White Blood Cell (WBC) Dyscrasias

Dr. Sundus Abdul Wadood

OMFS

Basrah Dental College

Classification and Features of White Blood Cell (WBC) Dyscrasias

Leucocytosis—Increased number of circulating WBCs
Leukopenia—Decreased number of circulating WBCs
Myeloproliferative disorders
(1) **Acute myeloid leukaemia**—Immature neoplastic malignancy of myeloid cells
(2) **Chronic myeloid leukaemia**—Mature neoplastic malignancy of myeloid cells
Lymphoproliferative disorders
(1) **Acute lymphoblastic leukaemia**—Immature neoplastic malignancy of lymphoid cells
(2) **Chronic lymphocytic leukaemia**—Mature neoplastic malignancy of lymphoid cells
(3) **Lymphomas**
(a) **Hodgkin's disease**—Malignant growth of B lymphocytes, primarily in lymph nodes
(b) **Non-Hodgkin's lymphoma**—B- or T-cell malignant neoplasms,
(c) **Burkett's lymphoma**—Non-Hodgkin's B-cell lymphoma involving bone and lymph nodes
(4) **Multiple myeloma**—Overproduction of malignant plasma cells involving bone

LEUKOCYTOSIS AND LEUKOPENIA

- The number of circulating **WBCs normally ranges from 4400 to 11,000/mm 3 in adults**.

- The differential WBC count is an estimation of the percentage of each cell type per cubic millimeter of blood.

- A normal differential count consists of:
  - **neutrophils**, 50% to 60%;
  - **eosinophils**, 1% to 3%;
  - **basophils**, less than 1%;
  - **lymphocytes**, 20% to 34%;
  - **monocytes,** 3% to 7%.

- The term leukocytosis is defined as an increase in the number of circulating WBCs (lymphocytes or granulocytes) to more than 11,000/mm 3

- leukopenia as a reduction in the number of circulating WBCs (usually to <4400/mm 3).

causes of leukocytosis

- **physiologic leukocytosis**: can lead to increased numbers of WBCs in the peripheral circulation
  Exercise, pregnancy, & emotional stress.

- **Pathologic leukocytosis** can be caused by
  infection, neoplasia, necrosis.
- **Pyogenic infections** induce a type of leukocytosis that is characterized by an **increased number of neutrophils.**
- **Tuberculosis, syphilis, and viral infections** produce a type of leukocytosis that is characterized by **increased numbers of lymphocytes**.
- **Protozoan infections** often produce a type of leukocytosis that **increases the numbers of monocytes.**
- **Allergies and parasitic infections** caused by certain helminths **increase the numbers of circulating eosinophils.**
- **Cellular necrosis** **increases the numbers of circulating neutrophils**.
- **Leukemia** (cancer of the WBCs) is characterized by a great **increase in the numbers of circulating immature leukocytes.**
- **Carcinoma of glandular tissues** may cause an **increase in the number of circulating neutrophils**.
- **Acute bleeding** also can result in leukocytosis.

causes of Leukopenia

- Occur in the **early phase of leukemia and lymphoma** as a result of bone marrow replacement through excessive proliferation of WBCs.
- Leukopenia also occurs **during agranulocytosis** (reduction of granulocytes)
- **Pancytopenia** (decreased WBCs and RBCs) that **result from toxic effects of drugs and chemicals.**
- Leukopenia is a **common complication that results from the use of chemotherapeutic (anticancer) drugs.**

Cyclic Neutropenia

- An important form of leukopenia involving the **cyclic depression of circulating neutrophils .**
- In this condition patients have a **periodic decrease** (at least a 40% drop) in the **number of neutrophils (about every 21 to 28 days).**
- During the period in which **few circulating neutrophils are present, the patient is susceptible to infection**

Leukemia and lymphoma

- **Account for about 8% of all new malignancies each year in the United States**, which amounts to approximately 101,000 cases per year.
- These patients **become gravely ill if they are not properly identified and do not receive appropriate medical care.**
- In addition, patients are usually **immunosuppressed as a result of the disease itself or because of the treatment used to control it.**
- Hence, they are **prone to develop serious infection** and often bleed easily because of thrombocytopenia.

