INTRODUCTION

Gynecomastia is a term derived from the Greek words "gyne" meaning woman, and "mastos" meaning breast. It means benign enlargement of the glandular and/or fatty components of the male breast and is the most common breast problem in men. Breast growth is primarily dependent on the balance between estrogens and androgens in serum and any physiological or pathological factor that interferes with this balance can result in gynecomastia. Gynecomastia may be due to an increase in the concentration of serum estrogens, a decrease in the concentration of serum androgens, or an increase in sensitivity of the breast tissue to the normal levels of circulating estrogens. When no cause is evident, gynecomastia is supposed to be idiopathic ***(El Noamani et al., 2010).***

There are many classifications of gynecomastia. ***Webster in 1946*** had classified gynecomastia into three grades. Grade 1: glandular, grade 2: fatty glandular, and grade 3: simple fatty gynecomastia, often called "pseudogynecomastia". Grade 1 comprising hypertrophy of the stromal and glandular breast tissue is the most common type of gynecomastia in adolescents ***(Webster, 1946)***.

On the other hand, the classification described by ***Simon and his colleagues in 1973*** was based on the amount of breast enlargement and graded the condition into four grades. Grade I: Minor breast enlargement without skin redundancy, grade IIa:

Moderate breast enlargement without skin redundancy, grade IIb: Moderate breast enlargement with minor skin redundancy, and grade III: Gross breast enlargement with skin redundancy that simulates a pendulous female breast ***(Simon et al., 1973)***.

Treatment of enlarged breast is indicated when causes sufficient pain, embarrassment or emotional distress that interfere with the patient's regular activities. The two treatment options are either medical therapy or surgical removal. Medical therapy is possibly most effective during the active proliferative phase of gynaecomastia. Danazol, clomiphene, testolactone and tamoxifen have been used. If medical therapy is unsuccessful or if the gynaecomastia has been present for a number of years and is troublesome to the patient, the breast glandular tissue should be removed surgically ***(Fruhstorfer et al., 2003)***.

According to ***Cordova and Moschella in 2008***, the aims of surgical treatment include flattening of the thorax, abolition of the inframammary fold, repositioning of nipple-areola complex (NAC), excision of skin redundancy, symmetrization, and scars containment. Many operative techniques have been described to correct gynecomastia. Nowadays surgical options concern the removal of excessive glandular tissue, fatty tissue, and skin by liposuction, subcutaneous mastectomy and breast reduction. The choice of surgical technique does not depend only on the grade of gynecomastia but also on the self-experience of the surgeon and other factors ***(Cordova et al., 2008).***

Adenectomy carried out by means of semicircular periareolar incision ***(Webster technique)*** is said to give good cosmetic results in cases of small or medium breast without skin redundancy. For the high-grade gynecomastia with ptosis and skin redundancy, the next methods can be used: reduction mammoplasty with free transplantation of the nipple areola complex, modified breast-reduction techniques (with I- or inverted-T pattern), and subcutaneous mastectomy with skin reduction. However, in such cases, a high rate of undesirable results is observed, mainly because of the residual scars and the nipple deformities. According to some authors, techniques such as suction-assisted lipectomy and ultrasound-assisted liposuction may be alternative to surgery, and they support ultrasound assisted liposuction use for all grades of gynecomastia, regardless of fibrous state ***(Fruhstorfer et al., 2003; Tashkandi et al., 2004; Kasielska et al., 2013)***.

AIM OF THE WORK

The study is to compare between circumareolar approach and inverted-T pattern aiming to reach the most effective, reliable and recommended technique in surgical management of high grades gynaecomastia.

Chapter 1
EMBRYOLOGY OF THE BREAST

The male and female breast is formed by the ectoderm and mesenchyme. The ducts and alveoli are derived from the ectoderm, while the mesenchyme is responsible for the formation of the connective tissue and its blood vessels ***(Skandalakis, 2004).***

The milk ridge or the milk line develops in the ventral area of the body and extends from the axilla to the inguinal area (Figure 1). However, sometimes it extends down to the triangle of Scarpa. The pectoral part of the milk line is responsible for the formation of the right and left mammary primordia while, the proximal and distal parts of the extrapectoral ridge disappear ***(Skandalakis, 2004).***

The breasts share the same ectodermal origin of skin glands (Figure 2). The ectodermal thickening of the mammary primordium grows into the dermis. This produces 16–24 solid cords of ectodermal cells growing within the underlying mesoderm (dermis). Later, these buds will become canalized and form the lactiferous ducts and alveoli. The epidermal surface of the future nipple is at first a shallow depression during the final trimester. Near term, it becomes everted and ready to accept the lactiferous ducts ***(Kopans, 2007)***.

