Congenital and Developmental Abnormalities of the Breast

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2.1 Embryology

Development in the prenatal breast is characterized by two main processes: formation of a primary mammary bud and development of a rudimentary mammary gland [1]. Formation of the glands in the embryo starts independent of gender in an identical way. Embryogenesis in the first trimester runs largely hormone independent [2, 3], whereas in the second trimester regulatory factors are important for development [4].

During the 4th and 6th week of gestation, two ridges called the mammary crests or milk lines are formed out of a pair of epidermal ectoderm. The ridges extend in a line between the fetal axilla and inguinal region, but rapidly regress except in the thorax. The primary bud forms by penetration in the chest wall mesenchyme. Out of this, diverse secondary buds rise and develop into lactiferous ducts and their branches. The fibrous stroma and fat of the mammary gland develop out of the surrounding mesenchyme. The small ducts and alveoli are formed out of the lactiferous ducts.

At the beginning of the second trimester, the nipple–areolar complex (NAC) starts to form out of differentiated mesenchymal cells. Hair follicles and sweat glands differentiate. By 20 and 30 weeks, canalization of the branched epithelial tissues is induced by placental sex hormones. Between 32 and 40 weeks, differentiation of the parenchyma into alveolar and lobular structures takes place. A shallow mammary pit is formed by depression of the epidermis, becoming the NAC onto which the lactiferous ducts open. At 34 weeks, the breast bud becomes palpable, sized approximately 3 mm at 36 weeks of age and 4–10 mm by 40 weeks.

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In exploring data concerning breast development, most sources agree that the secondary processes end in rudimentary lobular structures or end buds [1, 5, 6]. Contrary to this, some assume that there cannot be found any evidence of lobules breast at birth, only ductal structures with surrounding stroma [7].

2.2 Early Development of the Mammary Gland

In the neonate, the breast is usually palpable with variation in amount of tissue and no significant difference between the genders [8]. From four to seven days, postpartum neonates may show a unilateral or bilateral breast enlargement and/or transient secretion of colostral milk under the influence of maternal estrogens, also known as witch's milk [9].

The nipples evert soon after birth by proliferation of the underlying mesoderm and the pigmentation of the areolae increases [10]. Until puberty, the breast remains largely quiescent, independent of gender.

Nodular growth of one or both breasts in either gender before puberty is quite usual; up to 90 % of neonates may have palpable breast tissue that typically resolves spontaneously within few months [11]. Tumors of the infantile breast are benign in most cases. Nevertheless, observation is recommended due to rarely malignant occurrence [12].

One has to consider that biopsy of the pre-pubertal breast may irreversibly cause disruption of breast development [13], and therefore, application should be used with restrictions.

2.3 Thelarche

The physiological breast development in females is called thelarche, normally occurring at the age of eight years as a result of rising levels of estradiol. Estrogen stimulates ductal growth and branching, whereas progesterone influences lobular and alveolar development. Androgens as testosterone and

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dihydrotestosterone limit breast development [11]. Prolactin stimulates the alveolar buds. Increase of volume and elasticity of the connective tissues, vascularity, and fat deposition occurs resulting in progressive enlargement of the breasts.

The breast expansion normally lasts approximately until 25 years of age. This should be taken into consideration concerning interventional options including neoplasms and malformations. It has to be pointed out that every breast trauma (including iatrogenic intervention) before completed development can lead to developmental disorders.

2.4 Anatomy of the Breast

Superficial to the pectoralis major muscle on the anterior chest wall, the human breast is vertically located between the second anterior rib to the sixth anterior rib and horizontally between the lateral edge of the sternum and the mid-axillary line.

The breast tissue is formed by mammary gland, fat tissue, blood vessels, lymphatics, and nerves. The surface of the breast is attached by suspensory fibrous ligaments, called Cooper's ligament. These ligaments pass through the mammary gland from the superficial fascia to the deep fascia overlying the pectoralis major muscle. The extension of the breast varies in females, lasting from the midline to the near the mid-axillary line. Based on the embryological development, the maximum percentage of mammary gland is situated in the upper outer quadrant of the breast. Characteristically, the breast shows an elliptical base and a hemispheric shape.

The architecture of the breast is built by lactiferous ducts and lobes which are arranged radially around the nipple–areolar complex (NAC), opening on the nipple.

Functionally, the milk production takes place in the lobes, whereas the transport of the lactation products occurs by the ducts. Anatomically, each lobe consists of 20–40 lobules, containing 10–100 alveoli. During the lactation period, milk accumulates in the so-called lactiferous sinus, representing the excretory duct of each lactiferous duct. Breast parenchyma consists of connective tissue, including lymphatic and vascular components as well as fat.

Vascular supply of the breast is performed by the internal mammary and lateral thoracic arteries supplemented by lateral and anterior cutaneous branches of the intercostal arteries and subdermal vessels. The venous drainage primarily leads into the axilla and then further flows into the internal thoracic, lateral thoracic, and intercostal veins.

The lymphatic drain of the breast ends up in the regional lymph nodes, composed of axillary, supra-, and infraclavicular lymph nodes as well as the internal mammary lymph node chain, intrathoracic located in the parasternal space.

Innervation of the breast gland and overlying skin is performed mainly by the fourth lateral intercostal nerve.

2.5 Premature Thelarche

Premature thelarche is defined as premature breast tissue development unilaterally or bilaterally without other signs of sexual maturation. Common premature thelarche occurs between 6 and 24 months of age [14]. Most girls undergo puberty appropriately. Premature thelarche is in approximately 18 % of girls, the first manifestation sign of central precocious puberty [15]. Continued clinical observation every six months is recommended to distinguish from that differential diagnosis.

Higher levels of estrogen were found in girls with premature thelarche, measured by ultrasensitive bioassays compared to controls [16].

The etiology of premature thelarche is multifactorial. Endocrine disruptors, genetic, and nutritional factors can be proofed. It has been shown that some girls with exaggerated or fluctuating thelarche show an activating mutation in the GNAS gene, codifying for alpha subunit of G stimulating protein (Gsalpha) [16, 17].

Typical benign idiopathic thelarche is a self-limiting condition without need for treatment. If a precocious puberty is suspected, the referral to a pediatric endocrinologist is strongly recommended.

2.6 Accessory Breast Tissue: Polymastia/Polythelia

Based on the embryological development, accessory breast tissue can occur in the realm of the former embryonic milk line (Fig. 2.1). Occurrence is most often sporadic with an average in the general population between 0.22 and 6 % and a higher rate in women compared to men [18].

A distinction is drawn between polythelia, the most common type of accessory breast tissue and polymastia.

Polythelia describes the existence of supernumerary nipples or nipple–areolar complexes. It can be found in both males and females and may occur at any point along the embryonic milk line between axilla and groin. An association with nephrourologic abnormalities exists in sporadic cases [18, 19]. High blood pressure and conductive or rhythm disturbances represent cardiovascular problems which are associated with polythelia [19]. Surgery is requested for esthetic reasons or due to discomfort.