LEUKEMIA

1. Host factors

a. Heredity

(1) Generally not an inherited disease
(2) High concordance among identical twins if one twin develops the disease early
(3) A few leukemic families have been reported

b. **Chromosomal abnormalities** Increased risk in patients with the following:
(1) Down syndrome
(2) Turner's syndrome
(3) Klinefelter's syndrome
(4) Fanconi's anaemia

c. **Immunodeficiency syndromes** (hereditary types)

d. **Chronic bone marrow dysfunction**

2. Environmental factors

a. **Ionizing radiation**
(1) Radiation therapy
(2) Occupational exposure
(3) Atomic bomb survivors

b. **Chemical and drugs**
(1) Benzene (organic solvents)
(2) Chloramphenicol
(3) Phenylbutazone
(4) Arsenic pesticides
(5) Alkylating chemotherapeutic agents

c. **Viruses**
(1) HTLV-I (adult T-cell leukaemia)
(2) HTLV-II (atypical hairy cell leukaemia

TABLE 24-4 -- Comparison of Acute and Chronic Leukemias

Parameter	Acute	Chronic
Clinical onset	Sudden	Insidious
Course (untreated)	<6 months	2-6 years
Leukemic cells	Immature	Mature
Anemia	Mild to severe	Mild
Thrombocytopenia	Mild to severe	Mild
White blood cell count	Variable	Increased
Organomegaly	Mild	Prominent
Age	Adults and children	Adults

Modified from Perkins ML. Clinical Hematology and Fundamentals of Hemostasis. Philadelphia, FA Davis, 1992, pp 266-292.

ACUTE MYELOGENOUS LEUKEMIA

DEFINITION

AML is a neoplasm of myeloid (immature) WBCs that demonstrate uncontrolled proliferation in the bone marrow space and subsequently appear in the peripheral blood.

Etiology

AML arises primary in younger adults or secondarily in the elderly because of myelodysplasia.

Environmental or genetic factors (e.g., translocation and rearrangement of chromosomes) may cause cytogenetic abnormalities that affect transcriptional cascades of myeloid precursor cells and uncontrolled proliferation of these cells.

AML

SIGNS AND SYMPTOMS

AML produces a leukemic infiltration of marrow and organs that causes cytopenia and diverse nonspecific signs and symptoms, including :

- ❖ fatigue,
- ❖ easy bruising,
- ❖ bone pain.

Anemia and thrombocytopenia usually manifest as

- ❖ malaise,
- ❖ pallor,
- ❖ dyspnea on exertion,
- ❖ bleeding and small hemorrhage (petechial, ecchymosis) in the skin and mucous membranes

Because of granulocytopenia, at least one third of patients have

- ❖ recurrent infections (non healing wounds),
- ❖ Oral ulcerations,
- ❖ fever.
- ❖ Enlargement of the tonsils, lymph nodes, spleen, and gingiva occurs as a result of leukemic infiltration of these tissues.

Infiltration of the central nervous system (CNS) and the skin as a raised, nonpruritic rash is referred to as leukemia cutis.

ACUTE LYMPHOID LEUKEMIA

DEFINITION

ALL is the result of uncontrolled monoclonal proliferation of immature lymphoid cells in the bone marrow and peripheral blood. These neoplastic cells may also expand in the ***lymph nodes, liver, spleen, or CNS.***

Etiology

Although environmental, infectious, and genetic factors are considered likely causes of the disease, causal links for ALL have not been established. The disease is 20-fold more common in patients with Down syndrome (trisomy 21).

Signs and Symptoms

The clinical presentation of ALL can be acute or insidious. Presenting signs and symptoms relate to

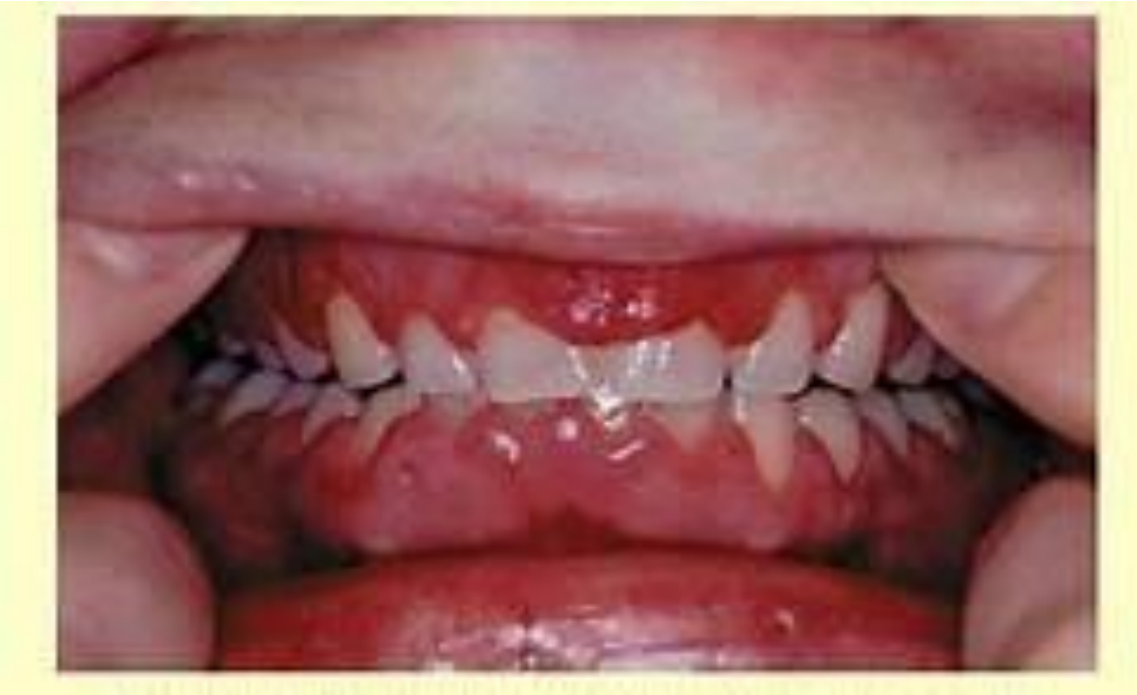
- anemia,
- thrombocytopenia,
- fever,
- Neutropenia
- bone and joint pain have effects on walking,
- infection.
- A higher propensity toward CNS disease occurs with ALL compared with AML.
- Patients may present with cranial nerve deficiencies.

