INTERNATIONAL HISTOLOGICAL CLASSIFICATION OF TUMOURS
No. 2

Histological Typing of Breast Tumours
SECOND EDITION

WORLD HEALTH ORGANIZATION
HISTOLOGICAL TYPING OF BREAST TUMOURS
SECOND EDITION
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SECOND EDITION

WORLD HEALTH ORGANIZATION
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No. 19. Histological typing of upper respiratory tract tumours (1978)
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No. 21. Histological typing of tumours of the central nervous system (1979)
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A coded compendium of the International Histological Classification of Tumours (1978)
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Colour photomicrographs
GENERAL PREFACE TO THE SERIES

Among the prerequisites for comparative studies of cancer are international agreement on histological criteria for the classification of cancer types and a standardized nomenclature. At present, pathologists use different terms for the same pathological entity, and furthermore the same term is sometimes applied to lesions of different types. An internationally agreed classification of tumours, acceptable alike to physicians, surgeons, radiologists, pathologists and statisticians, would enable cancer workers in all parts of the world to compare their findings and would facilitate collaboration among them.

In a report published in 1952, a subcommittee of the WHO Expert Committee on Health Statistics discussed the general principles that should govern the statistical classification of tumours and agreed that, to ensure the necessary flexibility and ease in coding, three separate classifications were needed according to (1) anatomical site, (2) histological type, and (3) degree of malignancy. A classification according to anatomical site is available in the International Classification of Disease.

The question of establishing a universally accepted classification by histological type has received much attention during the last 20 years and a particularly valuable Atlas of Tumor Pathology—already numbering more than 40 volumes—is being published in the USA by the Armed Forces Institute of Pathology under the auspices of the National Research Council. An Illustrated Tumour Nomenclature in English, French, German, Latin, Russian and Spanish has also been published by the International Union Against Cancer (UICC).

In 1956 the WHO Executive Board passed a resolution requesting the Director-General to explore the possibility that WHO might organize centres in various parts of the world and arrange for the collection of human tissues and their histological classification. The main purpose of such centres would be to develop histological definitions of cancer types and to facilitate the wide adoption of a uniform nomenclature. This resolution was endorsed by the Tenth World Health Assembly in May 1957 and the following month a Study Group on Histological Classification of Cancer Types met in Oslo to advise WHO on its implementation. The Group recommended criteria for selecting tumour sites

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for study and suggested a procedure for the drafting of histological classifications and testing their validity.

Since 1958, WHO has established 23 centres covering tumours of the lung; breast; soft tissues; oropharynx; bone; ovaries; salivary glands; thyroid; skin; male urogenital tract; jaws; female genital tract; stomach and oesophagus; intestines; central nervous system; liver, biliary tract and pancreas; upper respiratory tract; eye; and endocrine glands; as well as oral precancerous conditions; the leukaemias and lymphomas; comparative oncology; and exfoliative cytology. This work has involved more than 300 pathologists from over 50 countries.

The World Health Organization is indebted to the many pathologists who have participated and are participating in this large undertaking. The pioneer work of many other international and national organizations in the field of histological classification of tumours has greatly facilitated the task undertaken by WHO. Particular gratitude is expressed to the National Cancer Institute, USA, which, through the National Research Council and the USA National Committee for the International Council of Societies of Pathology, is providing financial support to accelerate this work. Finally, WHO wishes to record its appreciation of the valuable help it has received from the International Council of Societies of Pathology (ICSP) in proposing participants and in undertaking to distribute copies of the classifications to national societies of pathology all over the world.
PREFACE TO HISTOLOGICAL TYPING OF BREAST TUMOURS, SECOND EDITION

The first edition of Histological Typing of Breast Tumours was the result of a collaborative effort organized by the World Health Organization and carried out by the International Reference Centre for the Histological Definition and Classification of Breast Tumours, under the direction of Professor R. W. Scarff at the Bland-Sutton Institute of Pathology, Middlesex Hospital, London, England. The International Reference Centre was established in 1960 and the classification was published in 1968.

In order to keep the classification up to date, a WHO consultation was held in 1978 to discuss criticisms and utilization of the 1968 breast tumour classification. The information evaluated at that consultation had been collected by WHO from experts in this field and from literature surveys. Based on this material a new draft was elaborated and distributed to a number of pathologists for comments. Later in 1978 a WHO meeting was held to review proposals for revision (participants listed on page 13). At this meeting the present classification, definitions, and explanatory notes were formulated and recommended for publication. An editorial committee (see page 13) prepared and selected illustrations and made final preparations for the present version.

The histological classification of breast tumours, which appears on pages 15 and 16, contains the morphology code numbers of the International Classification of Diseases for Oncology (ICD-O) for tumours, and of the Systematized Nomenclature of Medicine (SNOMED) for tumour-like lesions.