Polymastia describes the presence of supernumerary breasts along the former milk line which may appear with or

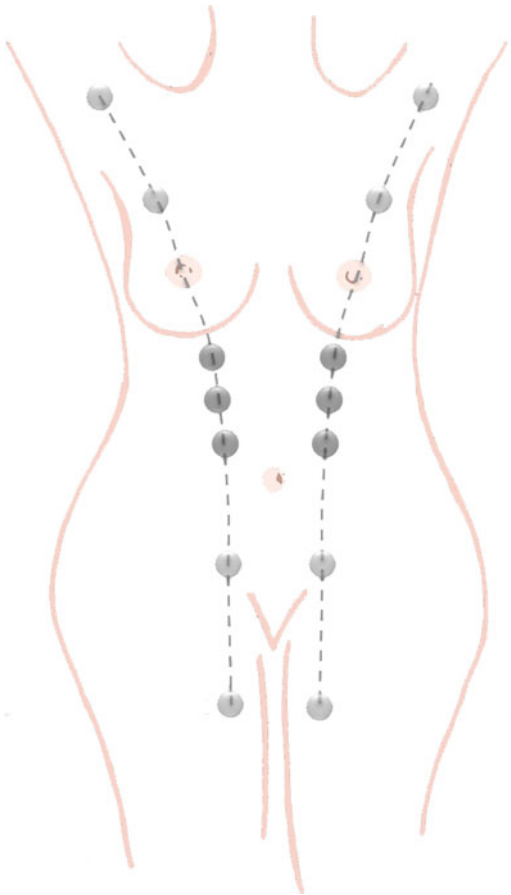

Fig. 2.1 Schematic illustration of the former embryonic milk line, where accessory breast tissue may appear

without nipples or areolae. The axilla is a common site of ectopic breast tissue [19] (Fig. 2.2); rarely, aberrant breast tissue can also be found at face, neck, torso, vulva, and lower extremities [18].

Symptomatic manifestation often occurs during menstrual periods or pregnancy when the breast tissue becomes tender, enlarged, or lactates. Reasons for surgical excision would include discomfort due to tenderness, milk secretion [19], or perhaps purely for esthetics.

2.7 Underdevelopment of the Mammary Corpus

Female breasts are typically not equal in size, especially during development, for unknown reasons. The left breast is statistically more often larger [20].

Depending on the underlying developmental disturbance, several forms can be discriminated [21]. The various developmental forms are addressed below.

2.7.1 Breast Hypoplasia

Hypoplasia can occur unilaterally, causing an asymmetric body image or bilateral. In some cases, it is associated with complex developmental syndromes such as the Poland syndrome, which is described later in this text.

Besides congenital causes, a variety of acquired reasons can lead to breast hypoplasia such as hormonal disorders or tumors. Iatrogenic causes, including medication, operations, radiation, and trauma, can also lead to hypoplasia [22, 23].

Depending on severity, breast hypoplasia may cause physical discomfort and significant psychological burden, especially in adolescence.

For the treating physicians, it is a huge challenge to determine the optimal timing for surgical intervention. The wishes of the adolescent girl for early adjustment must be considered in contrast to the risk of postoperative

