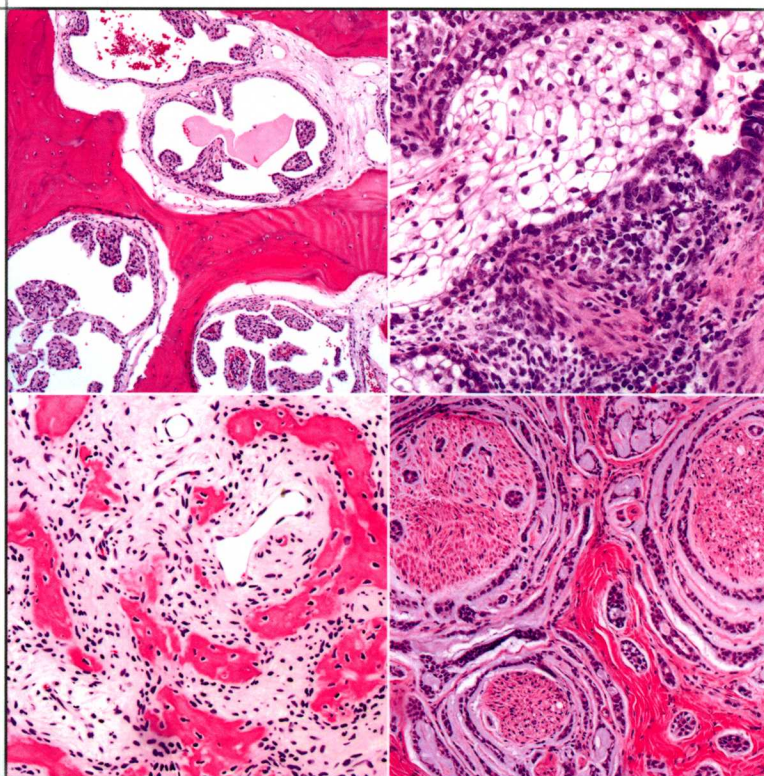


Activate your eBook

**DIFFERENTIAL DIAGNOSES IN
SURGICAL PATHOLOGY**

Head and Neck

William H. Westra
Justin A. Bishop



SERIES EDITOR
Jonathan I. Epstein



Wolters Kluwer

Differential Diagnoses in Surgical Pathology: **Head and Neck**

William H. Westra, MD

Professor of Pathology, Oncology, and Otolaryngology/Head and Neck Surgery
Associate Director, Surgical Pathology
Director, The Head and Neck Pathology Consultation Service
The Johns Hopkins Medical Institutions
Baltimore, Maryland

Justin A. Bishop, MD

Associate Professor of Pathology, Oncology, and Otolaryngology/Head and Neck Surgery
The Johns Hopkins Medical Institutions
Baltimore, Maryland

SERIES EDITOR

Jonathan I. Epstein, MD

Professor of Pathology, Urology and Oncology
The Reinhard Professor of Urological Pathology
Director of Surgical Pathology
The Johns Hopkins Medical Institutions
Baltimore, Maryland



Wolters Kluwer

Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Acquisitions Editor: Ryan Shaw
Product Development Editor: Kate Heaney
Production Project Manager: David Saltzberg
Manufacturing Coordinator: Beth Welsh
Marketing Manager: Dan Dressler
Design Coordinator: Teresa Mallon
Production Service: SPi Global

Copyright © 2017 Wolters Kluwer

All rights reserved. This book is protected by copyright. No part of this book may be reproduced or transmitted in any form or by any means, including as photocopies or scanned-in or other electronic copies, or utilized by any information storage and retrieval system without written permission from the copyright owner, except for brief quotations embodied in critical articles and reviews. Materials appearing in this book prepared by individuals as part of their official duties as U.S. government employees are not covered by the above-mentioned copyright. To request permission, please contact Wolters Kluwer at Two Commerce Square, 2001 Market Street, Philadelphia, PA 19103, via email at permissions@lww.com, or via our website at lww.com (products and services).

9 8 7 6 5 4 3 2 1

Printed in China

Library of Congress Cataloging-in-Publication Data

Names: Westra, William H., author. | Bishop, Justin A., author.

Title: Head and neck / William H. Westra, Justin A. Bishop.

Other titles: Differential diagnoses in surgical pathology series.

Description: Philadelphia : Wolters Kluwer, [2017] | Series: Differential diagnoses in surgical pathology | Includes index.

Identifiers: LCCN 2016028196 | ISBN 9781496309792

Subjects: | MESH: Head and Neck Neoplasms—diagnosis | Diagnosis, Differential | Pathology, Surgical—methods

Classification: LCC RC280.H4 | NLM WE 707 | DDC 616.99/491—dc23 LC record available at <https://lcn.loc.gov/2016028196>

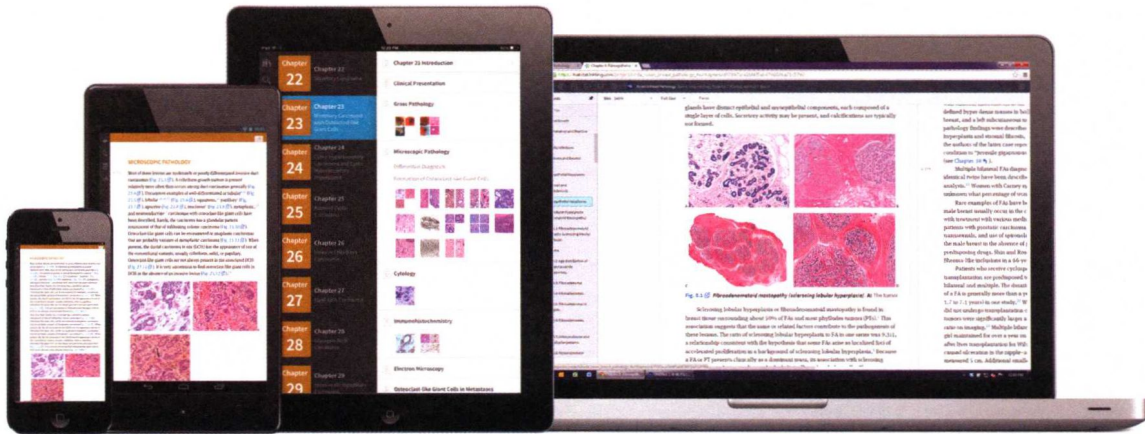
This work is provided “as is,” and the publisher disclaims any and all warranties, express or implied, including any warranties as to accuracy, comprehensiveness, or currency of the content of this work.

This work is no substitute for individual patient assessment based upon healthcare professionals’ examination of each patient and consideration of, among other things, age, weight, gender, current or prior medical conditions, medication history, laboratory data and other factors unique to the patient. The publisher does not provide medical advice or guidance and this work is merely a reference tool. Healthcare professionals, and not the publisher, are solely responsible for the use of this work including all medical judgments and for any resulting diagnosis and treatments.