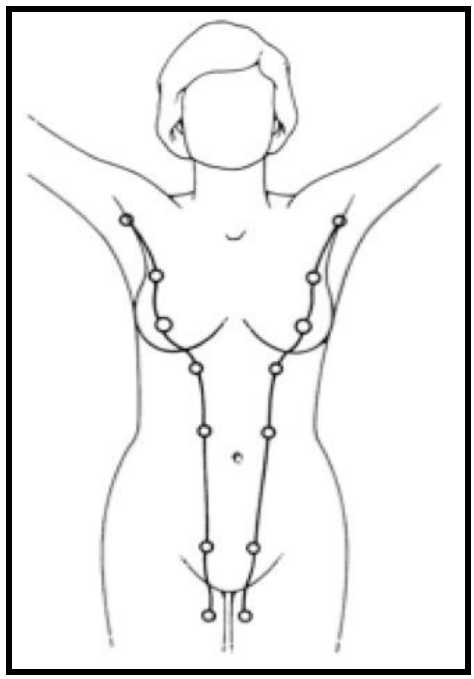

Figure (1): The milk line or ridge ***(Skandalakis et al., 2004).***

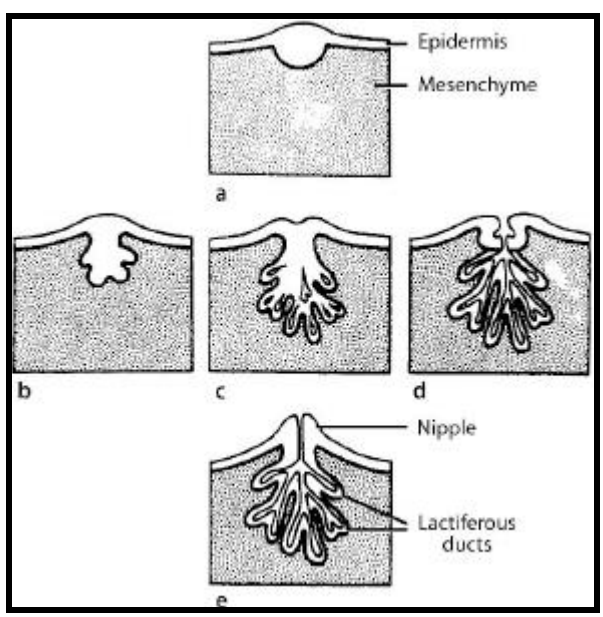

Figure (2): Development of the breast ***(Skandalakis et al., 2004).***

The molecular mechanisms that initiate breast formation have very little explanation about them. Some of the ridges remain in the pectoral area to aid in the genesis of both breasts ***(Howard and Gusterson, 2000)***.

Each mammary lobe has one lactiferous duct, which terminates at the nipple. The direction of the lobes is from the nipple to the connective and fatty tissue of the superficial fascia. So, the breast can be considered as a group of large glands that originate from the epidermis ***(Howard and Gusterson, 2000)***.

The breast forms in the superficial layer of fascia that lies just below the skin. It is not known if the superficial layer splits into a deep and superficial layer to form an incomplete envelope around the gland, or the elongating ducts invaginate the fascia (Figure 3), which then ends up enveloping the gland ***(Kopans, 2007).***

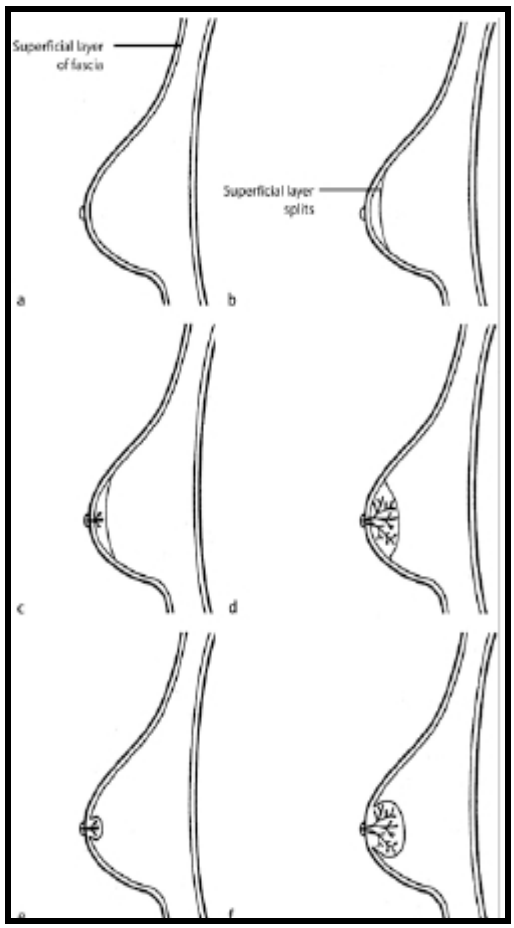

Figure (3): Mammary gland formation from superficial fascia or growing of ducts back into soft tissue ***(Kopans, 2007)***.

The chronology of breast development is presented in (Table 1).

Table (1): Timeline of breast development ***(Kopans, 2007).***

4th to 6th fetal Week	Milk lines or mammary (ectodermal) ridges are developed.
10th fetal week	Proximal and distal part of the milk lines become atrophied while the middle (pectoral) part forms the breast.
5th fetal month	The areola and 15–20 solid cords develop.
After 5th fetal month	Lactiferous ducts and mammary glands develop from the milk lines.
After birth	Nipple becomes visible.
At puberty	Acini appear at the ends of the ducts.