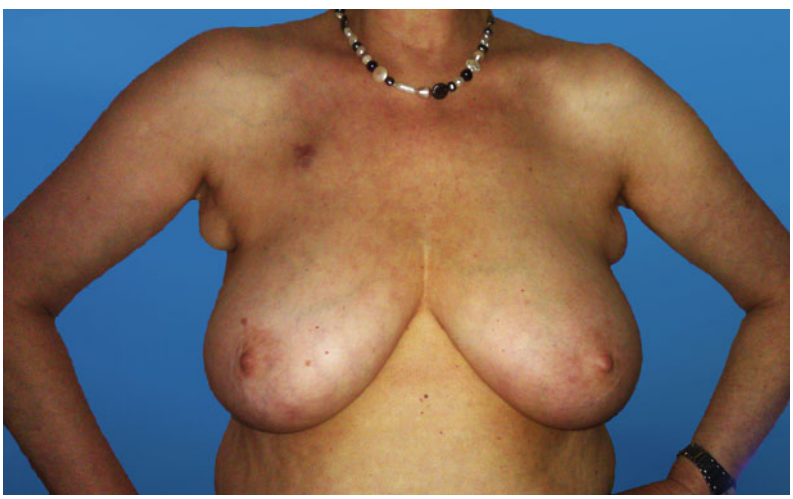

Fig. 2.2 Woman with polymastia with accessory axillary breast tissue

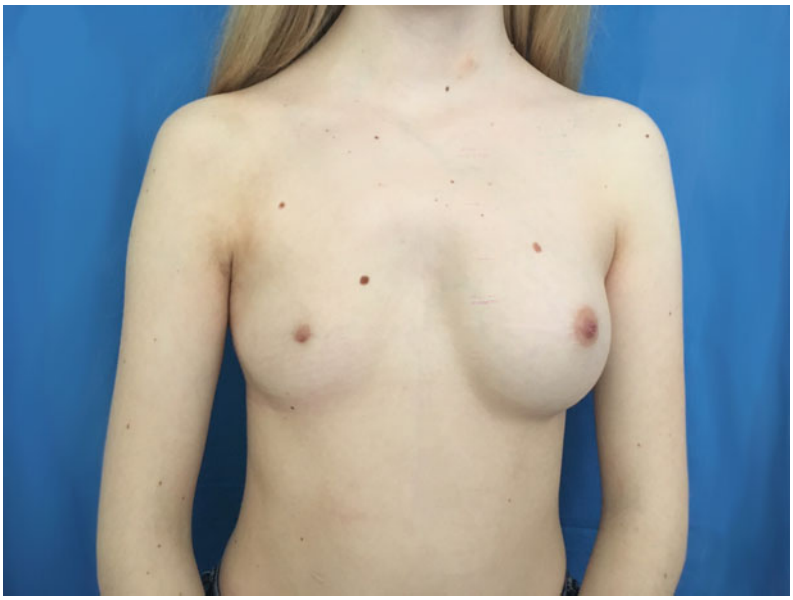

Fig. 2.3 Young woman with right breast micromastia

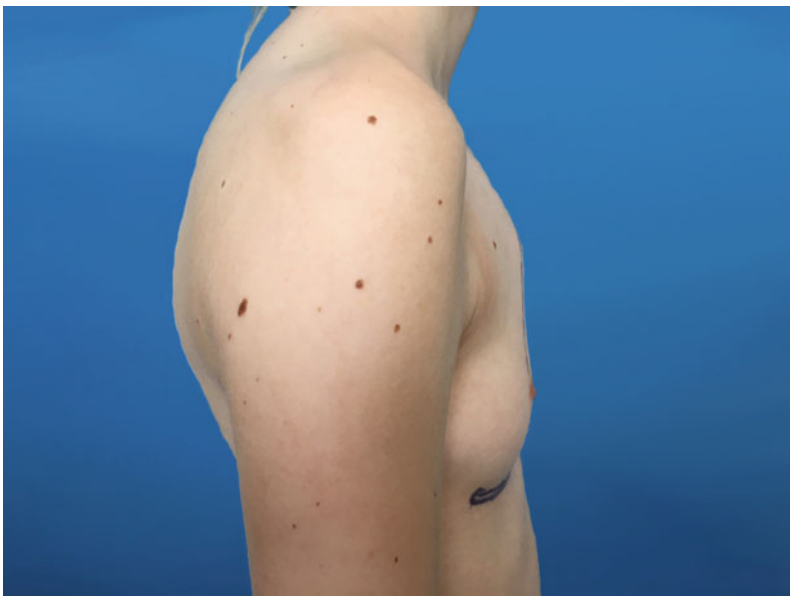

Fig. 2.4 Young woman with right breast micromastia

asymmetrical growth that may necessitate additional surgery and increase the risk of morbidity.

Still, many surgeons recommend protracting treatment until the breast development has finished or at least the patient shows stable adult weight and breast volume for one year [24, 25].

Generally, treatment of unilateral breast hypoplasia contains augmentation of the affected breast but remains a reconstructive challenge. Autologous versus heterologous techniques as one- and two-stage procedures with prior expansion of the overlying skin envelope should be carefully weighed against each other. The advantage of autologous reconstruction contains better long-term results but acceptance of longer operation time and additional donor site morbidity (Figs. 2.3, 2.4, 2.5 and 2.6).

2.7.2 Amastia/Athelia

Once the mammary ridges fail to develop or disappear completely [26], hypoplasia of mammary tissue results in varying specificity. Congenital disorders concerning breast development can be discriminated into amastia, amazia, and athelia.

- *Amastia* describes the complete absence of breast tissue, nipple, and areola.

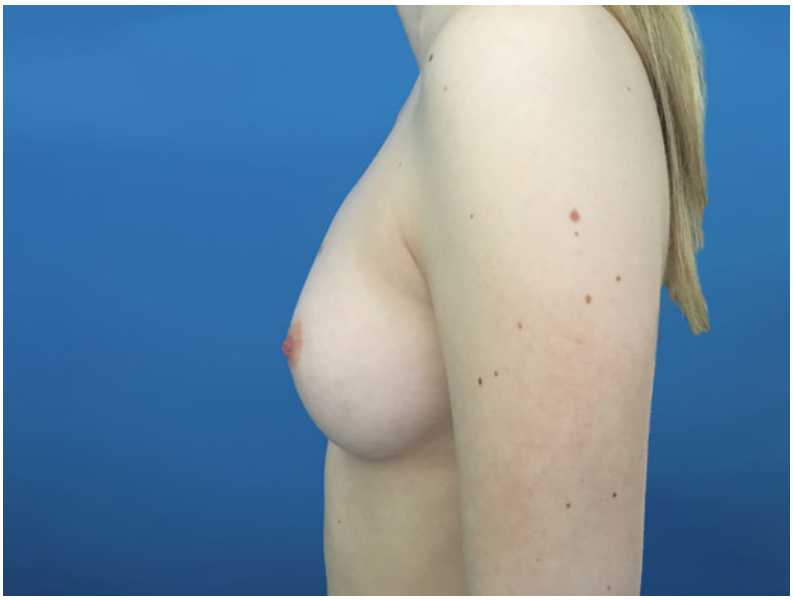

Fig. 2.5 Young woman with right breast micromastia

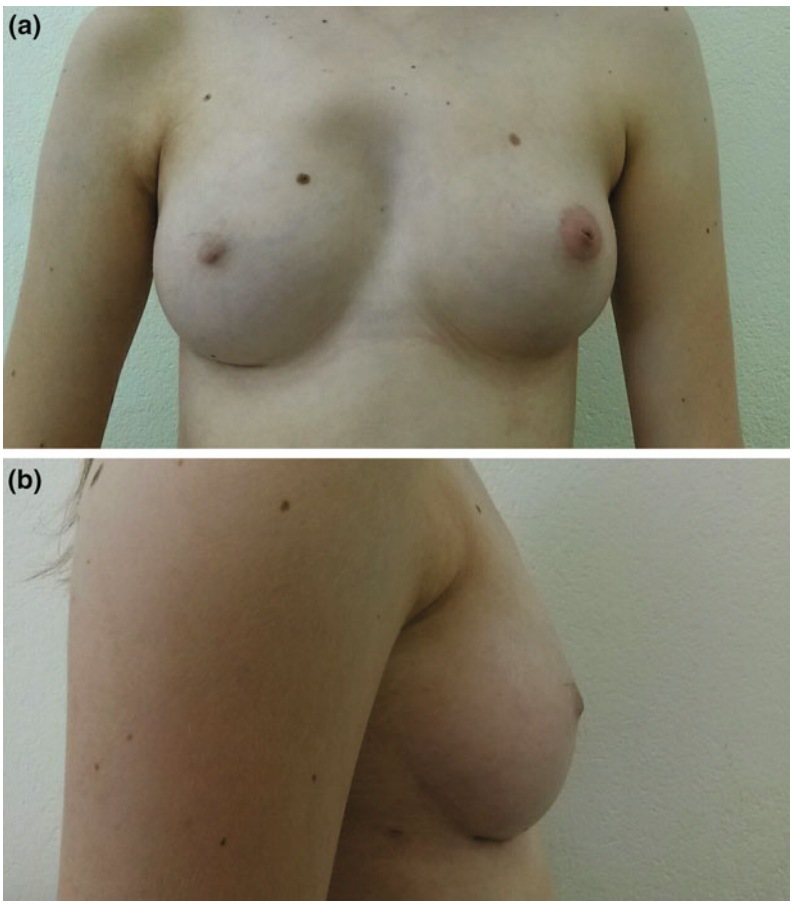

Fig. 2.6 **a** Young woman with *right* breast micromastia. **b** Postoperative result after performing *right* breast augmentation

Table 2.1 Classification of Poland syndrome

	Class I	Class II	Class III
Hypoplastic breast	√		
Hypoplastic NAC	√		
Hypoplastic pectoralis muscle	√		
Hypoplastic breast or absence of breast		√	√
Hypoplastic NAC or absence of NAC		√	√
Absence of pectoralis muscle (sternocostal portion)		√	√
Thoracic skeleton abnormalities minimal		√	
Thoracic skeleton abnormalities distinct			√
Others			√

Source Data from Hartrampf [37]

- *Amazia* is defined as the absence of one or both of the mammary glands without impairment of nipple and areola [27].
- *Athelia* is the congenital lack of one or both nipples.