It will, of course, be appreciated that the classification reflects the present state of knowledge, and modifications are almost certain to be needed as experience accumulates. Although the present classification has been adopted by the members of the group, it necessarily represents a view from which some pathologists may wish to dissent. It is nevertheless hoped that, in the interests of international cooperation, all pathologists will try to use the classification as put forward. Criticism and suggestions for its improvement will be welcomed; these should be sent to the World Health Organization, Geneva.

1 Scarff R. W. & Torloni H. Histological typing of breast tumours. Geneva, World Health Organization, 1968 (International Histological Classification of Tumours, No. 2)
The publications in the series International Histological Classification of Tumours are not intended to serve as textbooks but rather to promote the adoption of a uniform terminology of tumours that will facilitate and improve communication among cancer workers. For this reason the literature references have intentionally been kept to a minimum and readers should refer to standard works on the subject for extensive bibliographies.
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HISTOLOGICAL CLASSIFICATION OF BREAST TUMOURS

I. EPITHELIAL TUMOURS

A. BENIGN

1. Intraductal papilloma
2. Adenoma of the nipple
3. Adenoma
   a. Tubular
   b. Lactating
4. Others

B. MALIGNANT

1. Noninvasive
   a. Intraductal carcinoma
   b. Lobular carcinoma in situ
2. Invasive
   a. Invasive ductal carcinoma
   b. Invasive ductal carcinoma with a predominant intraductal component
   c. Invasive lobular carcinoma
   d. Mucinous carcinoma
   e. Medullary carcinoma
   f. Papillary carcinoma
   g. Tubular carcinoma
   h. Adenoid cystic carcinoma
   i. Secretory [juvenile] carcinoma
   j. Apocrine carcinoma

* These code numbers correspond to ICD-O and SNOMED morphology fields.

a—No specific code available for lactating adenoma.
k. Carcinoma with metaplasia
   i. squamous type 8570/3
   ii. spindle-cell type 8572/3
   iii. cartilaginous and osseous type 8571/3
   iv. mixed type b

l. Others
   3. Paget's disease of the nipple 8540/3 c

II. MIXED CONNECTIVE TISSUE AND EPITHELIAL TUMOURS

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D. GYNAECOMASTIA 71000
E. OTHERS

b—Code specific types.
c—Paget's disease and invasive ductal carcinoma is coded 8541/3.
d—Code behaviour: /0 = benign; /1 = uncertain whether benign or malignant; /3 = malignant.
DEFINITIONS AND EXPLANATORY NOTES

I. EPITHELIAL TUMOURS

A. BENIGN

1. Intraductal papilloma (Fig. 1–4): A discrete benign papillary tumour arising in a mammary duct.

This tumour is typically characterized by orderly, often glandular growth patterns; well-developed fibrovascular stalks; minimal cellular pleomorphism; little hyperchromasia; low mitotic activity; foci of apocrine metaplasia; little, if any, necrosis; and lack of a cribriform pattern. These criteria are helpful in distinguishing papillomas from intraductal carcinomas. Papillomas may be single or multiple. Solitary papillomas are mostly subareolar and have little or no relationship to carcinoma. Multiple papillomas are usually located at the periphery of the breast and are more frequently related to a carcinoma.

Multiple discrete papillomas should be distinguished from the diffuse intraductal papillary hyperplasia seen in mammary dysplasia.

A papilloma may occur within a widely dilated duct, which appears as a cyst, and has been referred to as intracystic papilloma.

2. Adenoma of the nipple (Fig. 5–7): A benign epithelial tumour arising in the nipple ducts and showing an intraductal proliferation, often combined, in the more advanced stages of its evolution, with a tubular component.

The intraductal growth may be papillary or solid. The cells show variations in size and shape. An outer layer of myoepithelial cells is often readily visible. The tubular component may appear to be infiltrative owing to distortion in the stroma. Extension to the surface of the nipple with erosion and discharge can lead to an erroneous clinical impression of cancer. Foci of intraductal or invasive carcinoma, however, may rarely develop in these tumours.

This lesion should be distinguished from discrete papillomas occurring in the subareolar area.

It is also known as subareolar duct papillomatosis and florid papillomatosis of the nipple.

3. Adenoma: An uncommon, well-demarcated, benign tumour composed of glandular elements and scant stroma.

Two forms are described:

a. Tubular adenoma (Fig. 8): An adenoma composed of regular tubules resembling the ductules of a resting (non-secreting) lobule.
b. Lactating adenoma (Fig. 9): An adenoma composed of tubulo-acinar structures with pronounced secretory changes as seen in pregnancy and lactation.

4. Others: Adenomas analogous to those arising in the salivary glands and in the sweat glands have rarely been reported to arise in the breast.

B. MALIGNANT

The classification that follows is based primarily on histological appearances rather than on histogenesis.

The pathologist responsible for the study of a tumour should convey sufficient information, particularly on the extent of disease, to assist in the establishment of a more reliable prognosis.