Given continuous, rapid advances in medical science and health information, independent professional verification of medical diagnoses, indications, appropriate pharmaceutical selections and dosages, and treatment options should be made and healthcare professionals should consult a variety of sources. When prescribing medication, healthcare professionals are advised to consult the product information sheet (the manufacturer’s package insert) accompanying each drug to verify, among other things, conditions of use, warnings and side effects and identify any changes in dosage schedule or contraindications, particularly if the medication to be administered is new, infrequently used or has a narrow therapeutic range. To the maximum extent permitted under applicable law, no responsibility is assumed by the publisher for any injury and/or damage to persons or property, as a matter of products liability, negligence law or otherwise, or from any reference to or use by any person of this work.

LWW.com

Get Your Free Enhanced eBook.



Your book purchase includes a complimentary download of the enhanced eBook for iOS, Android, PC & Mac. This eBook features:

- Complete content with enhanced navigation that can be accessed anywhere, even without a data connection
- Find information instantly using powerful search tools and smart navigation cross-links
- Highlighting tool for easier reference of key content throughout the text
- Ability to take and share notes with friends and colleagues
- Quick reference tabbing to save your favorite content for future use

Download Your eBook Now:

- 1 Go to <http://solution.lww.com/access>
- 2 Enter the Access Code to the right, and click Redeem Code.
- 3 Enter your information, click "Submit," and follow the on-screen instructions to start reading your eBook.

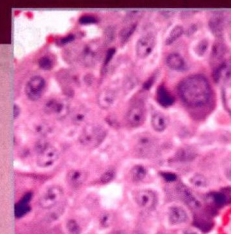
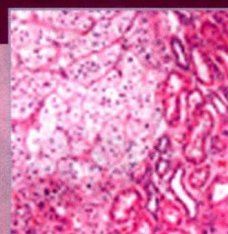
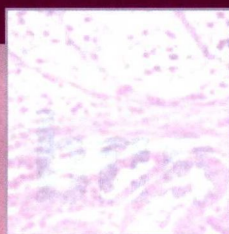
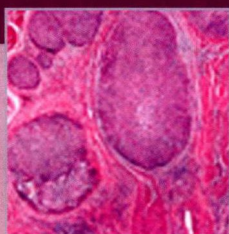
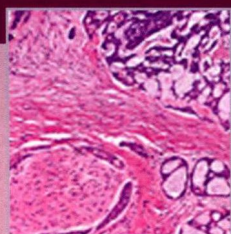
Scratch Off Below



Scratch off the sticker with care.

NOTE: Book cannot be returned once the panel is scratched off.

Continue learning with the full series of Differential Diagnoses in Surgical Pathology books.



Gastrointestinal System

Elizabeth A. Montgomery

Whitney M. Green

ISBN: 978-1-4511-9189-9

February 2015

Genitourinary System

Jonathan I. Epstein

George J. Netto

ISBN: 978-1-4511-8958-2

March 2014

Pulmonary Pathology

Rosane Duarte Achcar

Carlyne Cool

Steve Groshong

ISBN: 978-1-4511-9527-9

October 2016

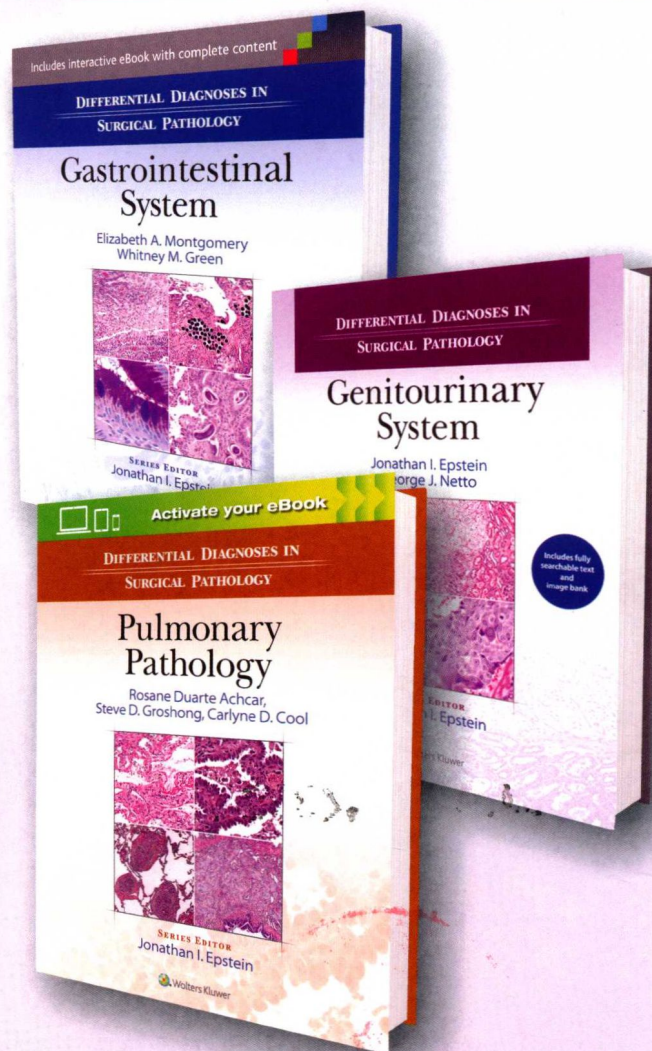
The Breast

Jean F. Simpson

Melinda E. Sanders

ISBN: 978-1-4963-0065-2

December 2016



Visit your local Wolters Kluwer website
or bookseller to learn more



DEDICATION



This book is for you, mom, and dad, in appreciation of your love and inspiration.
William Westra, MD

Dedicated to Ashley, Riley, and Avery.
Justin Bishop, MD

PREFACE

Most carcinomas of the head and neck are squamous cell carcinomas. Accordingly, the term “head and neck cancer” brings to the mind of the untested pathologist a uniform group of tumors that are easily recognized by an unwavering squamous morphology. But this naive approach to the practice of head and neck pathology is dissipated by any sustained exposure to the bewildering world of head and neck tumors and tumor-like conditions. Squamous cell carcinomas often deviate from their expected conventional appearance in ways that elude detection and even mimic benign processes. Conversely, benign process sometimes imitate squamous cell carcinomas. As for the nonsquamous tumors that arise from assorted tissues of the head and neck (e.g., craniofacial bones, salivary glands, soft tissues, thyroid), their sheer diversity is likely to overwhelm even the most capable pathologist. When dealing with a salivary gland epithelial neoplasm, for

example, the general pathologist is expected to single out a specific diagnosis from over 30 common and esoteric salivary gland tumors that, although clinically and biologically distinct, may look very similar under the microscope. To the experienced and tested pathologist, the term “head and neck cancer” brings to the mind the most challenging and treacherous group of tumors in all of surgical pathology.

