NEOPLASTIC MIMICS
in Genitourinary Pathology
Pathology of Neoplastic Mimics Series
Mark R. Wick and Peter A. Humphrey

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Neoplastic Mimics in Genitourinary Pathology

Peter A. Humphrey, MD, PhD
Ladenson Professor of Pathology and Immunology
Chief, Division of Anatomic and Molecular Pathology
Barnes-Jewish Hospital
Washington University School of Medicine
St. Louis, Missouri

J. Carlos Manivel, MD
Professor of Pathology and Urology
University of Minnesota
Veterans Administration Medical Center
Minneapolis, Minnesota

Robert H. Young, MD, FRCPath
Robert E. Scully Professor of Pathology
Director of Gynecological and Urological Pathology
Massachusetts General Hospital, Harvard Medical School
Boston, Massachusetts
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ISBN: 978-1-620700-20-4

Acquisitions Editor: Rich Winters
Compositor: diacriTech

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Library of Congress Cataloging-in-Publication Data

Humphrey, Peter A., author.
Neoplastic mimics in genitourinary pathology / Peter A. Humphrey, J. Carlos Manivel, Robert H. Young.
p.; cm.—(Pathology of neoplastic mimics series)
Includes bibliographical references and index.
RC280.G52
616.99'46—dc23
2013028152

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Printed in the United States of America by Courier.
13 14 15 16 17 / 5 4 3 2 1
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Contributors

Arbaz Samad, MD  
Oncologic Pathology Fellow  
Department of Pathology  
Memorial Sloan-Kettering Cancer Center  
New York, New York

Charanjeet Singh, MD  
Fellow  
Division of Cytopathology  
Department of Pathology & Laboratory Medicine  
University of Texas MD Anderson Cancer Center  
Houston, Texas

Mark R. Wick, MD  
Professor of Pathology  
Associate Director of Surgical Pathology  
University of Virginia Health System  
Charlottesville, Virginia
One of the most challenging and humbling aspects of surgical pathology practice is the well-known ability for disparate biological processes and diseases to resemble one another morphologically. This capacity for mimicry is seen within nonneoplastic and neoplastic processes. More problematically, and as discussed in this atlas, it is possible for nonneoplastic conditions to assume the guises of neoplasms at a macroscopic level, a microscopic one, or both. When that occurs, there is a real danger that pathologists will misinterpret these benign lesions, and incorrect treatment may result. It is critical that the diagnostic surgical pathologist generate a mental checklist of those pseudoneoplasms that can simulate malignancy, so that before diagnosing a neoplasm, the diagnostician rules out a benign lesion.

This book discusses and illustrates, in a comprehensive fashion, the many faces of these pseudoneoplasms in the genitourinary tract. It is important to appreciate the spectrum of appearance of these pseudoneoplasms, and accordingly this book provides, for many entities, several different images along that spectrum.

To make the life of the pathologist even more difficult, the converse issue may arise, namely deceptively benign appearing malignancies can be misdiagnosed as benign. In this book, these pseudoneoplasms and deceptively benign-appearing malignancies are discussed and illustrated together, often side-by-side.

It is our hope that this type of format will be a diagnostic aid for those who see these often challenging cases, with a reduction in errors in diagnosis.
Acknowledgments

Thanks to my wife Kay for unwavering support and understanding and to colleagues in Pathology and Urology at Washington University Medical Center. Special thanks to Dr. Robin Vollmer, who inspired my interest in genitourinary pathology, and to Dr. Mark Wick, who created the concept of a book focused on pseudoneoplasms. PAH

I am greatly indebted and profoundly grateful to the patients who have taught me over the years; the correct answer is always theirs. JCM

To my wife Terry for all her help and encouragement over the years and to our wonderful daughters Jessica and Jennifer for their understanding. As always I am indebted to the late Dr. Robert E. Scully for all he taught me and his great example. Thanks also to the other two great associates of my professional career, Dr. Philip B. Clement and Dr. Esther Oliva, for all they have done to enrich my academic life. RHY
"Pseudotumor" is an indefinite term that has typically been used to indicate the presence of a mass, which is felt to represent a neoplasm at some level of observation. In most circumstances it is a misnomer, because a mass actually exists; in that situation, the term “pseudoneoplasm” would be more appropriate. However, there may still be circumstances in which the term “pseudotumor” is appropriately used; some of those scenarios are presented below. Nonetheless, this discussion will be directed to those lesions that are best designated as “pseudoneoplasms.” They are typically represented by masses that have been biopsied or resected, but the microscopic slides do not show the presence of a neoplastic process.