Many carcinomas of the breast contain combinations of the growth patterns described below. If a component is a minor one only, the tumour should be classified by the predominant pattern. Extensive mixtures, however, require multiple diagnoses.

Histological grading of breast carcinomas has proved to be useful in evaluating prognosis. There are a number of acceptable grading systems; reference should be made to the original articles for details.

Microcalcifications are important findings in mammographs and specimen radiographs, but because they do not contribute to histological typing and are found in both benign and malignant lesions, they are not dealt with in this text.

1. Noninvasive

a. Intraductal carcinoma (Fig. 10–16): A carcinoma of mammary ducts which does not invade the surrounding stroma and is characterized by four growth patterns: solid, comedo, papillary, and cribriform.

Mixtures of these four patterns usually occur, although any one may predominate in a given tumour. The tumour may extend intraepithelially into the mammary lobules. Papillary carcinoma arising in, and limited to, a mammary cyst is referred to as noninvasive intracystic carcinoma.

Failure to identify stromal invasion simply means that it has not been demonstrated but does not rule it out, as rarely regional and distant metastases may occur with these tumours.

b. Lobular carcinoma in situ (Fig. 17–19): A carcinoma involving the intralobular ductules, which are obliterated and distended by loosely aggregated cells, without stromal invasion.

Although generally accepted as an intraepithelial carcinoma, it is considered by some as a precancerous lesion and has been referred to as lobular neoplasia.

The cells are usually relatively uniform and of small or moderate size, with faintly staining cytoplasm and finely structured, rounded nuclei, and
HISTOLOGICAL TYPING OF BREAST TUMOURS

show no or very few mitoses. The tumour cells may extend into extralobular ducts (pagetoid spread) and may replace the ductal epithelium. This lesion is frequently an incidental microscopic finding in breast tissue removed for another reason. It is, however, a dangerous lesion owing to its frequent multicentricity, bilaterality and association with invasive ductal and lobular carcinomas.

2. Invasive

a. Invasive ductal carcinoma (Fig. 20-25): The most frequently encountered malignant tumour of the breast, not falling into any of the other categories of invasive mammmary carcinoma.

The gross and microscopic appearances vary widely. Tumour cells are usually arranged as nests, cords and gland-like structures. Foci of intraductal carcinoma may be present. The tumour can be multicentric.

This category may be subdivided on the basis of the amount of diffuse fibrous stroma, or by the growth pattern as stellate or circumscribed. The significance of these subdivisions, however, needs further study.

This tumour has been referred to as infiltrating duct carcinoma not otherwise specified (NOS), carcinoma of no special type, infiltrating duct carcinoma with productive fibrosis, scirrhous carcinoma, infiltrating carcinoma and carcinoma simplex.

b. Invasive ductal carcinoma with a predominant intraductal component (Fig. 26-27): A carcinoma which is overwhelmingly intraductal and contains foci of stromal invasion.

The justification for delineating this group is that the aggressiveness of ductal carcinomas is to some extent dependent upon the relative amounts of noninvasive and invasive growth. Although additional studies are needed for clarification, it is suggested that this entity be restricted to cases in which the amount of the intraductal carcinoma is at least four times greater than that of the invasive component. The relative amounts of the noninvasive and invasive components should be indicated in diagnostic reports after appropriate sampling.

c. Invasive lobular carcinoma (Fig. 28-33): An invasive carcinoma composed of uniform cells resembling those of lobular carcinoma in situ and usually having a low mitotic rate.

The cells grow typically in a single-file, linear arrangement, or appear individually embedded in fibrous tissue. The stroma may be prominent enough to give a scirrhous appearance both grossly and microscopically. Infiltrating cells are often arranged concentrically around ducts, in a target-like pattern. Identification of remnants of lobular carcinoma in situ aids in the diagnosis. Tumour cells may appear in signet-ring shapes owing to distension with mucus. Other growth patterns have been described, e.g., tubulo-lobular and solid.

d. Mucinous carcinoma (Fig. 34-35): A carcinoma containing large amounts of extracellular epithelial mucus, sufficient to be visible
grossly, and recognizable microscopically surrounding and within
tumour cells.

These tumours usually have blunt rather than irregular borders. The
prognosis of pure mucinous carcinoma is considered to be better than that
of invasive ductal carcinoma.

Mucus can be found in most carcinomas of the breast, but the term
mucinous carcinoma should be restricted to those containing large amounts
of extracellular mucus.

This tumour has been referred to as colloid, gelatinous, mucoid, and
mucous carcinoma.

e. **Medullary carcinoma** (Fig. 36–39): A well-circumscribed carcinoma
composed of poorly differentiated cells with scant stroma and
prominent lymphoid infiltration.

The tumour cells are large with vesicular nuclei, prominent nucleoli, and
indistinct cytoplasmic outlines. Sheets and broad anastomosing cords without
gland-like structures are the usual growth form. Tumour borders should be
histologically blunt and "pushing", not insinuating and tentacular. Despite
poor differentiation and high mitotic rate, these tumours appear to have a
better prognosis than invasive ductal carcinoma.

f. **Papillary carcinoma** (Fig. 40–42): A rare carcinoma whose invasive
pattern is predominantly in the form of papillary structures.

