

chapter 10

LYMPHORETICULAR AND HEMATOPOIETIC DISEASES

HEMOPHILIA

Fig. 10.1

Hemophilia refers to a group of bleeding disorders that are caused by a hereditary deficiency of one of the clotting factors in the blood. The best known of these diseases are hemophilia A (classic hemophilia) and hemophilia B (Christmas disease), which are X-linked recessive disorders caused by a deficiency of Factor VIII and Factor IX, respectively. Because these conditions are X-linked recessive, females are typically only carriers of the disorders, which are then expressed in affected males in the family. Hemophiliacs are prone to severe, uncontrollable blood loss from any type of laceration or trauma, including surgical excisions, simple extractions, and periodontal scaling. Therefore, consultation with the patient's physician is mandatory before undergoing any dental or oral surgical procedure that may induce bleeding. If necessary, the missing clotting factor can be administered to the patient, plus epsilon-aminocaproic acid (EACA, an antifibrinolytic agent) can be used. Unfortunately, many hemophiliacs became infected with the human immunodeficiency virus when they received transfusions of concentrated clotting factors in the early 1980s. However, this risk has been eliminated with today's techniques, including purified factor concentrates and recombinant DNA technology.

THROMBOCYTOPENIA

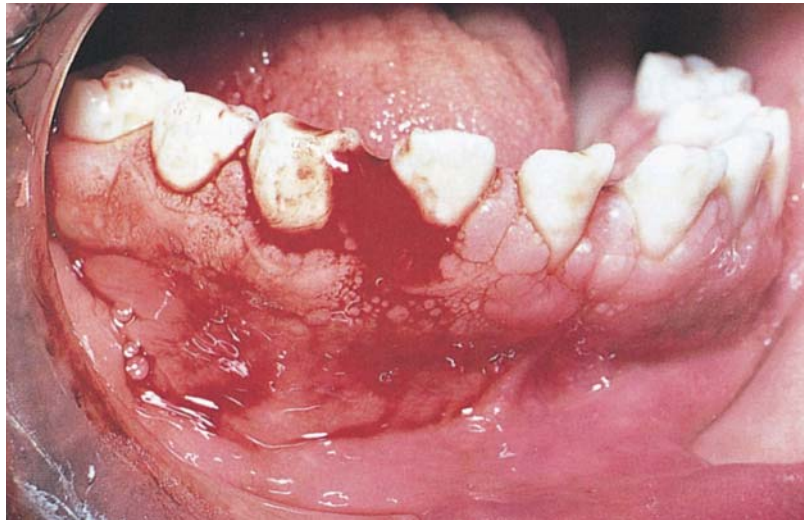
Figs. 10.2 and 10.3

Thrombocytopenia, or platelet deficiency, is a hemorrhagic disorder that may occur secondarily in a wide variety of conditions, or it may appear as a primary disease, probably of autoimmune origin, called "idiopathic thrombocytopenic purpura" (ITP). Acute ITP most commonly occurs in children and frequently follows an acute viral illness. It is usually a self-limiting disease of several days' to several months' duration, with full recovery and no further problems. On the other hand, chronic ITP is more common in adults, often exhibiting multiple remissions and exacerbations. Secondary causes of thrombocytopenia include marrow-suppressive drug therapy, abnormal drug reactions, marrow infiltration by tumor cells, systemic lupus erythematosus, and various infections.

Clinically, petechial and ecchymotic skin lesions may occur, as may epistaxis, hematuria, gastrointestinal tract bleeding, and intracranial hemorrhage. Oral manifestations include gingival bleeding, submucosal hemorrhage, and excessive bleeding following tooth extraction or surgery. Treatment for thrombocytopenia may include systemic corticosteroid therapy, platelet transfusions, and splenectomy. Control of secondary thrombocytopenia also must include management of the underlying cause. In one rare form of platelet deficiency known as "thrombotic thrombocytopenic purpura," gingival biopsy has been used as a diagnostic aid to demonstrate the presence of intravascular platelet thrombi.