This atlas was written to help the general pathologist navigate the troubled waters of head and neck pathology. It takes on some of the most commonly encountered differential diagnoses with the aims of pointing out diagnostic pitfalls and providing pathologic clues to guide diagnostic decisions. We hope that you find it helpful.

*William H. Westra, MD
Justin A. Bishop, MD*

CONTENTS

Preface vii

Chapter 1	SALIVARY GLANDS	1
Chapter 2	ODONTOGENIC/BONE	94
Chapter 3	EAR	123
Chapter 4	NECK/SOFT TISSUE	142
Chapter 5	ORAL CAVITY	183
Chapter 6	OROPHARYNX	224
Chapter 7	LARYNX	238
Chapter 8	SINONASAL TRACT/NASOPHARYNX	277
Chapter 9	THYROID AND PARATHYROID GLANDS	364

Index 435

Salivary Glands

- 1.1** Acinic cell carcinoma vs. Mammary analogue secretory carcinoma (MASC)
- 1.2** Adenoid cystic carcinoma vs. Epithelial–myoepithelial carcinoma
- 1.3** Adenoid cystic carcinoma vs. Basaloid variant of squamous cell carcinoma
- 1.4** Adenosquamous carcinoma vs. Mucoepidermoid carcinoma, high grade
- 1.5** Basal cell adenocarcinoma vs. Adenoid cystic carcinoma
- 1.6** Basal cell adenoma vs. Basal cell adenocarcinoma
- 1.7** Basal cell adenoma vs. Canalicular adenoma
- 1.8** Basal cell adenoma vs. Pleomorphic adenoma
- 1.9** Carcinoma ex-pleomorphic adenoma vs. Infarcted pleomorphic adenoma
- 1.10** Carcinoma ex-pleomorphic adenoma vs. Recurrent pleomorphic adenoma
- 1.11** Clear cell oncocytoma/nodular oncocytic hyperplasia vs. Metastatic renal cell carcinoma
- 1.12** Ectomesenchymal chondromyxoid tumor vs. Myoepithelioma
- 1.13** Epithelial–myoepithelial carcinoma vs. Pleomorphic adenoma
- 1.14** Hyalinizing clear cell carcinoma vs. Mucoepidermoid carcinoma, low grade
- 1.15** Hyalinizing clear cell carcinoma vs. Myoepithelial carcinoma
- 1.16** IgG4-associated chronic sclerosing sialadenitis (Kuttner tumor) vs. Lymphoepithelial sialadenitis (benign lymphoepithelial lesion)
- 1.17** Intranodal salivary gland tissue vs. Metastatic carcinoma
- 1.18** Lymphadenoma vs. Lymphoepithelial carcinoma
- 1.19** Mammary analogue secretory carcinoma (MASC) vs. Low-grade cribriform cystadenocarcinoma (low-grade salivary duct carcinoma)
- 1.20** Mucoepidermoid carcinoma vs. Necrotizing sialometaplasia
- 1.21** Membranous basal cell adenoma vs. Adenoid cystic carcinoma
- 1.22** Metaplastic warthin tumor vs. Mucoepidermoid carcinoma, oncocytic variant
- 1.23** Pleomorphic adenoma vs. Myoepithelioma
- 1.24** Oncocytoma vs. Nodular oncocytic hyperplasia
- 1.25** Oncocytoma vs. Mucoepidermoid carcinoma, oncocytic variant
- 1.26** Polymorphous low-grade adenocarcinoma vs. Adenoid cystic carcinoma
- 1.27** Polymorphous low-grade adenocarcinoma vs. Cribriform adenocarcinoma of the tongue and minor salivary glands
- 1.28** Polymorphous low-grade adenocarcinoma vs. Pleomorphic adenoma
- 1.29** Primary squamous cell carcinoma vs. Metastatic squamous cell carcinoma
- 1.30** Salivary duct carcinoma vs. Mucoepidermoid carcinoma, high grade
- 1.31** Carcinoma with tumor-associated lymphoid proliferation vs. Metastatic carcinoma

	Acinic Cell Carcinoma	Mammary Analogue Secretory Carcinoma (MASC)
<i>Age</i>	Wide range, with slight peak in seventh decade. Slight female predominance	Usually adults, mean 47 years
<i>Location</i>	Parotid gland (90%), submandibular gland (5%), and minor salivary glands (5%)	Parotid (70%), oral cavity (20%), and submandibular gland (5%)
<i>Symptoms</i>	Painless, slow-growing mass	Painless, slow-growing mass
<i>Signs</i>	Generally well-circumscribed mass, sometimes with cystic features	Generally well-circumscribed mass, sometimes with cystic features
<i>Etiology</i>	Unknown	Unknown
<i>Histology</i>	<ol style="list-style-type: none"> 1. Diagnostic feature is the serous acinar cell, a medium-to-large polygonal cell with blue–purple cytoplasmic granules (zymogen granules) (Fig. 1.1.1). The number of serous acinar cells is variable 2. Numerous additional cell types may be seen, including intercalated ductlike cells, vacuolated cells, clear cells, and nonspecific glandular cells (Fig. 1.1.2) 3. Numerous growth patterns can be seen, including solid, microcystic, papillary–cystic, and follicular (Figs. 1.1.3 and 1.1.4) 4. Secretory material within microcystic spaces may be seen occasionally 5. Often well circumscribed but may be infiltrative 6. Perineural invasion, necrosis, and elevated mitotic rates are not typical but are encountered in cases showing “high-grade transformation” 	<ol style="list-style-type: none"> 1. No serous acinar cells 2. Growth patterns overlap with acinic cell carcinoma. Microcystic is most common, but follicular, papillary–cystic, and solid patterns can be seen as well (Figs. 1.1.6, 1.1.7, and 1.1.8) 3. Cells have apocrine features including abundant eosinophilic granular cytoplasm and a large nucleus with a prominent nucleolus (Fig. 1.1.9) 4. Eosinophilic secretions are seen within the glandular spaces (Figs. 1.1.6, 1.1.7, and 1.1.9) 5. May be infiltrative or well circumscribed 6. Perineural invasion, necrosis, and elevated mitotic rates are not seen except in rare cases of high-grade transformation
<i>Special studies</i>	<ul style="list-style-type: none"> • Positive for DOG-1 and usually negative for S100, mammaglobin, and GATA3 (Fig. 1.1.5) • Negative for ETV6 translocation 	<ul style="list-style-type: none"> • Positive for S100, mammaglobin, and GATA3 (Figs. 1.1.10 and 1.1.11). Staining for DOG-1 is usually negative • Positive for ETV6 translocation (usually with partner gene NTRK3) similar to secretory carcinoma of the breast (Fig. 1.1.12)
<i>Treatment</i>	Surgery only, except in those rare cases showing high-grade transformation	Therapy is not well standardized. Most have been treated with surgery, with a subset receiving radiotherapy as well. For rare aggressive cases, targeted therapies (tyrosine kinase inhibitors) may be of value
<i>Prognosis</i>	Good, with 10-year survival >90%	Appears to be similar to acinic cell carcinoma

