Oral and Maxillofacial PATHOLOGY

A Rationale for Diagnosis and Treatment Second Edition

Volume I

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Preface to the Second Edition

What is the Same, What is New, and What is the Future

What is the Same

We were deeply honored and gratified that this book's first edition received the 2002 Medical Book of the Year Award from the American Medical Writers Association. However, we were even more honored and gratified by the reception and positive comments we received from our colleagues, residents, and students. Comments such as "just what was needed," "it is the only book of its kind," and "I use it all the time" affirmed the intent and goal of this book to be much more than microscopic pathology. The basic concept of clinical presentation, pathogenesis, diagnostic work-up, specific treatments and their rationale, as well as prognosis and follow-up guidelines, in addition to detailed histopathology is the best method to relate patient care–focused knowledge and remains the central theme of this second edition. Also the same is our attempt to simplify terminology and relate it more directly to the mechanism and causation of each disease.

What is New

What is new from a disease inclusion perspective is bisphosphonate-induced osteonecrosis, of which the inclusion in the first edition marked the very first identification of what is now recognized as a worldwide serious drug complication. Additionally, we have included sections on cavernous sinus thrombosis and Ludwig angina and expanded the chapters on salivary gland tumors, fibro-osseous diseases, and more. Also, the continuing evolution of immunopathology, molecular biology, and genetics, where pertinent, has been incorporated into the text. We have also added specific reconstructive techniques using recombinant human bone morphogenetic protein, stem cell concentrates, and tissue-engineering principles in those chapters dealing with the reconstruction of disease-related defects.

From a presentation perspective, we have added over 500 new pictures, incorporating the new imaging techniques of cone beam CT scans, 3D modeling, and PET scans as well as more representative photographs of several diseases and outcomes of treatments. These new images better illustrate the spectrum of clinical appearances of many diseases and document the efficacy of the treatment and reconstructive methods outlined. The reader may also note the expansion in antimicrobial drugs outlined with specific dosing and duration of treatments in the discussions and tables in chapter 3, along with the expansion in the drugs used to treat immune-based diseases in chapter 4, which were added to the treatment options of each disease and organized in table form for the reader.

Finally, we have added an appendix providing short biographies of the men and women attached to the disease names we have all come to know (eg, Ewing Sarcoma), cell names (eg, Antoni), and syndrome names (eg, Frey and Ramsay Hunt) so that the reader may appreciate their lives and contributions as well as their passion and dedication, which is mirrored in the present and is the hope for the future that this second edition embraces.

What is the Future

In our lifetime, we two authors have witnessed the extinction or near extinction of several diseases, such as smallpox, polio, diphtheria, leprosy, scarlet fever, which too often devastated and debilitated generations before us. However, these diseases have been replaced by the likes of HIV/AIDS, osteoradionecrosis, bisphosphonate-induced osteonecrosis, and multidrug-resistant tuberculosis, among others, which devastate and debilitate the current generations we treat today. Similar to Plato's remark made centuries ago that "only the dead have seen the last of war," today we can say that only the dead have seen the last of diseases. This should remind the reader that all diseases are dynamic and ever changing. It is therefore the challenge of this generation of health care providers to keep pace and not fall behind.

As it was in the first edition, it is the authors' sincere hope and intent that this second edition provides a reliable resource for each practitioner to keep up that pace so as to better treat and serve the people who entrust their care to our two specialties and in the end save a few lives and improve the lives of many more.

Preface to the First Edition

This book is intended to be a clinically oriented and forward-looking guide for oral and maxillofacial surgeons and other advanced dental and medical specialists who deal with pathologies in the oral cavity, midface, and neck. It focuses on the mechanism of each disease and how that dictates its clinical and radiographic presentation as well as the serious considerations on a sample differential diagnosis. It then progresses to specific treatment recommendations that the authors use or have researched as the most beneficial. Treatments avoid such vague phrases as "a wide local excision" and instead provide specific margins and anatomically based techniques. Generic medication protocols also are avoided for those conditions not treated with surgery; instead, specific drugs, doses, routes of administration, length of treatment, and alternative treatments are described in the context of how each works to affect the natural course of the disease. Discussion of each disease or condition concludes with the prognosis after treatment.

This book challenges some of the established concepts and dogmas currently prevailing in oral and maxillofacial pathology and surgery. It also is likely to challenge the reader's acceptance of dental and medical school teaching, which too often consists of a rushed and superficial presentation of these pathologies. It is the authors' hope that the evidence and rationales presented in this text are convincing of this change and of this approach to learning. This book is also specifically intended to simplify and streamline terminology. The reader will note numerous terminology changes from the past—changes that generally use only one name to describe and identify the specific underlying cause of each condition. This is reinforced in the last chapter of the book, "Where Have All the Great Terms Gone?," a concise review and explanation of why the original name for some diseases is inappropriate today.

Readers may use this text as a cover-to-cover course in clinical and histopathologic oral and maxillofacial pathology; as a reference text on a chapter-by-chapter basis to review the specifics of each disease category; or as a case reference to refresh their knowledge about a specific disease or the specific presentation of a new patient. In any case, it is the fond hope of both authors that clinicians will increase their knowledge and ability to care for their patients, who in turn will receive more accurate diagnoses and better treatment.

No book is created by its authors alone. The numerous individuals who have referred patients and biopsy material to our practices must be acknowledged, although their numbers preclude individual mention. These include our present and past hard-working and scientifically curious residents in oral and maxillofacial surgery whose "great cases" appear in the book. A specific and certainly sentimental acknowledgment must be given to our mentors: Stuart N. Kline and Robert P. Johnson, the mentors of Robert Marx; and Lester R. Cahn and Leon Eisenbud, the mentors of Diane Stern. Their teaching and self-sacrifice have not only advanced our knowledge but motivated us throughout our careers and personal lives. We have acknowledged the source of all illustrations that are not our own. It is possible that with the passage of time there may have been some whose source we no longer recall. If this is the case, we humbly apologize.

Photomicrographs require the work of several individuals. There is the histology technician who prepares the microscopic slide; many of these are the work of Maureen Frazel, ART. Others were prepared by the late Sarah Spector. Then there is the photographer who captures the microscopic image on film; the majority of these have been the work of Leroy Ivey of the Veterans Administration Medical Center, Miami. The drawings were executed by Hans Neuhart. Maria Ruiz typed the manuscript with her characteristic efficiency and good humor, to the benefit of everyone involved. To these skilled, dedicated, and patient individuals, our sincere thanks. Thanks also to Carlos Valdes, MD, whose generosity in sharing his dermatopathology material is greatly appreciated. We are most grateful to our publisher, represented by Tomoko Tsuchiya, who made the creation of this book as smooth and pleasant as is humanly possible; Susan Robinson, who handled the physical production of the book with true professionalism and grace; and Lisa Bywaters, our editor, whose infinite patience, knowledge, and understanding guided us to the final result. We are indeed fortunate to have been able to work with her.