► **Figure 10.1**
HEMOPHILIA

Persistent gingival bleeding following dental prophylaxis in a patient with hemophilia B. The patient also has Dilantin hyperplasia of the gingiva.



► **Figure 10.2**
IDIOPATHIC THROMBOCYTOPENIC PURPURA

Gingival hemorrhage.



► **Figure 10.3**
IDIOPATHIC THROMBOCYTOPENIC PURPURA

Palatal ecchymosis.

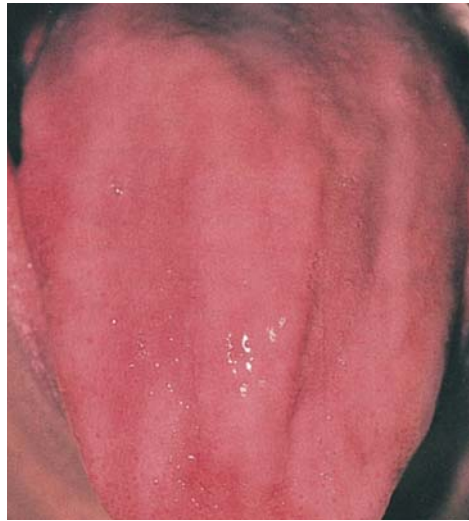


Figs. 10.4, 10.5, and 10.6

Megaloblastic anemia is characterized by a reduction in the number of red blood cells combined with abnormal enlargement of proliferating marrow cells. Most megaloblastic anemias arise from a deficiency of vitamin B12 (cobalamin) or folic acid. In pernicious anemia, vitamin B12 deficiency is caused by autoimmune-mediated atrophy of the gastric mucosa, with failure to produce intrinsic factor necessary for vitamin B12 absorption. Other causes of vitamin B12 and folic acid deficiency include improper diet, impaired intestinal absorption, gastric or intestinal resection, and gastric bypass or stapling procedures for obesity.

Clinical manifestations of megaloblastic anemia include pallor, weakness, fatigue, shortness of breath, headache, palpitations, and syncope. Neurologic symptoms may occur, such as paresthesia of the extremities, unsteadiness of gait, and loss of vibratory and positional sense. Oral features include atrophy and burning of the tongue, with loss of papillae sometimes imparting a “beefy red” appearance. Atrophic, erythematous lesions also may be seen on other oral mucosal surfaces. Treatment usually consists of appropriate vitamin supplementation; in vitamin B12 deficiency, parenteral administration is necessary in most cases.

► **Figure 10.4**
PERNICIOUS ANEMIA
Erythema and atrophy of the dorsal tongue.



► **Figure 10.5**
MEGALOBLASTIC ANEMIA
Burning macular, erythematous mucosal lesions in a patient with vitamin B12 deficiency secondary to gastric bypass for obesity. (Courtesy of Drummond JF, White DK, Damm DD. Megaloblastic anemia with oral lesions: a consequence of gastric bypass surgery. *Oral Surg Oral Med Oral Pathol* 1985;59:149.)



► **Figure 10.6**
MEGALOBLASTIC ANEMIA
Resolution of lesions in Figure 10.5 following vitamin B12 injections. (Courtesy of Drummond JF, White DK, Damm DD. Megaloblastic anemia with oral lesions: a consequence of gastric bypass surgery. *Oral Surg Oral Med Oral Pathol* 1985;59:149.)



Fig. 10.7

Plummer-Vinson syndrome consists of iron-deficiency anemia with associated dysphagia caused by the development of webs or strictures in the lower part of the hypopharynx or the upper part of the esophagus. The condition is seen almost exclusively in middle-aged women but is uncommon today because of improved nutrition. Patients with Plummer-Vinson syndrome usually present with a hypochromic, microcytic anemia with resultant pallor of the skin and atrophy of the mucous membranes. Atrophy of the tongue papillae may lead to a smooth, red, painful glossitis. Angular cheilitis also is frequently seen. The condition is significant in that patients exhibit a significantly increased prevalence of carcinoma of the esophagus, hypopharynx, and oral cavity.

