PATHOLOGY OF THE BREAST

Fortunately, most lesions of female breast are innocent, but as is well known, breast cancer was the foremost cause of cancer deaths in women in the United States until 1986, when it was replaced by carcinoma of the lung.

CONGENITAL ANOMALIES

*Supernumerary nipples or breasts* may be found along the embryonic ridge (milk line). *Congenital inversion of the nipple* is of significance because similar changes may be produced by an underlying cancer.

**GALACTOCOELE** is a cystic dilation of an obstructed duct that arises during lactation. Besides being painful, the cysts may rupture to provoke a local inflammatory reaction, which may yield a persistent focus of induration that may arouse suspicion of malignancy. (Fig. 11-1)

**FIBROCYSTIC CHANGES (FCC)** are a group of alterations in the female breast ranging from harmless to those associated with an increased risk of breast carcinoma. Some of these changes (stromal fibrosis and microcysts or macrocysts) produce palpable lumps. FCC are in essence an exaggeration and distortion of the cyclic breast changes that occur normally in the menstrual cycle. Traditionally, these breast alterations have been called fibrocystic disease. However, most of the changes included within this diagnosis have little clinical significance except that they cause nodularity; only a small minority (epithelial hyperplasia) are clinically important. Thus, the term FCC is preferred, since it does not mark the subject with a disease. The lumps produced by the various patterns of fibrocystic change must be distinguished from cancer, and the distinction between the unimportant variants and the more significant ones can be made by examination of fine-needle aspiration material or more definitively by biopsy and histologic evaluation. The alterations encountered within the FCC are subdivided into

1. **Nonproliferative** (simple FCC) that include cysts &/or fibrosis (the most common)
2. **Proliferative** that include a range of harmless to atypical epithelial cell hyperplasias of the ducts or ductules as well as sclerosing adenosis.

All the entities within FCC tend to arise during reproductive period of life but may persist after menopause. The various changes, particularly the nonproliferative ones, are so common, being found at autopsy in up to 80% of women, that they almost constitute physiologic variants.

**Cysts and Fibrosis**: (Fig. 11-2) characterized by an increase in fibrous stroma associated with dilation of ducts and formation of cysts of various sizes. *Grossly*, a single large cyst may form within one breast, but the disorder is usually multifocal and often bilateral. The cysts are up to 5 cm in diameter. Unopened, they are brown to blue (blue dome cysts) and are filled with serous, turbid fluid. *Microscopically*, in smaller cysts, the epithelium is cuboidal to columnar. In larger cysts it may be flattened. Frequently, cysts are lined by large cells with abundant granular, eosinophilic cytoplasm, with small, round, deeply stained nuclei; this is called *apocrine metaplasia*, which is virtually always benign.

**Proliferative Changes**

1. **Epithelial Hyperplasia** comprises a range of proliferative lesions within the ductules, the terminal ducts (ductal hyperplasia), and sometimes the lobules (lobular hyperplasia) of the breast. Some of the epithelial hyperplasias are mild and carry little risk of carcinoma, but others are the more florid and atypical that carry a significantly greater risk. *Microscopically*, the ducts, ductules, or lobules may be filled with cuboidal cells, within which small glands are disposed (fenestrations) (Fig. 11-3). The degree of hyperplasia can be mild, moderate, or severe. In some instances the hyperplastic cells show complex architectural patterns and approaching morphologically those of ductal carcinoma in situ, such hyperplasia is called *atypical*. The line separating the epithelial hyperplasias without atypia from atypical hyperplasia is difficult to define, just as it is difficult to clearly distinguish between atypical hyperplasia and carcinoma in situ.

**Atypical lobular hyperplasia** (Fig. 11-4) describes hyperplasias that cytologically resemble lobular carcinoma in situ, but the cells do not fill or distend more than 50% of the acini within a lobule. *Atypical lobular hyperplasia* is associated with an increased risk of invasive carcinoma.
Epithelial hyperplasia per se does not often produce a clinically discrete breast mass.