Frequently, foci of intraductal papillary growth are recognizable in the
breast. The papillary architecture is usually displayed in the metastases.

g. **Tubular carcinoma** (Fig. 43–44): A highly differentiated invasive
carcinoma whose cells are regular and arranged in well-defined
tubules typically one layer thick and surrounded by an abundant
fibrous stroma.

Tubular carcinoma should not be confused with invasive ductal carcinoma
with gland-like structures whose cells are less well differentiated.

Distinction from sclerosing adenosis may, at times, be difficult (see V).
Tubular carcinoma has a favourable prognosis.

h. **Adenoid cystic carcinoma** (Fig. 45–46): An invasive carcinoma
having a characteristic cribriform appearance.

These tumours are of the type seen more typically in the salivary gland.
They are uncommon in the breast. Their prognosis is much better than that
of invasive ductal carcinoma. This diagnosis should not be applied to the
cribriform pattern of intraductal carcinoma. Adenoid cystic carcinoma has
been referred to as adenocystic carcinoma and cylindroma.

i. **Secretory [juvenile] carcinoma** (Fig. 47–48): A carcinoma with
pale-staining cells showing prominent secretory activity of the type
seen in pregnancy and lactation. PAS-positive material is present
in the cytoplasm and in acinar-like spaces.
This type has a favourable prognosis, is found more frequently in children, and should not be confused with ductal carcinoma in pregnant women.

j. *Apocrine carcinoma* (Fig. 49–50): A carcinoma composed predominantly of cells with abundant eosinophilic cytoplasm reminiscent of metaplastic apocrine cells.

Foci of apocrine tumour cells may be seen in other types of mammary carcinoma. This tumour has been referred to as oncocytic carcinoma and sweat gland carcinoma.

k. *Carcinoma with metaplasia* (Fig. 51–53): Various types of metaplastic alterations can be observed in carcinomas otherwise recognizable as invasive ductal carcinomas, namely:
   i. squamous type
   ii. spindle-cell type
   iii. cartilaginous and osseous type
   iv. mixed type: containing mixtures of the above (i–iii).

1. Others

A variety of carcinomas have been described as occurring rarely in the breast.

*Lipid-secreting carcinoma* (Fig. 54–55) has cells with foamy cytoplasm and unusually large amounts of lipid, which should be confirmed by appropriate techniques.

Some tumours have been referred to as *small cell carcinoma* (Fig. 56–57) and others as *signet-ring cell carcinoma* (Fig. 58) when it is unclear whether they are of lobular or ductal origin.

Carcinomas with carcinoid features have been described as having argyrophilic granules. Additional studies are needed for further characterization.

* * *

*Inflammatory carcinoma* does not constitute a histological type but rather a clinical entity. It has been used for breast cancers which present with oedema, hyperaemia, tenderness, and rapid enlargement of the breast. This is associated with extensive invasion of dermal lymphatics by tumour.

Breast cancer occurs in the *male* much less frequently than in the female but shows similar histological types.

3. *Paget's disease of the nipple* (Fig. 59–60): A lesion in which large pale-staining cells are present within the epidermis of the nipple, predominantly in its deep half.

The Paget cells typically contain mucin and rarely melanin granules. The cells are arranged singly or in nests and do not invade the dermis. Paget cells may likewise be present in lactiferous ducts and skin appendages. These changes may secondarily extend to the areola and adjacent skin. Paget's
disease of the nipple is almost invariably found to be associated with an intraductal carcinoma and less frequently with an invasive carcinoma. The case should be classified both as Paget's disease and according to the nature of the associated lesion. Paget's disease should be clearly separated from cases of direct invasion of the skin by a mammary carcinoma.

II. MIXED CONNECTIVE TISSUE AND EPITHELIAL TUMOURS

A. FIBROADENOMA (Fig. 61): A discrete benign tumour showing evidence of both connective tissue and epithelial proliferation.

The distinction between intracanalicular and pericanalicular types is not considered to be significant. The epithelial elements on occasion may show marked proliferation or apocrine metaplasia. The connective tissue component is made up of specialized fibroblasts immersed in an abundant myxoid ground substance rich in acid mucopolysaccharides. Involution of the lesion leads to fibrosis, hyalinization and, less frequently, calcification and ossification of the stroma, and to atrophy of the epithelium. Fibroadenomas may be multiple.

Sometimes, especially in adolescents, fibroadenomas may grow rapidly to a very large size (giant fibroadenoma) without having the histological features of phyllodes tumour.

Rarely, carcinoma in situ has been reported to occur in fibroadenoma; mostly, this has been of the lobular type.