NEUTROPENIA

Figs. 10.8 and 10.9

Neutropenia refers to a marked reduction of circulating neutrophils in the blood. It may be congenital or hereditary, a result of marrow infiltration by tumor cells, or caused by radiation, cytotoxic drugs, or an abnormal response to a variety of noncytotoxic drugs. Neutropenia also may be a consequence of a variety of viral or bacterial infections. Cyclic neutropenia is an interesting variant in which there are periodic severe reductions in neutrophil counts accompanied by clinical manifestations of infection. Although variable, these episodes of severe neutropenia occur about every 3 weeks, although the neutrophil counts are still generally low during asymptomatic periods.

Neutropenia is characterized clinically by generalized constitutional symptoms including fever, malaise, weakness, and sore throat, plus increased susceptibility to a variety of infections. Oral manifestations include gingivitis, severe periodontal bone destruction, and necrotizing ulcers that may resemble aphthous stomatitis. Management of neutropenia includes prompt diagnosis and aggressive treatment of secondary infections, plus correction of any underlying cause, if possible. Steroid therapy and white blood cell transfusions are sometimes used. Recombinant human granulocyte colony-stimulating factor, a cytokine that stimulates growth and differentiation of neutrophils, has recently shown great promise in the management of patients with neutropenia. Meticulous oral hygiene and periodontal care are needed for oral lesions.

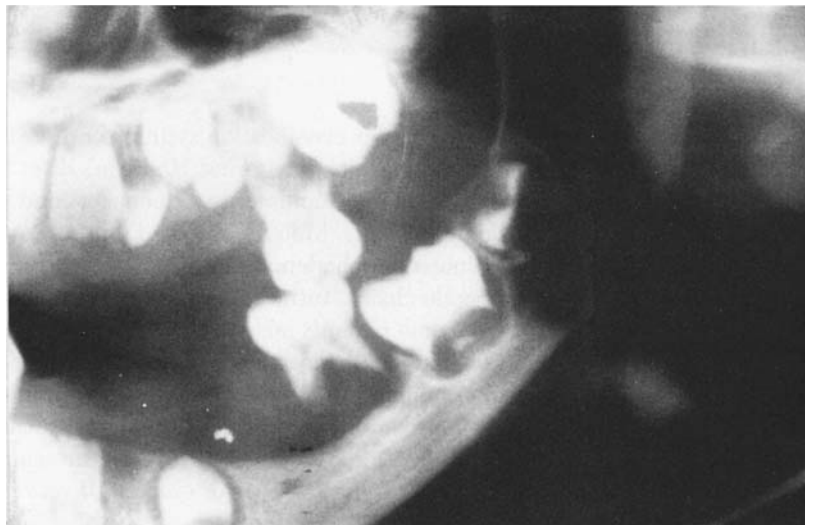
- ▶ **Figure 10.7**
PLUMMER-VINSON SYNDROME
Erythema of tongue with atrophy of filiform papillae.



- ▶ **Figure 10.8**
NEUTROPENIA
Hyperplastic gingivitis.



- ▶ **Figure 10.9**
CYCLIC NEUTROPENIA
Severe periodontal bone destruction in a child.



LEUKEMIA

Figs. 10.10 and 10.11

Leukemias are malignancies of the hematopoietic tissues. The disease is characterized by overproduction of white blood cells with replacement of the normal bone marrow, circulation of abnormal cells in the blood, and infiltration of other tissues. The clinical features may be acute or chronic, and cells of either the lymphocytic or myeloid series are most commonly involved. Acute lymphocytic leukemia is one of the more common childhood malignancies; chronic lymphocytic leukemia, the most common form of leukemia, primarily affects older adults. Acute myeloid leukemia affects a broad age range that includes children and adults; chronic myeloid leukemia shows a peak incidence in the third and fourth decades of life.