The breast shows changes in both intrauterine and extrauterine period. ***Russo and Russo in (2004)*** classify these changes into two phases:

a) Developmental phase: Early stages of gland morphogenesis from nipple epithelium to lobule formation.

b) Differentiation phase: Differentiation of mammary epithelium.

A small portion of ridges remains in the pectoral region in order to form a single pair of glands ***(Bland and Romrell, 2006).***

The stages of human mammary development (Table 2) consisting on the importance of the mammary fat pad in formation of the mammary epithelium. The fat pad sends signals that allow ductal morphogenesis and alveolar differentiation ***(Neville et al., 1998, 2001).***

Table (2): Stages of mammary development (*GH* growth hormone, *IGF-1* insulin-like growth factor-1, *HGF* human growth factor, *TGF-ß* transforming growth factor-ß, *PRL* prolactin, HER heregulin, FIL feedback inhibitor of lactation) ***(Neville, 1998***).

Developmental stage	Hormonal Regulation	Local factors	Description
Embryogenesis	—	Fat pad necessary for ductal extension.	Epithelial bud forms in 18 to 19 fetal week, extending to mammary fat pad with blind ducts that become canalized; some milk secretion may be seen at birth.
Pubertal Development	—	—	—
Before onset of Menses	Estrogen, GH.	IGF-1, HGF, TGFß.	Ducts extend into the mammary fat pad.
After onset of Menses	Estrogen, progesterone, PRL.	—	Lobules develop and terminal ducts lobular units form.
Development in pregnancy	Progesterone, PRL, placental Lactogen.	HER.	Alveolus formation; partial cellular Differentiation.
Transition: Lactogenesis	Progesterone withdrawal, PRL, glucocorticoid.	Unknown.	Onset of milk secretion: Stage I: midpregnancy Stage II: parturition.
Lactation	PRL, oxytocin.	FIL, stretch.	Milk secretion and ejection**.**
Involution	Withdrawal of Prolactin.	Milk stasis.	Apoptosis and remodeling of alveolar epithelium; gland reverts to prepregnant condition.

Congenital Anomalies of the breast

The congenital anomalies of the breast appear when the typical harmony between ectoderm and mesenchyme fades away ***(Howard and Gusterson, 2000)***.

1. Amastia

Amastia is the absence of one or both breasts and is very rare anomaly. Complete mammary ridge involution, including the pectoral ridge on the affected side is the cause of amastia ***(Greydanus et al., 2006).***

In females, the most common form of amastia is unilateral breast absence. Bilateral amastia has been found in both males and females ***(Skandalakis et al., 1994).***

Trier in 1965 classified amastia into three types:

a) Bilateral amastia

b) Unilateral amastia

c) Bilateral amastia with congenital ectodermal defects

Familial inheritance present in this anomaly and other congenital malformations (skeletal, facial, renal and genital) occur in about 40% of cases. Considered numbers of syndromes are also associated with this condition ***(Merlob, 2003).***

2. Athelia

The congenital absence of one or both nipples and areolas of the breast is called athelia. It may be the rarest of all breast anomalies ***(Huffman et al., 1981).***

It results from failure of development of the lower cervical and upper thoracic somites ***(Lawrence and Lawrence, 2005).***

The precursor of the mammary fat pad is the fatty stroma and the overlying breast tissue cannot grow without a good stroma **(*Ishida et al., 2005).***

Athelia is always associated with other anomalies and amastia is the most associated anomaly with it. So, athelia cannot be classified in distinct groups ***(Ishida et al., 2005).***

Latham and his colleagues in (2006) highlight that athelia is better to be differentiated from amastia (absence of breast tissue and nipple–areolar complex) and amazia (absence of breast tissue). They also spot on that athelia doesn`t mean inverted nipple.

The persistence of fibers from the original invagination of the mammary pit (dimple) is the cause of this anomaly ***(Lawrence and Lawrence, 2005).***

3. Polythelia

Polythelia is the presence of more than two nipples, each with an areola.

Every supernumerary nipple has an areola but not every supernumerary areola has a nipple. Along the embryonic milk line, supernumerary nipples may be found ***(Schmidt, 1998).***

Polythelia may be sporadic, familial, or syndromic ***(Merlob, 2003).***

4. Polymastia

It is the presence of more than two breasts that usually located at the mammary ridge.

Darwin (1889) viewed both polymastia and polythelia as old terms that represent human phylogeny.

Incidence of supernumerary breasts and nipples is a 1–2% of all live births ***(Bland and Romrell, 2006)***.

The reported cases of supernumerary breasts on the vulva, legs, or dorsal trunk are of unknown etiology. Ectopic and normally located breasts may be equally due to pathological causes (mastitis, cancer, etc) ***(Dixon and Mansel, 1994).***

Complete examination of the patient with polymastia is advised to exclude other problems like renal anomalies because