2. Sclerosing Adenosis: a significant variant of FCC because its clinical and morphologic features may be deceptively similar to those of carcinoma. Grossly, the lesion has a hard, rubbery consistency, and thus simulates that of breast cancer. Microscopically, there is proliferation of lining epithelial cells and myoepithelial cells in small ducts and ductules, yielding masses of distorted small glands within a fibrous stroma (Fig. 11-5). Aggregated glands or proliferating ductules may be virtually back to back (adenosis). Marked stromal fibrosis, which may compress and distort the proliferating glandular structures, is always associated with the adenosis; hence, the designation sclerosing adenosis. This overgrowth of fibrous tissue may completely compress the lumina of the acini and ducts, so that they appear as solid cords or trabeculae of cells. This pattern may then be difficult to distinguish histologically from an invasive scirrhous carcinoma. The presence of double layers of epithelium and the identification of myoepithelial elements are helpful in suggesting a benign diagnosis. Sclerosing adenosis is associated with only a minimally increased risk of progression to carcinoma.

**Relationship of Fibrocystic Changes to Breast Carcinoma**

This is a controversial issue. With respect to the relationship of the various patterns of fibrocystic change to cancer, the statements below currently represent the most widely accepted opinions:

1. **Minimal or no increased risk of breast carcinoma:**
   - Fibrosis,
   - Cystic changes (microscopic or macroscopic)
   - Apocrine metaplasia
   - Mild hyperplasia
   - Fibroadenoma

2. **Slightly increased risk (1.5-2 times):**
   - Moderate to florid hyperplasia (without atypia)
   - Ductal papillomatosis
   - Sclerosing adenosis.

3. **Significantly increased risk (5 times):**
   - Atypical hyperplasia, ductular or lobular. Proliferative lesions may be multifocal, and the risk of subsequent carcinoma extends to both breasts.

A family history of breast cancer may increase the risk in all categories (e.g., to about 10-fold with atypical hyperplasia).

Fortunately, most women who have lumps related to fibrocystic change can be reassured that there is little or no increased predisposition to cancer.

**INFLAMMATIONS**

Acute mastitis develops when bacteria gain access to the breast tissue through the ducts; when there is inspissation of secretions; through fissures in the nipples, which usually develop during the early weeks of lactation; or from various forms of dermatitis involving the nipple. Staphylococcal infections induce single or multiple abscesses accompanied by the typical clinical acute inflammatory changes. They are usually small, but may be large. (Fig. 11-6) Streptococcal infections generally spread throughout the entire breast, causing pain, marked swelling, and breast tenderness.

Tuberculosis of the breast (Fig. 11-7)

Both clinical and radiological features of tuberculous mastitis are not diagnostic and easily can be confused with either breast cancer or pyogenic breast abscess by clinicians. The involvement is usually unilateral without pulmonary involvement. Multiple draining sinuses may occur.

Although rare, this entity should be taken into consideration; remember the fact that the prognosis for complete cure with appropriate antituberculous drug therapy is excellent.

Definitive diagnosis of the disease is based on identification of TB bacilli (ZN stain) within typical histological features under microscopy or detection of the tubercle bacilli with mycobacterial culture. For breast cancer patients with granulomatous axillary lymphadenitis, PCR may be required to rule out TB in endemic regions.

Idiopathic Granulomatous mastitis (IGM) (Fig. 11-8)

This rare condition is seen mainly in young women, usually after pregnancy. Patients present with firm tender mass. It may be complicated by overlying skin ulcerations & multiple draining sinuses. The diagnosis is by
excluding other granulomatous conditions e.g. TB, fungal infections, sarcoidosis, fat necrosis, foreign body reaction, etc. Its distinctive microscopic feature is that the noncaseating granulomas are centered on the lobules.

**Mammary duct ectasia (MDE)** (Fig. 11-9) is characterized by inspissation of breast secretions in the main excretory ducts. Ductal dilation with subsequent rupture leads to nonbacterial chronic inflammation in the surrounding periductal breast tissues. MDE is usually encountered in multiparous women in their 40s and 50s. Patients present with a poorly defined palpable periareolar mass, sometimes with skin retraction, often accompanied by thick, white nipple secretions. **Grossly**, sectioning shows ropelike dilated ducts from which thick, cheesy secretions can be extruded. **Microscopically**, the dilated ducts are filled by granular debris, sometimes containing lipid-laden macrophages. The most distinguishing features are
1. The prominence of a lymphocytic and plasma cell infiltration
2. The occasional granulomas in the periductal stroma.

Mammary duct ectasia is important because of its clinical confusion with malignancy as it leads to induration of the affected tissue and, more significantly, to retraction of the skin or nipple.