Clinical manifestations of leukemia include fatigability, anemia, lymphadenopathy, hepatosplenomegaly, bone and abdominal pain, secondary infection, and hemorrhagic lesions secondary to thrombocytopenia. Oral lesions are more common in myelocytic/monocytic leukemias and may include gingival leukemic infiltrates, severe periodontal bone loss, oral ulcerations, and hemorrhagic lesions. Treatment and prognosis depend on the specific type of leukemia. Therapy primarily centers around chemotherapy and, in some cases, bone marrow transplantation. Children with acute lymphocytic leukemia now can expect a 50 to 70% 5-year survival rate, with 45% being long-term survivors. The myeloid leukemias have a more guarded prognosis. Although chronic myeloid leukemia may begin as a mild disease, the cells eventually undergo an acute blast transformation that can rapidly result in the patient's death. Acute myeloid leukemia has a 5-year survival rate of 10 to 30%. Although there is no cure for chronic lymphocytic leukemia, the disease is often slowly progressive; some patients have a life expectancy of greater than 10 years, depending on the stage of the disease.

HODGKIN'S DISEASE

Fig. 10.12

Although complex classification systems have evolved to categorize lymphomas, they can be divided into two major categories: Hodgkin's disease and non-Hodgkin's lymphomas. Hodgkin's disease is seen most commonly in teenagers and young adults, with a second prevalence peak in middle-aged adults. Men are affected more frequently than women. Patients generally present with painless lymphadenopathy, usually involving the cervical, supraclavicular, and mediastinal lymph node chains. Intraoral involvement is exceedingly rare. Additional clinical symptoms noted in some patients include weight loss, fever, night sweats, and pruritus (itching). The treatment for Hodgkin's disease depends on the stage of involvement but typically involves radiation therapy, chemotherapy, or both. Patients with limited stage I or stage II disease are often managed with radiation therapy alone and have a nearly 90% 10-year survival rate. Patients with advanced stage III and stage IV disease require chemotherapy, possibly in conjunction with radiation therapy, and have a 50% or greater 10-year survival rate.

► **Figure 10.10**
LEUKEMIA
Hemorrhagic gingival leukemic infiltrate.
(Courtesy of Dr. M. Nazif.)



► **Figure 10.11**
LEUKEMIA
Ulceration of the palate. (Courtesy of
Dr. B. Roberts.)



► **Figure 10.12**
HODGKIN'S DISEASE
Cervical and supraclavicular
lymphadenopathy.