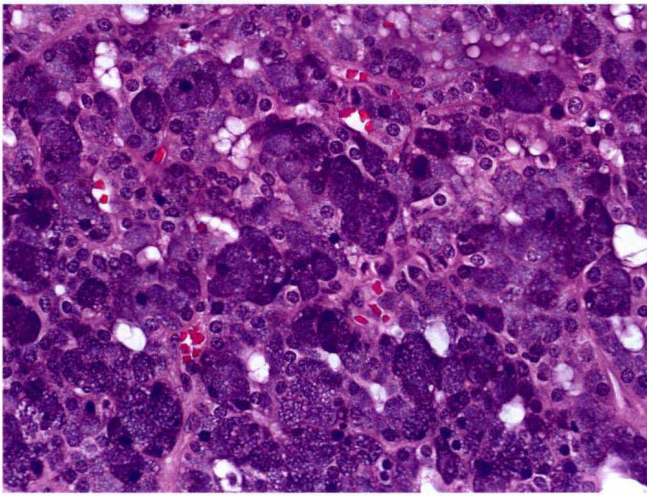


Figure 1.1.1 Acinic cell carcinoma with a solid proliferation of serous acinar cells that contain numerous blue–purple granules in their cytoplasm.

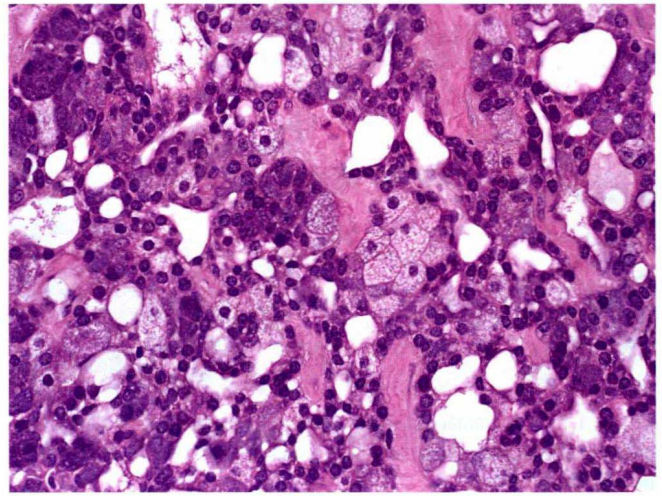


Figure 1.1.2 Acinic cell carcinomas may show a combination of cell types including serous acinar cells, vacuolated cells, clear cells, and nonspecific glandular cells.

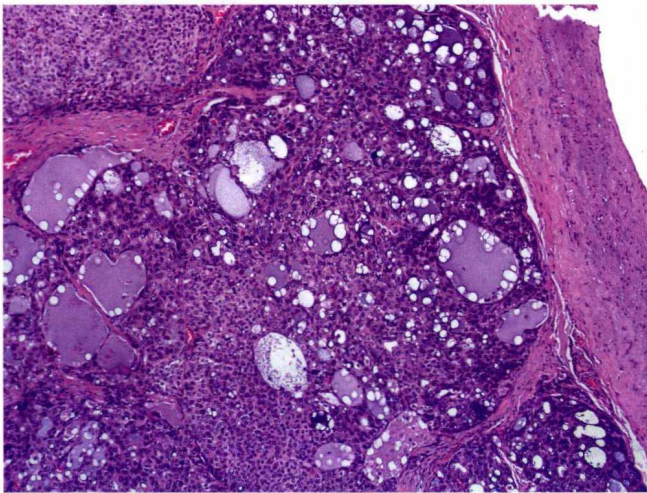


Figure 1.1.3 Acinic cell carcinoma demonstrating a mixture of solid, microcystic, and follicular growth patterns.

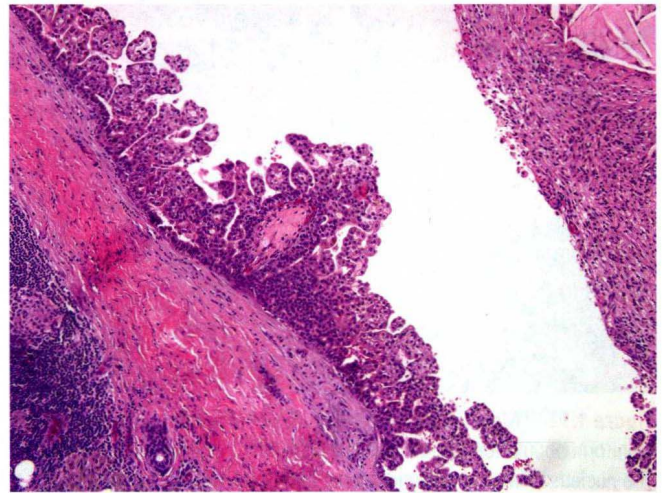


Figure 1.1.4 Acinic cell demonstrating a papillary–cystic growth pattern.

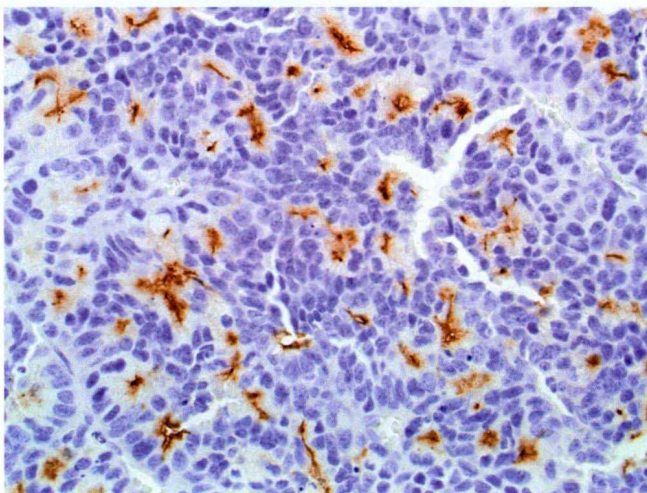


Figure 1.1.5 Acinic cell is consistently positive for DOG-1 in a membranous, canalicular-type distribution.