From whence did the designation of “pseudotumor” arise? Ophthalmology has been cited as the first specialty in which the literature used the descriptor of “pseudotumor” (1); in 1930, Birch-Hirschfeld employed it in discussing orbital lesions that had produced proptosis (2). Thereafter, other authors appended such adjectives as “inflammatory,” “lymphoid,” “granulomatous,” “xanthomatous,” and “fibrous,” in attempts to subdivide “pseudotumors” into pathologically recognizable subgroups (3). The word “pseudotumor” has also been synonymized with such alternatives as “postinflammatory tumor” (4,5), “histiocytoma” (6), “plasma cell granuloma” (7–9), “mast cell granuloma”
(10), “xanthoma” (11–14), “xanthogranuloma” (15), “plasma cell/histiocytoma complex” (16), and, in some reports (17), “sclerosing hemangioma” as well.

This brief introductory discussion further considers definitions and presentations of “pseudotumors.” Because this discussion is especially aimed at pathologists, it will generally be based on the morphologic characteristics of the lesions being considered.

**PHILOSOPHICAL AND PRACTICAL ISSUES**

The visual images of the same cases that are seen by internists, surgeons, radiologists, and pathologists are potentially quite dissimilar. For example, the surgeon may believe that he or she has identified a palpable mass in the breast, but the radiologist may see little or nothing on mammogram. Similarly, a pathologist examining a fine needle aspiration biopsy of the putative lesion may find nothing that is abnormal. In this scenario, the second and third physicians may question the claims of the surgeon, but unless they physically examine the patient themselves, they can never really determine whether a “tumor” is actually present at a clinical level of assessment. Nonetheless, the palpable lesion is certainly a potential neoplasm in the mind of the surgeon.

Radiologists and pathologists are subject to the same tricks of nature. For example, it is well known that hemorrhages in hemophiliac patients may simulate neoplasms in radiographs, although none is seen through the microscope after excision (18,19). Comparable comments apply to the roentgenographically defined images of “tumefactive biliary sludge” (20), duodenal ampullary “mucosal rosettes” (21), tumor-like musculoskeletal variations (22), and pulmonary “rounded atelectasis” (23–26). From the perspective of the pathologist, adnexal nevi of the skin (27–29), cutaneous reactions to Monsel’s reagent (a styptic solution) (30,31), and the presence of the organ of Chievitz (a histologic imitator of squamous carcinoma) in biopsies of the oral mucosa (32–36) are singular pseudoneoplastic conditions with no associated clinical or radiographic aberrations. Only by face-to-face collegial interaction can all information on such cases be integrated, to yield a clear delineation of the processes or anatomic variations in question (37). Indeed, practically speaking, interpretative disagreement between specialists—as stressful as that can be—is often the principal clue to the existence of a pseudoneoplastic condition.

A considerable number of “pseudotumors” can simulate neoplasms on all levels of analysis—clinical, radiologic, and pathologic—and they consequently represent particular diagnostic pitfalls, which can lead to therapeutic...
misdirection. Those are the entities to which this book is principally directed. In addition, other pathologic conditions are included that histopathologists alone—and not clinicians or radiologists—may uniquely misinterpret as neoplastic in nature.