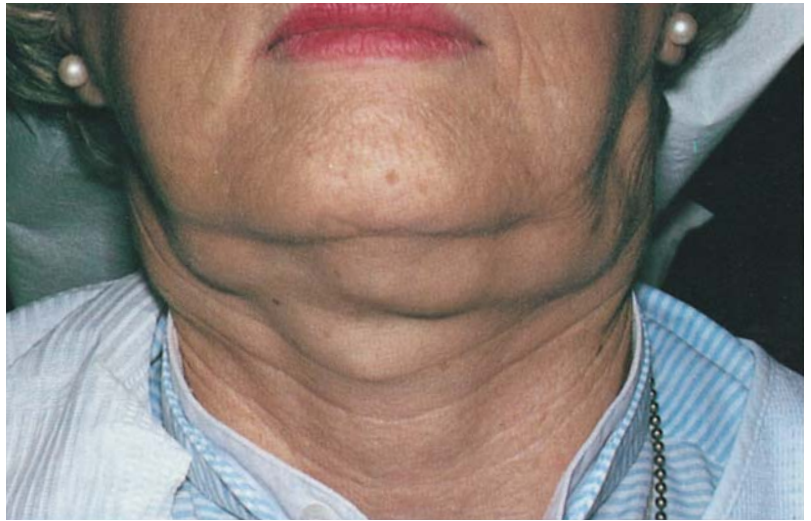
Figs. 10.13, 10.14, and 10.15

Non-Hodgkin's lymphomas are a heterogeneous group of lymphoid malignancies with clinical symptoms that are similar to Hodgkin's disease, although widespread systemic involvement is seen more frequently. They most frequently arise from cells of the B-lymphocyte series but also can arise from T-lymphocytes and rarely from histiocytes. Like Hodgkin's disease, non-Hodgkin's lymphomas are more common in men but with a peak prevalence in middle-aged and older adults. The condition usually begins as a slowly enlarging, nontender mass of one or more lymph nodes but sometimes occurs in extranodal locations. As the lymph nodes enlarge, they may become fixed to adjacent structures or matted together.

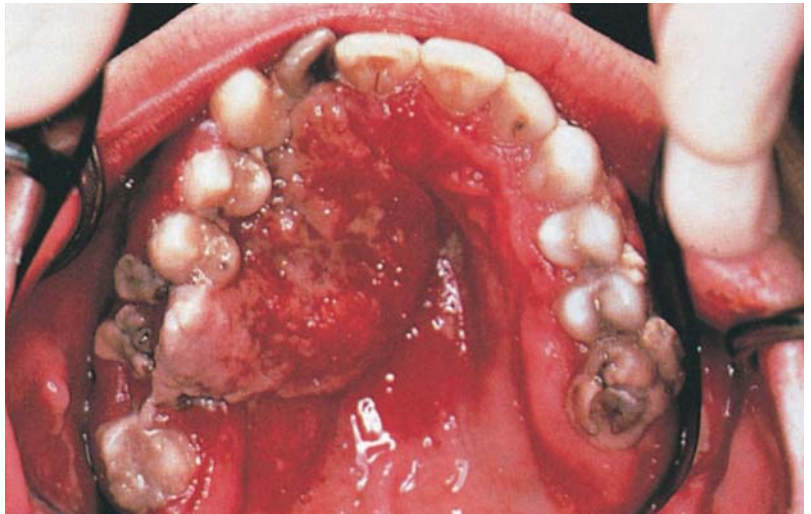
Oral involvement is most commonly seen in the lymphoid tissue of Waldeyer's ring, the hard palate, and buccal vestibule and gingiva. Oral lymphomas often present as a rapidly growing mass of short duration that may or may not be ulcerated. Primary bone involvement also may be seen. Concomitant systemic involvement may or may not already be present at the time of diagnosis.

Non-Hodgkin's lymphomas are usually treated with chemotherapy, radiation therapy, or both. The prognosis is highly variable, depending on the histopathologic subtype of lymphoma. Some low-grade tumors are slowly progressive, with many patients surviving for 10 years or more with limited therapy. The prognosis for high-grade tumors is more guarded, with a 60% mortality rate within 5 years of diagnosis.

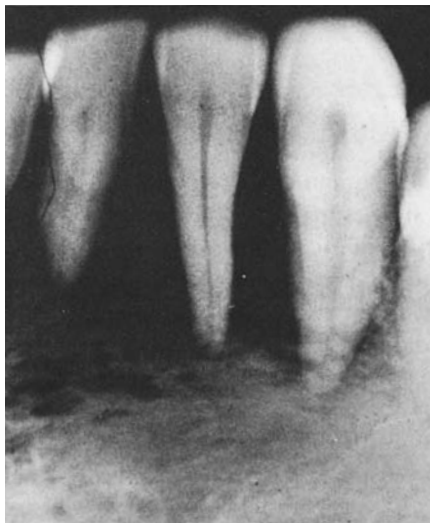
► **Figure 10.13**
NON-HODGKIN'S LYMPHOMA
Bilateral submandibular lymphadenopathy.



► **Figure 10.14**
NON-HODGKIN'S LYMPHOMA
Ulcerated mass of the hard palate.



