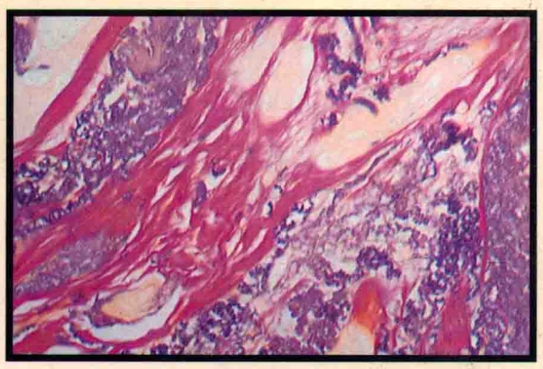
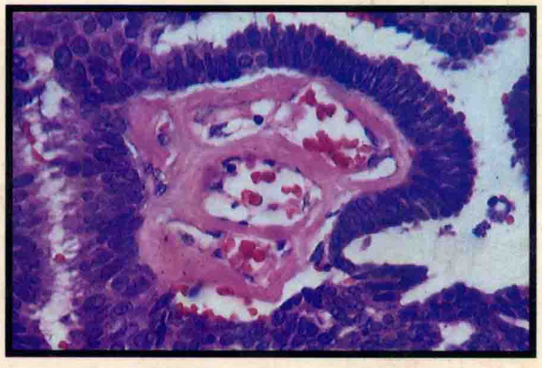
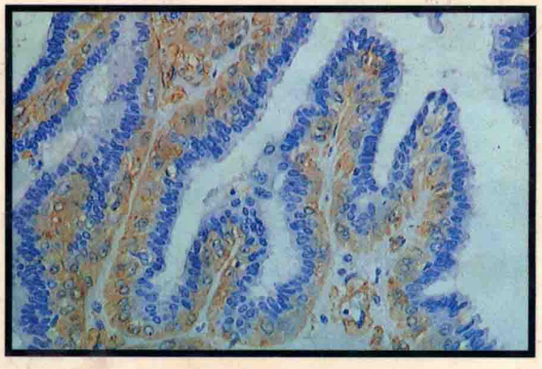
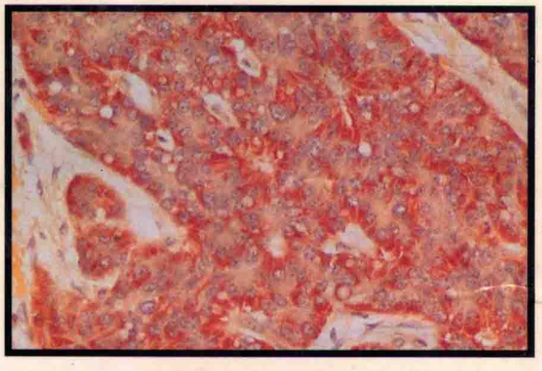
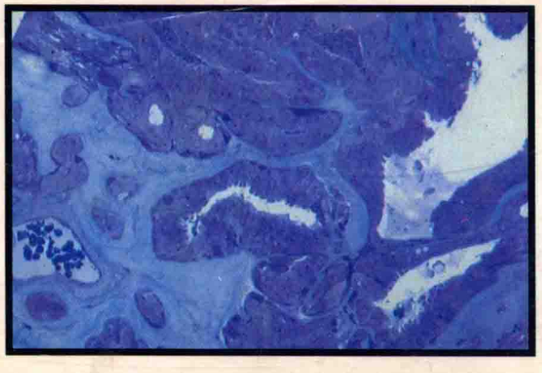
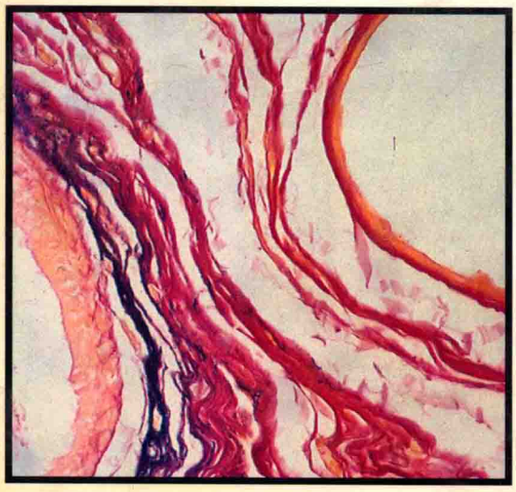
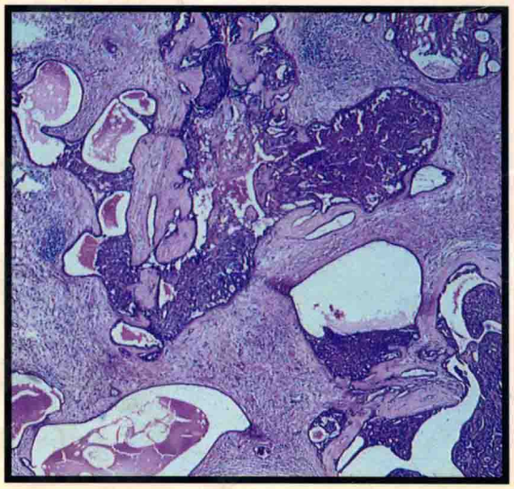
Diagnostic Breast Pathology

A TEXT AND COLOUR ATLAS

ALI AHMED

Churchill Livingstone

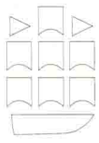
Diagnostic Breast Pathology

A TEXT AND COLOUR ATLAS

Ali Ahmed MD

Honorary Fellow, Department of Pathological Sciences
University of Manchester
Formerly Senior Lecturer in Pathology
University of Manchester
Honorary Consultant Histopathologist
Manchester Royal Infirmary

Consultant in Breast Pathology
Clinique Sainte Catherine, Avignon



CHURCHILL LIVINGSTONE
EDINBURGH LONDON MADRID MELBOURNE AND NEW YORK 1992

CHURCHILL LIVINGSTONE
Medical Divison of Longman Group UK Limited

Distributed in the United States of America by Churchill Livingstone Inc., 650 Avenue of the Americas, New York, N.Y. 10011, and by associated companies, branches and representatives throughout the world.

First published 1992

ISBN 0-443-03185-1

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library.

Library of Congress Cataloging in Publication Data
Ahmed, Ali.
Diagnostic breast pathology: a colour atlas and text / Ali Ahmed.
– 1st ed.
p. cm.
Includes bibliographical references and index.
ISBN 0–443–03185–1
1. Breast–Diseases–Diagnosis–Atlases. 2. Breast–Cancer–Atlases.
3. Breast–Tumors–Atlases. I. Title.
[DNLM: 1. Breast–pathology–atlases. 2. Breast Neoplasms–pathology–atlases.
WP 17 A286d]
RC280.B8A38 1992
618.1'90754–dc20
DNLM/DLC
for Library of Congress 91–34218
CIP

For Churchill Livingstone:

Publisher Timothy Horne
Project Editor Dilys Jones
Production I Macaulay Hunter
Designer Design Resources Unit
Sales Promotion Executive Hilary Brown

Diagnostic Breast Pathology

A TEXT AND COLOUR ATLAS

Preface

The aim of this book is to provide a concise illustrated account of diagnostic breast pathology. The book is primarily intended for practising and trainee histopathologists and should be of value to surgeons, radiologists and oncologists responsible for the management of breast disease.

During the last decade, there have been many changes in the practice of the diagnosis and management of breast disease. The advent of breast screening and the wide use of mammography have resulted in the excision of small and often early breast lesions. Limited surgical procedures and special biopsy techniques have further reduced the amount of tissue available for histopathological examination. In order to obtain the maximum information from these small biopsies, it will become increasingly necessary to employ new techniques, such as immunohistochemistry, as an adjunct to the routine haematoxylin and eosin stain. The use, therefore, of monoclonal antibodies to assess accurately the cellular and structural configuration of breast lesions has been emphasised throughout the atlas. As electron microscopy can also facilitate the full understanding of light microscopic appearances, selected electron micrographs have been included where appropriate.

In recent years, a considerable amount of literature on breast disorders has been devoted to diagnostic categories that grossly and microscopically mimic cancer. Such lesions, as well as epithelial hyperplasia and in-situ carcinoma, are described and illustrated in a comparative format which is a unique feature of this atlas. This arrangement should assist in the study and easy comparison of these lesions.

Each main topic includes a section devoted to the diagnostic and clinical aspects and is intended to reflect the increasing and important role of the histopathologist in the management of breast disease.

I am grateful to the following colleagues who have kindly allowed me access to their cases and material: Dr S. Banik, Dr S. S. Banerjee, Dr J. Davson, Dr Indu Gupta, Dr N. Y. Haboubi, Dr M. Harris, Dr A. W. Jones, Dr W. Fiona Knox, Dr R. F. T. McMahon, Dr Lorna J. McWilliam, Dr A. R. Mainwaring, Dr Caroline M. Nicholson, Dr Marion B. Reid and Dr N. L. Reeve.

I wish to thank the staff in Surgical Histology at the Manchester Royal Infirmary and in particular Graham Bigley for his technical assistance. My thanks are also due to Mrs Rita Parkinson for typing the manuscript.

The critical help provided by the staff at Churchill Livingstone, during the production of the atlas, is greatly appreciated.

Chateaurenard, Provence
1992

Contents

1 The normal breast

Resting breast

The glandular tissue in the breast is located mainly in the upper outer quadrant[1] and central area. The major component of the breast is fibrous tissue and fat.

The normal resting lobule consists of a collection of small, blind-ending epithelial structures termed acini, alveoli or terminal ductules.[2] Each terminal ductule is connected to a small ductule, sometimes termed terminal duct. This terminal ductal lobular unit[3] (Fig. 1.1) is surrounded by loose, vascular connective tissue. The extralobular terminal ducts lead to larger ducts (Fig. 1.2) and eventually to a segmental duct. These segmental ducts extend towards the nipple and connect to lactiferous ducts (Fig. 1.3) and collecting ducts at the surface of the nipple. Numerous apocrine and sebaceous glands are present in the vicinity of the nipple (Fig. 1.4). Irregularly arranged bundles of smooth muscle are also a prominent feature in the nipple (Fig. 1.5).