Certain caveats must be stated in addressing this general topic. First, the lesions known as “inflammatory pseudotumors” (IPs) are generally recognized as representing true neoplasms (“inflammatory myofibroblastic tumors,”) although sometimes considerable difficulty may still be encountered in separating them from selected inflammatory conditions (38). Similar comments are applicable to angiomyolipomas, certain melanocytic nevi, and proliferative but reactive lymphoid lesions (“pseudolymphomas”). After the application of such techniques as immunohistochemistry, cytogenetics, and gene rearrangement analyses, the majority of true neoplasms in these lesional groups can be defined correctly. Furthermore, as more knowledge continues to accrue from those studies, the classification of “pseudoneoplasms” will undoubtedly undergo continuing revision.

TOPOGRAPHIC DISTRIBUTION AND BIOLOGIC NATURE OF “PSEUDOTUMORS”

Virtually all topographic sites in the human body may play host to lesions that simulate neoplasms. Perhaps because they are especially sensitive to etiologic influences that cause pseudotumors, some locations appear to be particularly prone to them. Principal examples are the lungs, urinary tract, and gut. In addition, pseudoneoplastic processes are partially overlapping and partly distinctive as seen in various anatomic sites. That observation may again reflect the relative influences of different pathogenetic factors on different tissues, but other variables probably have an effect as well. The following sections consider “pseudoneoplasms,” as related to their putative causal categories.

IDIOPATHIC PSEUDONEOPLASMS

True (IP) has an unknown cause in most cases and is most commonly seen in the pulmonary parenchyma (38–61). It may conceivably represent a localized remnant of unresolved or organizing pneumonia (62,63).

There are several other IP variants that represent etiologically heterogeneous entities, with at least one variant of each being idiopathic. Examples include nodular lymphoid hyperplasias of the lung, gut, skin, mucosal surfaces, soft tissue, and lymphoreticular system (64–72); and fibro-osseous lesions of
craniofacial and small tubular bones (73–76). In these groups of disorders, some potentially identifiable pathogenetic factors may include viral infection or autoimmune diseases (71,72); and fibrous dysplasia or familial “cherubism” (75,77). However, other morphologically identical IPs are associated with no causal explanations and are therefore classified as idiopathic.

Still other conditions are “quasi-neoplastic,” in that they feature etiologically unknown, and, to some extent, autonomous, cellular proliferations in various locations without a definable stimulus. Radial scar of the breast (78–81); urethral (prostatic utricular) polyp (82); fibroepithelial polyp of the urinary bladder (83); some “tumefactive fibroinflammatory lesions” of soft tissue (84,85), metaphysial fibrous defects (“nonossifying fibromas”) of bone (86,87); and aneurysmal bone cysts (88–92) belong to that category. They differ biologically from true neoplasms because they are self-limited or even spontaneously regressing.

Other pseudoneoplastic processes do have a linkage with underlying disease processes, but the ultimate etiology of the latter conditions can be idiopathic. Proliferative Paget’s disease of bone (osteitis deformans) (93–96), tumefactive plaques of active multiple sclerosis (97–100), and lymphoma-like Hashimoto’s thyroiditis (101,102) are representative examples.

**REPARATIVE/POSTTRAUMATIC PSEUDONEOPLASMS**

Exaggerated host responses to tissue insults arguably comprise the most common mechanisms underlying the formation of pseudoneoplasms. In many of those instances the injury may have been trivial and subclinical—to wit, unnoticed by the patient—and the resulting reparative process might therefore be regarded initially as idiosyncratic. That situation likely applies to nodular fasciitis (103,104), proliferative fasciitis and myositis (105–108), giant-cell reparative granuloma of bone (109–112), fibrohyaline pleuritis (113,114), and xanthogranulomatous inflammation in several sites (115–120).