► **Figure 10.15**
NON-HODGKIN'S LYMPHOMA
Moth-eaten bone destruction in a primary bone lymphoma.



BURKITT'S LYMPHOMA

Figs. 10.16 and 10.17

Burkitt's lymphoma is a characteristic form of non-Hodgkin's lymphoma that was originally described in Central Africa and accounts for 50% of pediatric malignancies in that region. It has since been recognized throughout the world, including North America, although much less frequently. Burkitt's lymphoma is primarily a tumor of childhood, with occasional cases seen in young adults. The mean age of African cases is 7 years, compared with 11 years for American cases. The Epstein-Barr virus has been implicated in the etiology; evidence of Epstein-Barr virus DNA is found within the tumor cells in 95% of African cases, but virus can be demonstrated in only 15% of American cases. Approximately 60% of the African cases occur in the jaws; abdominal involvement is the most common presentation for American Burkitt's lymphoma, with only 15 to 18% of cases affecting the jaws. Jaw involvement is more frequent in younger patients.

Burkitt's lymphoma is more common in the maxilla than in the mandible, and it typically presents as a rapidly growing mass associated with bone destruction, loosening of teeth, and extension into adjacent soft tissues. Concomitant maxillary and mandibular involvement is not unusual. Loss of the lamina dura may be an early radiographic sign. Chemotherapy is the treatment of choice and usually results in disease remission, although recurrence is not uncommon. Prognosis depends on the clinical stage of the disease, with prolonged survival achieved in 50% of cases.

MIDLINE LETHAL GRANULOMA (MIDLINE MALIGNANT RETICULOSIS, POLYMORPHIC RETICULOSIS, IDIOPATHIC MIDLINE DESTRUCTIVE DISEASE, ANGIOCENTRIC LYMPHOMA)

Fig. 10.18

Midline lethal granuloma is a progressive, destructive condition involving the nasal cavity, paranasal sinuses, palate, pharynx, and face. Clinically, it presents with ulceration of the palate or nasal septum, often in association with nasal stuffiness. This process gradually progresses, often resulting in total palatal destruction, bone sequestration, and destruction of facial tissues. Patients may become extremely debilitated, with death resulting from hemorrhage caused by large vessel erosion. Because of the variable histopathologic features of midline lethal granuloma, considerable confusion and controversy have arisen concerning the etiopathogenesis and classification of this condition. Most investigators currently believe that it represents a T-cell lymphoma with an affinity for involving blood vessels ("angiocentric"). Epstein-Barr virus has been implicated as a possible cause. Microscopically, some cases exhibit a benign-appearing inflammatory process ("idiopathic midline destructive disease"), whereas others show a proliferation of atypical mononuclear cells ("midline malignant reticulosis," "polymorphic reticulosis") or an obvious malignant lymphoma. Radiation therapy seems to be the most effective treatment, but chemotherapy is also sometimes used. Prolonged survival is achieved in 15 to 33% of cases.

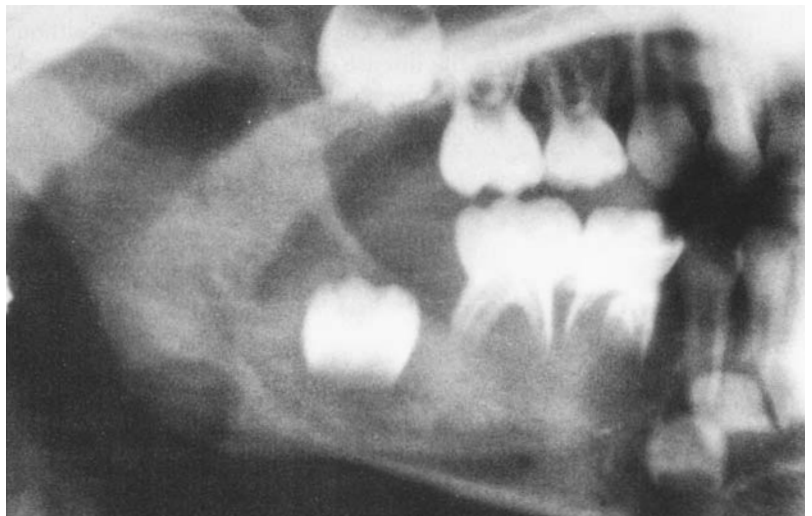
► **Figure 10.16**
BURKITT'S LYMPHOMA

Right facial swelling. (Reprinted with permission from Budnick SD. Handbook of pediatric oral pathology. Chicago: Year Book, 1981.)