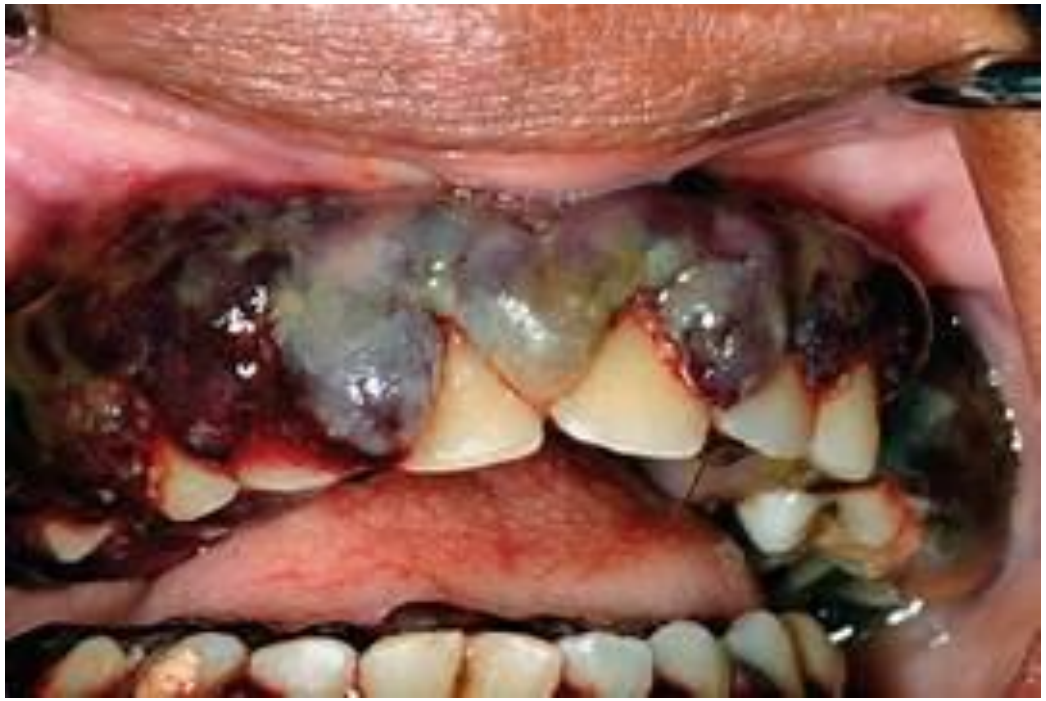
Oral Manifestations of Acute Leukemia

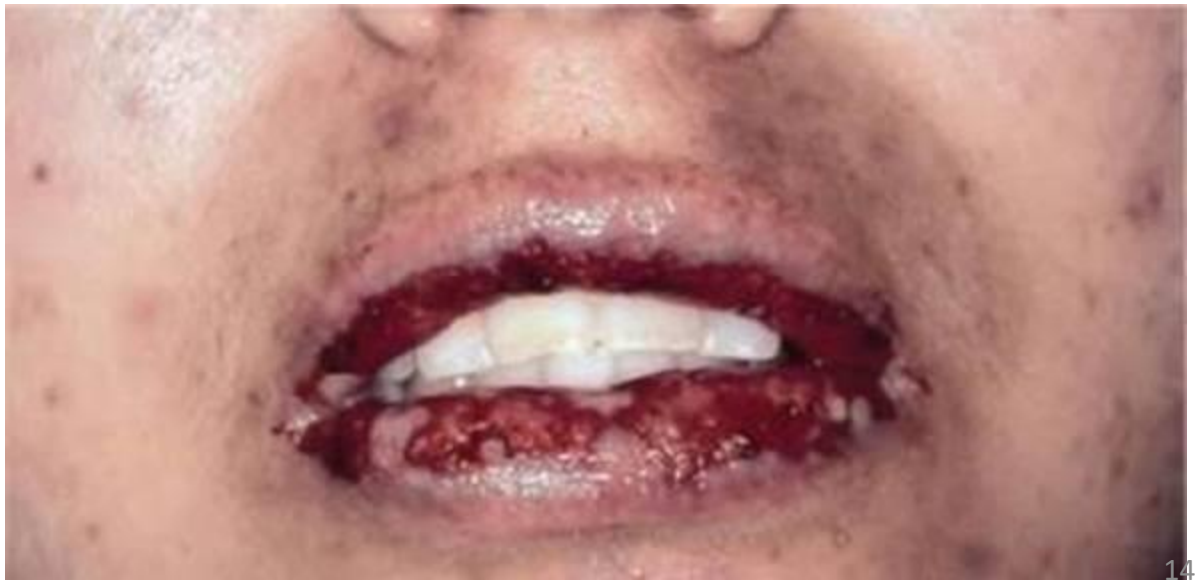
It occurs in up to 36% of those with acute leukemia (most frequently with the acute myelomonocytic types) and in about 10% of those with chronic leukemia.

Leukemic patients are prone to develop :

1. **gingival enlargement,**
2. **Ulceration,**
3. **Oral infection.**
4. **Localized or generalized gingival enlargement is caused by inflammation and infiltration of atypical and immature WBCs**
5. **The gingiva is boggy and bleeds easily, and multiple tooth sites are typically affected.**
6. **Generalized gingival enlargement is more common and is particularly prevalent when oral hygiene is poor and in patients who have AML (particularly the monocytic type**
7. **The combination of poor oral hygiene and gingival enlargement contributes to gingival bleeding**
8. **Gingival bleeding is exacerbated by the presence of thrombocytopenia.**
9. **A localized mass of leukemic cells (in the gingiva or other sites) is specifically known as a granulocytic sarcoma or chloroma .**
10. **These extra medullary tumors have been observed in the maxilla and the palate**







CHRONIC MYELOID LEUKEMIA

DEFINITION

Chronic myeloid leukemia (CML) is a neoplasm of mature myeloid WBCs.

Etiology

The etiology is unknown, but **radiation exposure** increases risk for the disease.
The genetic defect

SIGNS AND SYMPTOMS

In nearly 90% of patients, CML is diagnosed during the chronic phase. Up to half of these patients are asymptomatic, and diagnosis is based on their complete blood count. Common symptoms are:

- fatigue,
- weakness,
- abdominal (upper left quadrant) pain,
- abdominal fullness,
- weight loss,