Our families have sustained us with their love, support, and encouragement despite the considerable infringement that this book has made on their time.

Finally, without the patients we have treated in the past, this book could not have been written. We hope that it will ultimately benefit those who are patients today and in the future.

CHAPTER 2

Inflammatory and Reactive Diseases of the Oral and Maxillofacial Region

"The desire to take medicine is perhaps the greatest feature which distinguishes man from animals."
–Sir William Osler

Inflammation and Repair

Inflammation and repair are fundamental processes that pervade the spectrum of pathology. They represent a continuum of constant change, so that any given microscopic view represents only a "freeze-frame" in the evolution of any inflammatory lesion.

Inflammation is the reaction of vascular tissue to injury, be it physical, chemical, bacterial, or immunologic in nature. The result is a dilation of capillaries (Fig 2-1a), swelling of endothelium, margination of polymorphonuclear leukocytes (Fig 2-1b), and subsequent migration of neutrophils followed by plasma through the vessel wall (ie, a vascular change followed by exudation). This is characteristic of the acute phase of inflammation. The neutrophils remove and digest the inciting agent. Repair may follow with the formation of granulation tissue, which is the result of ingrowth by capillaries and fibroblasts (Fig 2-1c).

The inflammatory process may resolve completely with no adverse effect on the host. Alternatively, there may be a "walling off" with focal destruction of tissue secondary to the action of the neutrophils. This may result in abscess and/or scar formation (Fig 2-1d). If there is failure to eliminate the injurious agent, the more prolonged state of chronic inflammation occurs, which typically results in fibrosis and scarring (Fig 2-1e). Although inflammation and repair are sequential in acute inflammation, in chronic inflammation they are simultaneous. The short-lived neutrophils are reinforced and eventually replaced by the much hardier macrophages with the addition of lymphocytes and plasma cells, and the inflammatory process hence acquires an immunologic component.

In some specific cases, the inciting agent persists; it may be sequestered within macrophages, where, if it exists in particulate form or in high concentrations, it can stimulate the formation of a granuloma in which the "wandering" macrophage becomes immobile and mature (Fig 2-2a). If it induces a strong delayed hypersensitivity response, the macrophages may develop into so-called epithelioid cells (Fig 2-2b). These cells are not really epithelial cells but mature macrophages from the blood monocyte lineage that vaguely take on an appearance resembling an epithelial cell. There may also be cytoplasmic fusion of macrophages with the resultant formation of multinucleated giant cells. Granulomas may show necrosis if the inciting agent is highly toxic or if it induces a marked delayed hypersensitivity reaction through the action of T8 cells. There may also be a superimposed infiltration of neutrophils, eosinophils, and/or lymphocytes. Once destruction of the injurious agent has occurred, the macrophage dies, disperses, or reverts to a less mature form. The granuloma then resolves to form fibrotic (scar) tissue.

Many factors influence the specific inflammatory picture. Trauma-induced inflammation tends to have a sparser cellular component than that caused by microorganisms. The type of agent may alter the nature of the cellular component, as for example the marked eosinophilic response that is seen secondary to parasites.

Inflammation itself also acts as a stimulus to repair. Platelets begin the repair process by secreting platelet-derived growth factors (PDGFaa, PDGFab, PDGFbb), which induce cellular proliferation and capillary angiogenesis, as well as stromal-derived activation factor 1α (SDAF-1α), which is a signaling device to chemoattract stem cells, and several transforming growth factor–βs (TGF-βs), which promote not only cellular proliferation and angiogenesis but connective tissue differentiation. They also secrete vascular endothelial growth factor (VEGF), which promotes capillary angiogenesis, and epithelial growth factor (EGF), which promotes epithelial proliferation to cover an open wound. Within the area of

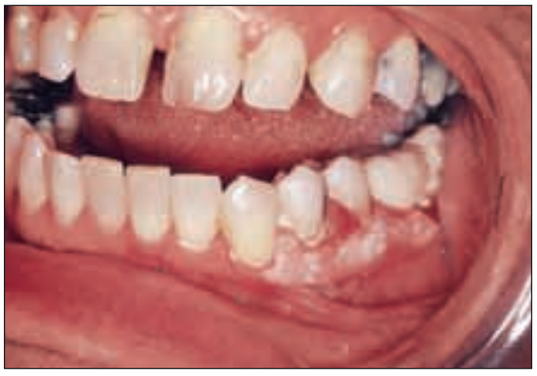

Fig 8-66 Gingival carcinoma is slow growing and limited to the attached gingiva. It is nevertheless a treacherous carcinoma because of its diffuse extension along the attached gingiva, its inclusion of both the buccal and lingual gingiva, and its potential to invade the crestal bone through the abundant local lymphatic and blood vessels.

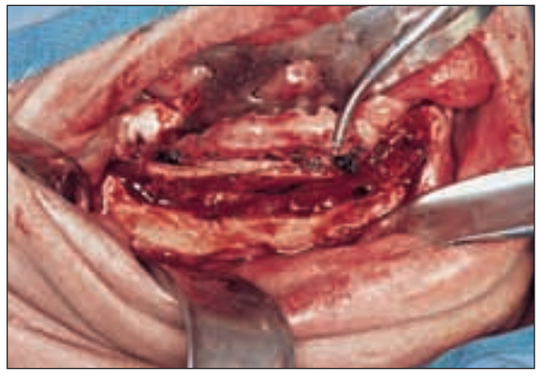

Fig 8-67a Excisions of gingival carcinoma must include the attached gingiva and alveolar bone to at least the mid-root level.

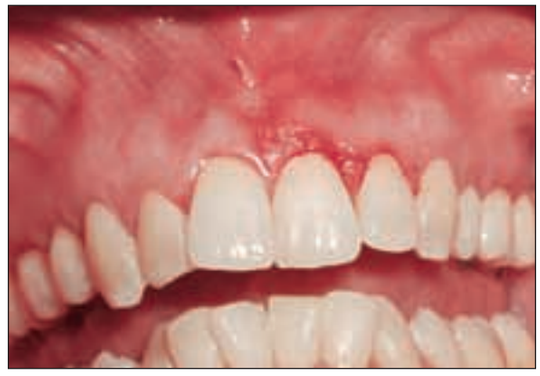

Fig 8-67b Gingival carcinoma of the anterior maxilla.