Still other pseudoneoplasms are caused by documented episodes of injury, but again with an amplified and idiosyncratically robust reparative response. Pseudocarcinomatous (“pseudoepitheliomatosus”) hyperplasias of the skin (121,122), acroangiodermatitis (123), myositis ossificans (124,125), “atypical (ischemic) decubital fibroplasia” (126–129), tumefactive synovial chondrometaplasia (130), necrotizing sialometaplasia (131–133), gliosis in the central nervous system (134), nephrogenic urothelial metaplasia (135), inflammatory polyps of the anorectal region (136), colitis cystica profunda (137), florid reactive mesothelial proliferations (138–141), tumor-like chronic pancreatitis (142), therapy-induced tissue reactions (143–146) (which are
simultaneously reparative and iatrogenic, causally), and polypoid-papillary cystitis (147) are all examples of lesions in this category. Why some individuals develop exaggerated repair mechanisms and others do not is an open question at present.

■ DEVELOPMENTAL PSEUDONEOPLASMS

The most straightforward group of pseudoneoplasms relates to individual abnormalities in development, or, alternatively, ignorance on the part of pathologists regarding the details of normal organogenesis and embryology. Choristomas (148), hamartomas (149–153), and cutaneous nonmelanocytic nevi are members of the first of those categories, because they are classified as flaws of morphogenesis. Tissue remnants or heterotopias relating to embryologic development constitute another large subgroup in the same cluster, including examples of vaginal adenosis (154–156), cervical mesonephric remnants (157), “adenomyoma” (“Brunner’s gland hamartoma”) of the duodenum (158), and cutaneous “rudimentary meningocele” (159) and glial heterotopias (160,161).

On the other hand, the juxtaoral organ of Chievitz is a normal structure rather than an embryologic vestige or developmental anomaly (32–36). However, it is so uncommonly seen in mucosal biopsies that some observers may fail to recognize it and confuse this inclusion with a malignant neoplasm (squamous carcinoma).

Malformations related to congenital or Mendelian syndromes also may resemble neoplasms. The tumefactive lesions of osseous fibrous dysplasia (94,109,162,163) and the paraventricular glial nodules of tuberous sclerosis (161) are representatives.

■ “FUNCTIONAL” PSEUDONEOPLASMS

Some pseudoneoplasms are caused by a dysfunctional pathophysiologic state, often relating to endocrine abnormalities. “Dominant” nodules often arise in parenchymal hyperplasias of the thyroid or parathyroid glands (102) and that those lesions can simulate adenoma or even carcinoma microscopically. Comparable comments apply to localized unilateral nodular adrenocortical hyperplasia (164). Other pseudoneoplastic conditions with an endocrine underpinnings include prostatic hyperplasias (165,166), focal nodular hyperplasia of the liver (167–169), ovarian stromal hyperthecosis (170), the Arias-Stella reaction in epithelium of the gynecologic tract (171–173), osteitis fibrosa
cystica ("brown tumor" of hyperparathyroidism) (174–178), and uterine cervical microglandular hyperplasia (179).

**IATROGENIC PSEUDONEOPLASMS**

Several medical procedures may produce subsequent tissue reactions that imitate the appearance of neoplasms. Most are reparative in nature and could also have been included in the corresponding section above. However, because of their clearly iatrogenic nature, they deserve special recognition. Instrumentation or surgery of the genitourinary tract may cause an idiosyncratic tumefaction comprising variably atypical spindle cells, known simply as “postoperative spindle cell nodule” (180–186). In the same vein, application of the iron salt-based cutaneous styptic, Monsel’s solution, has caused idiosyncratic cellular proliferations resembling spindle-cell tumors of the skin and uterine cervix (30,31).

Other pseudoneoplasms in this category may be able to imitate an infiltrative or in-situ neoplasm. These include urothelial cytotoxicity caused by cyclophosphamide, simulating in-situ urothelial carcinoma (187); epithelial atypia in the breast after chemotherapy (188); and florid vaginal adenosis in women whose mothers took diethylstilbestrol during pregnancy (155). Iatrogenic pseudoneoplasms are particularly problematic for pathologists (189), because we often are not supplied with pertinent—and sometimes crucial—information concerning prior treatments or interventions.