Causes can be congenital as well as iatrogenic. The first case of amastia was reported in 1939 by Froriep. Because of the rare occurrence, data refer to only few cases. There are three subgroups of amastia. (1) congenital ectodermal defects leading to bilateral amastia; (2) unilateral amastia; and (3) bilateral amastia with variable associated anomalies, including hypertelorism, anomalous pectoral muscles, cleft palate, upper limb deformities, and abnormalities of the genitourinary tract [28].

Associations between special congenital syndromes and amastia or athelia have been reported. This includes ectodermal dysplasia, the Mayer–Rokitansky–Kuster–Hauser syndrome, where a failure of development of the Müllerian duct leads to accompanying vaginal-uterine agenesis [29, 30], as well as the Poland Syndrome (see Sect. 2.7.3). Exclusive occurrence of athelia is extremely rare. It is described in some various congenital syndromes.

Surgical correction is normally performed in several steps, for instance, the procedure commences with tissue expansion and finishing with placing the definitive implant or use of autogenous tissue such as abdominals or gluteal flaps.

2.7.3 Poland Syndrome

In 1841, Alfred Poland described a rare congenital anomaly characterized by unilateral underdevelopment or absence of the pectoralis major muscle. It was first named in 1962 by Clarkson [31]. It may occur with different gravity, with range from mild to severe which makes the classification difficult. Ribs, sternum, other muscles of thorax and abdomen, skin, breast, and nipple can all be abnormal, missing, or underdeveloped [32–35].

Most often, occurrence is sporadic with an incidence ranging from one in 20,000–30,000 live births [12, 35]. Males and the right side are more often affected with a ratio of 3:1 [32, 36].

One possible classification was described by Hartrampf, who defined three classes occurring since the 1980s (Table 2.1).

The cause of Poland syndrome is unknown. Etiology is hypothesized to be related to an intrauterine interruption of the embryonic blood supply to the subclavian arteries at about the 46th day of embryonic development, disrupting normal development of the chest wall and upper limb [35, 38]. The range of signs and symptoms that occur in Poland syndrome may be explained by variations in the site and extent of the disruption.

Another theory postulates an abnormal migration of embryonic tissues. Development of the primitive limb bud that later forms the pectoralis muscle takes place in the 9-mm embryo; later, the bud splits into clavicular, pectoral, and sternal components in the 15-mm embryo. An explanation for Poland deformity could be defective attachment or failure of attachment of that bud to the upper rib cage and sternum [39, 40].

The aim of surgical reconstruction treatment is to conceal the deformity and create an esthetic, natural appearing décolleté and breast according to the unaffected opposite side.

Possible techniques include the use of breast implants, tissue expanders, and autologous tissue (pedicled or free). While planning the reconstruction, one ought to consider that volume discrepancies might lead to displacement of the NAC and a new symmetric inframammary fold has to be formed. If necessary, a contralateral adjustment of breast might be indicated to achieve an esthetic and satisfactory goal.

2.7.4 Tubular/Tuberous Breast

Rees and Aston [41] first shaped the term tuberous breast, describing a hypoplastic breast deformity with a narrowed

breast base diameter, malposition of the constricted inframammary fold, and herniation of breast tissue through the areola (Fig. 2.7).

Due to the reduced transverse breast diameter and base constriction, the breast seems to herniate into the NUC. This unique appearance caused the descriptive term "*Snoopy-nose deformity*" [42].

The condition may be unilateral or bilateral. In many cases, there exists a significant asymmetry between both breasts. Exact incidence and etiology remains unknown.

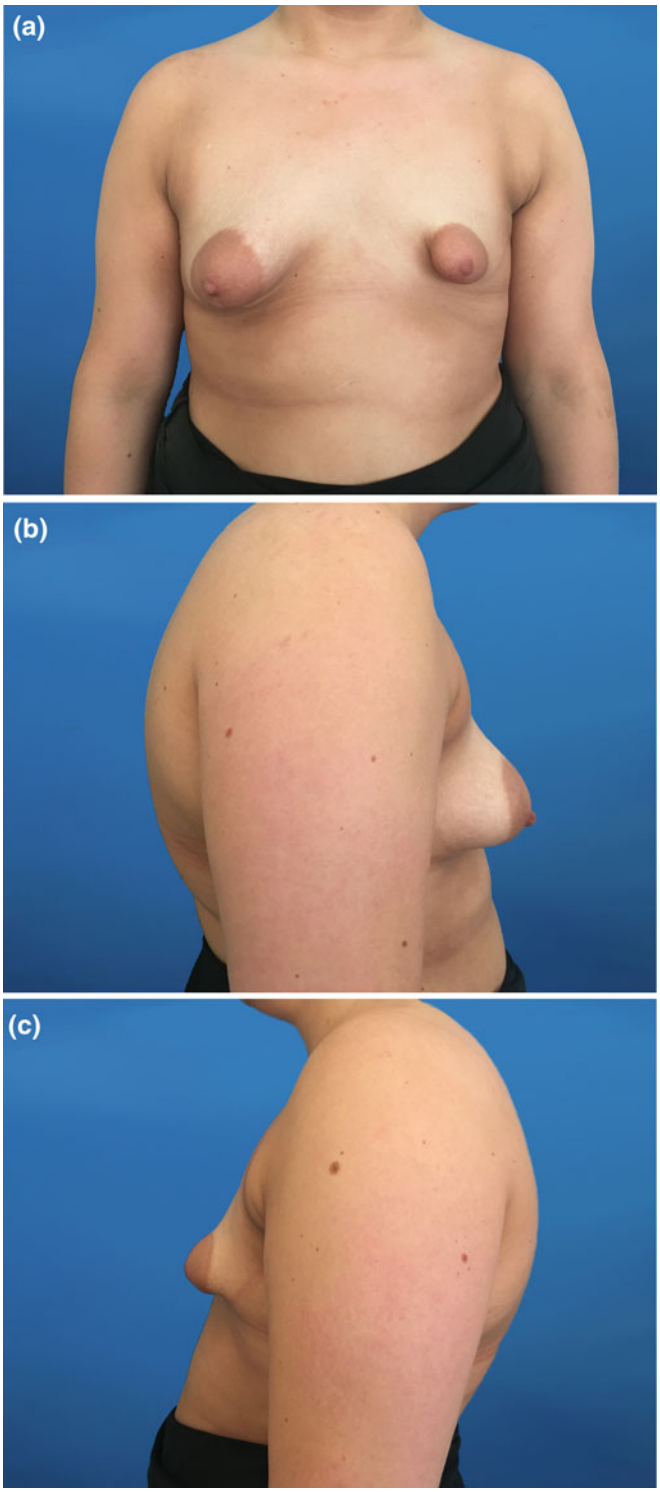

Fig. 2.7 Young woman with tubular breast deformity, characterized by a narrowed base diameter and pseudo-herniated breast tissue through the enlarged nipple–areolar complex (NAC). **a** anterior view, **b** right, and **c** left facing lateral view

Table 2.2 Classification of tuberous breast

Type I	Hypoplasia of the lower medial quadrant
Type II	Hypoplasia of the lower medial and lateral quadrants sufficient skin in the subareolar region
Type III	Hypoplasia of the lower medial and lateral quadrants deficiency of skin in the subareolar region
Type IV	Severe breast constriction with minimal breast base

Source Data from von Heimburg [43]

Classifications graduate the different occurrence of malformation. An example is shown in Table 2.2.