**Traumatic fat necrosis** is significant only because of its confusion clinically with carcinoma. Most women give a history of antecedent trauma to the breast. The lesion consists of a central focus of necrotic fat cells surrounded by neutrophils and lipid-laden macrophages. (Fig. 11-10)

**TUMORS OF THE BREAST**

**Fibroadenoma** is the most common benign neoplasm of the female breast. An increase in estrogen activity is thought to contribute to its development, and indeed similar lesions may appear with fibrocystic changes (fibroadenomatoid changes). The peak incidence is in the third decade of life.

**Gross features**
- The tumor is usually solitary, well-defined, and freely movable.
- It is variable size & may reach up to 10 cm in diameter. Larger tumors are referred to as **giant fibroadenoma**.
- It is firm, with a uniform tan-white color on cut section (Fig. 11-11 A).

**Microscopically**
- There is a loose fibroblastic stroma containing epithelium-lined duct-like spaces of various forms and sizes.
- The duct-like spaces are lined with single or multiple layers of benign epithelial cells having an intact basement membrane.
- In some lesions the ductal spaces are open, and fairly regular (**pericanalicular fibroadenoma**), whereas in others they are compressed by the proliferation stroma and thus appear as slits or irregular, star-shaped structures (**intracanalicular fibroadenoma**) (Fig. 11-11 B).

Cytogenetic studies reveal that the stromal cells are monoclonal and thus represent the neoplastic element of these tumors. It is possible that the neoplastic stromal cells secrete growth factors that induce proliferation of epithelial cells.

**Phyllodes Tumors** are much less common than fibroadenomas. They are thought to arise from the periductal stroma. Most grow to large, possibly massive size; the patient typically has a history of a rapidly growing palpable breast mass. **Grossly**, the tumors are lobulated and cystic and on sectioning exhibit leaf-like clefts and slits. The latter feature is responsible for the naming them as phyllodes tumors (Greek for "leaflike"). Microscopically, there is expansion and increased cellularity of the stromal component. (Fig. 11-12) **The most ominous change is the appearance of anaplasia and high mitotic activity, usually with invasion of adjacent breast tissue (malignant phyllodes tumor)**. Most of these tumors remain localized and are cured by excision; malignant lesions may recur, but they also tend to remain localized. **Only the most malignant, about 15% of cases, metastasize to distant sites**.

**Intraductal Papilloma** is a neoplastic papillary growth within a duct. Most lesions are solitary, found within the principal lactiferous ducts or sinuses, thus present clinically with a serous or bloody nipple discharge and/or a small subareolar nodule and rarely, nipple retraction. **Grossly**, the tumors are usually solitary and less than 1 cm in diameter. **Microscopically**, they consist of delicate, branching papillary growths within a dilated duct (or...
cyst). Each papillary projection has a connective tissue core covered by double layer of cells; outer cuboidal epithelial cells that overlies myoepithelial cells. (Fig. 11-13) Some cases display multiple papillomas (intraductal papillomatosis). The latter sometimes become malignant, whereas the solitary papilloma is virtually benign. Papillary carcinoma is distinguished by
1. The absence of a myoepithelial component and
2. The epithelial cells show either severe cytologic atypia or monotonous ductal morphology.

CARCINOMA (BRCA)
Breast carcinoma in the USA ranks second only to lung cancer as a cause of cancer death in women; in our country it probably ranks first. Despite advances in diagnosis and treatment, almost 25% of women who develop these neoplasms will die of the disease. This has incite an intense study of the possible causes and origins of this form of cancer and of ways to diagnose it early enough to permit cure. 75% of women with breast cancer are older than age 50; only 5% are younger than the age of 40.