Squamous Cell Carcinoma of the Gingiva

Clinical presentation

Gingival carcinoma (Fig 8-66) is an indolent, slow-growing carcinoma that has a low propensity to metastasize. Accounting for a little less than 10% of all intraoral carcinomas, it will present as a red-white thickening of the marginal and interdental gingiva. It will often have a history of several years and is frequently confused with various periodontal conditions. This carcinoma may not be very infiltrative, but its surface spread may be extensive. Frequently, the entire gingiva on each side of the midline is involved, but it includes only the attached gingiva.

Differential diagnosis

The most common condition from which to differentiate gingival carcinoma is chronic persistent periodontitis. In addition, lichen planus of the attached gingiva and the pemphigoid forms that involve the attached gingiva may each mimic gingival carcinoma by their red-white appearance and scar formation. Because gingival carcinoma usually is not ulcerated but appears as a thickened red-white gingiva, verrucous carcinoma and the benign hyperkeratosis form of clinical leukoplakia are also distinct considerations.

Diagnostic work-up

An oral and head and neck examination is required, including a mirror or fiberoptic examination of the laryngopharynx followed by a TNM staging and an incisional biopsy. It is important to biopsy such lesions to include the periosteum. Because of the thick epithelium and the thinness of the connective tissue in this area, biopsies that do not include the periosteum will be difficult to assess for the presence and depth of invasive carcinoma. Periapical radiographs and a panoramic radiograph are also required to assess areas of possible bony invasion.

Histopathology

Carcinomas of the gingiva are typically well differentiated; however, distinguishing a hyperplastic response from a carcinoma may be particularly difficult at this site unless a sufficiently deep biopsy is taken. This is primarily due to the lack of marked cellular atypia and the thinness of the tissue, which makes evidence of infiltration difficult to appreciate.

Treatment

Gingival carcinoma is treated with a specific type of surgical excision. The surgeon must resist the temptation to perform only a soft tissue gingivectomy. While the carcinoma rarely extends beyond the attached gingiva, the abundant lymphatics and vasculature of the alveolar bone–gingival complex necessitate an excision of soft tissue including the alveolar bone (Fig 8-67a). The excision must include both buccal and lingual gingiva. The bone is resected to the level of the root apices superior to the mandibular canal in the posterior mandible and short of the sinus floor or nasal floor in the maxilla (Figs 8-67b to 8-67e). Frozen sections are extremely important because of the diffuse spread along the marginal gingiva. In fact, even if clear margins are established, biopsy of the gingiva posterior to the clear margins is recommended because of satellite lesions and "skip" areas of uninvolved gingiva with this cancer. Because of the low propensity of gingival carcinoma to undergo local lymph node metastasis, a prophylactic neck dissection is not recommended.

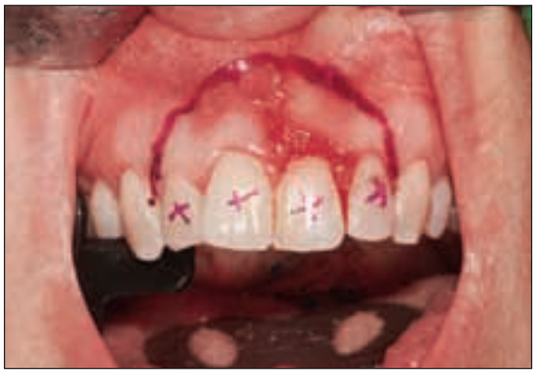

Fig 8-67c Outline of alveolar resection for gingival carcinoma of the anterior maxilla.

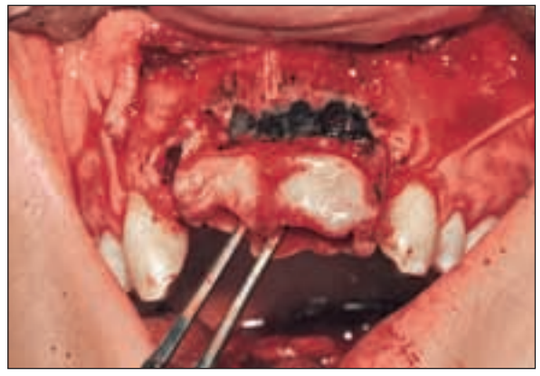

Fig 8-67d Subnasal alveolar resection inclusive of labial and palatal gingiva.

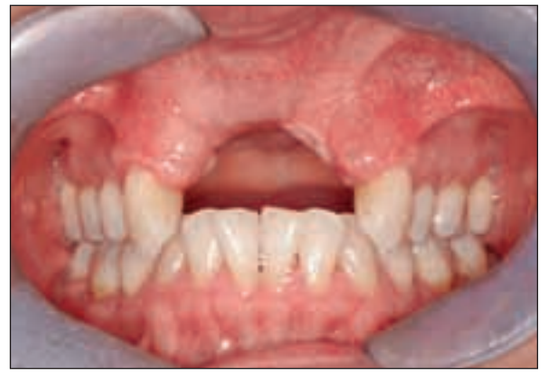

Fig 8-67e Resultant defect from subnasal alveolar resection.

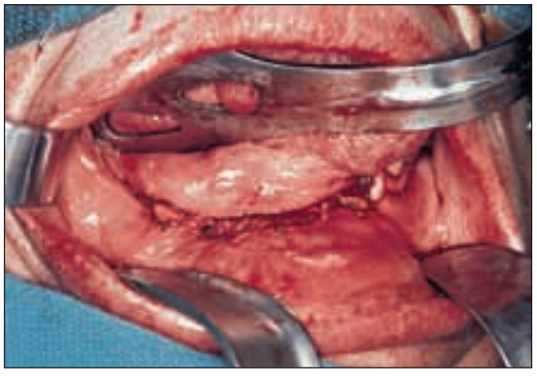

Fig 8-67f Excision of the attached gingiva will cause an obliteration of the buccal and lingual vestibules upon closure. A vestibuloplasty 3 to 4 months later is usually required.

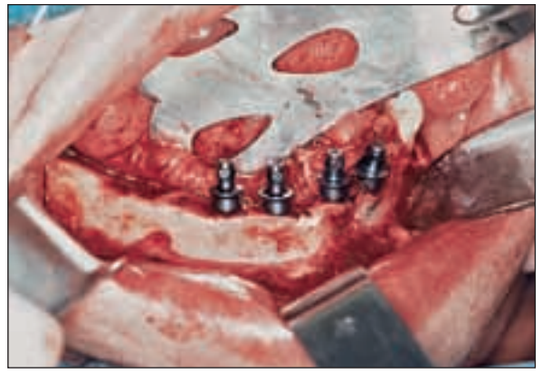

Fig 8-67g Because excision of gingival carcinoma includes teeth and some alveolar bone, immediate placement of dental implants is a feasible option that results in early dental rehabilitation. Dental implants may be placed whether or not postoperative radiotherapy is planned provided its onset is at least 6 weeks after surgery.