**INFECTIOUS PSEUDONEOPLASMS**

Particularly because of Acquired Immunodeficiency Syndrome (AIDS), tissue reactions to several infectious pathogens have been increasingly recognized that are capable of simulating neoplasms. Conjointly cytopathic and reparative responses to infection with cytomegalovirus, Epstein–Barr virus (EBV), papovavirus, selected species of mycobacteria, and other bacilli can produce proliferations in several organ systems with a histological likeness to sarcomas, gliomas, or lymphomas (71,190–194).

That phenomenon is not restricted to individuals with AIDS. It is also the basis for malakoplakia (195–199), “histoid” cutaneous infections caused by *Mycobacterium leprae* (200,201), and florid lymphoid hyperplasia of the terminal ileum in patients infected with *Yersinia* (202).

As outlined in the foregoing material, pseudoneoplastic proliferations are potentially encountered in virtually all the topical areas of anatomic pathology,
including cytopathology. Thus, they represent a truly protean challenge with many possible implications for patient care and case outcome. All pathologists must bear those facts in mind during day-to-day practice, in order to guard against misinterpretations.

Table 1.1 presents a synopsis of this discussion in tabular form, separated into organ systems.

This volume, Neoplastic Mimics in Genitourinary Pathology, provides comprehensive descriptions, discussion, and images of the lesions summarized in the table as pertinent to the male and female genitourinary tract. For similar coverage of the other systems and categories of lesions shown in the table, the reader is referred to the other volumes of the Neoplastic Mimics series of atlases.

**TABLE 1.1** Selected Examples of Pseudoneoplastic Lesions and Related Neoplastic Imitators

<table>
<thead>
<tr>
<th>SKIN</th>
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<tbody>
<tr>
<td>Cutaneous nonmelanocytic nevi and hamartomas (epithelial and mesenchymal neoplasms)</td>
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<tr>
<td>Lymphoid hyperplasias (lymphomas)</td>
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<tr>
<td>Reactions to Monsel’s solution (sarcomas; sarcomatoid carcinomas)</td>
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<tr>
<td>Acroangiodermatitis (Kaposi’s sarcoma)</td>
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<tr>
<td>Proliferating scars and posttraumatic spindle-cell nodules (mesenchymal tumors; sarcomatoid carcinomas)</td>
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<tr>
<td>Intravascular papillary endothelial hyperplasia (Masson’s lesion) (vascular neoplasms)</td>
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<tr>
<td>Pseudocarcinomatous epithelial hyperplasia (squamous cell carcinoma)</td>
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<td>“Rudimentary meningocele” (mesenchymal neoplasms)</td>
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<td>Ruptured ganglion cyst (acral myxoinflammatory sarcoma)</td>
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<tr>
<td>Mycobacterial pseudotumors (mesenchymal neoplasms)</td>
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<tr>
<td>Bacillary angiomatosis (hemangiomas)</td>
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<td>Kimura disease (hypereosinophilic syndrome; eosinophilic leukemia)</td>
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<th>SOFT TISSUE</th>
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<tr>
<td>Neuromuscular choristoma (peripheral nerve sheath neoplasms)</td>
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<tr>
<td>Fibrolipomatous hamartoma (lipomas)</td>
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<tr>
<td>Nodular fasciitis (sarcomas)</td>
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<tr>
<td>Proliferative myositis (sarcomas)</td>
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<tr>
<td>Myositis ossificans (osteosarcoma)</td>
</tr>
<tr>
<td>“Tumefactive fibroinflammatory lesions” (fibromatoses)</td>
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<tr>
<td>Florid (tumefactive/focal) lymphocytic myositis (lymphoma)</td>
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<tr>
<td>“Atypical decubital” (ischemic) fibroplasia (sarcomas)</td>
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</tbody>
</table>

(Continued)
**TABLE 1.1 Selected Examples of Pseudoneoplastic Lesions and Related Neoplastic Imitators (Continued)**