Epidemiology and Risk Factors
A large number of risk factors have been identified that modify the likelihood of developing BRCA (Fig. 11-14). These risk factors are divided into well-established and less well-established groups.
1. Geographic Variations: the risk for BRCA is significantly higher in North America and northern Europe than in Asia and Africa. For example, the incidence and mortality rates are five times higher in the United States than in Japan. These differences seem to be environmental rather than genetic in origin, because migrants from low-incidence to high-incidence areas tend to acquire the rates of their adoptive countries, and vice versa. Diet, reproductive patterns, and nursing habits are thought to be involved.
2. Age: BRCA is uncommon in women younger than age 30. Thereafter, the risk steadily increases throughout life reaching a plateau after menopause.
3. Genetics and Family History: up to 10% of BRCA are related to specific inherited mutations. Women are more likely to carry a BRCA susceptibility gene if they have
   a. BRCA before menopause
   b. bilateral cancer
   c. other associated cancers (e.g., ovarian cancer)
   d. a significant family history (i.e., multiple relatives affected before menopause)
   About 50% of women with hereditary BRCA have mutations in gene BRCA1, and an additional 30% have mutations in BRCA2. Both BRCA 1 & 2 seem to be involved in DNA repair and act as tumor suppressor genes. Cancer arises when both alleles are inactive (defective); one due to a germ-line mutation and the second by a subsequent somatic mutation. It is possible that other mechanisms, such as methylation of regulatory regions, act to inactivate the genes in sporadic (nonhereditary) cancer.
4. Prolonged exposure to exogenous estrogens: short-term use of combined estrogen plus progestin as hormonal replacement therapy in postmenopausal women is associated with an increased risk of breast cancer. However, a large study concluded that birth control pills do not increase the risk of breast cancer.
5. Ionizing radiation: e.g. to the chest increases the risk of breast cancer. Only women irradiated before age 30, during breast development, seem to be affected. For example, 20% to 30% of women irradiated for Hodgkin lymphoma in their teens and 20s develop breast cancer, but the risk for women treated later in life is not elevated.
6. Other less well-established risk factors, such as obesity, alcohol consumption, and a diet high in fat, have been implicated in the development of breast cancer on the basis of population studies. Obesity is a recognized risk factor in postmenopausal women.

Pathogenesis
The exact cause of breast cancer remains unknown. However, three sets of influences seem to be important:
1. Genetic changes
2. Hormonal influences
3. Environmental factors
Genetic Changes
In addition to those producing the well-established familial BRCA, genetic changes have also been implicated in the genesis of sporadic (nonfamilial) breast cancer. Mutations affecting proto-oncogenes and tumor suppressor genes in breast epithelium contribute to the malignant transformation process. Overexpression of the
HER2/NEU proto-oncogene has been found to be amplified in up to 30% of invasive breast cancers. This gene is a member of the epidermal growth factor receptor family, and its overexpression is associated with a poor prognosis. Similarly, amplification of RAS and MYC genes has also been reported in some human breast cancers. Mutations of the well-known tumor suppressor genes RB and p53 may also be present. Multiple acquired genetic alterations seem to be involved in the sequential transformation of a normal epithelial cell into a cancerous cell.

**Hormonal Influences**

Endogenous estrogen excess clearly has a significant role. This is supported by the following observations:

1. Many of the risk factors mentioned (long duration of reproductive life, nulliparity, and late age at birth of first child) imply increased exposure to estrogen peaks during the menstrual cycle.
2. Functioning ovarian tumors that elaborate estrogens are associated with breast cancer in postmenopausal women.
3. Estrogens stimulate the production of growth factors by normal breast epithelial cells and by cancer cells. It seems that estrogen (and progesterone) receptors normally present in breast epithelium, and often in breast cancer cells, may interact with growth promoters (such as transforming growth factor α) produced by human breast cancer cells, to create an autocrine mechanism of tumor development.

**Environmental factors** are suggested by the variable incidence of breast cancer in genetically identical groups and the geographic differences in prevalence. Other important environmental variables include irradiation and exogenous estrogens, described earlier.

**Pathological features of BRCA**

- About 4% of women with breast cancer have bilateral primary tumors. The locations of the tumors within the breast are:
  
  - Upper outer quadrant: 50%
  - Central sector (subareolar): 20%
  - Upper inner: 10%
  - Lower outer: 10%
  - Lower inner: 10%

- Of these, **invasive ductal carcinoma is the most common**. Because it usually has an abundant fibrous stroma, it is also referred to as **scirrhous carcinoma**. There are two types of noninvasive breast carcinoma: ductal carcinoma in situ (DCIS) and **lobular carcinoma in situ (LCIS)** both usually arise from the terminal duct lobular unit (TDLU). Both are confined by a basement membrane and do not invade into stroma or lymphovascular channels.

- **DCIS**
  - Has several of histologic appearances. Architectural patterns are often mixed and include solid, comedo, cribriform, papillary, etc.
  - Nuclear appearance ranges from bland and monotonous (low nuclear grade) to pleomorphic (high nuclear grade).
  - The **comedo subtype** is distinctive and is characterized by cells with high-grade nuclei distending spaces with extensive central necrosis (Fig. 11-15). The name derives from the toothpaste-like necrotic tissue that can be extruded from transected ducts with gentle pressure.