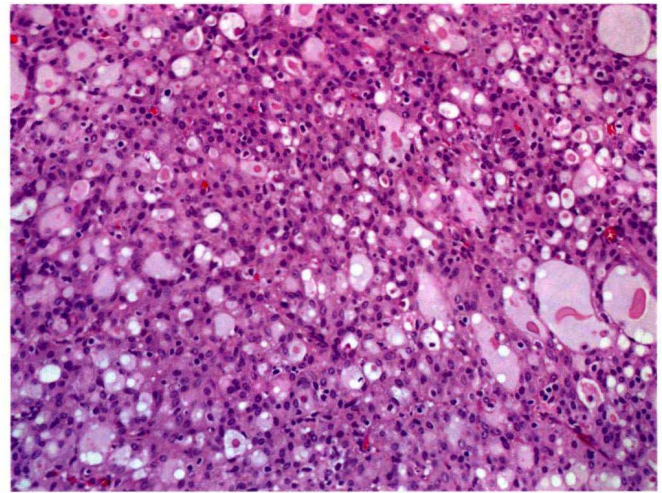


Figure 1.1.6 Mammary analogue secretory carcinoma exhibiting microcystic growth. This is the most common growth pattern of mammary analogue secretory carcinoma.

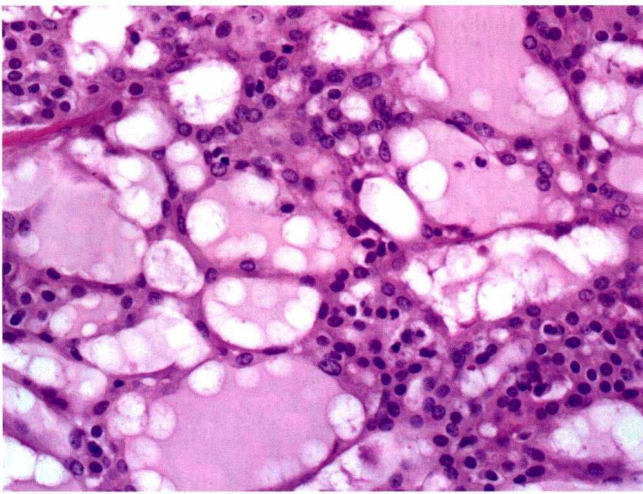


Figure 1.1.7 Mammary analogue secretory carcinoma with a follicular growth pattern. With the colloid-like eosinophilic secretions, the tumor mimics thyroid tissue.

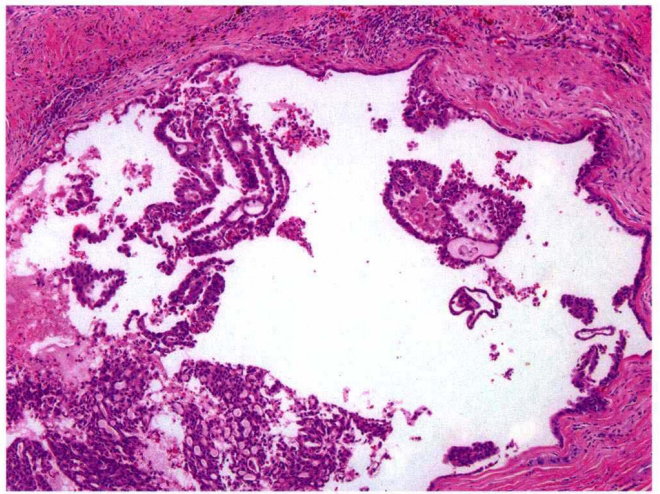


Figure 1.1.8 Mammary analogue secretory carcinoma with papillary cystic growth.

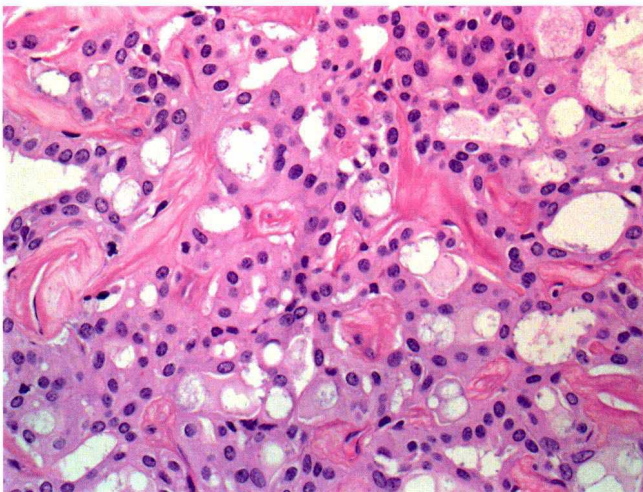


Figure 1.1.9 Mammary analogue secretory carcinoma is comprised of a uniform population of cells with an abundant, eosinophilic cytoplasm. The nucleus is large and round to oval and has a visible nucleolus.

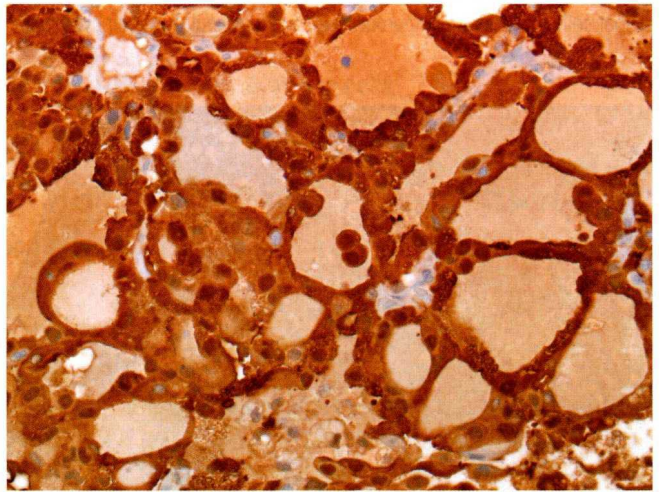


Figure 1.1.10 Mammary analogue secretory carcinoma is consistently positive for S100.

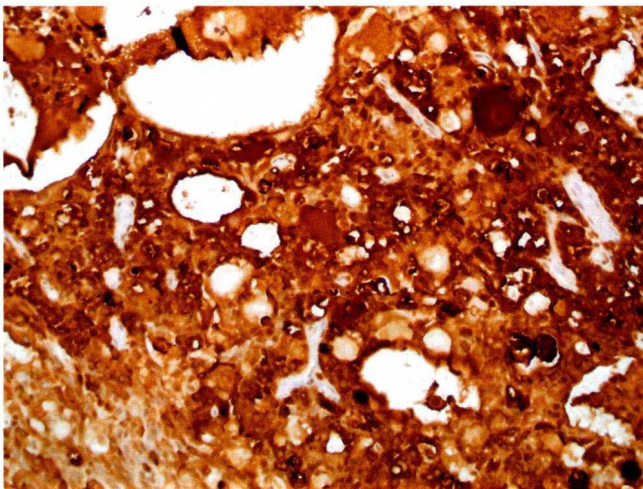


Figure 1.1.11 Mammary analogue secretory carcinoma is consistently positive for mammaglobin.