Regarding reconstruction, this deformity remains a challenge. While planning the concept of surgery, all abnormal elements in breast shape have to be taken into consideration.

Depending on tissue volume, autologous reconstruction with internal flaps or combinations with heterologous materials such as implants or expanders are used.

Furthermore, the ideal timing for surgery should be identified and the possible advantage of a two-stage reconstructive approach should be debated for gradually expanding the skin envelope.

In order to correct the tuberous breast deformity, surgical objectives include remodeling of the existing breast tissue to expand the base circumference and when indicated, also the skin. Incision of the tethering bands releases the constriction through the base of the breast, allowing the breast to re-expand. Conclusively, the inframammary fold has to be reformed at a lower, anatomically correct position [44, 45]. Taking a peri-areolar access allows modification of the

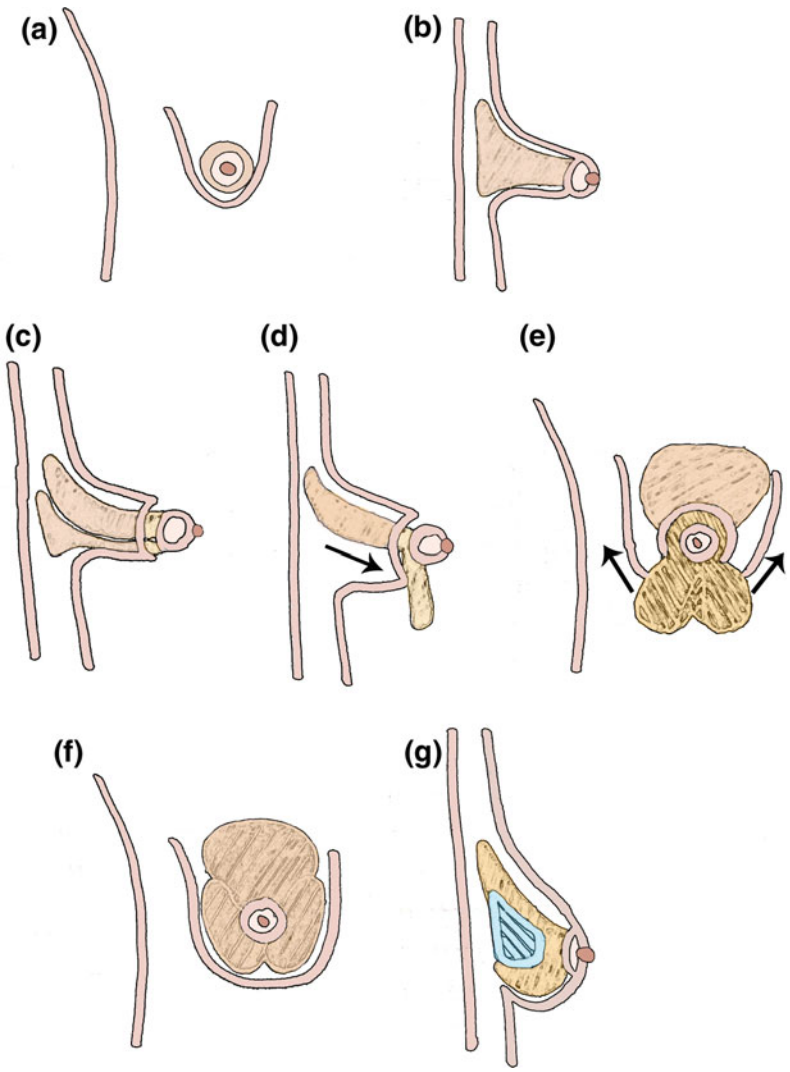

Fig. 2.8 Schematic illustration of the operational technique of tubular breast (modified according to Puckett and Mandrekas [46, 49]). **a** Tubular breast frontal, peri-areolar approach. **b** Tubular breast side view. **c** Tissue flap from the deep superior portion of the breast. **d** Elevation of glandular flap tissue and forming a new inframammary fold by extending the skin envelope. **e** Glandular flap tissue divided vertically to dispense the constriction and fill out the lower pole. **f** Reduction of the areola. **g** Breast contouring, e.g., by using an implant if needed