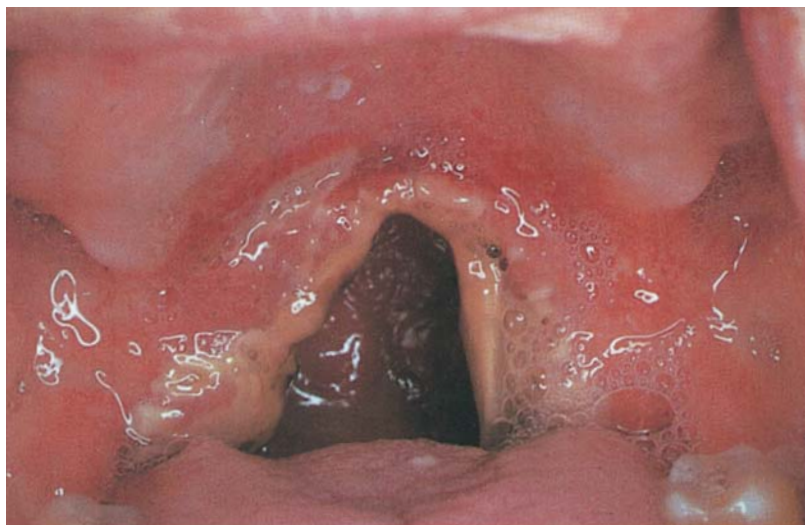
► **Figure 10.17**
BURKITT'S LYMPHOMA

The tumor has destroyed the alveolar bone supporting the primary molar teeth. The teeth were mobile and painful. (Courtesy of Dr. Gregory Anderson.)



► **Figure 10.18**
MIDLINE LETHAL GRANULOMA

Necrotic destruction of the soft palate.



Figs. 10.19, 10.20, and 10.21

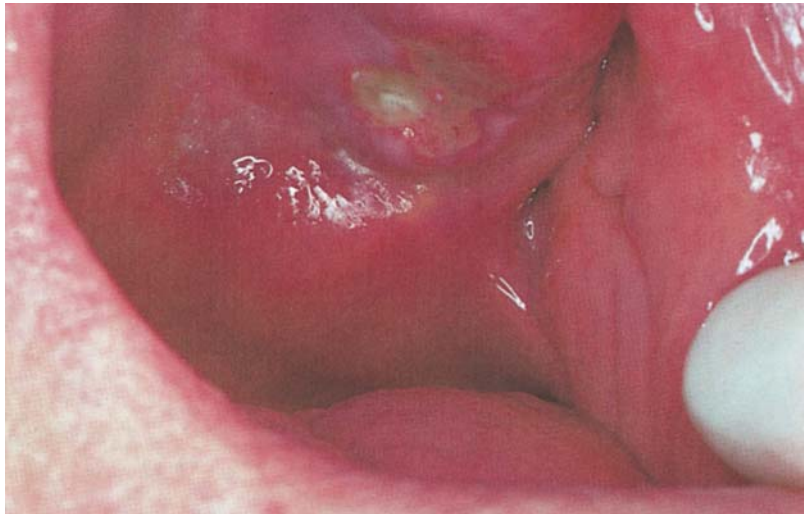
Mycosis fungoides is a T-cell lymphoma that preferentially involves the skin, tending to spare the lymph nodes and internal organs until late in the course of the disease. It occurs most commonly in middle-aged and older adults and affects men more often than women. Classic mycosis fungoides of the skin develops over many years, progressing through three stages: scaly patches, infiltrative plaques, and, finally, tumors. These stages often overlap, so that all three types of lesions may be present at the same time. Diagnosis in the early stages of the disease may prove difficult, with initial lesions being mistaken for psoriasis or eczema. In the fourth and final stage, tumor cells spread to lymph nodes and internal organs, causing death. Sézary syndrome is considered a leukemic variant of the disease in which patients exhibit circulating abnormal T-lymphocytes.