B. PHYLLODES TUMOUR [cystosarcoma phyllodes] (Fig. 62–66): A more or less circumscribed neoplasm having a foliated structure and composed of connective tissue and epithelial elements analogous to a fibroadenoma but characterized by a greater connective tissue cellularity. It may also contain myxoid, adipose, osseous, and chondroid foci. The tumour is usually large, but size alone is not a diagnostic criterion.

Prediction of biological behaviour based on histological appearance is difficult, but it is considered useful to separate cases into three categories, benign, borderline, and malignant, based on the following criteria: frequency of mitoses, infiltrative margins, cellular atypia, and cellularity.

Local recurrence is much more frequent than metastasis. Recurrences usually show both epithelial and connective tissue elements, whereas metastases typically have only the malignant connective tissue component.

When a specific sarcoma, such as liposarcoma, occurs within a phyllodes tumour, separate diagnoses should be made.

C. CARCINOSARCOMA: A true carcinosarcoma of the breast is extremely rare when carcinomas with spindle cell, cartilaginous or osseous metaplasia, and phyllodes tumours are excluded.
A. SOFT TISSUE TUMOURS

A variety of soft tissue tumours may occur in the breast. For a detailed classification, definitions, and illustrations, see elsewhere.\textsuperscript{1}

The rare and highly malignant \textit{angiosarcoma} of the breast may be difficult to diagnose because of sampling errors and the occurrence of deceptively benign appearing areas. Postmastectomy angiosarcomas are typically lesions of the soft tissue of the arm rather than of the breast. \textit{Haemangiomas} occur rarely in the female breast. They are of microscopic size and almost always are in a perilobular location.

The term \textit{stromal sarcoma} has been applied to a tumour having microscopic features of a histologically malignant phyllodes tumour but lacking the epithelial component.

\textit{Granular cell tumour} ["myoblastoma"] can mimic carcinoma clinically and grossly. Histologically it should not be confused with carcinomas which contain granular cells.

B. SKIN TUMOURS

A variety of benign and malignant cutaneous tumours may affect the skin of the breast. For a detailed classification, definitions, and illustrations, see elsewhere.\textsuperscript{2}

C. TUMOURS OF HAEMATOPOIETIC AND LYMPHOID TISSUES

Malignant lymphomas and myeloid sarcoma in the mammary region rarely occur in the apparent absence of involvement of other organs. For a detailed classification, definitions, and illustrations, see elsewhere.\textsuperscript{3}

IV. UNCLASSIFIED TUMOURS

Benign and malignant tumours which cannot be placed in any of the above categories.

V. MAMMARY DYSPLASIA/FIBROCYSTIC DISEASE (Fig. 67–83)

A condition characterized by a spectrum of proliferative and regressive alterations of mammary tissues, with an abnormal interplay of epithelial and

\textsuperscript{1} \textsc{Enzinger, F. M.} \textit{et al.}, \textit{Histological typing of soft tissue tumours}. Geneva, World Health Organization, 1969 (International Histological Classification of Tumours, No. 3).

\textsuperscript{2} \textsc{Ten Seldam, R. E. J.} \textit{et al.}, \textit{Histological typing of skin tumours}. Geneva, World Health Organization, 1974 (International Histological Classification of Tumours, No. 12).

\textsuperscript{3} \textsc{Mathe, G.} \textit{et al.}, \textit{Histological and cytological typing of neoplastic diseases of haematopoietic and lymphoid tissues}. Geneva, World Health Organization, 1976 (International Histological Classification of Tumours, No. 14).
connective tissue elements. These alterations combine variously and may produce a palpable lump.

The individual components almost never appear alone. Those dominating should be recorded with special regard to the presence of epithelial hyperplasias.

Most epithelial proliferations probably begin in the terminal ducts and manifest themselves as hyperplastic changes of extralobular ducts (ductal hyperplasia) and/or intralobular ductules (lobular hyperplasia).

Ductal hyperplasia (Fig. 67–73) is characterized by an intraductal proliferation of epithelial cells leading to partial or total obliteration of the lumen. The proliferating cells form varying degrees of solid masses, gland-like structures or papillary fronds. The hyperplasia is frequently diffuse or multifocal and has been referred to as papillomatosis or epitheliosis. Small size and uniformity of cells and nuclei, lack of mitoses, presence of a readily recognizable myoepithelial layer and of apocrine cells favour the diagnosis of a benign condition.

The architecture and cellular details vary greatly. When atypia is pronounced, the term atypical ductal hyperplasia is used. In the most atypical forms, a distinction from intraductal carcinoma may be impossible.

Lobular hyperplasia (Fig. 74–78) is of two types: that resulting from an increase in the number of ductules, i.e., adenosis (see below); and that resulting from a proliferation of epithelial cells within intralobular ductules. The latter can take a form similar to that seen in extralobular ducts (papillomatosis, epitheliosis).