The ducts and ductules are lined by two cell types. The inner layer is composed of columnar or cuboidal epithelium (Fig. 1.6). The outer layer consists of myoepithelial cells which are arranged in a discontinuous layer in the ductules and a continuous layer in the ducts (Fig. 1.2). In H & E preparations, the accurate characterisation of the two cell types can often prove difficult. Immunohistochemical techniques can facilitate both the appreciation and the identification

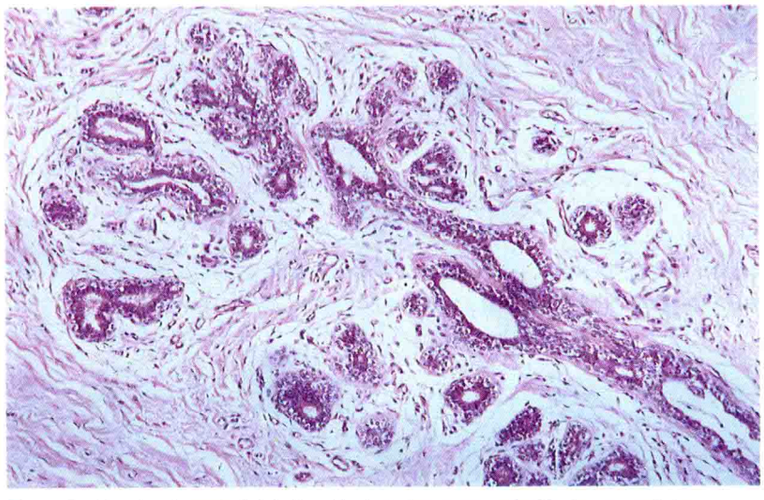


Fig. 1.1 Resting breast. A lobule with ductules surrounded by loose, cellular stroma.

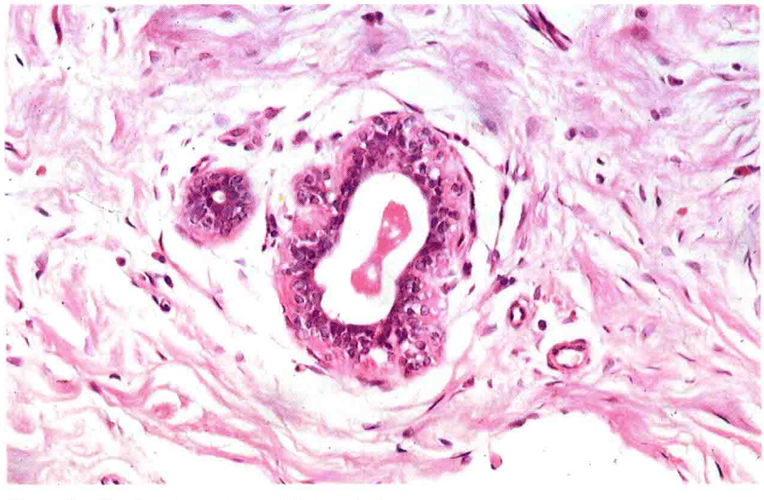


Fig. 1.2 Resting breast. Interlobular duct.

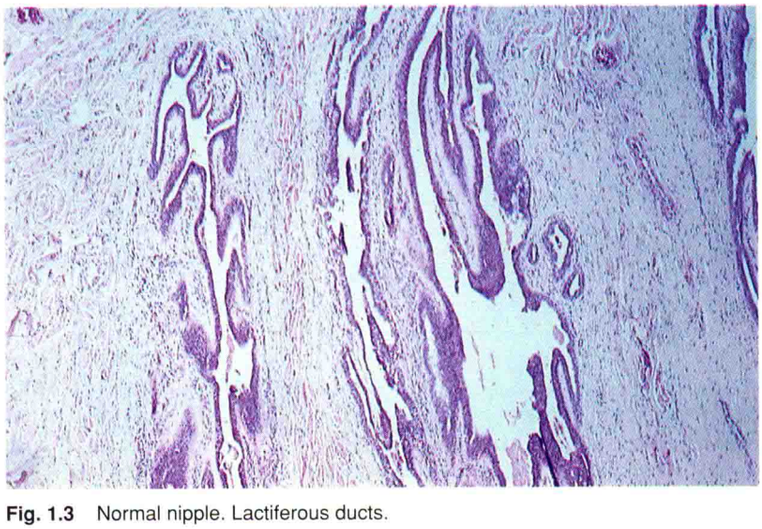


Fig. 1.3 Normal nipple. Lactiferous ducts.

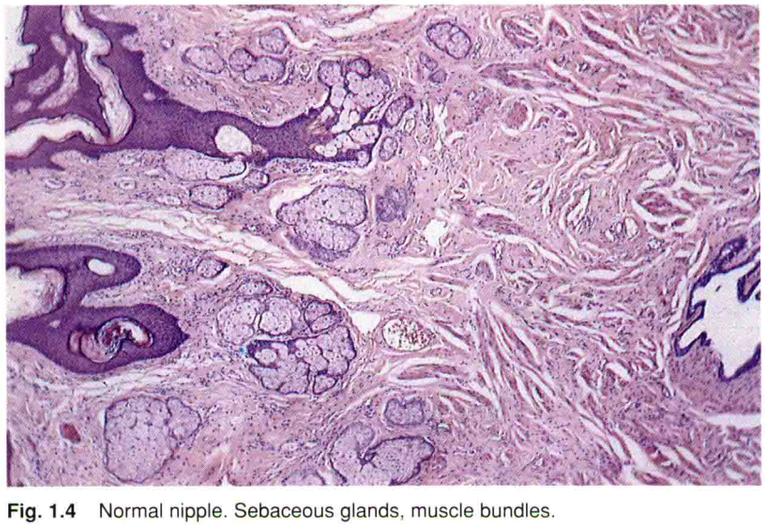


Fig. 1.4 Normal nipple. Sebaceous glands, muscle bundles.

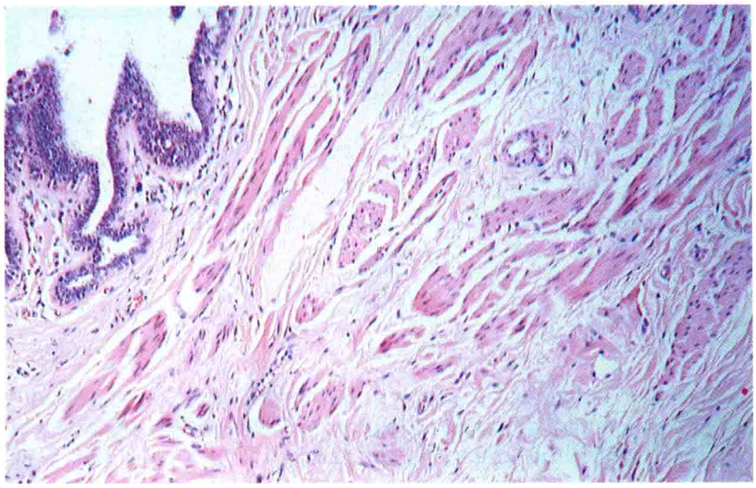


Fig. 1.5 Normal nipple. Bundles of smooth muscle cells.

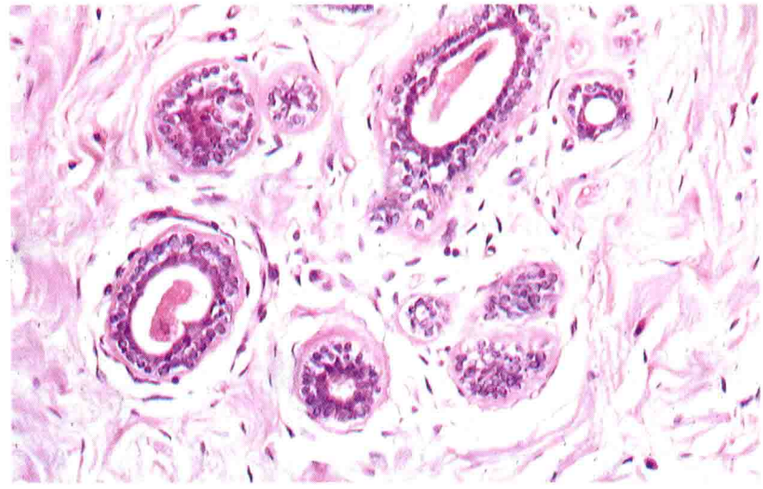


Fig. 1.6 Ductules. Two cell types.

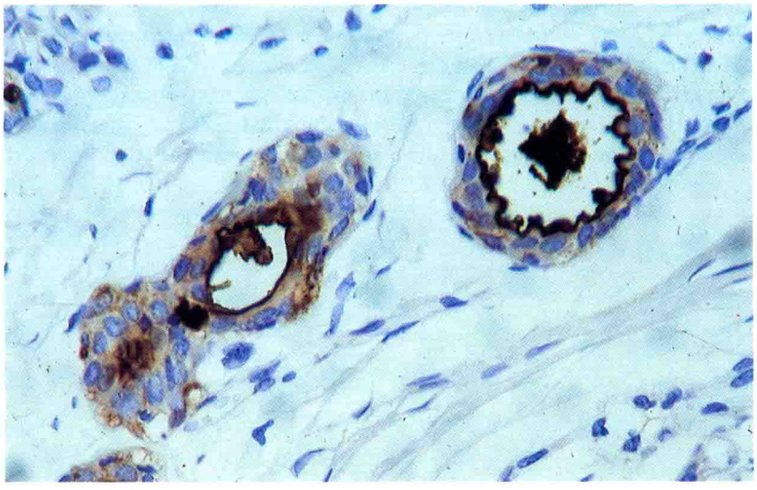


Fig. 1.7 Epithelial cells. EMA stain.

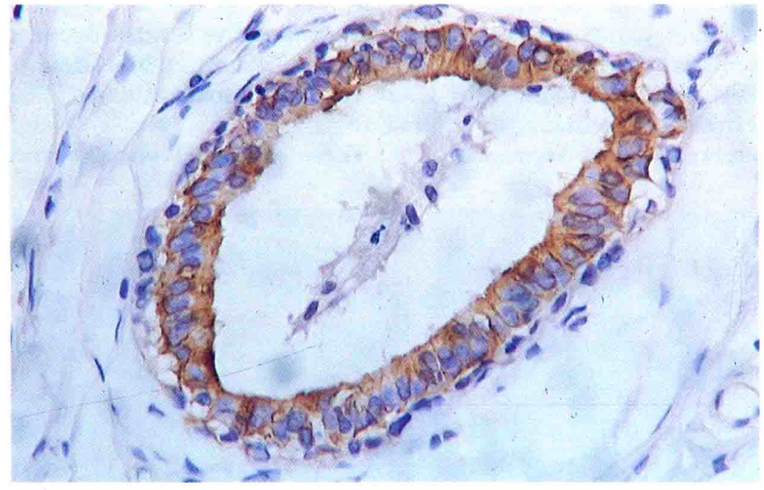


Fig. 1.8 Epithelial cells. Cytokeratin antibody stain.

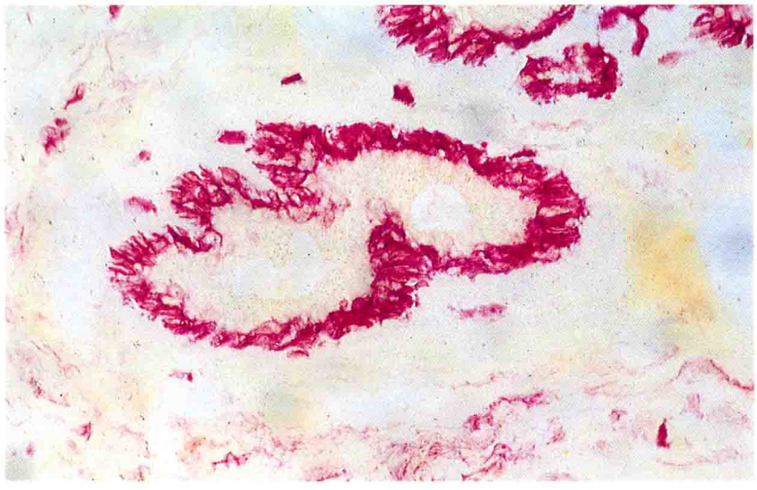


Fig. 1.9 Myoepithelial cells. Alkaline phosphatase stain. Frozen section.