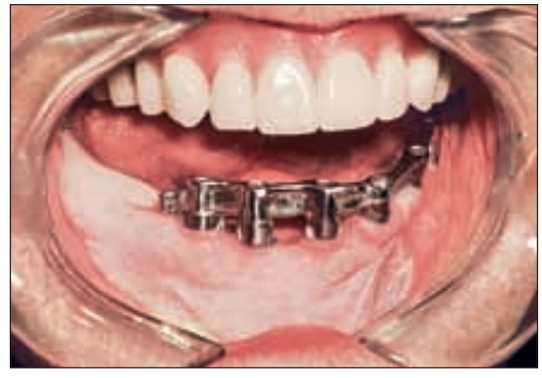

Fig 8-67h Fully osseointegrated dental implants and a healed split-thickness skin graft create an ideal situation for a functional and retentive prosthesis.

Prognosis and follow-up

The excision of bone and soft tissues will obliterate the vestibule for future prosthetic use (Fig 8-67f). Most patients require a secondary vestibuloplasty for prosthetic rehabilitation. The remaining bone is also sufficient in health and height for osseointegrated implants, which can be placed at the time of resection (Figs 8-67g and 8-67h).

The prognosis associated with gingival carcinoma is excellent compared to other areas where intraoral carcinoma may occur. Adjusted 5-year survival rates are between 85% and 90%.

Follow-up is the same as that for the other locations of intraoral carcinoma because its purpose is to detect not only recurrence from disease re-emergence but also new primary tumors.

Squamous Cell Carcinoma of the Alveolar Ridge

Clinical presentation

Carcinoma of the alveolar ridge may be confused with gingival carcinoma because their anatomical locations are similar (Fig 8-68). The major difference is that alveolar ridge carcinoma is not limited to the attached gingiva. It will enter into either the buccal or lingual vestibule (Fig 8-69), the floor of the mouth (Fig 8-70) if the tumor is on the mandibular alveolar ridge, or the palate if the tumor is on the maxillary alveolar ridge (Figs 8-71a and 8-71b). This distinction is important because alveolar ridge carcinoma is

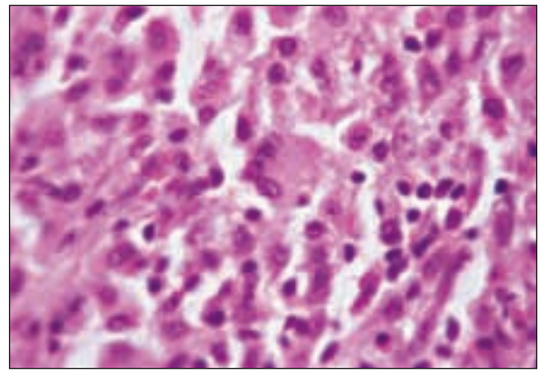

Fig 10-11a A juvenile xanthogranuloma with giant cells and inflammatory cells, including eosinophils.

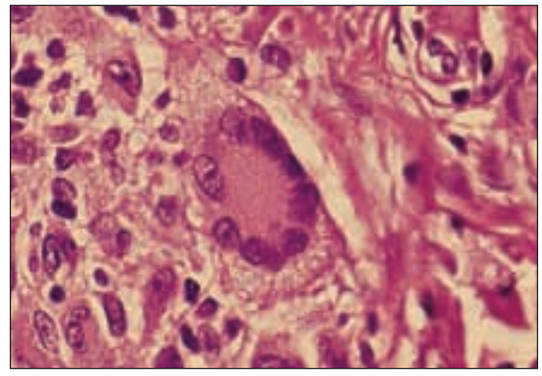

Fig 10-11b Touton giant cell with nuclei in a wreath-like arrangement, beyond which is a lipid-containing foamy cytoplasm.

cause multiple skin lesions that are flat but may be slightly raised because of a collection of lipid-laden histiocytes.

One infectious disease worthy of including on a differential list is molluscum contagiosum, a viral disease known to produce 2- to 4-mm self-regressing skin lesions and few systemic toxic symptoms. However, as these mature lesions regress, they form a white waxy plug, which represents virally altered epithelial cells.

Diagnostic work-up

Excision of a representative lesion will confirm the diagnosis, although obvious clinical lesions can be diagnosed without biopsy.

Histopathology

The lesions are usually defined but unencapsulated masses composed of histiocytes with some scattered lymphocytes and eosinophils (Fig 10-11a). Over time, the histiocytes become foamy because of the presence of lipid, and Touton giant cells are seen. These are characterized by a peripheral wreath-like arrangement of nuclei surrounded by a rim of foamy cytoplasm (Fig 10-11b). Subsequently, interstitial fibrosis takes place, such that the lesion may resemble a fibrous histiocytoma.

Treatment

No specific treatment is required except parental reassurance of the anticipated regression of even the larger, deep-seated lesions. Information about residual scarring and some hyperpigmentation is advised. Follow-up facial skin resurfacing procedures may be necessary.

Prognosis

Once lesions regress, they do not recur. Regression occurs over several months to a year for each lesion.

Solitary Fibrous Tumor

Clinical presentation and pathogenesis

This newly defined tumor was part of the spectrum of hemangiopericytomas and is now recognized as a separate neoplasm. Approximately 80% are benign, but 20% are malignant. Most cases (80%) arise from the lung pleura, with the remaining 20% occurring in various other sites. In the head and neck area, the most common site is the orbital region.

Like hemangiopericytomas, the mass will be deeply seated, usually within fascia or muscle. Therefore, the mass may be only slightly mobile or seem fixed. It is painless and, on rare occasions, may be pulsatile.

Differential diagnosis

A deep-seated mass in the infraorbital area is suggestive of mesenchymal malignancies such as a fibrosarcoma, malignant fibrous histiocytoma, and rhabdomyosarcoma. Additionally, an aggressive fibromatosis, nodular fasciitis, or a rare benign fibrous histiocytoma or true hemangiopericytoma may be considered.