The term atypical lobular hyperplasia is used to designate lesions which are similar to but quantitatively and qualitatively insufficient to support a diagnosis of lobular carcinoma in situ. The persistence of luminal spaces, lack of marked ductular distension, and the persistence of a readily recognizable myoepithelial cell layer help to distinguish atypical lobular hyperplasia from lobular carcinoma in situ.

Proliferation of ductules leading to an increase of tubular profiles is known as adenosis (Fig. 79–83). When this is accompanied by prominent intertubular fibrous tissue, the term sclerosing adenosis is applied. This lesion may be in the form of microscopic foci scattered in the breast parenchyma or may produce an isolated palpable mass which has been referred to as adenosis tumour. In either form, it may simulate infiltrating ductal carcinoma or tubular carcinoma, because of the distortion of ductal structures by the sclerotic stroma. The retention of a lobular pattern, best seen at low magnification, and lack of infiltration into the surrounding fat favour a benign process.

Cysts are usually multiple and vary in size from microscopic to grossly visible. Apocrine metaplastic cells are often found lining mammary cysts. The epithelium may be atrophic or show degrees of hyperplasia and papillary growth.

Focal fibrosis [fibrosclerosis, fibrous disease]. Most of the dysplasias listed above will show some degree of fibrosis, but focal fibrosis is restricted to those cases that show a tumour-like mass due mainly to the production of comparatively acellular hyalinized fibrous tissue encompassing atrophic ducts and ductules.
Ill-defined foci of connective tissue and epithelial proliferations resembling fibroadenomas may be seen in mammary dysplasia/fibrocystic disease. This pattern has been referred to as fibroadenomatous hyperplasia.

VI. TUMOUR-LIKE LESIONS

A. Duct Ectasia (Fig. 84–85): A progressive dilatation of the mammary duct system producing a mass usually located in the subareolar region.

Debris and lipids characteristically fill the lumen. The periductal stroma is infiltrated by inflammatory cells and, as the disease progresses, it becomes more and more fibrotic. When the continuity of the epithelial lining is broken, lipid material enters the stroma and provokes a foreign body reaction. This lesion is also known as periductal mastitis, plasma cell mastitis, and comedomastitis.

B. Inflammatory Pseudotumours (Fig. 86–87) secondary to foreign body reactions can be observed following intramammary injections of various prosthetic materials (mostly paraffin); or as talc granulomas from previous surgical interventions; or more frequently from traumatic fat necrosis, which consists of a focal foreign body reaction to necrotic fat with varying amounts of scar tissue. It forms a hard mass that retracts the surrounding tissues and may mimic carcinoma clinically and grossly. Local infection may lead to inflammatory pseudotumours.

C. Hamartoma (Fig. 88): A well-demarcated mass of mammary ducts and lobules containing varying amounts of fibrous and adipose tissue and producing a distinctive mammographic image. Its nature is unclear.

It differs from mammary dysplasia because of its sharp circumscription and from fibroadenoma by its architecture and the presence of adipose tissue.
Lesions with abundant adipose tissue have been referred to as adenolipoma.

D. Gynaecomastia (Fig. 89–90): A condition of the male breast characterized by proliferation of ducts and increased periductal stroma which may have a myxoid appearance.

Epithelial hyperplasia may be observed. Lobules are rarely present.

E. Others

Asynchronous involution in a lactating breast may produce a localized tumour-like lesion.

Diffuse hyperplasia, so-called virginal hyperplasia, may be unilateral or bilateral and occurs in children and adolescents. Focal or diffuse hyperplasia and lactation changes may occur in nonpregnant women, particularly after the administration of hormones or tranquillizers.
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Unless otherwise stated, all the preparations shown in the photomicrographs reproduced on the following pages were stained with haematoxylin-eosin.
Fig. 1. Intraductal papilloma  
× 40

Fig. 2. Intraductal papilloma  
× 40
Fig. 3. Intraductal papilloma
Variety of papillary and gland-like patterns. × 40

Fig. 4. Intraductal papilloma
 Stromal fibrosis with architectural distortion. Regular epithelium and foci of apocrine metaplasia. × 40
Fig. 5. Adenoma of the nipple
Papillary proliferation within nipple duct. X 75

Fig. 6. Adenoma of the nipple
Same tumour as Fig. 5 with prominent tubular component. X 120
Fig. 7. Adenoma of the nipple
Same tumour as Fig. 5. No significant epithelial atypia. × 300

Fig. 8. Tubular adenoma
Uniform tubules separated by scant stroma. × 40
Fig. 9. Lactating adenoma
Prominent cytoplasmic vacuoles and dilated lumens similar to lactating breast tissue. × 240

Fig. 10. Intraductal carcinoma
Dilated ducts containing tumour cells showing solid and comedo patterns. No stromal invasion present. × 40
Fig. 11. Intraductal carcinoma
Solid and comedo growth patterns. × 120

Fig. 12. Intraductal carcinoma
Papillary growth pattern. × 40
Fig. 13. Intraductal carcinoma
Papillary pattern. Large moderately pleomorphic tumour cells form papillary projections devoid of a fibrovascular stalk. x 300