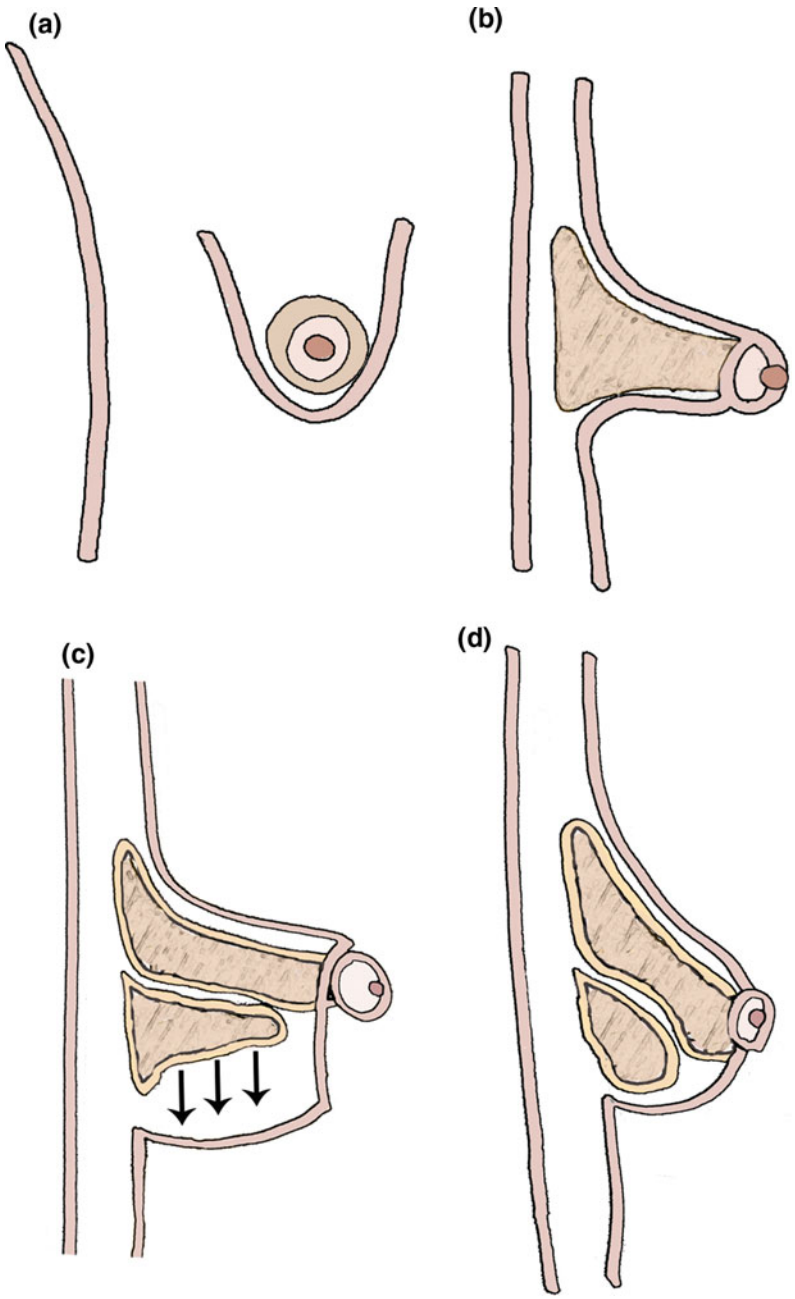

Fig. 2.9 Schematic illustration of the operational technique of tubular breast (modified according to Ribeiro [47]). **a** Tubular breast frontal, peri-areolar approach. **b** Tubular breast side view. **c** Forming a tissue flap petiolate to the pectoralis muscle and forming a new inframammary fold by extending the skin envelope. **d** Breast contouring by turning over the tissue flap

areolar diameter as well as expansion and dilatation of breast tissue for increased breast base diameter.

For compensation of deficient breast volume, a tissue expander or implant can be placed under the divided breast tissue [43, 48–50] (Figs. 2.8 and 2.9).

2.8 Inverted Nipples

An inverted nipple is defined as a condition where a part or the complete nipple is covered below the level of the areola. In some cases, the nipple might temporarily protrude after stimulation, whereas in others the retraction persists. Depending on the constellation of how easily the nipple can be pulled out, the grade of breast fibrosis, and the degree of damage caused to the milk ducts, severity codes can be defined.

This state was primarily described in 1840 by Cooper. Nipple inversion appears with a reported prevalence ranging from 1.8 to 3.3 % quite frequently [51, 52] and occurrence is most frequently bilateral [52, 53]. The cause is congenital in most cases or can be caused by, e.g., repeated inflammation and breast surgery or can occur after sudden and major weight loss. Some syndromes such as Robinow syndrome

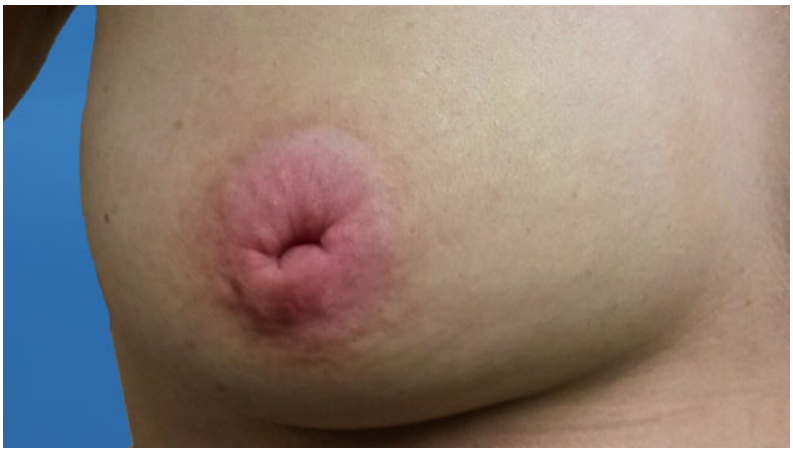

Fig. 2.10 Woman with inverted nipple

and carbohydrate-deficient glycoprotein syndrome go along with inverted nipples [54, 55].

The clinical presentation of patients with inverted nipples (Fig. 2.10) is characterized by a relatively short lactiferous duct which is attached to the nipple via dense and highly inelastic connective fibers [51, 56]. That condition may result in psychological, esthetic, and functional problems such as inconvenience with breast-feeding.

The first adjusting operation was reported by Kehrer in 1879. Over time, numerous methods have been proposed to correct this deformity. Commonly, techniques forming bilateral triangular dermal flaps crossing under the nipple in modified forms are used [57–60]. Figure 2.11 illustrates the use of subcutaneous turnover flaps for creation of a tent suspension-like effect [61]. Nevertheless, a remaining problem portrays the postoperative incomplete correction as well as a high rate of recurrence. Further problems can include change in nipple sensory, vascular compromise, scarring, and obliterated ducts with defective lactation.

2.9 Hyperplasia of the Breast

Hyperplasia of the breast, also called macromastia or gigantomastia, describes a rare medical condition with growing of excessive breast tissue (Fig. 2.12).

Breast hypertrophy might be caused by abnormally elevated hormone levels or increased end organ hypersensitivity toward female sex hormones, growth factors or prolactin, or a combination of both [62]. Histologically, hypertrophy of breast tissue represents a benign situation, which can occur unilaterally or bilaterally. Depending on etiology and chronological occurrence, different subgroups can be described.

Associated symptoms contain bra grooving, pain of shoulder, neck and back, postural problems, breathing difficulties while in supine position, and skin necrosis [63].

Juvenile (or *virginal*) *hypertrophy* describes a rare condition of an atypical and rapid breast growth during puberty often defined as a 6-month period of extreme breast

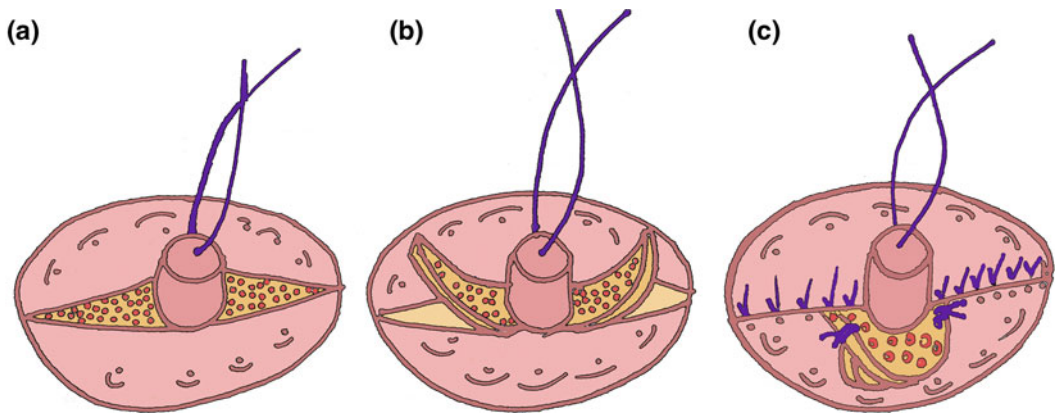

Fig. 2.11 Schematic illustration of an operative technique, showing the use of subcutaneous turnover flaps to create a tent suspension-like effect [61]. **a** Two bilateral de-epithelized subcutaneous triangular flaps are formed. Subcutaneous tunneling below the areolar level by vertical blunt dissection is performed. **b** The triangular flaps are rotated about 90° and then crossed through the vertical slit. **c** Postoperative view. The space under the nipple is filled by reposition of the flaps in the vertical direction and fixation. Finally, nipple skin and surrounding areolar skin is re-draped