DCIS only rarely presents as a palpable or radiologically detectable mass. If detection is delayed, a palpable mass or nipple discharge may develop. The cells in the better differentiated tumors express estrogen and, less often, progesterone receptors. The prognosis for DCIS is excellent, with over 97% long-term survival after simple mastectomy.

- **Paget disease of the nipple** is caused by the extension of DCIS up to the lactiferous ducts and into the contiguous skin of the nipple. The clinical appearance is usually of a unilateral crusting exudate over the nipple and areolar skin. (Fig. 11-16) In about half of cases, an underlying invasive carcinoma will also be present. Prognosis is based on the underlying carcinoma and is not worsened by the presence of Paget disease.
• In LCIS the cells are small & monomorphic with bland, round nuclei and occur in loosely cohesive clusters within distended lobular ductules & acini (Fig. 11-17). Intracellular mucin vacuoles (signet ring cells) are common. LCIS is virtually always an incidental finding, and, unlike DCIS, it does not form masses. Approximately one-third of women with LCIS will eventually develop invasive carcinoma. Unlike DCIS, subsequent invasive carcinomas arise in either breast at significant frequency. Current treatment requires either close clinical and radiologic follow-up of both breasts or bilateral prophylactic mastectomy.

• **Invasive (Infiltrating) Carcinoma (IDC)** is a term used for all carcinomas that cannot be subclassified into one of the specialized types described below; it does not indicate that this tumor specifically arises from the ductal system. Carcinomas of "no special type" or "not otherwise specified"(NOS) are synonyms for ductal carcinomas. The majority (75%) of BRCA fall into this group. This type of cancer is usually associated with DCIS. Most ductal carcinomas produce a desmoplastic response, which replaces normal breast fat and forms a hard, palpable mass. The microscopic appearance is quite variable, ranging from tumors with well-developed tubule formation and low-grade nuclei to tumors consisting of sheets of anaplastic cells. The tumor margins are usually irregular (Fig. 11-18). Advanced cancers may cause dimpling of the skin, retraction of the nipple, or fixation to the chest wall (Fig. 11-19). About two-thirds express estrogen or progesterone receptors, and about one-third overexpress HER2/NEU.

• **Inflammatory carcinoma** is defined clinically by an enlarged, swollen, erythematous breast, usually without a palpable mass. The underlying carcinoma is generally poorly differentiated and diffusely invades the breast parenchyma. The blockage of numerous dermal lymphatic spaces by carcinoma results in the clinical appearance. (Fig. 11-20) True inflammation is minimal or absent. Most of these tumors have distant metastases, and the prognosis is extremely poor.

• **Invasive lobular carcinoma** consists of cells morphologically identical to & is usually associated with LCIS. The cells invade individually into stroma and are often aligned in strands (Indian file). Occasionally they surround cancerous or normal-appearing acini or ducts, creating a so-called bull's-eye pattern. (Fig. 11-21) Lobular carcinomas are also more frequently multicentric and bilateral (10% to 20%). Almost all of these carcinomas express hormone receptors, but HER2/NEU overexpression is very rare or absent. These tumors comprise fewer than 20% of all breast carcinomas.

• **Medullary carcinoma** is a rare subtype, constituting 1% of cases. These cancers consist of sheets of large anaplastic cells with pushing, well-circumscribed borders. There is also a pronounced lymphoplasmacytic infiltrate. These carcinomas uniformly lack hormone receptors and do not overexpress HER2/NEU.

• **Colloid (mucinous) carcinoma** is also a rare subtype. The tumor cells produce abundant quantities of extracellular mucin that dissect into the surrounding stroma. Grossly the tumors are usually soft and gelatinous.

• **Tubular carcinoma** rarely presents as palpable masses but account for 10% of invasive carcinomas smaller than 1 cm found with mammographic screening. It consists of well-formed tubules with low-grade nuclei. Lymph node metastases are rare, and prognosis is excellent.