Fig. 14. Intraductal carcinoma
Cribriform pattern. x 180
Fig. 15. **Intraductal carcinoma**

Solid growth pattern. Mitosis. Pleomorphism and size of nuclei are in contrast with lobular carcinoma in situ; compare with Fig. 19, × 480

Fig. 16. **Intraductal carcinoma**

Intraepithelial extension of intraductal carcinoma into adjacent lobule. No evidence of stromal invasion. This appearance should not be confused with lobular carcinoma in situ. × 120
Fig. 17. Lobular carcinoma in situ
Tumour cells fill ductules at left. Lobule at right is without tumour. \( \times \) 120

Fig. 18. Lobular carcinoma in situ
Ductules greatly distended by tumour cells. No evidence of invasion. \( \times \) 300
Fig. 19. **Lobular carcinoma in situ**
Tumour cells are uniform in size, shape and staining. Cell membranes are well delineated. × 480

Fig. 20. **Invasive ductal carcinoma**
Pleomorphic tumour cells invading breast stroma. No distinctive growth pattern. × 120
Fig. 21. Invasive ductal carcinoma
Tumour cells are much larger than those of residual normal duct. Moderate amount of loose and dense stroma. × 180

Fig. 22. Invasive ductal carcinoma
Abundant dense fibrous stroma (scirrhous growth pattern). × 150
Fig. 23. Invasive ductal carcinoma
Well-differentiated cells arranged in gland-like structures. Unlike tubular carcinoma, there are multiple layers of cells which are irregular. Compare with Fig. 44. x 330

Fig. 24. Invasive ductal carcinoma
Moderately differentiated cells. Scant stroma. Resembles medullary carcinoma but is without lymphoid infiltrate. Compare with Fig. 38 and 39. x 375
Fig. 25. Invasive ductal carcinoma
Poorly differentiated cells. Pleomorphism and high mitotic activity. x 375

Fig. 26. Invasive ductal carcinoma with a predominant intraductal component
Small nests of tumour cells invade the stroma between two tumour-filled ducts. Remainder of specimen was free of invasion. x 75
Fig. 27. Invasive ductal carcinoma with a predominant intraductal component
Focal stromal invasion around tumour-lined duct. This was the only area of invasion found in several sections. × 100

Fig. 28. Invasive lobular carcinoma
Small individually oriented tumour cells within fibrofatty stroma. Foci of residual lobular carcinoma in situ. × 120
Fig. 29. Invasive lobular carcinoma
High magnification from Fig. 28. × 480

Fig. 30. Invasive lobular carcinoma
Uniform tumour cells arranged in single file separated by scant stroma. The tumour cells are smaller and less pleomorphic than those of typical invasive ductal carcinoma. × 480
Fig. 31. Invasive lobular carcinoma
Tumour cells infiltrate the stroma around an uninvolved duct. × 240

Fig. 32. Invasive lobular carcinoma
Tumour cells arranged in irregular masses. × 300
Fig. 33. Invasive lobular carcinoma
Prominent dense fibrous bands separate tumour cells arranged in single file. × 300

Fig. 34. Mucinous carcinoma
Small clumps of uniform tumour cells immersed in abundant epithelial mucin separated by thin strands of stroma. × 100
Fig. 35. Mucinous carcinoma
× 240

Fig. 36. Medullary carcinoma
Circumscribed tumour with "pushing" margin. × 40
Fig. 37. Medullary carcinoma
Prominent lymphoid infiltrate. Scant fibrous stroma. × 150

Fig. 38. Medullary carcinoma
Vesicular nuclei. × 240
Fig. 39. Medullary carcinoma
Nuclear pleomorphism and atypical mitoses. x 375

Fig. 40. Papillary carcinoma
Invasive tumour with a predominantly papillary pattern. x 90
Fig. 41. Papillary carcinoma
Well-differentiated tumour cells without a myoepithelial layer. × 300

Fig. 42. Papillary carcinoma
Papillary growth pattern retained in lymph node metastasis. × 60
Fig. 43. Tubular carcinoma
Small uniform tubules infiltrate the stroma and adipose tissue. No evidence of tubular arrangement.  
× 100

Fig. 44. Tubular carcinoma
The neoplastic tubules are lined by a single layer of small uniform cells. The stroma is dense. Compare with Fig. 23.  
× 300
Fig. 45. Adenoid cystic carcinoma
Masses of infiltrating tumour cells arranged in a typical cribriform pattern. This should not be confused with intraductal carcinoma with cribriform features (Fig. 14). \( \times 120 \)

Fig. 46. Adenoid cystic carcinoma
\( \times 180 \)
Fig. 47. Secretory carcinoma
Nests of tumour cells invade adipose tissue. X 150

Fig. 48. Secretory carcinoma
Large cytoplasmic vacuoles. X 300
Fig. 49. Apocrine carcinoma
Many of the tumour cells have abundant eosinophilic and finely granular cytoplasm. × 300