Oral involvement in mycosis fungoides is unusual and typically is discovered during the late stages of the disease. Oral lesions most commonly occur on the tongue and palate and usually are described as ulcerative or nodular in appearance. Treatment for mycosis fungoides depends on the stage of the disease and may include skin irradiation, topical chemotherapy, or systemic chemotherapy. The prognosis is variable, although some patients survive for 10 years or more before the disease disseminates and results in death. However, once oral involvement has been diagnosed, most patients die of disease complications within 3 years.

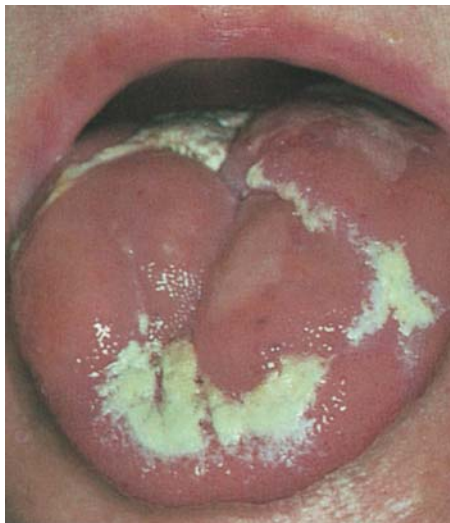
▶ **Figure 10.19**
MYCOSIS FUNGOIDES
Ulcerated plaque of the skin.



▶ **Figure 10.20**
MYCOSIS FUNGOIDES
Ulcerated tumor of the hard and soft palate. (Courtesy of Damm DD, White DK, Cibull ML, et al. Mycosis fungoides: initial diagnosis via palatal biopsy with discussion of diagnostic advantages of plastic embedding. Oral Surg Oral Med Oral Pathol 1984;58:413.)



▶ **Figure 10.21**
MYCOSIS FUNGOIDES
Ulcerated lesions of the tongue.



Figs. 10.22 and 10.23

Multiple myeloma is a malignancy of plasma cells usually occurring in middle-aged and older adults. Men are more frequently affected than women. The disease is characterized by multiple destructive, “punched-out” bone lesions accompanied by pain and, sometimes, pathologic fracture. Typically, only one group of plasma cells becomes malignant, producing abnormal immunoglobulin proteins detectable on serum electrophoresis as a monoclonal gammopathy. In addition, excess immunoglobulin light chains may spill over into the urine as Bence Jones proteins. Other clinical features include anemia, thrombocytopenia, hypercalcemia, amyloidosis, and renal failure.

The spine, ribs, skull, and pelvis are common sites of involvement; the jaws are affected in 15 to 30% of cases. Jaw involvement may be characterized clinically by swelling, pain, paresthesia, tooth mobility, and root resorption. Treatment for multiple myeloma consists primarily of chemotherapy. Autologous stem cell transplantation and allogeneic bone marrow transplantation also have been used. Unfortunately, the prognosis is poor: the 5-year survival rate is about 25% and the median survival time is 3 years.

▶ **Figure 10.22**
MULTIPLE MYELOMA

Punched-out lesions of the skull. (Courtesy of Meyer I, Waldron C. Clinical pathologic conference. Oral Surg Oral Med Oral Pathol 1957;10:175.)



▶ **Figure 10.23**
MULTIPLE MYELOMA

Moth-eaten bone destruction and root resorption.



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