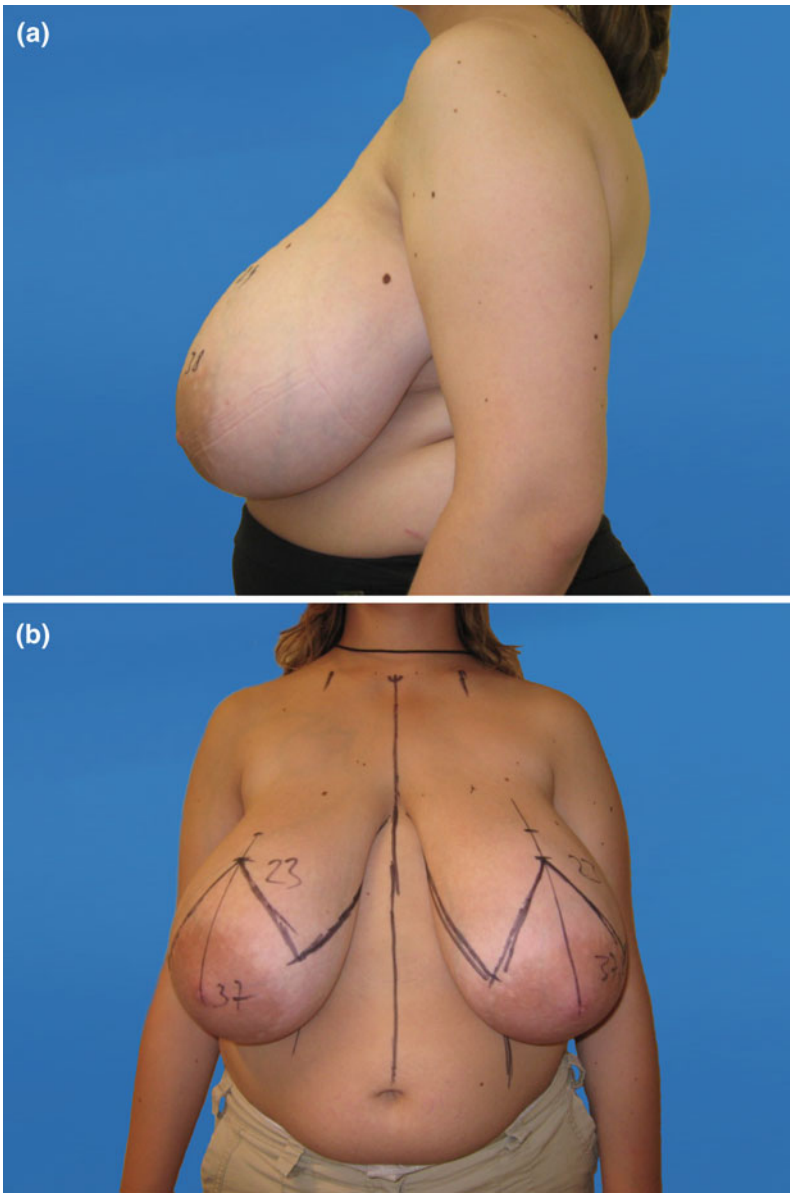

Fig. 2.12 Two women with bilateral gigantomastia from **a** lateral view and **b** anterior view

enlargement, followed by a longer period of slower, but lasting breast growth [63, 64]. Occurrence can be unilateral or bilateral. Serum levels of estrogen, progesterone, gonadotropins, and growth hormone are normal in previous studies [65]. Pharmacotherapeutic attempts include drugs such as tamoxifen, danazol, or bromocriptine to control this condition [66, 67]—safety and efficacy still unknown [68]. More common is to perform volume reduction mammoplasty as soon as the breast growth is completed (Fig. 2.13).

Cause of *adolescent macromastia* is multifactorial and usually idiopathic. It is normally associated with hormonal imbalances or obesity and develops throughout puberty with steadily ongoing breast growth. It can have significant long-term medical and psychological impacts.

Gravid-induced gigantomastia describes a very rare condition that is similar to virginal hypertrophy, where excessive breast growth occurs during pregnancy [69, 70]. It is related to breast hypersensitivity to elevated circulating hormone levels such as estrogen and prolactin. Bromocriptine can be used as a therapeutic option after delivery to induce breast involution by lowering secretion of prolactin [71].

Drug-induced gigantomastia can occur after taking several medications or drugs such as hormonal therapy, corticosteroids, marijuana, D-penicillamine, cimetidine, and the antiepileptic sulpiride. It can result in unilateral or bilateral hyperplasia. An optional treatment for D-penicillamine-induced gigantomastia has been reported with danazol [72]. Medications either stimulate hormones or act locally. If

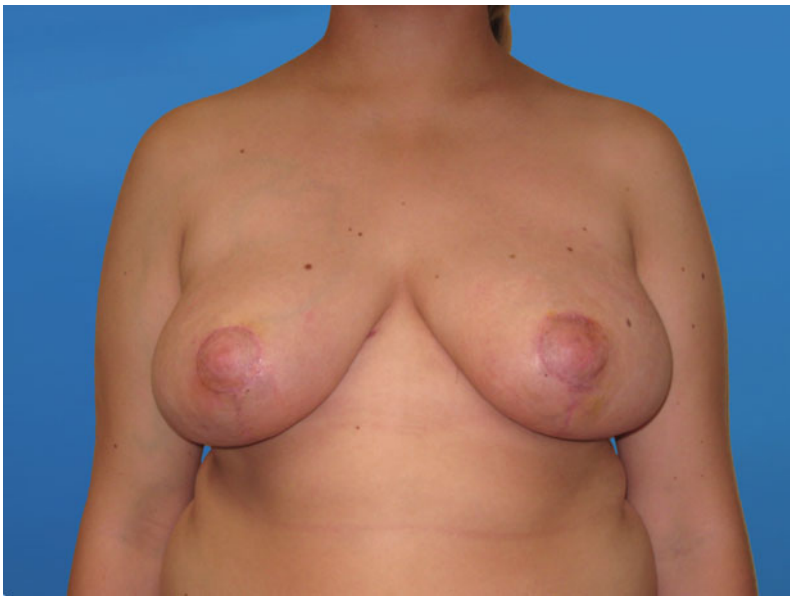

Fig. 2.13 Postoperative result of bilateral reduction mammoplasty

possible, the first attempt should cease the potentially triggering medication to reverse gigantomastia.