Fig. 50. Apocrine carcinoma
PAS-positive cytoplasmic granules. × 375
Fig. 51. Carcinoma with metaplasia
Squamous type. × 180

Fig. 52. Carcinoma with metaplasia
Spindle-cell type. × 180
Fig. 53. Carcinoma with metaplasia
Cartilaginous and osseous type. × 40

Fig. 54. Lipid-secreting carcinoma
Majority of cells have abundant pale cytoplasm. × 150
Fig. 55. Lipid-secreting carcinoma
Finely vacuolated cytoplasm. Lipids were demonstrated by fat stains on frozen sections. X 375

Fig. 56. Small cell carcinoma
Individually oriented, moderately pleomorphic tumour cells. X 300
Fig. 57. Small cell carcinoma
Lymph node metastasis of tumour in Fig. 56. Alcian blue-positive mucin is in many cells. The tumour resembled a malignant lymphoma in haematoxylin-eosin stained sections. x 300

Fig. 58. Signet-ring cell carcinoma
The invasive signet-ring cells are adjacent to a focus of lobular carcinoma in situ. x 240
Fig. 59. Paget's disease of the nipple
Numerous pale Paget cells are in the epidermis of the nipple. An infiltrating ductal carcinoma was present in the breast. × 100

Fig. 60. Paget's disease of the nipple
The Paget cells have abundant clear cytoplasm. There is no invasion of the dermis. × 240
Fig. 61. Fibroadenoma
x 100

Fig. 62. Phyllodes tumour
Typical foliated architecture. X 30
Fig. 63. Phyllodes tumour
Pronounced stromal cellularity. × 120

Fig. 64. Phyllodes tumour
Small, uniform duct cells. The connective tissue cells are crowded but have a regular fibroblast-like morphology. × 300
Fig. 65. Phyllodes tumour
Pleomorphism of the neoplastic connective tissue cells. x 150

Fig. 66. Phyllodes tumour
Cellular atypia and abnormal mitosis. x 300
Fig. 67. Ductal hyperplasia
Dilated duct containing hyperplastic papillary projections with fibrovascular stalks. × 75

Fig. 68. Ductal hyperplasia
In addition to the papillary component there are gland-like structures. × 75
Fig. 69. Ductal hyperplasia
Regular epithelium and fibrovascular stalks. Myoepithelial cells are evident in several areas. × 180

Fig. 70. Ductal hyperplasia
Hyperplastic cells virtually fill and distend the lumen of a duct but do not show atypical features. × 150
Fig. 71. Ductal hyperplasia
Mild cellular atypia. × 150

Fig. 72. Ductal hyperplasia
Moderate cellular and architectural atypia. × 300
Fig. 73. Atypical ductal hyperplasia
Marked nuclear atypia and architectural abnormalities approaching a cribriform pattern. × 300

Fig. 74. Lobular hyperplasia
Greatly enlarged lobule due to increased number of ductules, i.e., adenosis. × 120
Fig. 75. Lobular hyperplasia
Higher magnification of Fig. 74 showing regularity of the ductular cells. × 300

Fig. 76. Lobular hyperplasia
Proliferation of epithelial cells within intralobular ductules. × 120
Fig. 77. Lobular hyperplasia
   Same lesion as Fig. 76 showing lack of cellular atypia. × 300

Fig. 78. Atypical lobular hyperplasia
   Nuclear atypia and pronounced hypercellularity but there is preservation of lumens. × 300
Fig. 79. Sclerosing adenosis
Although distorted the lobular architecture is retained. No extension into the adipose tissue. X 50

Fig. 80. Sclerosing adenosis
Ductular epithelial hyperplasia. At this stage the fibrosis is inconspicuous. X 300
Fig. 81. Sclerosing adenosis
Distortion of lobular architecture by early fibrosis. X 150

Fig. 82. Sclerosing adenosis
High magnification of Fig. 81 shows architectural resemblance to invasive carcinoma but the epithelial cells are not atypical. X 300
Fig. 83. Sclerosing adenosis
Marked interstitial fibrosis. × 300

Fig. 84. Duct ectasia
Dilated ducts filled with inspissated secretions and surrounded by dense fibrous tissue. × 40
Fig. 85. **Duct ectasia**
Atrophic duct epithelium, periductal fibrosis and lymphoid infiltration. × 150

Fig. 86. **Inflammatory pseudotumour**
Foreign body reaction to talc produced a clinically suspicious mass at site of previous biopsy. × 180
Fig. 87. Fat necrosis
   Foreign body reaction to necrotic adipose tissue. × 75

Fig. 88. Hamartoma
   Well-demarcated mass of distorted ducts and ductules with fibrous stroma lacking the typical features of fibroadenoma. × 60
Fig. 89. Gynaecomastia
x 100

Fig. 90. Gynaecomastia
Epithelial hyperplasia and loose periductal stroma. x 240