**Features Common to All Invasive Cancers:** in all forms of BRCA discussed previously, progression of the disease leads to certain local morphologic features. These include a tendency to become adherent to the pectoral muscles or deep fascia of the chest wall, with consequent fixation of the lesion, as well as adherence to the overlying skin, with retraction or dimpling of the skin or nipple. The latter is an important sign, because it may be the first indication of a lesion, observed by the woman herself during self-examination. Involvement of the lymphatic pathways may cause localized lymphedema. In these cases the skin becomes thickened around exaggerated hair follicles, a change known as *peau d'orange* (orange peel) (Fig. 11-20).

**Spread of Breast Cancer**
Spread eventually occurs through lymphatic and hematogenous channels. Lymph node metastases are present in about 40% of cancers presenting as palpable masses but in fewer than 15% of cases found by mammography. Outer quadrant and centrally located lesions typically spread first to the axillary nodes. Those in the inner quadrants often involve the lymph node along the internal mammary arteries. The supraclavicular nodes are sometimes the primary site of spread, but they may become involved only after the axillary and internal mammary nodes are affected. More distant dissemination eventually ensues, with
metastatic involvement of almost any organ or tissue in the body. *Favored locations are the lungs, skeleton, liver, and adrenals and (less commonly) the brain.* However, no site is immune. Metastases may appear many years after apparent therapeutic control of the primary lesion, sometimes 15 years later.

**Clinical Course**

When BRCA is discovered by the woman or her physician, it is felt as a deceptively discrete, solitary, painless, and movable mass. At this time, the carcinoma is typically 2 to 3 cm in size, and involvement of the regional lymph nodes (most often axillary) is already present in about half of patients. With mammographic screening, carcinomas are frequently detected before they become palpable. The average invasive carcinoma found by screening is around 1 cm in size, and only 15% of these have nodal metastases. In addition, in many women DCIS is detected before the development of invasive carcinoma.

**Prognosis**

This is influenced by the following (note that the first three are components of tumor stage):

1. **The size of the primary carcinoma.** Invasive carcinomas smaller than 1 cm have an excellent prognosis in the absence of lymph node metastases.
2. **Lymph node involvement and the number of lymph nodes involved by metastases.** With no axillary node involvement, the 5-year survival rate is close to 90%. The survival rate decreases with each involved lymph node and is less than 50% with 16 or more involved nodes.
3. **Distant metastases.** At this stage the disease is rarely curable, although chemotherapy may prolong survival.
4. **The grade:** Well-differentiated carcinomas have a significantly better prognosis as compared with poorly differentiated carcinomas.
5. **The histologic type:** all specialized types of breast carcinoma (tubular, medullary, and mucinous) have a somewhat better prognosis than carcinomas NOS.
6. **Estrogen or progesterone receptors status:** determining the presence or absence of these receptors is to predict the response to therapy and thus indirectly the prognosis. The highest rate of response is to anti-estrogen therapy (oophorectomy or tamoxifen) is seen in women whose tumors have both estrogen and progesterone receptors. Lower rates of response are seen if only one of the receptors is present. If both are absent, very few patients respond.
7. **The proliferative rate of the cancer** as measured by mitotic counts. Mitotic counts are included as part of the grading system. High proliferative rates are associated with a poorer prognosis.
8. **Aneuploidy i.e.** carcinomas with an abnormal DNA content; these have a slightly worse prognosis.
9. **Overexpression of HER2/NEU** is caused by amplification of the gene. Overexpression is associated with a poorer prognosis. However, the importance of evaluating HER2/NEU is to predict response to a monoclonal antibody ("Herceptin") to the gene product. This is one of the first examples whereby an antitumor antibody therapy has been developed on the basis of a specific gene abnormality present in the tumor.

**MALE BREAST**

**Gynecomastia** refers to enlargement of the male breast, which may occur in response to absolute or relative estrogen excesses. The most important cause of such hyperestrinism in the male is cirrhosis of the liver, with consequent inabilty of the liver to metabolize estrogens. Other causes include Klinefelter syndrome, estrogen-secreting tumors, estrogen therapy, and digitalis therapy. Grossly, a button-like, subareolar swelling develops, usually in both breasts but occasionally in only one. **(Fig. 11-22)**

**Carcinoma** is a rare, with a frequency ratio to breast cancer in the female of 1: 125. It occurs in advanced age. Because of the scant amount of breast substance in the male, the tumor rapidly infiltrates the overlying skin and underlying thoracic wall. Both morphologically and biologically, these tumors resemble invasive carcinomas in the female.