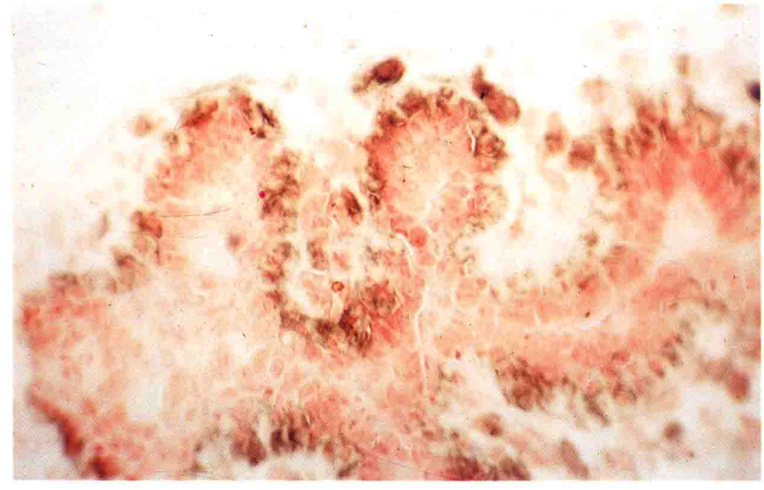


Fig. 1.10 Myoepithelial cells. Adenosine triphosphatase stain. Frozen section.

of epithelial and myoepithelial cells. Epithelial cells are stained by antibody to epithelial membrane antigen (EMA) and anticytokeratin antibodies especially to 'low molecular weight' cytokeratin[4] (Figs 1.7 and 1.8). Myoepithelial cells can be clearly demonstrated in fresh tissue, frozen sections with alkaline phosphatase[5] (Fig. 1.9) and adenosine triphosphatase[6] stains (Fig. 1.10). In routinely formalin fixed, paraffin embedded tissue, α-smooth muscle actin is a very useful marker for myoepithelial cells (Fig. 1.11). It is important to note that anti-actin antibodies can also stain myofibroblasts[7,8] and vascular smooth muscle. S100 protein can also be localised in myoepithelial cells (Fig. 1.12) but the distribution is variable and may also be found in epithelial cells and breast cancer cells.[9] Other

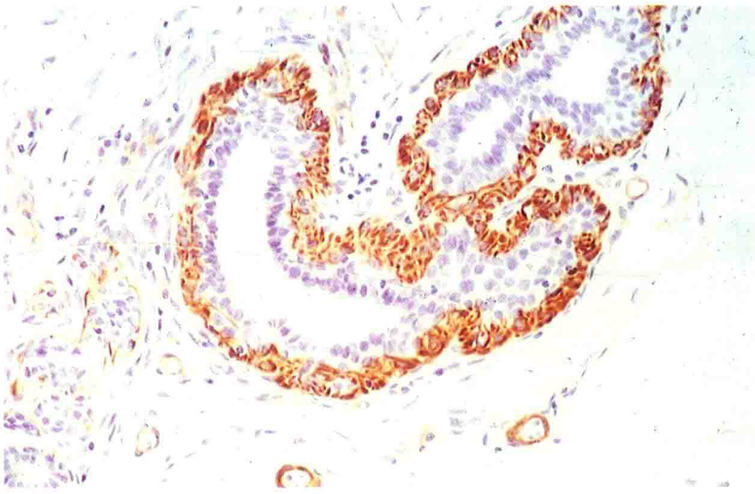


Fig. 1.11 Myoepithelial cells. α-smooth muscle actin antibody. Paraffin embedded section.

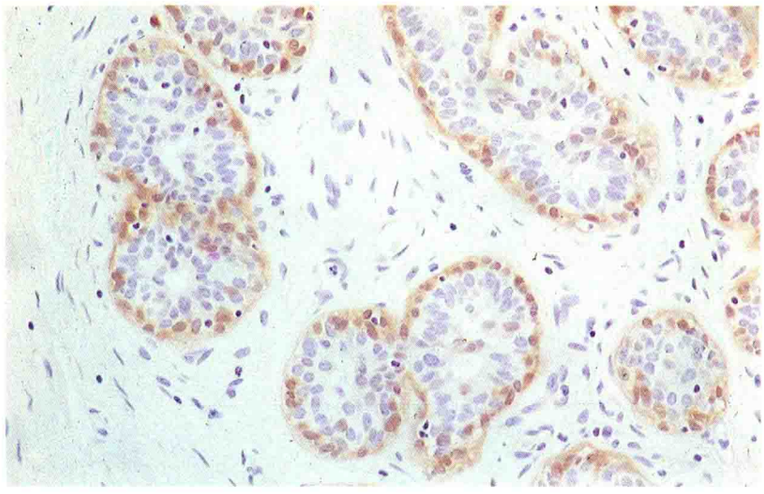


Fig. 1.12 Myoepithelial cells. S100 protein.

markers used in the identification of myoepithelial cells include anti-actin antibody[10] and anti-Common Acute Lymphoblastic Leukaemia Antigen (CALLA) antibody[11] on fresh tissue, and anti-muscle actin–specific antigen on formalin fixed paraffin embedded tissue.[12]

Electron microscopy is also useful in the characterisation of epithelial and myoepithelial cells. In the resting *epithelium*, the cytoplasm is relatively sparse in organelles which include free ribosomes, a few scattered profiles of rough surfaced endoplasmic reticulum, occasional mitochondria and inconspicuous Golgi complex (Fig. 1.13). The *myoepithelium*, situated between the epithelial cells and the basal lamina, is characterised by the presence of cytoplasmic filaments with dense bodies (Fig. 1.14). The cytoplasmic organelles are confined to the paranuclear and apical zones. The nucleus is irregular in shape with deep indentations of the nuclear envelope characteristic of potentially contractile cells. The basal plasma membrane presents distinctive club-like processes covered with hemidesmosome (Figs 1.14, 1.15), a feature which is absent in epithelial cells in contact with the basal lamina (Fig. 1.16). The basal as well as apical plasma membranes of the myoepithelial cell also characteristically possess pinocytotic vesicles (Fig. 1.15). Cytoplasmic filaments with dense bodies, hemidesmosomes and pinocytotic vesicles represent distinctive ultrastructural features of myoepithelial cells.

Occasional intraepithelial lymphocytes and macrophages are also seen between epithelial and myoepithelial cells[13,14] (Fig. 1.17).

The periductal connective tissue consists mainly of scattered collagen fibres among which there is a layer of extremely attenuated fibroblasts termed 'delimiting fibroblasts' (Fig. 1.16).[15]

Physiological variations

Menstrual cycle. Morphological changes in the functional unit of lobule and terminal duct during the menstrual cycle have been described.[16,17,18,19] During the proliferative phase the ductules are

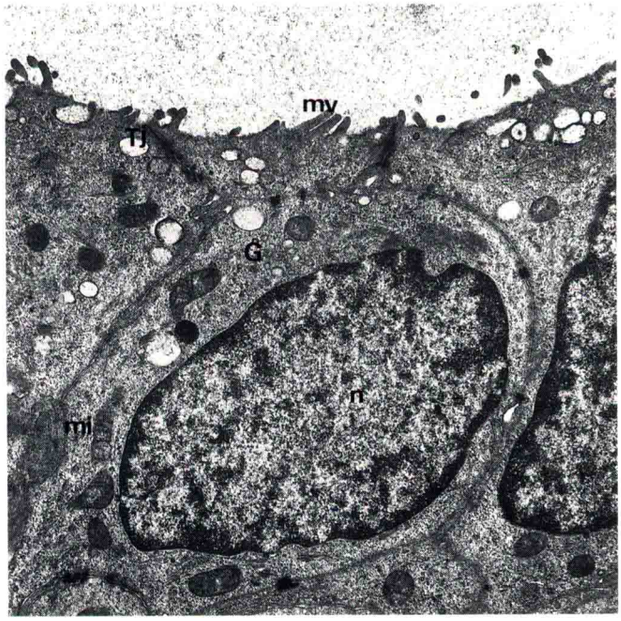


Fig. 1.13 Epithelium. Electron micrograph. The nucleus (n) is round. Occasional mitochondria (mi) and Golgi complex (G) are seen. Microvilli (mv) and tight junction (Tj) are present along the luminal surface.

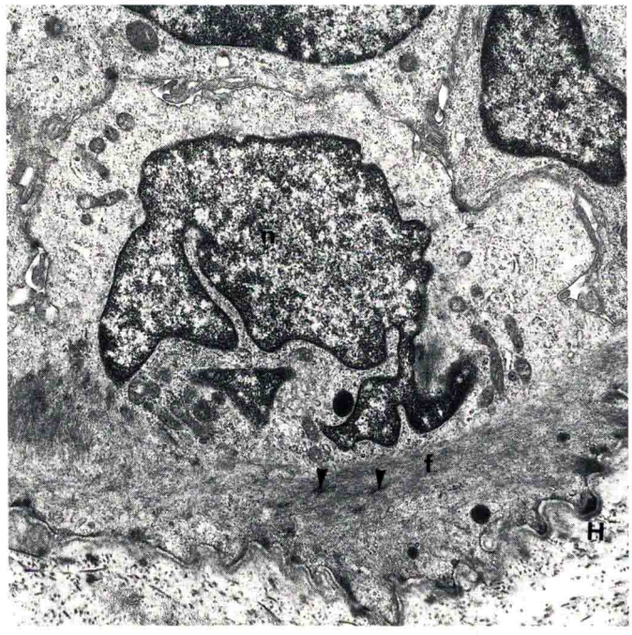


Fig. 1.14 Myoepithelium. Electron micrograph. The nucleus (n) is irregular with deep indentations. The cytoplasmic filaments (f) with characteristic dense bodies (arrowheads) are located in the basal zone. Hemidesmosomes (H) are present on the club-like processes.