Diagnostic work-up

An incisional biopsy, or an excisional biopsy if feasible, is the best diagnostic approach. A computed tomography (CT) or magnetic resonance imaging (MRI) scan may be needed to determine the extent of the mass and its relationship to adjacent structures. However, only histopathology with immuno-

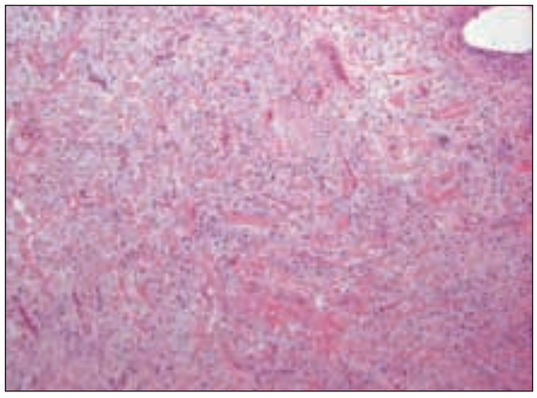

Fig 10-12a Low power shows a patternless proliferation of cells with scattered areas of hyalinization. Slit-like blood vessels can be seen.

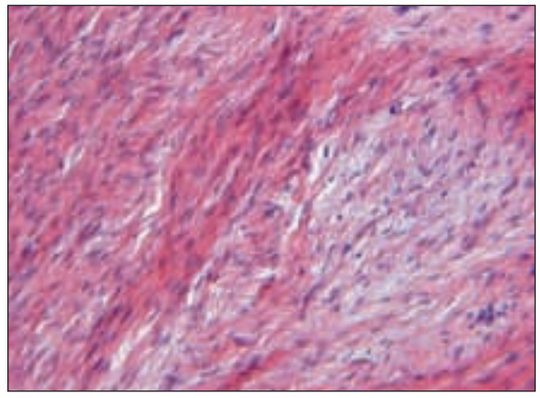

Fig 10-12b This high-power view shows an area of hypercellularity and adjacent hypocellularity.

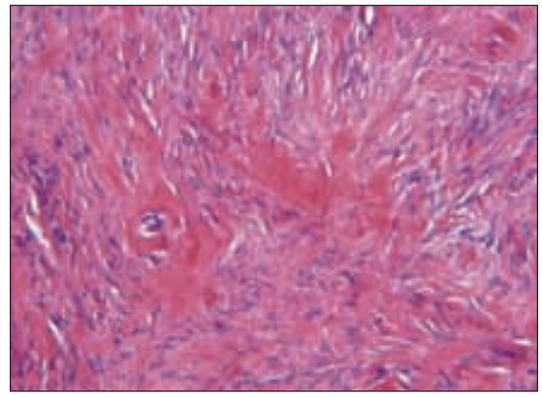

Fig 10-12c Typical hyalinized collagen is present.

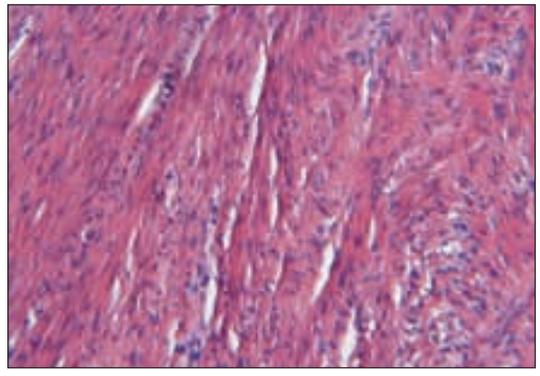

Fig 10-12d Artifactual separation between collagen fibrils and/or cellular elements is often present.

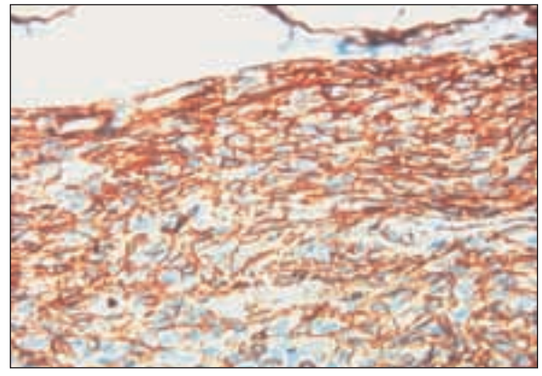

Fig 10-12e Solitary fibrous tumor staining strongly positive for CD34.

Histopathology

histochemistry studies to determine cell membrane markers such as CD34 and CD99 can make the diagnosis.

Typically, the solitary fibrous tumor is well circumscribed and may have a partial capsule. It is composed mainly of spindle-shaped cells with vesicular nuclei that are arranged either in short fascicles or randomly (Fig 10-12a). There are areas of hypercellularity and hypocellularity (Fig 10-12b). An important and striking feature is hyalinized collagen (Fig 10-12c). Artifactual separation may occur between the cellular element and the collagenized tissue or between the collagen fibers (Fig 10-12d). The vascular component may form narrow slits or branching staghorn hemangiopericytoma-like vessels (see Fig 10-12a). Myxoid areas may be present, and mast cells are common. Little or no mitotic activity is seen in the typical case. Characteristically, these tumors express CD34 (Fig 10-12e), and the majority will also express CD99.

The solitary fibrous tumor is usually benign, but some are aggressive. The aggressive tumors usually are hypercellular, infiltrative, and likely to show marked cellular atypia, and they have increased mitotic activity (>4 mitoses per 10 high-power fields [HPFs]). Necrosis and hemorrhage also may be seen in these cases.

Treatment and prognosis

The truly benign solitary fibrous tumor may be excised for cure with 5-mm margins. The rarer malignant counterpart requires a resection with 1.0- to 1.5-cm margins. Even then, the 2-year survival rate is only 50%, requiring surveillance examinations every 4 months for this malignant counterpart or any of the benign lesions in which the biologic behavior is uncertain.

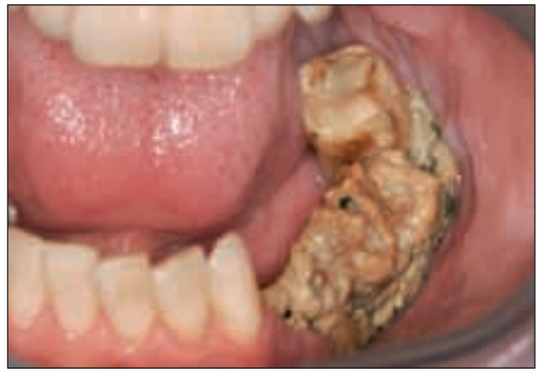

Fig 17-40 Stage I BIONJ by virtue of one quadrant of exposed bone.