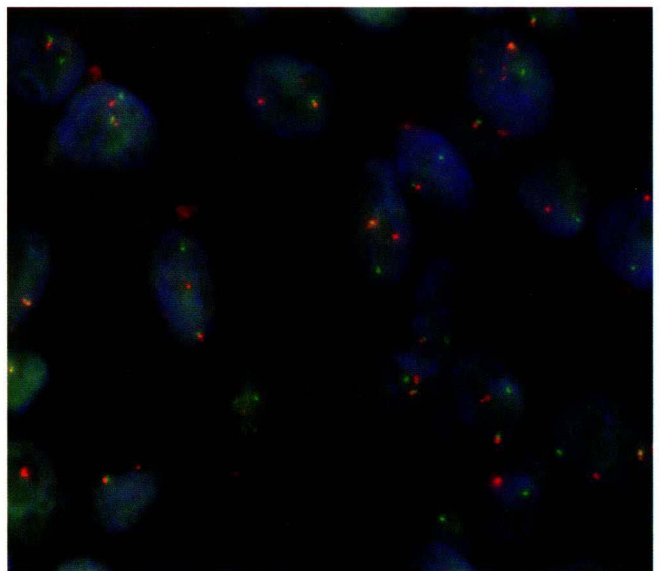


Figure 1.1.12 Mammary analogue secretory carcinoma harbors translocations for ETV6. In this break apart FISH assay, one copy of the ETV6 gene is intact (*red* and *green* signals together) and one is rearranged (*red* and *green* signals apart).

	Adenoid Cystic Carcinoma	Epithelial–Myoepithelial Carcinoma
<i>Age</i>	Typically adults, peak incidence in sixth decade	Typically adults, peak incidence in sixth to seventh decades
<i>Location</i>	Parotid gland is most common (40%–50%) but may arise in any major or minor salivary gland	Usually parotid gland (up to 80%) and uncommonly submandibular gland or minor salivary glands
<i>Symptoms</i>	Slow-growing swelling sometimes pain or paralysis due to perineural invasion. Other site-specific symptoms in minor salivary locations (e.g., nasal obstruction or epistaxis for sinonasal tumors)	Slowly growing, painless mass
<i>Signs</i>	Palpable nodules that may become fixed to surrounding tissues	Typically circumscribed, small mass. May be grossly cystic
<i>Etiology</i>	Unknown	Unknown
<i>Histology</i>	<ol style="list-style-type: none"> 1. Variable mixture of tubules, cribriform structures, and solid nests (<i>Fig. 1.2.1</i>) 2. Cribriform pattern is most common, with cylindromatous microcystic spaces (false ducts) filled with basophilic mucoid or hyaline basement membrane-like material (<i>Fig. 1.2.2</i>) 3. Two cell types: ductal and myoepithelial. The myoepithelial cells predominate. They are monotonous and basaloid and have hyperchromatic, angulated nuclei with indistinct nucleoli and small amounts of clear to eosinophilic cytoplasm. The ducts are often subtle and are comprised of cuboidal cells with eosinophilic cytoplasm (<i>Fig. 1.2.2</i>) 4. Sometimes, the proportion of ducts to myoepithelial cells may be higher. These areas may closely resemble epithelial–myoepithelial carcinoma (<i>Fig. 1.2.3</i>) 5. Highly infiltrative, with perineural invasion very commonly identified 6. Tumor necrosis and elevated mitotic rates are uncommon but are more common in tumors with a predominant solid pattern 	<ol style="list-style-type: none"> 1. Tightly coupled biphasic tumor cell population with ducts surrounded by a row of myoepithelial cells, typically with clear cytoplasm (<i>Fig. 1.2.4</i>) 2. Cribriform growth is absent or, at most, very focal 3. Compared to adenoid cystic carcinoma, tumor cells exhibit nuclei with more open chromatin and more prominent nucleoli (<i>Fig. 1.2.5</i>) 4. Ductal cells are usually evident, but in some cases, the myoepithelial cell component can overgrow the ducts in a sheetlike manner (<i>Fig. 1.2.6</i>) 5. Often relatively circumscribed, though infiltrative growth is present at least focally in the form of nodular growth or invasion of benign tissues (<i>Fig. 1.2.7</i>). Perineural invasion may be seen but less frequently than in adenoid cystic carcinoma 6. Tumor necrosis and elevated mitotic rates are not seen, except in rare cases of high-grade transformation

	Adenoid Cystic Carcinoma	Epithelial–Myoepithelial Carcinoma
<i>Special studies</i>	<ul style="list-style-type: none"> Actin, calponin, S100, p63, and p40 highlight myoepithelial cells, while c-kit and EMA typically stain ductal component Approximately 50% of adenoid cystic carcinomas harbor a (6;9) translocation resulting in MYB–NFIB gene fusion 	<ul style="list-style-type: none"> Actin, calponin, S100, p63, and p40 highlight myoepithelial cells, while c-kit and EMA typically stain ductal component Negative for MYB rearrangements. Some cases harbor RAS mutations
<i>Treatment</i>	Wide local resection with adjuvant radiotherapy. Neck dissection not usually performed because lymph node metastases are uncommon. Chemotherapeutic agents are generally ineffective	Surgical excision. Adjuvant radiation considered if there are aggressive histologic features (e.g., perineural invasion, large tumor size)
<i>Prognosis</i>	Patients have a good 5-year survival (75%–80%) but poor 15-year survival (25%–30%) due to slow but relentless growth. Tumors metastasize to lung, bone, liver, and brain. Tumors with a predominantly solid pattern are more aggressive	Good. Recurrences and metastases are uncommon

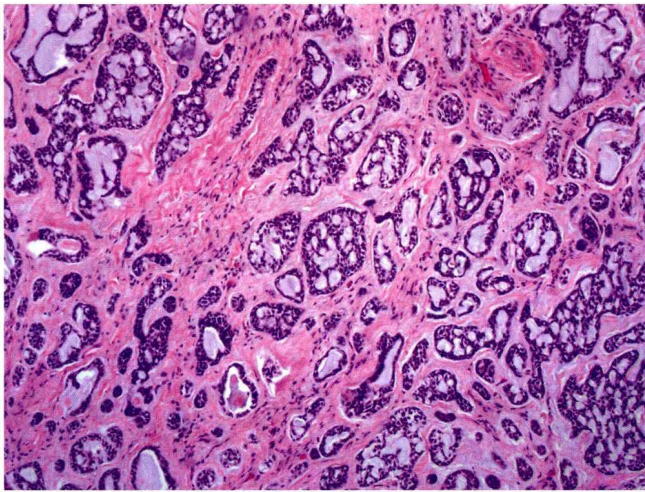


Figure 1.2.1 Adenoid cystic carcinoma with an infiltrative collection of tubules and cribriform structures.