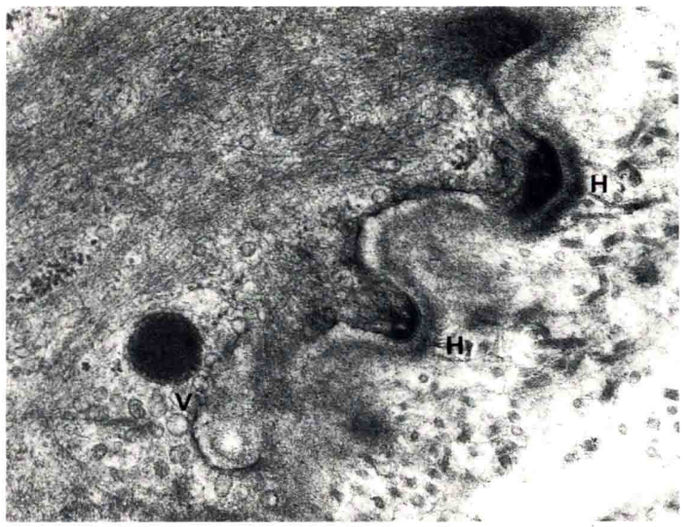


Fig. 1.15 Myoepithelium. Hemidesmosomes (H) and pinocytotic vesicles (V).

small and are surrounded by condensed intralobular stroma containing plasma cell infiltrate.[17] During the secretory phase, there is parenchymal proliferation and the lobules and ductules increase in size. The stroma becomes loose and oedematous.[19] During the late secretory phase, there is vacuolation of the basal cells[19] which may be due to the accumulation of glycogen. The frequency of mitoses during the menstrual cycle has been assessed and appears to be particularly prominent in the premenstrual phase.[18] Lymphocytic infiltrate[19] and apoptosis[18] are dominant features at the onset of menstruation.

Pregnancy. There is marked proliferation and enlargement of lobules during pregnancy. There is progressive obliteration of both intralobular and interlobular stroma (Fig. 1.18) and ductules or acini are arranged in close proximity (Fig. 1.19). The myoepithelial cells become compressed and elongated and can be difficult to identify in H & E preparations (Fig. 1.19). Such myoepithelial cells

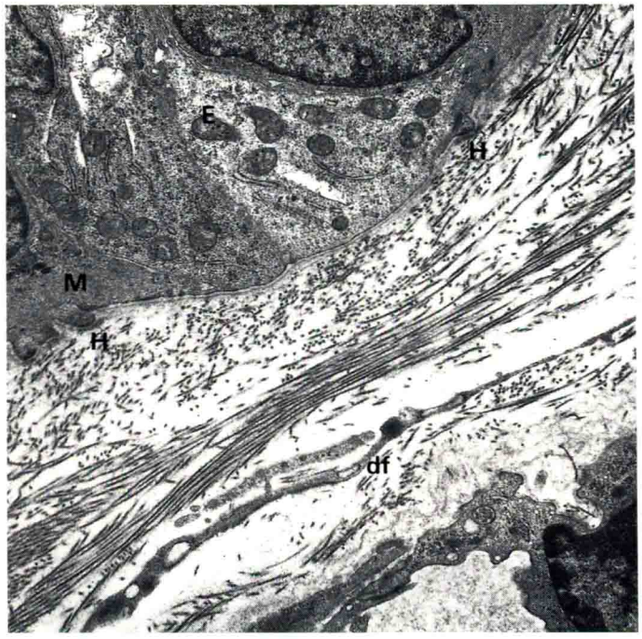


Fig. 1.16 Epithelial cell (E) lacks hemidesmosomes (H) as seen in the adjacent myoepithelial cell (M). Delimiting fibroblasts (df) are present as an attenuated layer.

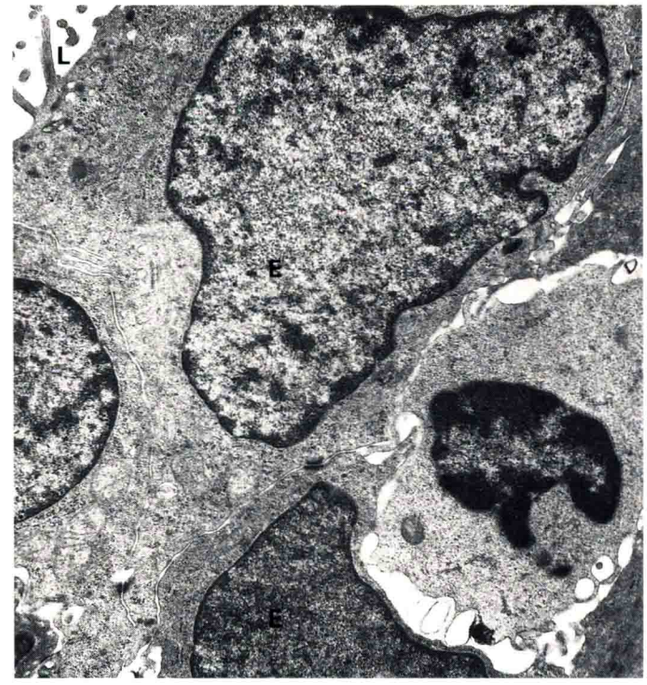


Fig. 1.17 Intraepithelial lymphocyte is seen adjacent to epithelial cell (E). Microvilli project towards the lumen (L).

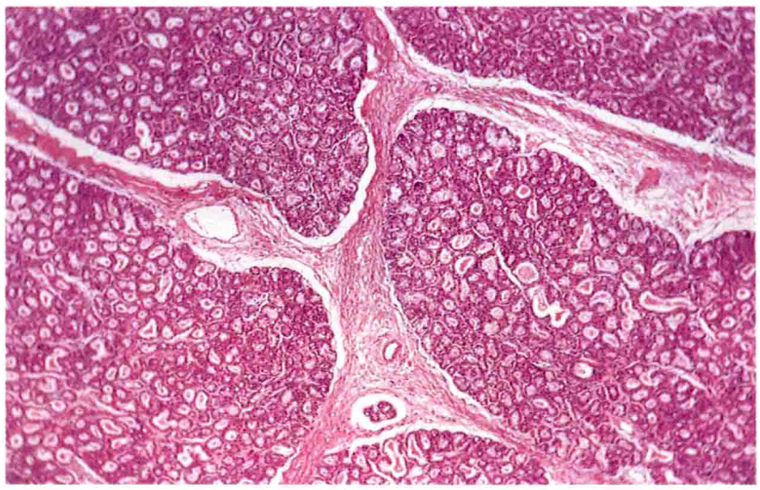


Fig. 1.18 Pregnancy. Lobular proliferation and enlargement.

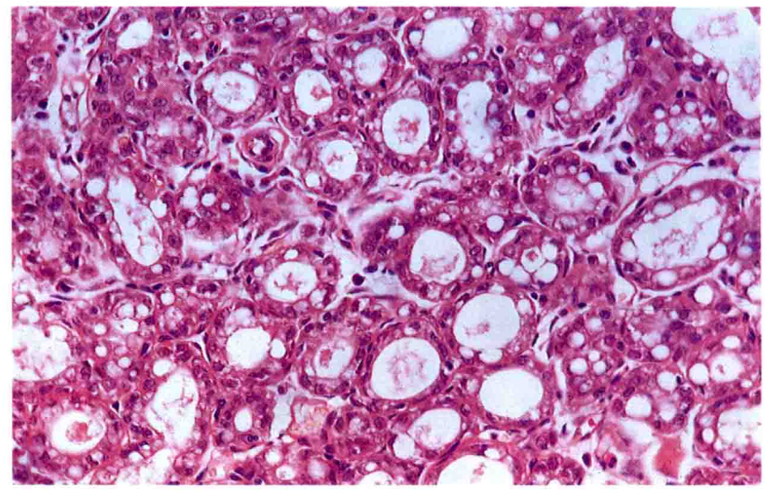


Fig. 1.19 Pregnancy. Ductules or acini in close approximation. Myoepithelial cells are difficult to identify.

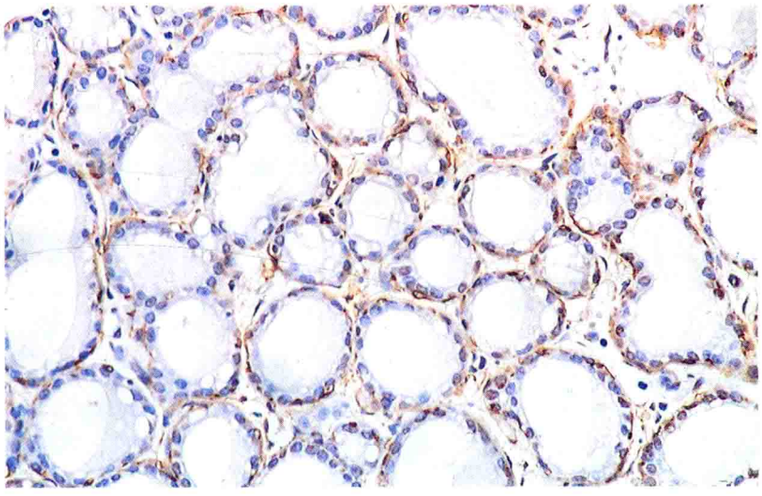


Fig. 1.20 Pregnancy. Myoepithelial cell stained with α-smooth muscle actin antibody.

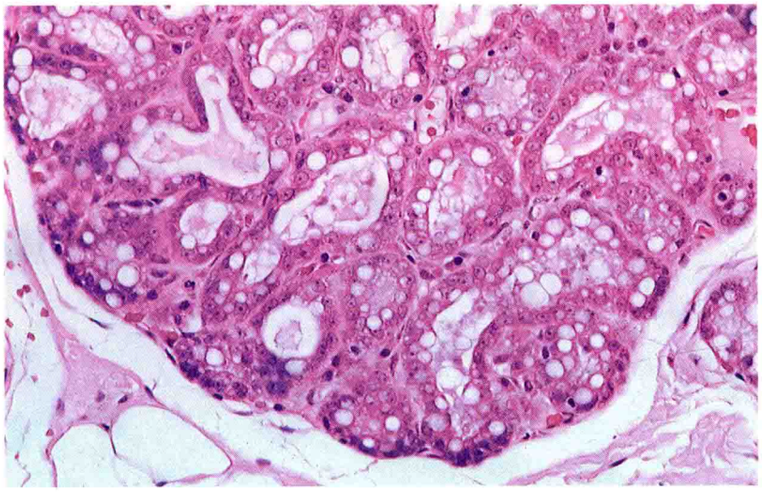


Fig. 1.22 Lactation. Ductules show variable size. Note the cytoplasmic vacuolation.

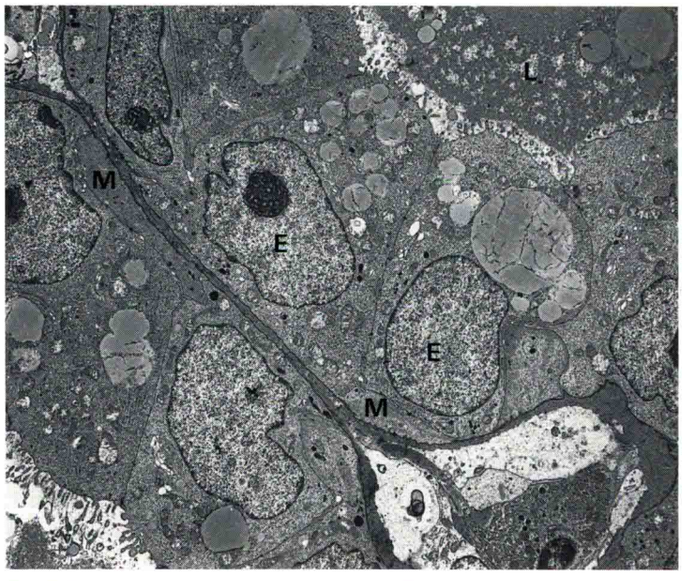


Fig. 1.21 Pregnancy. Electron micrograph. The closely situated ductules are lined by organelle-rich epithelial cells (E). Note the fat globules and adjacent compressed myoepithelial cells (M). Secretions are also present in the lumen (L).