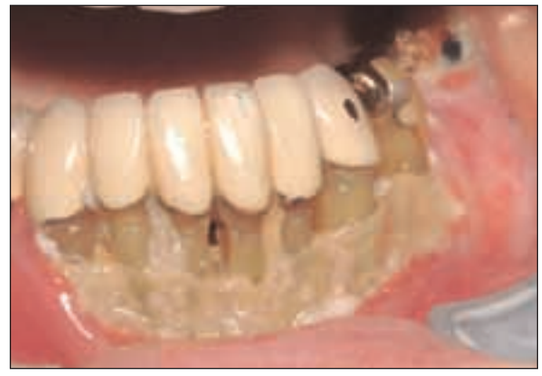

Fig 17-41 Stage II BIONJ by virtue of two quadrants of exposed bone.

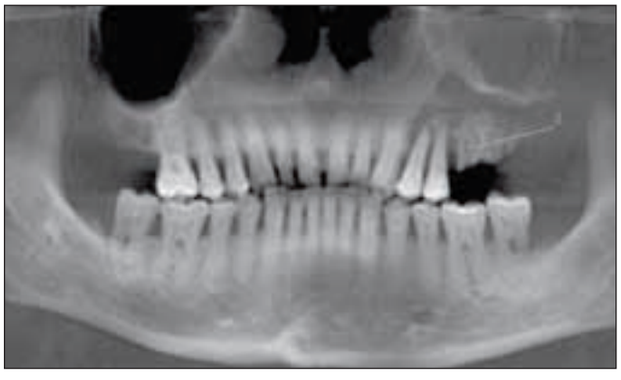

Fig 17-42 Stage III BIONJ by virtue of exposed bone extending into the maxillary sinus.

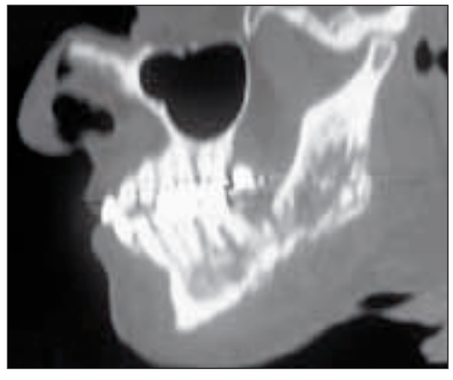

Fig 17-43 Stage III BIONJ by virtue of exposed bone with osteolysis to the inferior border of the mandible.

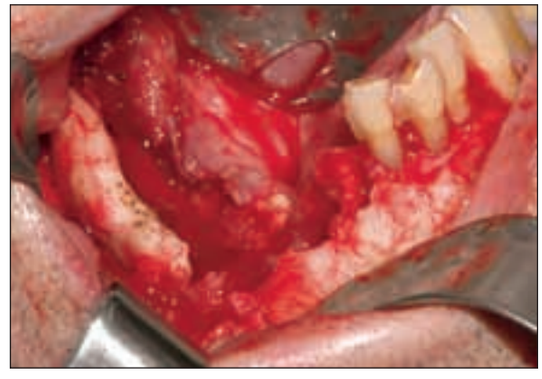

Fig 17-44 Stage III BIONJ by virtue of a pathologic fracture (see also Figs 17-39b and 17-39c).

- Stage I: Exposed bone in one quadrant (Fig 17-40)
- Stage II: Exposed bone in two quadrants (Fig 17-41)
- Stage III: Exposed bone in three or more quadrants or exposed bone extending into the maxillary sinus (Fig 17-42) or exposed bone with osteolysis to the inferior border (Fig 17-43) or exposed bone with a pathologic fracture (Fig 17-44)

Treatment and prognosis

Intravenous BIONJ

Intravenous BIONJ is most often nonresolvable by office-based debridements, and these procedures may even worsen the condition. Although extensive resections of the mandible with titanium plate reconstructions and possible flap surgery can resolve most cases of intravenous BIONJ, it is only necessary to resort to such aggressive surgery in the Stage III presentations (Figs 17-45a to 17-45h) or in those patients whose condition is repeatedly refractory to control by nonsurgical means. For intravenous BIONJ in the maxillary sinus, a submucosal posterior maxillary resection with a buccal fat pad advancement can be curative (Figs 17-46a to 17-46e). Because the exposed bone is likely to be permanent and the patient's metastatic cancer treatments take priority, the goal is control and management of the Stage I and Stage II patients to relieve pain and prevent extension of the exposed bone. For the patient who presents with exposed bone but no pain, maintenance therapy with 0.12% chlorhexidine, oral swish and spit three times daily, is recommended. If pain is present, penicillin VK 500 mg four times daily is added to the chlorhexidine regimen. Because penicillin VK is nontoxic and unassociated with a significant incidence of side effects with long-term use, it can be used as ongoing

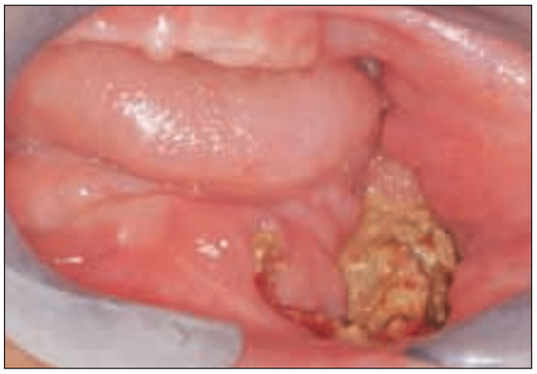

Fig 17-45a Stage III BIONJ with secondary infection and a cutaneous fistula in a cancer patient who received Zometa.

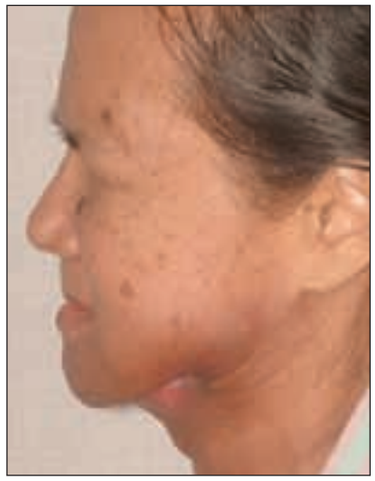

Fig 17-45b Soft tissue loss seen as secondary infection has caused soft tissue lysis, erythema, and exposed necrotic bone through the skin.

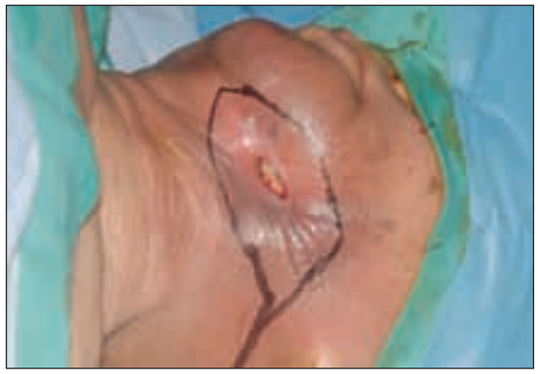

Fig 17-45c Excision of affected skin and flap surgery to replace the soft tissue was necessary in this advanced case.