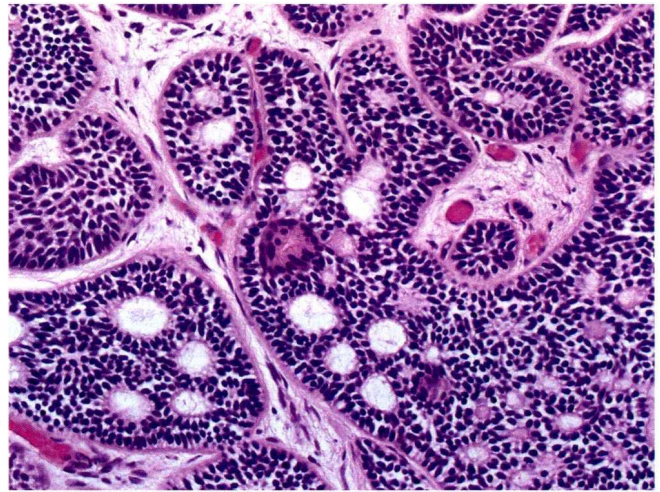


Figure 1.2.2 Adenoid cystic carcinoma with cribriform growth. There is a true duct (**center**) with eosinophilic cytoplasm, numerous pseudoducts, and a predominance of myoepithelial cells with dark, angulated nuclei.

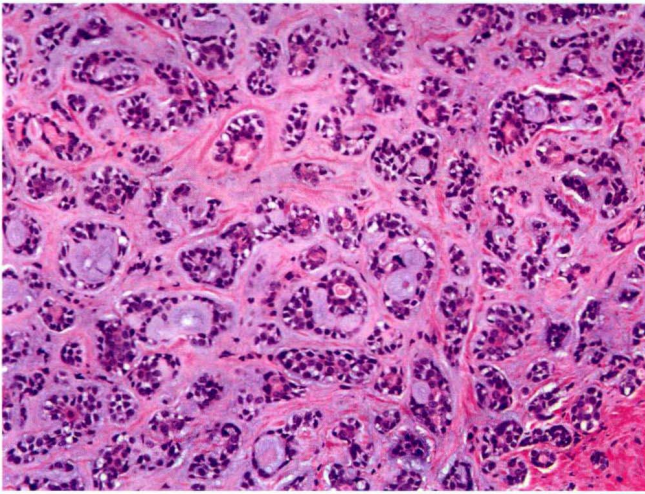


Figure 1.2.3 Adenoid cystic carcinomas are comprised of true ductal cells and myoepithelial cells. In this tumor, the eosinophilic ductal cells are surrounded by a zone of clear myoepithelial cells in a way that resembles epithelial–myoepithelial carcinoma. The basophilic stromal matrix is characteristic of adenoid cystic carcinoma.

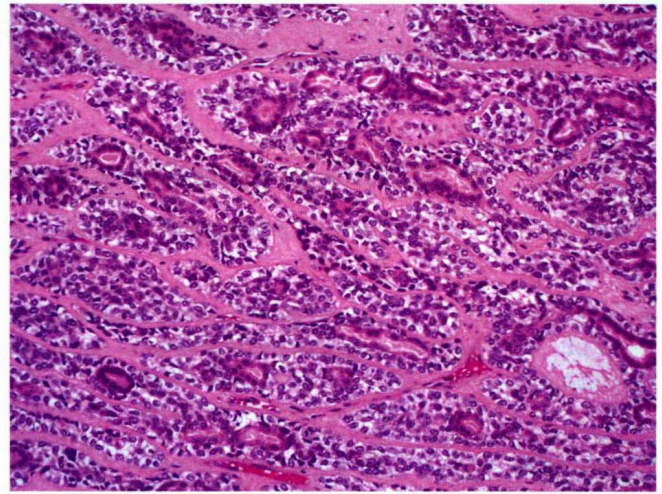


Figure 1.2.4 Epithelial–myoepithelial carcinoma with numerous eosinophilic ducts that are tightly coupled with a surrounding layer of myoepithelial cells with abundant clear cytoplasm.

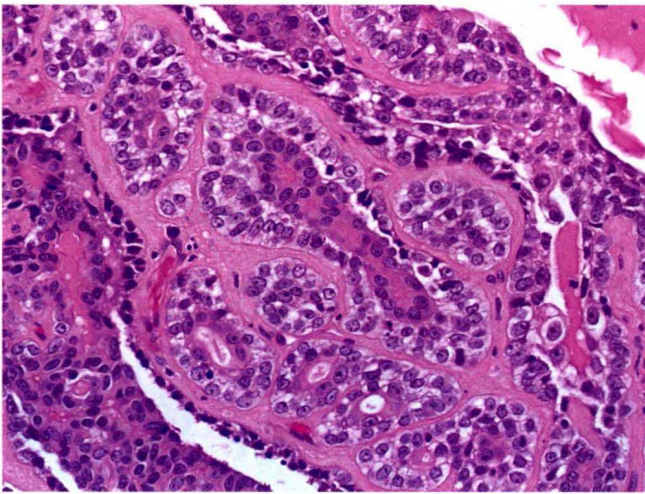


Figure 1.2.5 Epithelial–myoepithelial carcinoma with myoepithelial cells demonstrating large nuclei with open chromatin and prominent nucleoli.

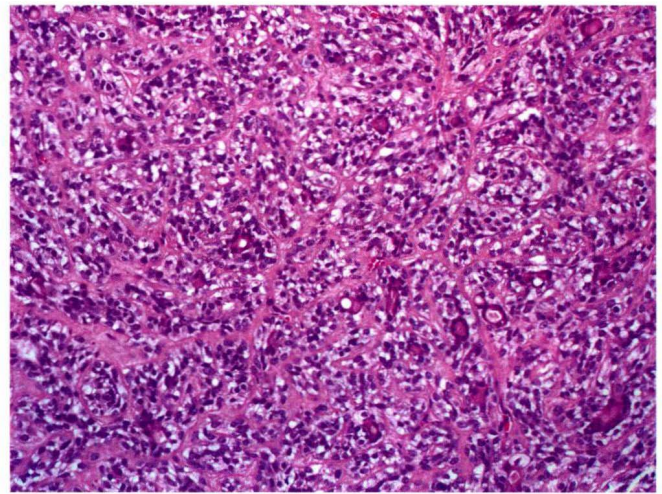


Figure 1.2.6 Epithelial–myoepithelial carcinoma with a predominance of myoepithelial cells.

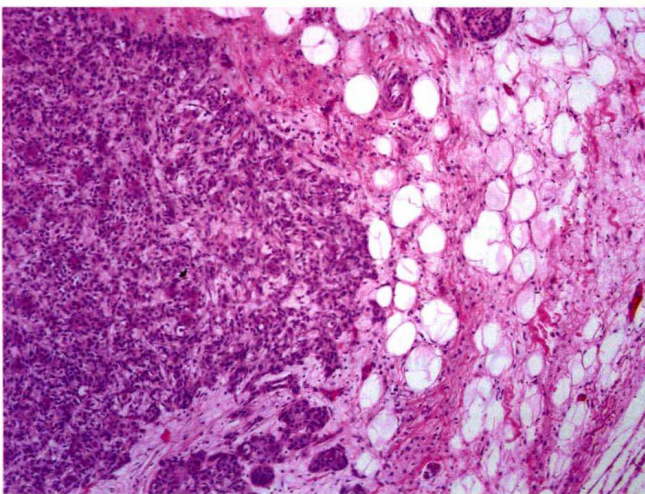


Figure 1.2.7 Epithelial–myoepithelial carcinoma demonstrating irregular infiltration into surrounding fat.