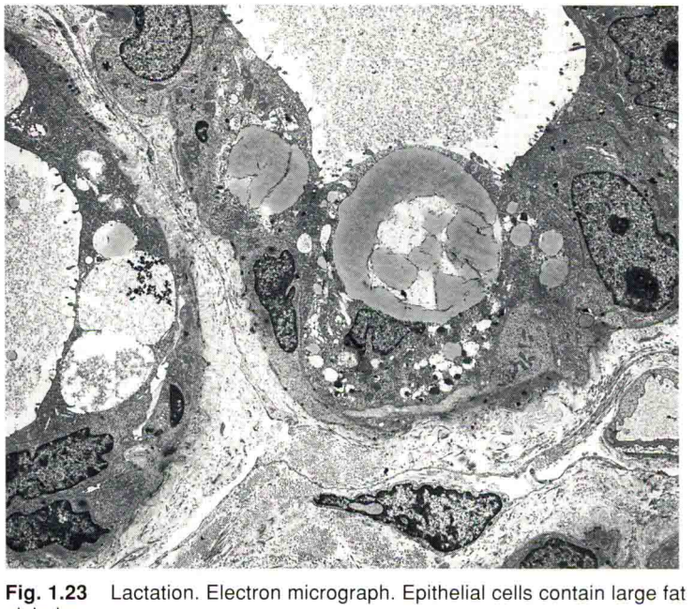


Fig. 1.23 Lactation. Electron micrograph. Epithelial cells contain large fat globules.

can be demonstrated with α-smooth muscle actin antibody (Fig. 1.20). The proliferative changes during pregnancy can be particularly appreciated at the cellular level. In contrast to the resting epithelium (Fig. 1.13) the progressive cellular development produces an abundance of cytoplasmic organelles as well as lipid-bodies, fat globules and secretory vesicles (Fig. 1.21).

Lactation. During lactation, there is increased secretory activity in the ductules with a variable distension of the glandular lumina (Fig. 1.22). These changes are not uniform and groups of glandular structures may show minimal secretory changes. At the ultra-structural level, the epithelial cell cytoplasm is filled with large fat globules and secretory vesicles (Fig. 1.23). Occasionally, secretory material may be observed being discharged into the lumen (Fig. 1.24).

Post-menopausal involution. Involutionary changes take place after pregnancy and lactation. The term involution, however, is normally used to describe post-menopausal atrophic changes involving lobules, ducts and the stroma. There is gradual decrease in the amount of lobular component after the menopause, although the process may begin well before the onset of the menopause.[1,20] The glandular structure becomes smaller with loss of lumina (Fig. 1.25). There is hyalinisation of the specialised intralobular stroma and a gradual increase in the amount of fatty tissue in the interlobular stroma. Eventually there may be complete disappearance of lobules.[2] The ducts also show atrophy and shrinkage with relative prominence of myoepithelial cells (Fig. 1.26). Microcystic changes are often a feature (Fig. 1.27) and should not be confused with fibrocystic change. Dilated ducts, in involution, have been termed varicose or ectatic in order to prevent a false diagnosis of cystic 'disease'.[20] Persistence of mature lobules after the menopause is considered to be a risk factor for co-existent or subsequent cancer.[21]

Focal pregnancy-like change. Focal pregnancy-like change also termed lactational foci can involve a single lobule or part of lobule.[22] The affected lobule is enlarged and the ductules or acini are dilated (Fig. 1.28). The cells project into the lumina and exhibit vacuolated cytoplasm. The nuclei are large and hyperchromatic, and are often located apically, producing a hobnailed appearance (Fig. 1.29). The nuclear hyperchromasia and apparent pleomorphism may occasionally be confused with a malignant lesion.

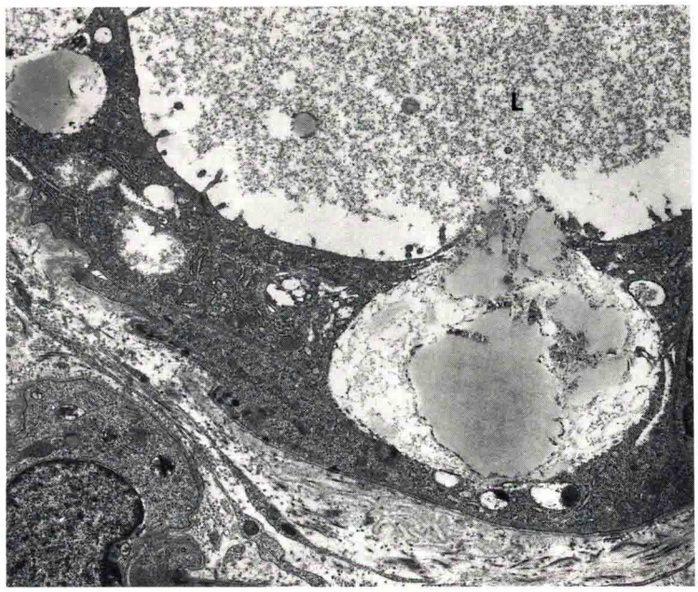


Fig. 1.24 Lactation. A large secretory globule appears to be discharging into the lumen (L).

There are morphological similarities to lactation. Alpha-lactalbumin has been demonstrated in lactional foci.[23] At the ultrastructural level, the epithelial cells are rich in cytoplasmic organelles and contain lipid droplets of varying sizes (Fig. 1.30).

The lesion can occur in young or elderly women. There is no definite relationship between pregnancy or lactation, and pregnancy-like change has been described in nulliparous women.[23,24] A possible association has been suggested with anti-hypertensive, anti-psychotic and hormone preparations.[24]

There is no relationship between focal pregnancy-like change and carcinoma or any specific benign breast lesion.

Clear cell change. Clear cell change is an incidental finding involving the whole or part of a lobule.[25] The lobule is usually enlarged and the ductules are expanded with large, clear cells (Fig. 1.31). These clear cells are polygonal with distinct borders. Nuclei are small, round and eccentrically located (Fig. 1.32). The cytoplasm is clear and abundant but may also contain eosinophilic granules which are PAS-positive and diastase resistant (Figs 1.33, 1.34). At the ultrastructural level some of the granules are membrane-bound and electron-dense whilst others are amorphous.[25] Glycogen granules have not been identified.[24]

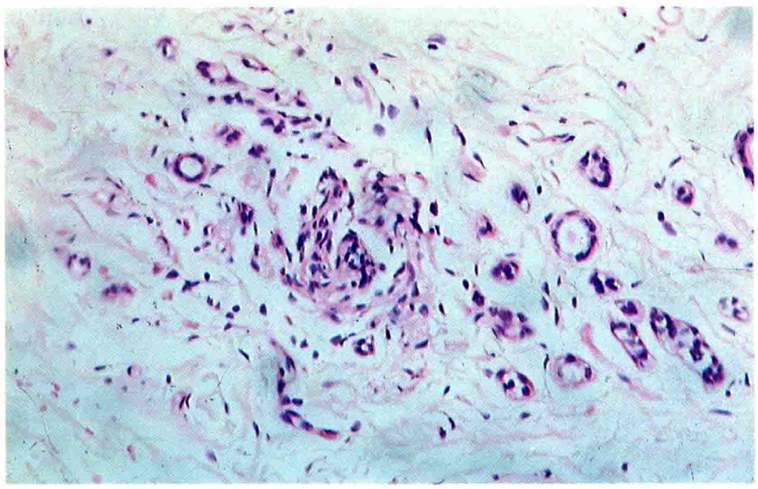


Fig. 1.25 Post-menopausal involution.

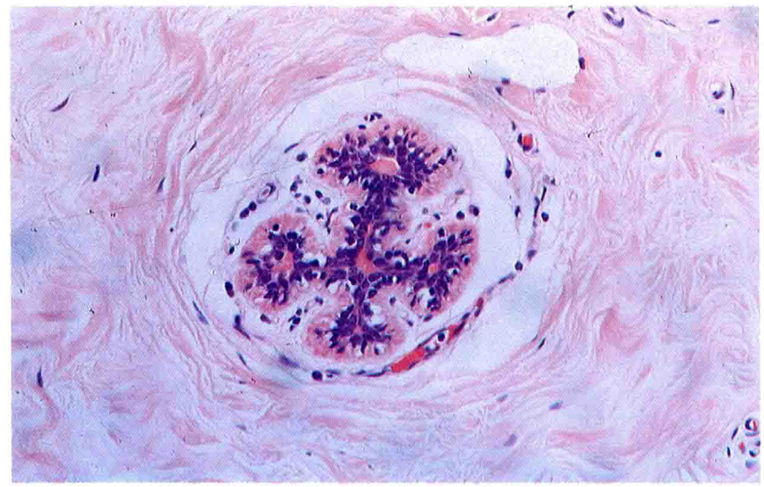


Fig. 1.26 Post-menopausal involution.

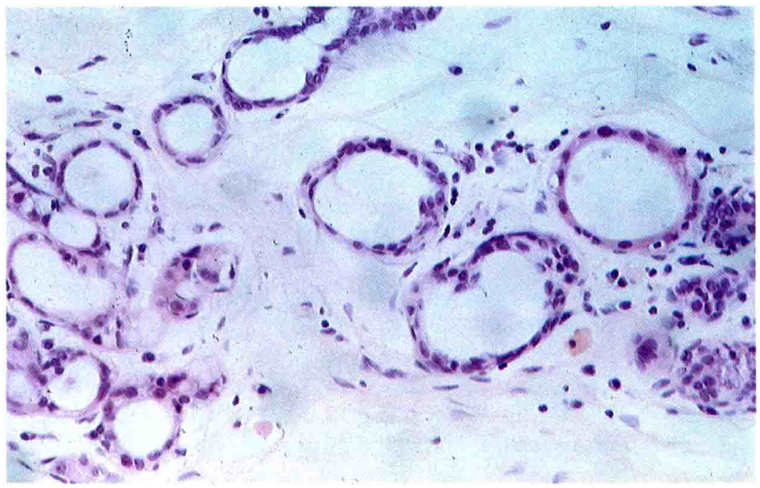


Fig. 1.27 Post-menopausal involution. Microcystic change.