**BONES AND JOINTS**

- “Bizarre osteochondromatous proliferations” (Nora’s lesion) of digits (cartilaginous neoplasms)
- Synovial chondrometaplasia/chondrocalcinosis (cartilaginous neoplasms)
- Fibrous dysplasia and “fibro-osseous lesions” (osteosarcoma and fibrosarcoma)
- Proliferative-phase Paget’s disease of bone (osteosarcoma)
- Aneurysmal bone cyst (telangiectatic osteosarcoma)
- Giant-cell reparative granuloma (giant cell tumor)
- Avulsion fractures of ischial tuberosities (osteosarcoma)
- “Brown tumor” of hyperparathyroidism (osteitis fibrosa cystica) (giant cell tumor)

**BREAST**

- Radial scar (low-grade ductal adenocarcinoma)
- Choristoma (Hamartoma) (metaplastic carcinoma)
- Proliferative adenosis (low-grade ductal adenocarcinoma)
- Extramedullary hematopoiesis (invasive lobular carcinoma)
- Collagenous spherulosis (adenoid cystic carcinoma or intraductal carcinoma)
- Pseudoangiomatous stromal hyperplasia (angiosarcoma)

**NERVOUS SYSTEM**

- Gliosis (low-grade gliomas)
- Active-phase plaques of multiple sclerosis (gliomas)
- Progressive multifocal leukoencephalopathy (gliomas)
- Paraventricular glial nodules of tuberous sclerosis (gliomas; gangliogliomas)
- Viral encephalitides (lymphoma)

**ENDOCRINE SYSTEM**

- Sclerosing and proliferative Hashimoto’s thyroiditis (anaplastic carcinomas and lymphomas)
- Nodular thyroid hyperplasia (thyroid adenomas and differentiated thyroid carcinomas)
- Nodular parathyroid hyperplasia (parathyroid adenoma)
- Nodular adrenal hyperplasia (adrenocortical adenoma)
- Adrenal myelolipoma (liposarcoma)

**LYMPHORETICULAR SYSTEM**

- Selected lymphoid hyperplasias (lymphomas)
- Florid oligoclonal hyperplasia in bone marrow recovery (myelodysplasia; leukemia)
- Infection-related hemophagocytic syndrome (T-cell lymphoma)
- Epstein–Barr virus-related atypical lymphoid hyperplasias (large-cell lymphoma)
- Mycobacterial pseudotumors (dendritic-cell tumors)
NEOPLASTIC MIMICS: GENERAL CONSIDERATIONS

TABLE 1.1 Selected Examples of Pseudoneoplastic Lesions and Related Neoplastic Imitators (Continued)

UPPER AIRWAY
- Pseudocarcinomatous epithelial hyperplasia (squamous cell carcinoma)
- Oral organ of Chievitz (low-grade squamous or mucoepidermoid carcinoma)
- Necrotizing sialometaplasia (squamous or mucoepidermoid carcinoma)
- Radiation effects on mucosal epithelia (squamous carcinoma)
- Benign lymphoepithelial lesion of salivary gland (lymphomas)
- Traumatized antral/choanal polyps (polypoid sarcomatoid carcinomas)
- Glial heterotopias (peripheral nerve sheath tumors)
- Benign fibro-osseous lesions (low-grade fibrosarcoma or osteosarcoma)

LOWER AIRWAY
- Pseudocarcinomatous epithelial hyperplasia (squamous cell carcinoma and adenocarcinoma)
- Atypical bronchiolar and alveolar epithelial hyperplasia (carcinomas)
- Fibrohyaline plaques of pleura and fibrohyaline pleuritis (desmoplastic mesothelioma)
- Florid mesothelial hyperplasia (epithelial mesothelioma)
- Localized tumefactive organizing pneumonia (inflammatory sarcomatoid carcinoma)
- Selected examples of lymphocytic interstitial pneumonia (lymphoma)
- Pulmonary chondroid/lipomatous/muscular hamartomas (metaplastic carcinomas)

MEDIASTINUM
- Sclerosing mediastinitis (sclerosing carcinomas, lymphomas, or germ-cell tumors)
- Thymic dysplasia (thymoma)
- Proliferating thymic cyst (thymic squamous carcinoma)
- Benign mesothelial inclusions in mediastinal lymph nodes (metastatic carcinoma)