	Adenoid Cystic Carcinoma	Basaloid Variant of Squamous Cell Carcinoma
<i>Age</i>	Typically adults, peak incidence in sixth decade	Adults, peak in seventh decade
<i>Location</i>	Parotid gland is most common (40%–50%) but may arise in any major or minor salivary gland	Mucosal head and neck sites, most commonly larynx. Does not arise the major salivary glands, though rarely may involve them by metastatic spread
<i>Symptoms</i>	Slowly growing swelling, sometimes pain or paralysis due to perineural invasion. Other site-specific symptoms in minor salivary locations (e.g., nasal obstruction or epistaxis for sinonasal tumors)	Depends on site. In larynx, patients present with hoarseness and dysphagia
<i>Signs</i>	Palpable nodules that may become fixed to surrounding tissues	Ulcer and ill-defined mucosal-based mass
<i>Etiology</i>	Unknown	Strongly related to tobacco and alcohol consumption
<i>Histology</i>	<ol style="list-style-type: none"> 1. Variable mixture of tubules, cribriform structures, and solid nests (Fig. 1.3.1) 2. Cribriform nests have cylindromatous microcystic spaces (false ducts) (Fig. 1.3.2) 3. Two cell types: ductal and myoepithelial. The myoepithelial cells predominate; they are monotonous, basaloid, and have hyperchromatic, angulated nuclei with indistinct nucleoli and small amounts of clear to eosinophilic cytoplasm. The ducts are often subtle and are comprised of cuboidal cells with eosinophilic cytoplasm (Fig. 1.3.2) 4. Squamous differentiation is absent 5. Highly infiltrative. Perineural invasion is very common 6. Tumor necrosis and elevated mitotic rates are uncommon but are more common in tumors with a predominant solid pattern 	<ol style="list-style-type: none"> 1. Rounded nests of cells separated by thin lines of hyalinized stroma, creating a jigsaw puzzle-like pattern (Fig. 1.3.5) 2. Frequent deposition of hyaline basement membrane-like material, creating pseudoglandular spaces and a cribriform-like appearance (Fig. 1.3.6) 3. No true ducts 4. Nests exhibit peripheral palisading, and tumor cells have high nuclear to cytoplasmic ratios 5. Squamous differentiation is present within the tumor itself where it is often abrupt or in the form of overlying squamous cell carcinoma in situ (Fig. 1.3.7) 6. Highly infiltrative. Perineural invasion is very common 7. Tumor necrosis and elevated mitotic rates are present

	Adenoid Cystic Carcinoma	Basaloid Variant of Squamous Cell Carcinoma
<i>Special studies</i>	<ul style="list-style-type: none"> Actin, calponin, p63, S100, and p40 highlight myoepithelial cells, while c-kit and EMA typically highlight the ductal component (<i>Figs. 1.3.3 and 1.3.4</i>) Approximately 50% of adenoid cystic carcinomas harbor a (6;9) translocation resulting in MYB–NFIB gene fusion 	<ul style="list-style-type: none"> The tumor is diffusely positive for p63, CK5/6, and CK903 (<i>Fig. 1.3.8</i>). S100 and actin are negative. C-kit may be positive Negative for (6;9) translocation
<i>Treatment</i>	Wide local resection with adjuvant radiotherapy. Neck dissection not usually performed because lymph node metastases are uncommon. Chemotherapeutic agents are generally ineffective	Surgery, radiotherapy, and chemotherapy. Neck dissection often performed, as nodal metastases are common
<i>Prognosis</i>	Patients have a good 5-year survival (75%–80%) but poor 15-year survival (25%–30%) due to slow but relentless growth. Tumors metastasize to lung, bone, liver, and brain. Tumors with a predominantly solid pattern are more aggressive	Poor. Basaloid variant appears to be more aggressive than conventional squamous cell carcinoma

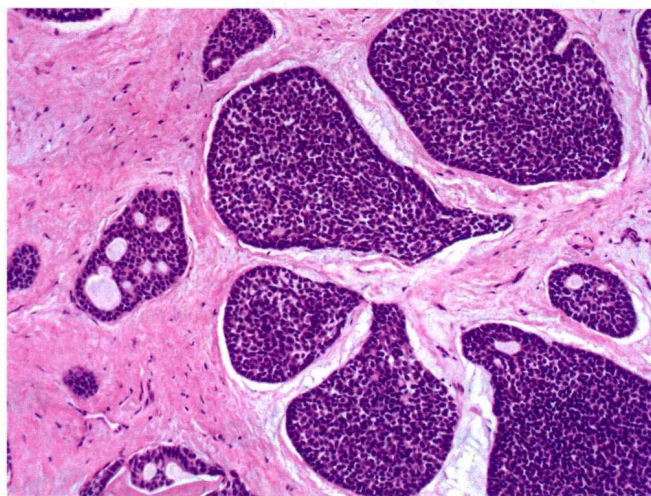


Figure 1.3.1 Adenoid cystic carcinoma growing as cribriform and solid nests of basaloid cells.

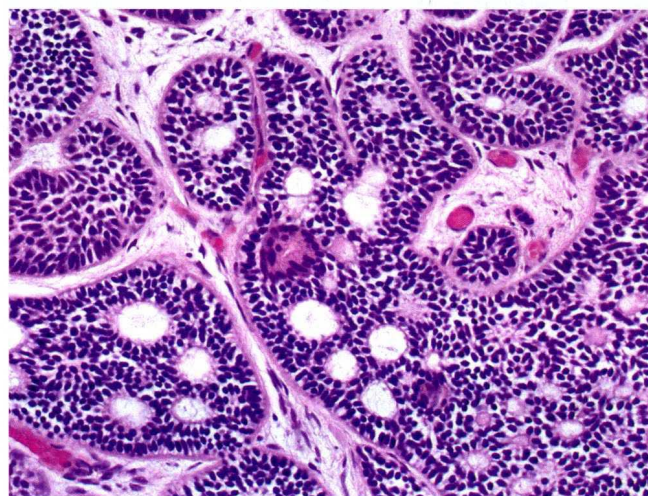


Figure 1.3.2 Adenoid cystic carcinoma consisting predominantly of myoepithelial cells characterized by hyperchromatic and angulated nuclei. They form rounded cylindromatous (pseudoglandular) spaces. A few true ducts lined by cells with eosinophilic cytoplasm are also present.