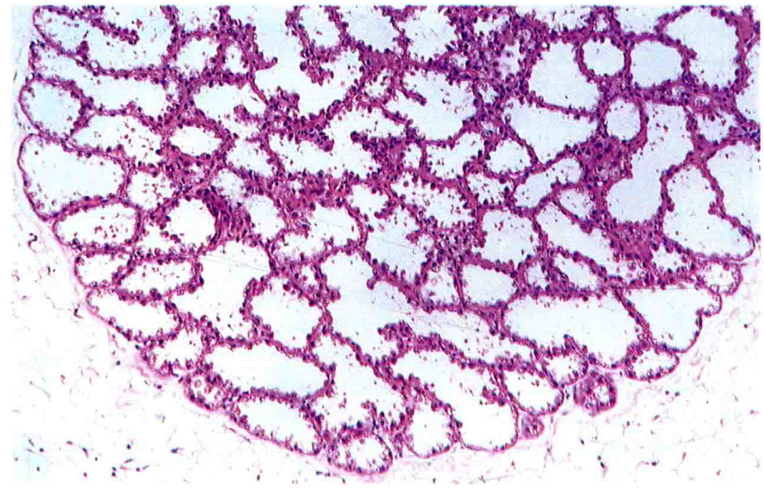


Fig. 1.28 Focal pregnancy-like change. Enlarged ductules containing secretions.

Clear cell change can involve both epithelial and myoepithelial cells[24,25] and when affecting the entire lobule may be confused with lobular carcinoma in situ.

Morphological and immunohistochemical similarities have been demonstrated between clear cell change and eccrine sweat glands and the alternative term 'eccrine metaplasia' has been suggested for this lesion.[26]

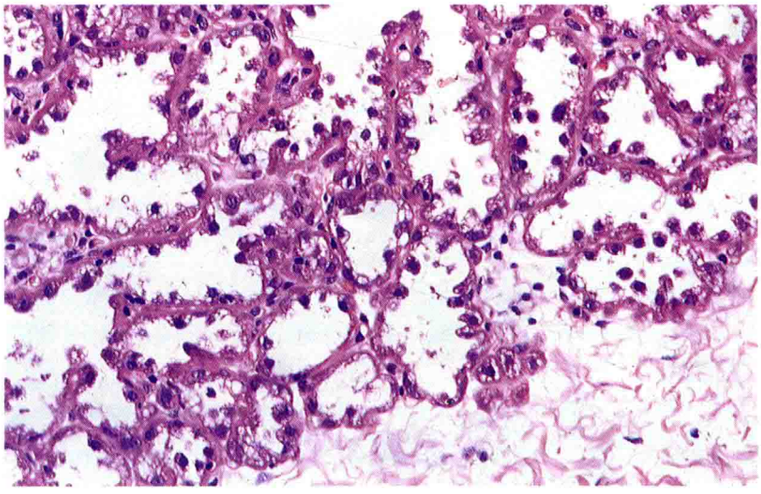


Fig. 1.29 Focal pregnancy-like change. Large, hobnailed nuclei.

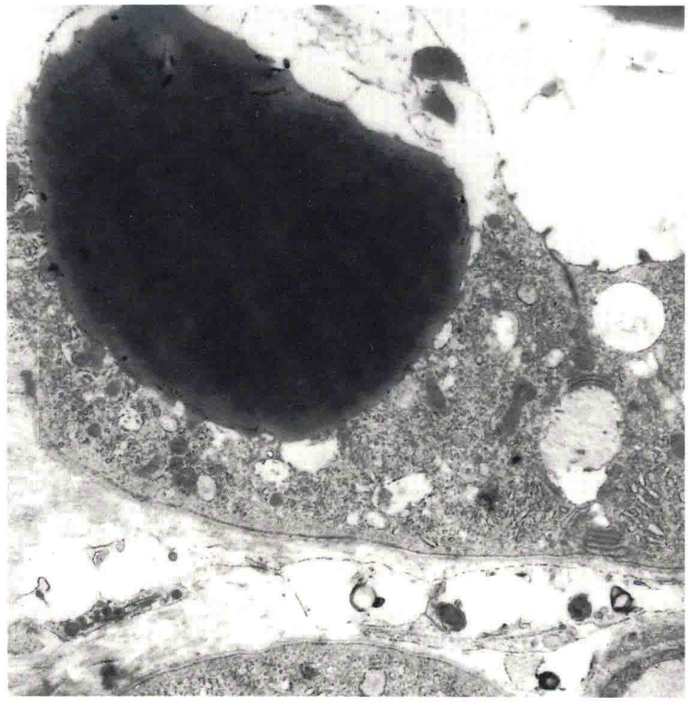


Fig. 1.30 Focal pregnancy-like change. Electron micrograph. Epithelial cells contain abundant organelles and lipid droplets. Myoepithelial cells are compressed.

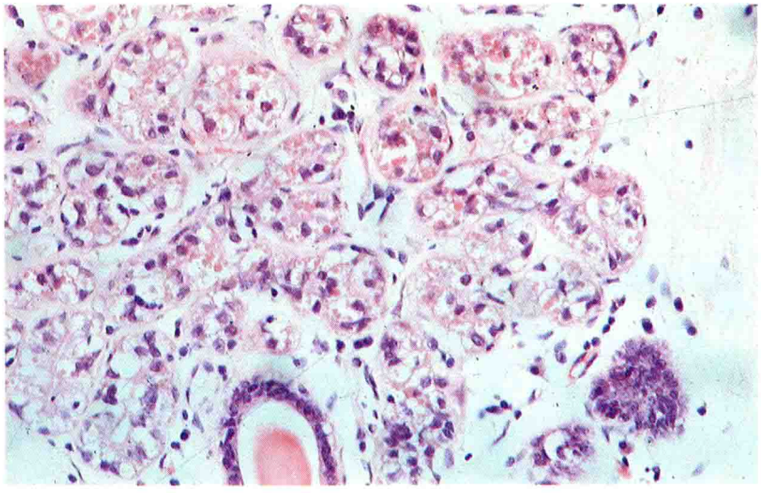


Fig. 1.31 Clear cell change.

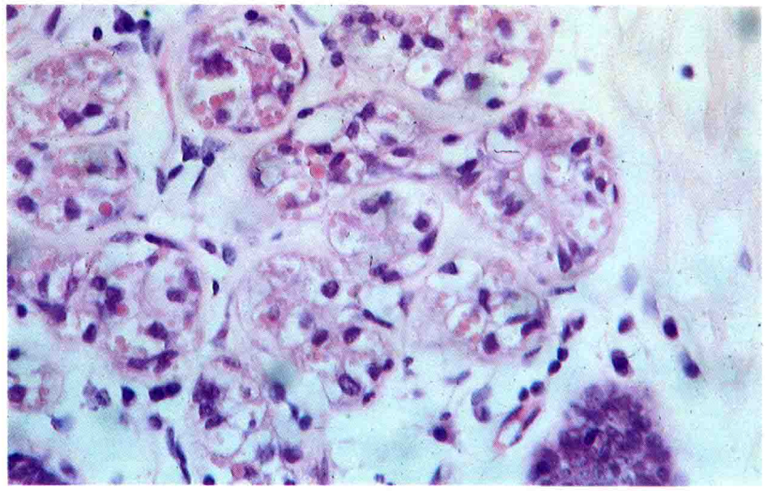


Fig. 1.32 Clear cell change. Small, round nuclei.

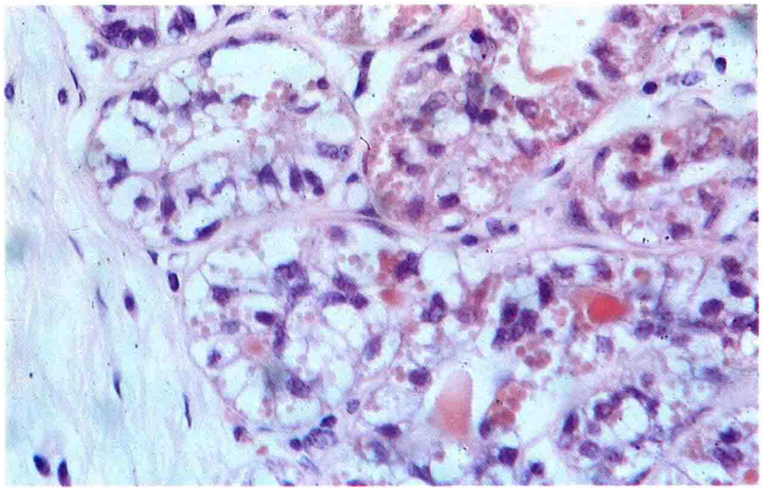


Fig. 1.33 Clear cell change. Eosinophilic granules of varying size.

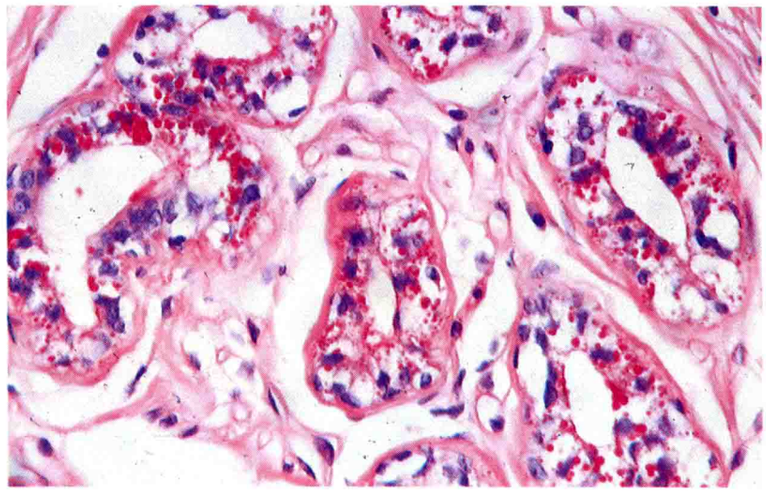


Fig. 1.34 Clear cell change. Cytoplasmic granules. PAS-diastase stain.