2.10 Gynecomastia

The excessive development of male breast tissue is a common phenomenon and appears in 32–65 % of healthy men [73–75] (Fig. 2.14).

Deriving from the Greek, the term gynecomastia combines gyne (woman) and mastos (breast), describing a female-like enlargement of the male breast which leads to glandular proliferation [11].

Gynecomastia appears in 75 % of all cases bilateral and asymmetric [76]. It is often clinical asymptomatically but may also cause local pain or psychical disturbance.

The classification of gynecomastia is based on the amount of glandular tissue. Division has to be made between the glandular, true gynecomastia, and simple fatty gynecomastia which is often found in obese man, also known as pseudo-gynecomastia. In true gynecomastia, glandular tissue can be palpated and verified via ultrasound.

The pathological process involves a relative increase in the ratio of free estrogen to androgen locally in the breast [74, 77]. Etiological factors can range from physiologic to pathologic conditions; several illnesses (e.g., hyperthyroidism; benign Leydig cell tumor, liver, and renal failure) or medication (e.g., spironolactone and drugs) could be causal. Possible causes of gynecomastia are shown in Table 2.3.

In newborns, bilateral proliferation of breast tissue is induced by maternal and placental estrogens, resolving within a few weeks after birth.

During adolescence—usually at 13 or 14 years of age—a physiological pubertal gynecomastia appears, lasting up to 6 months. Causal is a relative increase in estrogens derived mostly from peripheral aromatization of testicular and adrenal androgens. In late puberty, testicular testosterone production increases, resulting in spontaneous regression [78].

In any case, careful taking of a patient's history, as well as physical examination, should be utilized to evaluate and define the cause. The differential diagnosis of breast cancer should always be taken into consideration. If suspected, mammography, mamma sonography, and diagnostic fine needle or core biopsy should be performed, showing a 90 % sensitivity and specificity for distinguishing benign from malignant [79].

Laboratory diagnostics should include hCG (human chorionic gonadotropin), luteinizing hormone (LH), testosterone, and estradiol [75]. Due to the circadian rhythm of hormone secretion, measurement is recommended in the morning at time of the maximum release.

As soon as hypogonadism—which is increasing in elderly patients—is detected, a symptomatic therapy with testosterone should be performed.

Without question, if drug-induced gynecomastia is suspected medication should be stopped or adapted if possible.

A therapeutic approach is the use of tamoxifen, a selective estrogen receptor modulator. A daily orally dose of 20 mg tamoxifen for up to 3 months shows good results in randomized and not randomized trials. Regression of gynecomastia is shown in up to 80 % of patients. It has to be qualified that data on tamoxifen therapy are limited due to small cohorts. Adverse side effects, including epigastric

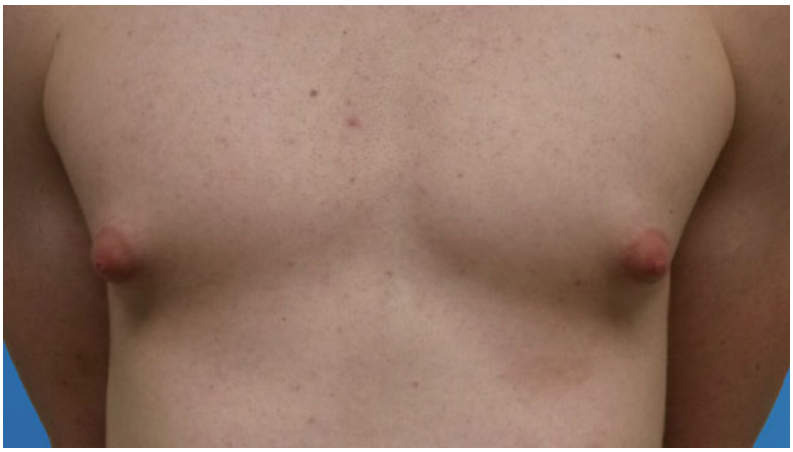

Fig. 2.14 Adolescent male presenting with bilateral gynecomastia

Table 2.3 Causes of gynecomastia

Physiologic	• Antiandrogens • Antibiotics • Antihypertensive agents • Gastrointestinal (GI) agents • Hormones • Illicit drugs • Psychiatric drugs
Decreased androgen production	• Primary (testicular) hypogonadism • Secondary (central) hypogonadism
Decreased androgen effect or synthesis	• Androgen insensitivity syndrome • 5α-reductase deficiency • 17-β-hydroxysteroid dehydrogenase deficiency
Increased estrogen production	• Adrenal tumor • Testicular tumor • hCG-secreting tumor • Familial aromatase excess syndrome
Other	• Liver disease • Thyrotoxicosis • Obesity • Renal disease • Malnutrition

Source Data from Morcos and Kizy [77]

distress and a post-traumatic deep-vein thrombosis, are rarely reported [80, 81].

Use of anastrozole, an aromatase inhibitor did not show more effectiveness than placebo in boys with pubertal gynecomastia [82].

If gynecomastia persists more than 12 months, a fibrosis remodeling takes place. Therefore, the effect of endogen treatment by testosterone or tamoxifen is limited.

Indications for surgical intervention include psychosocial stress and pain as well as cosmetic discomfort. Therapeutic interventions include liposuction, breast tissue resection, and reduction mammoplasty considering size of hypertrophic tissue and expertise of the surgeon. Aim is to remove the hypertrophic glandular tissue and not only fat. Sometimes a combination of methods can be effective. In particular, liposuction can be used after open excisional surgery for contouring the chest wall to achieve a nice shape [83, 84].

2.11 Conclusion

In their entirety, breast deformities or developmental breast disorders represent a small group of patients. Nevertheless, people affected by these body shape malformations often suffer from relevant psychological strain, which can cause isolation and withdrawal from social situations. In our increasingly sexualized society, idealized archetypes gain progressive influence. Therefore, an obstacle for receiving support may be the avoidance of consulting a physician due to embarrassment.

Plenty of these deformities are congenital and result from false processes in development. Therefore, underlying systemic disorders or syndromes should be excluded. Other breast deformities are iatrogenic, so the potential damage of surgery to the developing breast must be considered when contemplating an intervention on the chest of an infantile or adolescent patient.

The aim of the reconstructive breast surgeon includes preservation of breast structures while achieving improved symmetry for better appearance. Even if exact symmetry cannot be achieved, self-esteem is often much improved after accomplished surgery. At the present time, plenty of therapeutic options exist. Individualized counseling has to be achieved, leading to a unique concept for all patients.

Knowing all that one should, however, keep in mind: breasts are sisters, not twins!

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