ALIMENTARY TRACT
- Pseudocarcinomatous epithelial hyperplasia (adenocarcinoma)
- Enteritis/colitis cystica profunda (adenocarcinoma)
- “Adenomyoma” of duodenum (well-differentiated adenocarcinoma)
- Tumefactive chronic pancreatitis (well-differentiated ductal adenocarcinoma or lymphoma)
- Mycobacterial pseudotumors (sarcomas)
- Bacillary angiomatosis (hemangiomas)
- Florid lymphoid hyperplasia (lymphomas)
- Hepatic bile duct hamartoma (metastatic adenocarcinoma)
- Hepatic focal nodular hyperplasia (fibrolamellar hepatocellular carcinoma)
- Inflammatory “cloacogenic” polyp (tubulovillous adenoma)
- Chronic tumefactive pancreatitis (low-grade pancreatic ductal adenocarcinoma)
- Xanthogranulomatous cholecystitis (sarcomatoid carcinoma)

(Continued)

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TABLE 1.1  Selected Examples of Pseudoneoplastic Lesions and Related Neoplastic Imitators (Continued)

<table>
<thead>
<tr>
<th>GENITOURINARY TRACT (MALE AND FEMALE)</th>
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<tr>
<td>Pseudocarcinomatous epithelial hyperplasia (carcinomas)</td>
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<td>Postoperative spindle cell nodules (sarcomas; sarcomatoid carcinoma)</td>
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<td>Drug effects (e.g., cytoxan cystitis mimicking carcinoma in-situ) (urothelial carcinoma)</td>
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<td>Proliferative prostatic urethral (utricular) polyp (adenocarcinoma)</td>
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<tr>
<td>Paratesticular mycobacterial pseudotumor (sarcomas)</td>
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<td>Lymphoplasmacytic orchitis (lymphoma)</td>
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<tr>
<td>Xanthogranulomatous nephritis/cystitis/orchitis/endometritis/oophoritis (carcinomas)</td>
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<td>Adenomatous and basal-cell prostatic hyperplasia (adenocarcinoma)</td>
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<td>Cribiform intraductal prostatic hyperplasia (adenocarcinoma)</td>
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<td>Nodular stromal prostatic hyperplasia (low-grade sarcoma)</td>
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<td>Prostatic sclerosing adenosis (adenocarcinoma)</td>
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<td>Radiation effect on prostatic epithelium (residual adenocarcinoma)</td>
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<tr>
<td>Granulomatous prostatitis/orchitis (sclerosing carcinoma and sclerosing seminoma)</td>
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<td>Vaginal adenosis (adenocarcinoma)</td>
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<tr>
<td>Uterine cervical mesonephric remnants (adenocarcinoma)</td>
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<tr>
<td>Uterine cervical microglandular adenosis (adenocarcinoma)</td>
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<tr>
<td>Ovarian stromal hyperplasia/hyperthecosis (ovarian stromal neoplasms)</td>
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<tr>
<td>Nephrogenic metaplasia of bladder and urethra (adenocarcinoma)</td>
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<tr>
<td>Endometriosis (adenocarcinoma)</td>
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<tr>
<td><strong>CARDIOVASCULAR SYSTEM</strong></td>
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<tr>
<td>Rhabdomyomatous hamartomas of myocardium (true “adult” rhabdomyoma)</td>
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<td>Endodermal choristoma of interatrial cardiac septum (metastatic adenocarcinoma)</td>
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<tr>
<td>Lipomatous hypertrophy of the heart (lipoma)</td>
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<td>Mesothelial-monoeyctic intracardiac excrescences (metastatic adenocarcinoma or mesothelioma)</td>
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<td>Endocardial and myocardial lymphocytic infiltrates after transplantation (lymphomas)</td>
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<td>Florid pericardial mesothelial hyperplasia (epithelial mesothelioma and metastatic adenocarcinoma)</td>
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