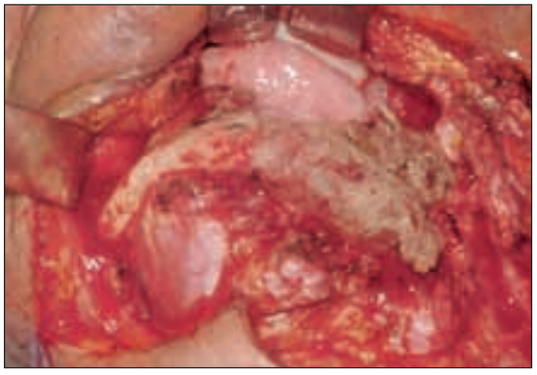

Fig 17-45d Extensive necrotic bone with a line of demarcation with unaffected bone.

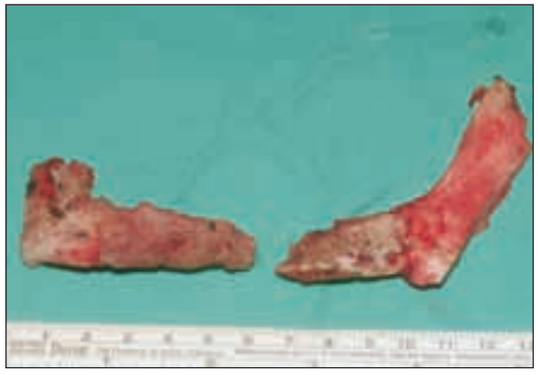

Fig 17-45e Resection specimen identifies the necrotic bone, a pathologic fracture, and margins of unaffected bone.

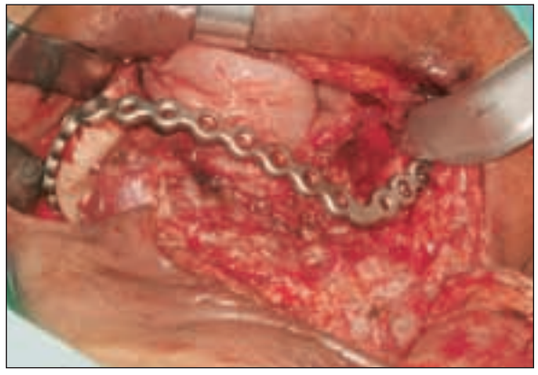

Fig 17-45f Titanium plate reconstruction of the defect.

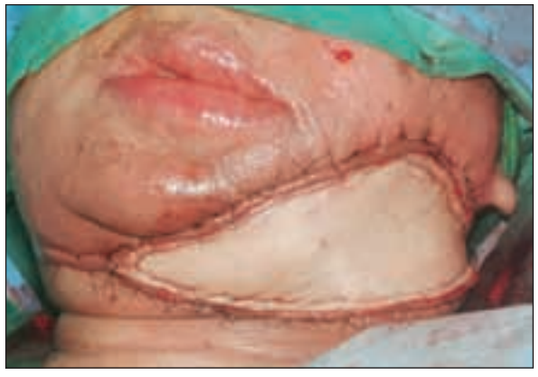

Fig 17-45g A large trapezius myocutaneous flap was necessary to replace the affected soft tissue component.

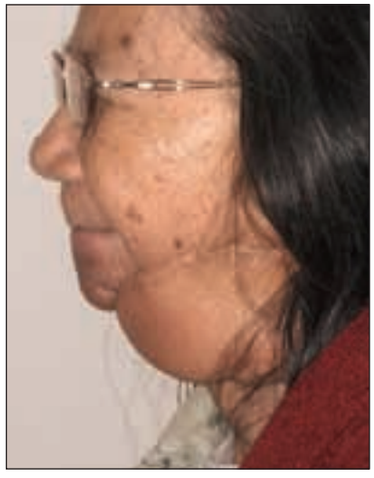

Fig 17-45h Healed flap and resolution of Stage III BIONJ.

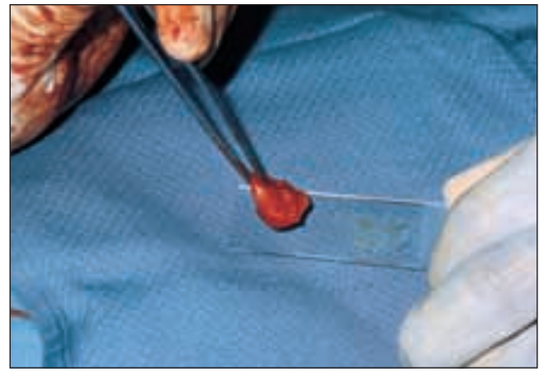

Fig 20-6d A "touch prep" can be accomplished by sectioning the lymph node and imprinting the cut edge on a clean, dry glass slide.

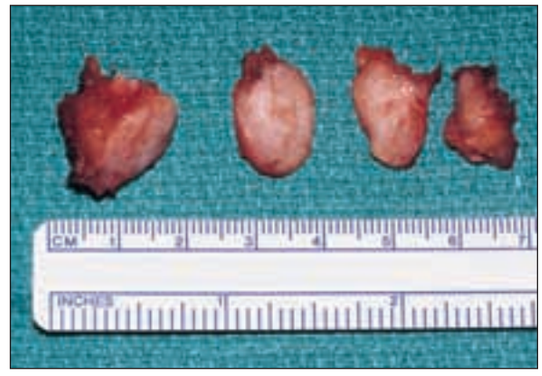

Fig 20-6e It is best to slice lymph nodes into 2-mm-thick sections. One section should be subdivided and sent for various cultures and the others placed into the fixative or fixatives preferred by the pathologist.

Specimens removed and submitted in this manner will allow for a more rapid and unequivocal diagnosis and, therefore, early treatment. The need to rebiopsy lymph nodes can be virtually eliminated. Because the newer classifications of lymphoma rely heavily on cytologic detail, nuclear shape, and cell membrane markers, fresh or well-preserved representative tissue specimens are especially critical.

Once the diagnosis of a lymphoma is made, it is best to refer the patient to a medical oncologist/hematologist, who will develop and organize the staging work-up and treatment course. It is important that the oral and maxillofacial specialist continue to follow the patient through the treatment course and the follow-up period to offer a trained eye for potential treatment failures or new disease foci and to manage some of the treatment complications.