References

1. Hutson SW, Cowen PN, Bird CC (1985) Morphologic studies of age related changes in normal breast and their significance to evolution of mammary cancer. J Clin Pathol 38, 281–287.
2. Azzopardi JG (1979) Problems in breast pathology. Saunders, Philadelphia, pp. 11–17 and 17–21.
3. Wellings SR, Jensen HM, Marcum RG (1975) An atlas of subgross pathology of the human breast with special references to possible precancerous lesions. J Natl Cancer Inst 55, 231–273.
4. Ellis GK, Gowen AM (1990) New applications of monoclonal antibodies to the diagnosis and prognosis of breast cancer. Pathol Annu 25(2), 193–235.
5. Ahmed A (1974) The myoepithelium in cystic hyperplastic mastopathy. J Pathol 113, 209–215.
6. Ahmed A (1974) The myoepithelium in human breast carcinoma. J Pathol 113, 129–135.
7. Sappino AP, Skalli O, Jackson B, Schurch W, Gabbiani G (1988) Smooth muscle 'differentiation' in stromal cells of malignant and non-malignant breast tissue. Int J Cancer 41, 707–712.
8. Ahmed A (1990) The myofibroblast in human breast disease. Pathol Annu 25(2), 237–286.
9. Dwarakanath S, Lee AKC, Delellis RA et al (1987) S100 protein activity in breast carcinoma: a potential pitfall in diagnostic immuno-histochemistry Hum Pathol 18, 1144–1148.
10. Bussolati G (1980) Actin-rich (myoepithelial) cells in lobular carcinoma in situ of the breast. Virchows Arch B 32, 165–176.
11. Gusterson BA, Monaghan P, Mahendran R, Ellis J, O'Hare MJ (1986) Identification of myoepithelial cells in human and rat breasts by anti-common acute lymphoblastic leukaemia antigen antibody A12. J Natn Cancer Inst 77, 343–349.
12. Gottlieb C, Raju U, Greenwald KA (1990) Myoepithelial cells in the differential diagnosis of complex benign and malignant breast lesions: an immunohistochemical study. Mod Pathol 3, 135–140.
13. Stirling JW, Chandler JA (1976) The fine structure of the normal resting terminal ductual lobular unit of the female breast. Virchows Arch A 372, 205–206.
14. Ferguson DJP (1985) Intraepithelial lymphocytes and macrophages in the normal breast. Virchows Arch A 407, 369–378.
15. Ozzello L (1970) Epithelial-stromal junction of normal and dysplastic mammary gland. Cancer 25, 586–600.
16. Fanger H, Ree HJ (1974) Cyclic changes of human mammary gland epithelium in relation to the menstrual cycle – an ultrastructural study. Cancer 34, 571–585.
17. Vogel PM, Georgiade NG, Fetler BF, Vogel FS, McCarty KS Jr (1981). The correlation of histologic changes in the human breast with the menstrual cycle. Am J Pathol 104, 23–34.
18. Anderson TJ, Ferguson DJP, Raab G (1982) Cell turnover in the 'resting' human breast: influence of parity, contraceptive pill, age and laterality. Br J Cancer 46, 367–382.
19. Longacre TA, Bartow SA (1986) A correlative morphologic study of human breast and endometrium in the menstrual cycle. Am J Surg Pathol 10, 382–393.
20. Cowan DF, Herbert TA (1989) Involution of the breast in women aged 50 to 104 years: an histopathological study of 102 cases. Surg Pathol 2, 323–333.
21. Wellings SR, Jensen HM, De Vault MR (1976) Persistent and atypical lobules in the human breast may be precancerous. Experientia 32, 1463–1465.
22. Kiaer HW, Andersen JA (1977) Focal pregnancy-like changes in the breast. Acta Pathol Microbiol Scand (A) 85, 931–941.
23. Bailey AJ, Sloane JP, Trickey BS, Ormerod MG (1982) An immunocytochemical study of α-lactalbumin in human breast tissue. J Pathol 137, 13–23.
24. Tavassoli FA, Yeh IT (1987) Lactational and clear cell changes of the breast in non-lactating, non-pregnant women. Am J Clin Pathol 87, 23–29.
25. Barwick KW, Kashgarian M, Rosen PP (1982) 'Clear-cell' change within duct and lobular epithelium of the human breast. Pathol Annu 17, 319–328.
26. Vina M, Wells CA (1989) Clear cell metaplasia of the breast: a lesion showing eccrine differentiation. Histopathology 15, 85–92.

2 Inflammatory lesions

Inflammatory lesions of the breast are relatively rare. Acute inflammation is often seen in the form of an abscess occurring during lactation. Chronic inflammatory lesions of the breast include tuberculosis, sarcoidosis and lobular granulomatous mastitis. Another inflammatory lesion of the breast that may occasionally be encountered is recurrent subareolar abscess (mammary duct fistula).

Tuberculosis

Tuberculosis of the breast is a rare disease and is usually seen in women of childbearing age group. There is an increased susceptibility to tuberculous mastitis during pregnancy and lactation.[1] Cases of tuberculosis of the breast have been described in older women and occasionally in the male breast.

Mammary tuberculosis can result from the spread of infection by the haematogenous or lymphatic routes or sometimes by direct extension from the underlying pleura or rib cage.

Clinically, tuberculosis of the breast presents as one or more nodules (nodular form), which can eventually progress to chronic discharging sinuses. The less common type (sclerosing form) occurs in older patients and involves the entire breast, with fibrosis as the dominant feature. The sclerosing form of tuberculosis may be extremely difficult to differentiate from breast carcinoma.

Histological examination shows the typical caseating granulomata

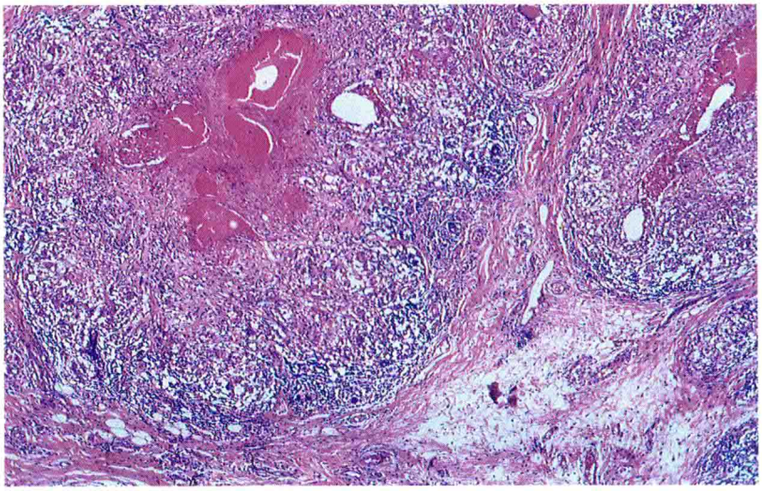


Fig. 2.1 Tuberculosis. Caseating granulomata in lobule.

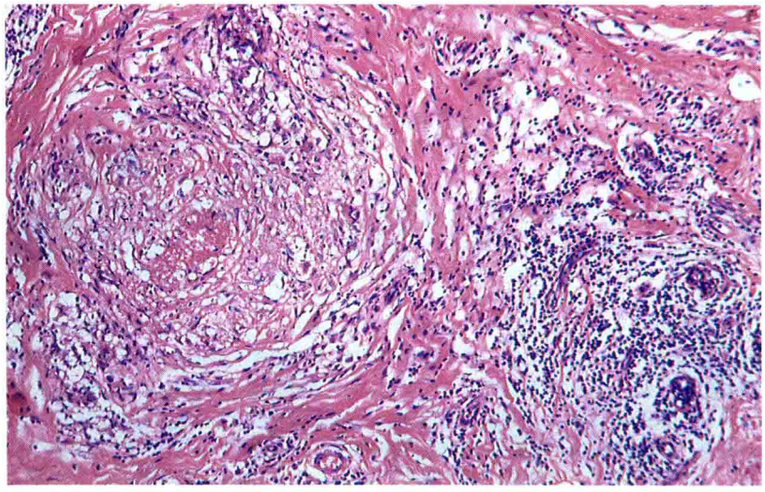


Fig. 2.2 Tuberculosis. Granulomata in interlobular stroma.

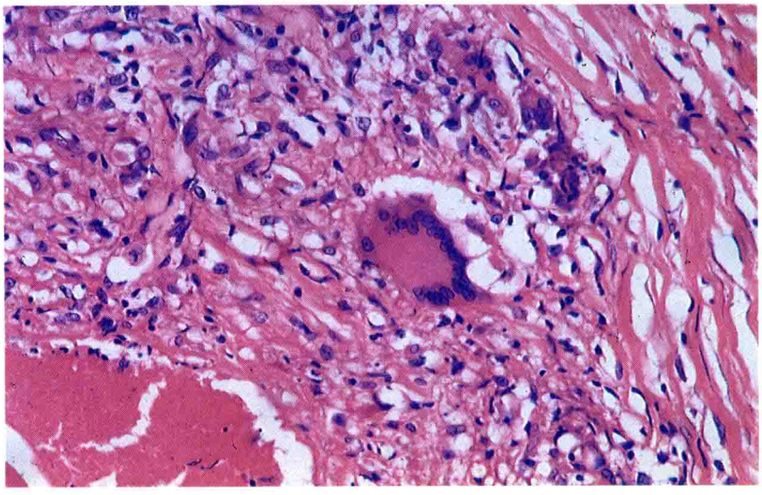


Fig. 2.3 Tuberculosis. Caseating granuloma with Langhan's giant cell.

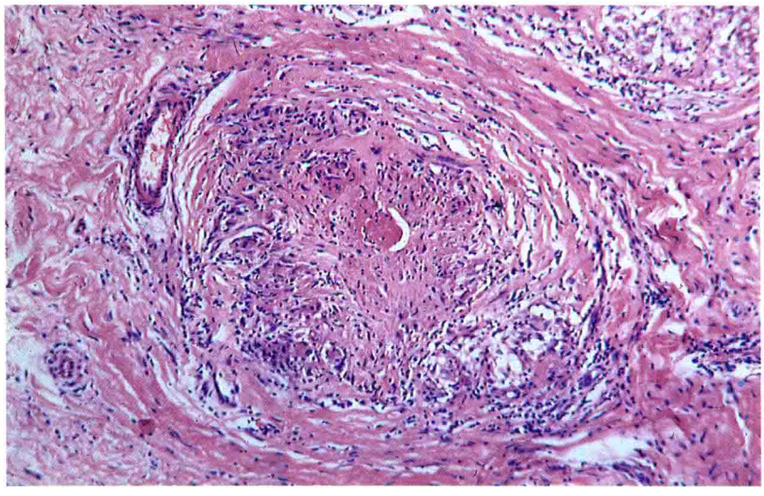


Fig. 2.4 Tuberculosis. Caseating granuloma with partial central fibrosis.

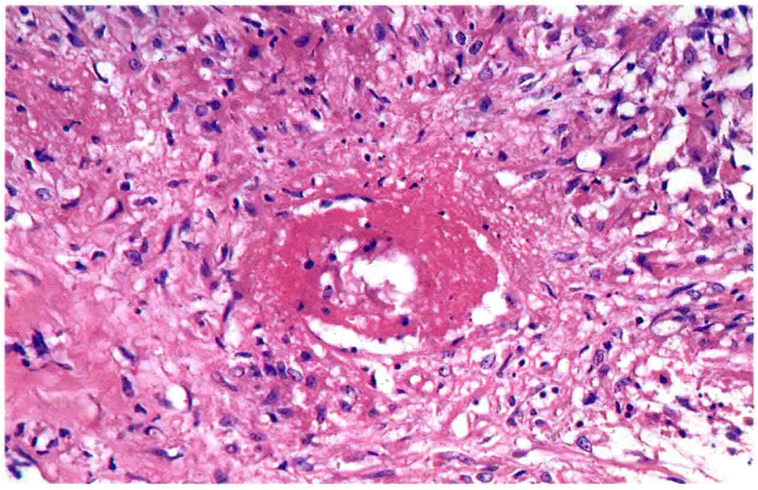


Fig. 2.5 Tuberculosis. Granuloma with fibrosis.

located within the lobules as well as in the interlobular stroma, and composed of epithelioid cells, Langhan's giant cells and peripheral lymphocytes (Figs 2.1, 2.2, 2.3). There is progressive fibrosis of the granulomata (Figs 2.4, 2.5). This fibrosis is most marked in the sclerosing form of tuberculosis and results in extensive replacement by fibrous tissue.

Tuberculosis may spread along the ductal system and the involved ducts can exhibit epithelial proliferation, necrosis and periductal fibrosis.

