to extend along the chest wall, especially when used for very large breasts.¹⁴ The free areola-nipple grafting technique by Thorek, also used in this case, has certain disadvantages. These include a flat, non-erectile, and numb nipple, loss of lactation, color changes in the nipple due to grafting, epidermolysis, graft failure, and the risk of NAC necrosis.^{9,14}

In the literature, both superior and central pedicle techniques have been attempted in the surgical treatment of gigantomastia.^{9,14} However, due to the presence of varicose veins observed during inspection, which increase the risk of venous insufficiency and NAC (nipple-areola complex) necrosis, pedicle-based breast reduction techniques were avoided in this patient. Additionally, since the excision was planned to be performed from the superolateral area of the breast, which is the highest risk zone for breast cancer, the dermoglandular flap was chosen from the superomedial side.

In the case presented, due to the distance from the sternal notch to the nipple being over 40 cm and the patient not planning any future pregnancies, a Thorek free nipple transfer with reduction mammoplasty was planned. However, because of the excessively large and heavy breasts, the majority of the breast tissue had descended below the current IMF (inframammary fold) level. Therefore, it was believed that sufficient breast volume and projection could not be achieved with a standard Thorek breast reduction. The superomedial pedicle-like dermoglandular flap was adapted to the upper margin of the areola to provide autoaugmentation, and the nipple graft was directly adapted to this flap. Furthermore, to prevent long-term breast ptosis, the superomedial dermoglandular flap was also adapted to the pectoral fascia.

Modifications similar to Thorek mammoplasty have been observed in the literature. Firat et al. described autoaugmentation-autoprosthesis techniques, where the inferior dermoglandular flap was passed under the pectoral muscle to ensure projection, instead of using the superomedial dermoglandular flap as used by us in Thorek mammoplasty.¹⁵ However, as stated above, this modification was considered to minimize tissue retention in the lower pole of the breast.

Conclusion

Gigantomastia is a condition with significant physical and psychological negative effects, and its etiological factors are not fully understood.⁸ Although it is thought to result from an altered breast response to normal hormone concentrations, the abnormal relationship between hormone receptors and the target organ remains unclear.⁵ The choice of surgical approach in treatment is complex and should be tailored to the patient's desires, the specific characteristics of the breasts, and any presence of breast asymmetry.⁶ Thorek reduction mammoplasty remains an appropriate option for immediate symptom relief and improved quality of life. However, in cases where the breasts are very heavy and the breast footprint has descended below the IMF level, modifications that provide autoaugmentation should be added to the surgery. Lastly, as there may be blood loss during surgery, adequate preparation for blood and blood products should be ensured.⁴

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