Hodgkin Lymphomas

Classification

Until recently, the classification for HL had not changed dramatically since the Rye classification of 1966, which in turn was based on the Lukes-Butler classification. The Reed-Sternberg cell and its variants are considered to be the malignant component of HL. They are of lymphoid origin and almost always B cell, although rarely they are of T-cell origin. The majority of the cell population represents a reactive proliferation of immune and inflammatory cells rather than malignant cells, and in fact the classification depends essentially on the nature of the reactive component. However, the REAL and World Health Organization (WHO) classifications have instituted a significant change. These classifications separate NLPHL from the other types, which are defined as classic Hodgkin lymphoma (cHL). NLPHL is clinically, morphologically, and immunophenotypically distinct from the cHLs and has a far better prognosis.

Histopathology common to all types of Hodgkin lymphoma

The diagnosis of HL depends on the identification of characteristic neoplastic cells (Reed-Sternberg cell and its variants) against the proper cellular background. It is now recognized that the Reed-Sternberg cell is indeed a lymphoid cell. It is almost always of preaptotic germinal center B-cell origin, but it loses its B-cell identity, thus delaying the identification of its histogenesis. Rarely, the cell may originate from T cells. The unique nature of HL is the paucity of neoplastic cells that are found against an inflammatory, reactive background. This background is of considerable importance: Not only is its nature pivotal to the proper classification within HL, but also, because Reed-Sternberg–like cells may be seen in reactive lymphoid proliferations other than HL (eg, infectious mononucleosis), it becomes essential for the basic diagnosis. It is also apparent that the specific background and the morphology of the neoplastic cells are interrelated. In evaluating HL on morphologic grounds, there are three elements that must be considered: the neoplastic cells (Reed-Sternberg cell and its variants), the reactive inflammatory cells, and the stromal component.

The classic Reed-Sternberg cell is a giant cell (about 60 to 80 μm; a normal lymphocyte is 12 to 16 μm) with a bilobed or multilobed nucleus that has a large eosinophilic nucleolus surrounded by a clear

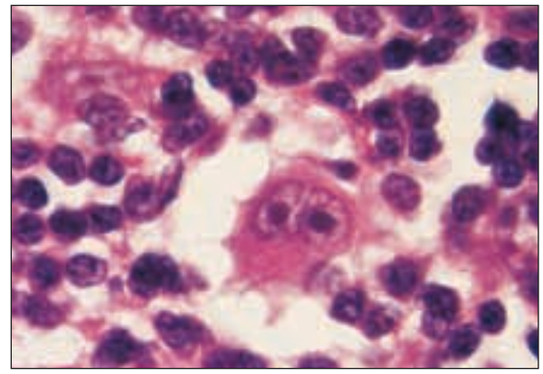

Fig 20-7a Classic Reed-Sternberg cell with a bilobed nucleus and very large nucleoli.

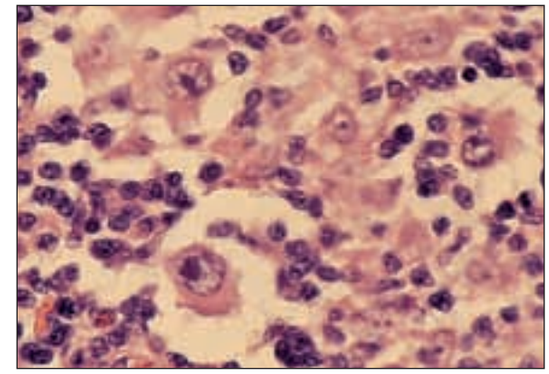

Fig 20-7b A Hodgkin cell has a single nucleus and a large nucleolus.

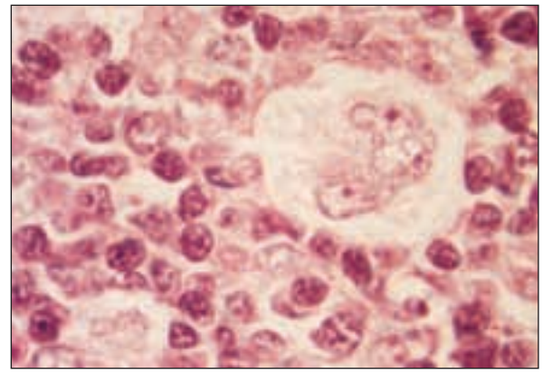

Fig 20-7c Lacunar cell with a multilobed nucleus and relatively small nucleoli. The cell appears to be floating in space.

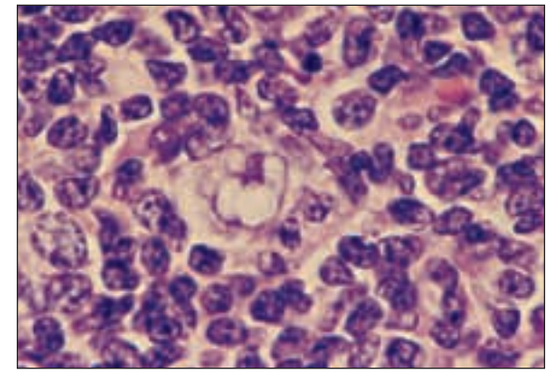

Fig 20-7d L&H cells resembling popcorn, thus giving rise to the term *popcorn cells*.

zone and a prominent nuclear membrane. It has a mirror-image appearance and is often said to resemble the eyes of an owl (Fig 20-7a). The cytoplasm is eosinophilic. It is this type of cell that has been required for the initial diagnosis of HL. However, more recently this has been questioned, particularly in cases of NLPHL. The neoplastic variants include the following:

- Hodgkin cells, which are mononuclear but otherwise resemble the classic Reed-Sternberg cell with its large nucleolus (Fig 20-7b).
- Lacunar cells, which are multilobed with abundant cytoplasm and nucleoli that are far less conspicuous than those in the classic Reed-Sternberg cell. They appear to lie in spaces or "lacunae," which are actually the consequence of formalin fixation. Their appearance can be variable, depending on the background (Fig 20-7c).
- Lymphocytic and histiocytic (L&H) cells, which have multilobed, convoluted nuclei; small, peripherally placed nucleoli; and no perinucleolar halos. They are called *popcorn cells* because of this appearance (Fig 20-7d).

While the Reed-Sternberg cell and the Hodgkin cell appear to be fully neoplastic, the lacunar and L&H cells may actually represent transformed or aberrant cells that have the potential to transform into neoplastic Reed-Sternberg cells. The variable quantities of these cells in the different types of HL correlate with the aggressiveness of the tumor.

The background component includes lymphocytes, which are usually small with nuclei that are round and regular. The majority are T cells. In addition, there may be histiocytes, which can include epithelioid histiocytes, eosinophils, neutrophils, and plasma cells, along with fibroblasts. Acellular col-

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