References

1. Ikard RW, Perkins D (1977) Mammary tuberculosis a rare modern disease. Sthn Med J 70, 208–212.

Sarcoidosis

Sarcoidosis, a systemic granulomatous disorder, can rarely involve the breast.[1,2,3] The clinical presentation is usually as a painless firm mass[1,2] which may suggest a malignancy.[2]

The histological appearances reveal numerous, non-caseating epithelioid granulomata with multi-nucleated giant cells, scattered throughout the breast parenchyma and the interlobular stroma (Figs 2.6, 2.7). Sarcoid granulomata lack acidophilic granular necrosis and the giant cells contain abundant 'glassy' cytoplasm (Fig. 2.8). Occasionally, granulomata may be located in the lobules[3] as described in lobular granulomatous mastitis (Fig. 2.9). In sarcoidosis, however, there is no lobular inflammation or micro-abscess formation, both features considered to be hallmarks of lobular granulomatous mastitis.[4]

The diagnosis of sarcoidosis requires the exclusion of tuberculosis and of fungal infection. Kveim test, chest radiography and the measurement of serum angiotension converting enzyme (ACE) and serum lysozyme have been used to confirm the diagnosis of mammary sarcoidosis.[3]

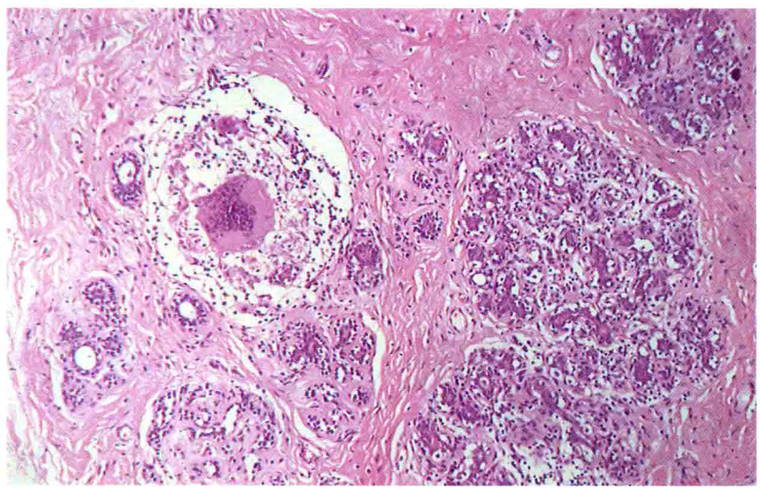


Fig. 2.6 Sarcoidosis. Sarcoid granuloma in interlobular stroma.

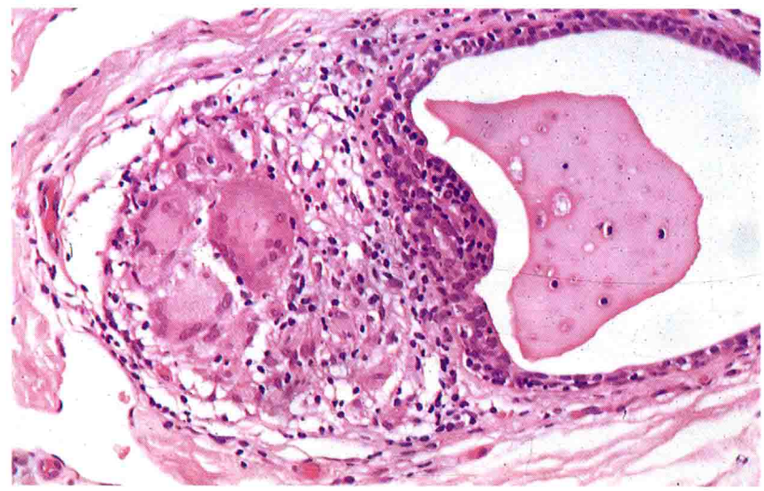


Fig. 2.7 Sarcoidosis. Typical sarcoid granuloma near an interlobular duct.

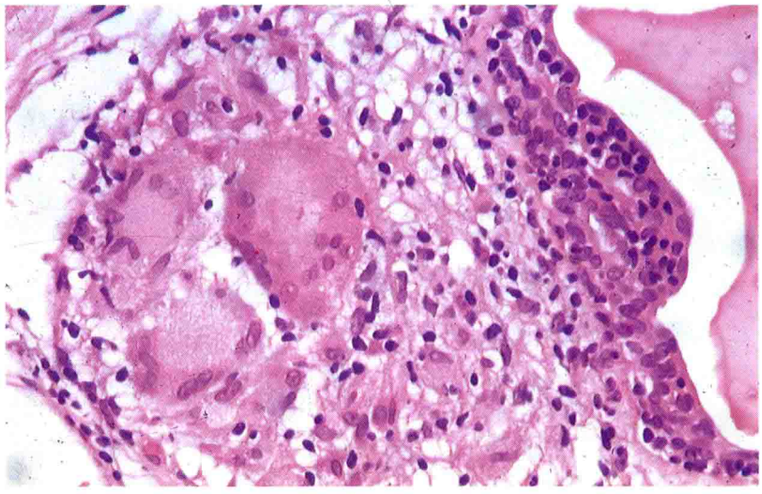


Fig. 2.8 Sarcoidosis. Sarcoid granuloma.

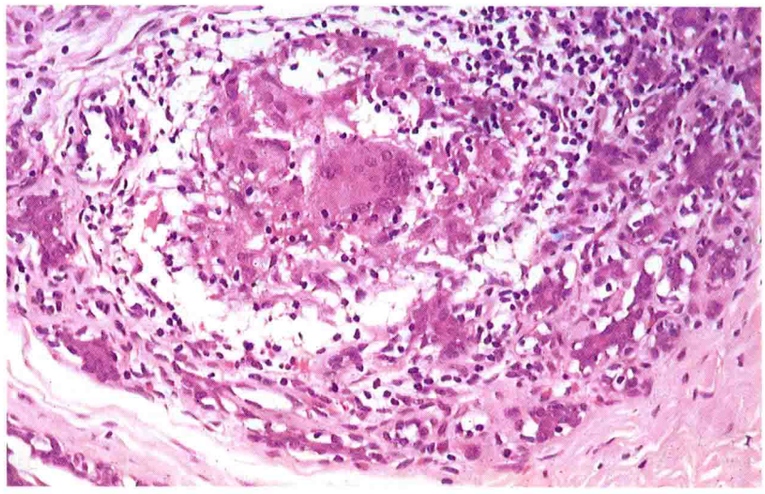


Fig. 2.9 Sarcoidosis. Sarcoid granuloma in lobule.

References

1. Ross MJ and Merino JM (1985) Sarcoidosis of the breast. Hum Pathol 16, 185–187.
2. Fitzgibbons PL, Smiley DF and Kern WH (1985) Sarcoidosis presenting initially as breast mass: report of two cases. Hum Pathol 16, 851–852.
3. Banik S, Bishop PW, Ormerod LP and O'Brien TEB (1986) Sarcoidosis of the breast. J Clin Pathol 39, 446–448.
4. Kesseler EI and Katzav JA (1990) Lobular granulomatous mastitis. Surg Pathol 3, 115–120.

Lobular granulomatous mastitis

Lobular granulomatous mastitis is a distinct form of mastitis which affects women of childbearing age.[1,2] The lesion is often related to recent pregnancy and lactation and thus the term 'post-partum lobular granulomatous mastitis' has been suggested.[3] Clinically the lesion often presents as a firm, tender mass, which can be mistaken for carcinoma.[1,2,4]

The main histological feature is the predominantly lobular inflammatory process. Numerous, discrete, non-caseating granulomata composed of epithelioid cells and multi-nucleated giant cells are centred on lobular units (Figs 2.10, 2.11). Lymphocytes and polymorphs may also be present (Fig. 2.12). The inflammatory process can sometimes be sufficiently acute and intense with resultant microabscess formation (Fig. 2.13). There may be damage and atrophy of the adjacent ductules (Fig. 2.14) and ducts (Fig. 2.15).[4,5]

The exact cause of lobular granulomatous mastitis is not known. A possible immunological cause is suggested by the resemblance of the lesion to granulomatous orchitis and to thyroiditis.[2]

The firm diagnosis of lobular granulomatous mastitis requires the exclusion of tuberculosis and fungal infection. Sarcoidosis of the breast can present a similar histological picture,[6] and has also to be excluded by Kveim testing, chest radiography and the measurement of serum angiotension converting enzyme and lysozyme, both of which are raised in sarcoidosis.[6]

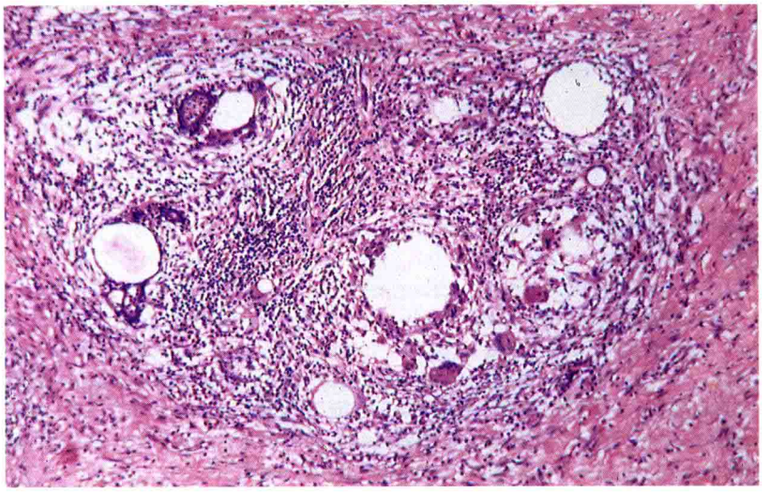


Fig. 2.10 Lobular granulomatous mastitis. Non-caseating granulomata in lobules.

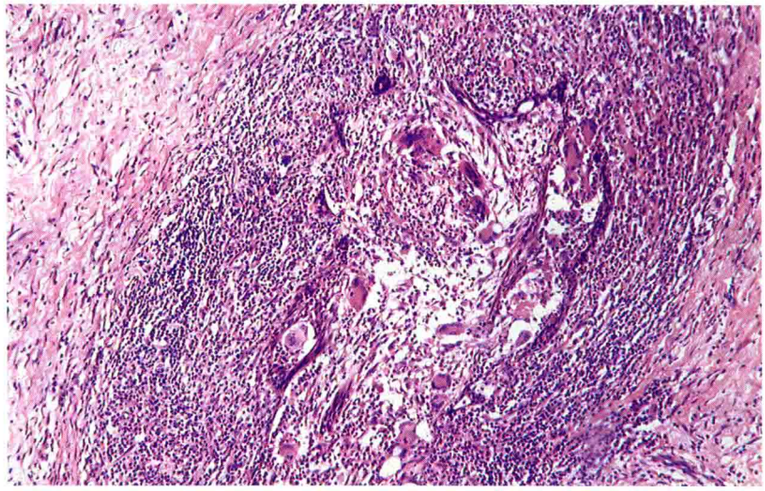


Fig. 2.11 Lobular granulomatous mastitis. Another area of lobular granulomatous mastitis.