- night sweats due to anemia,
- an enlarged and painful spleen (splenomegaly),
- altered hematopoiesis.
- Hyper viscosity of the blood may cause a stroke.

Oral Manifestations
Chronic myeloid leukaemia (CML)

Chronic forms of leukemia are **less likely to demonstrate oral manifestations** than are acute forms of leukemia.

1. Generalized lymphadenopathy,
2. pallor of the oral mucosa,
3. soft tissue infection may be present.

CHRONIC LYMPHOCYTIC LEUKEMIA

DEFINITION

Chronic lymphocytic leukemia (CLL) is a neoplasm of mature clonal CD5+ B lymphocytes

Etiology

The etiology of CLL is unknown, and risk factors are more related to familial inheritance than to exposure to harmful environmental agents. Neoplastic B cells have various genetic aberrations, most commonly, gene deletions (e.g., on chromosome 11, 13, or 17) that lead to loss of cell cycle control.

CLL

SIGNS AND SYMPTOMS

Most patients with CLL are asymptomatic at presentation. When symptoms occur,

- fatigue,
- anorexia, and
- weight loss are the most common complaints.
- Patients have an enlarged spleen,
- lymphadenopathy
- decreased serum immunoglobulin levels (hypogammaglobulinemia) that contribute to
- susceptibility to infection.
- develop autoantibodies against red blood cells (RBCs) or platelets that produce hemolytic anemia or thrombocytopenia.

In about 15% of patients, CLL become more aggressive malignancy with increasing

- lymphadenopathy,
- hepatosplenomegaly,
- fever,
- abdominal pain,
- weight loss,
- progressive anemia, and thrombocytopenia.

TABLE 24-3 -- Clinical Factors in Acute and Chronic Leukemias

	Type of Leukemia			
Factor	**ALL**	**AML**	**CLL**	**CML**
Age	Children (75%)	Adults (85%)	Over 40 years	30-50 years
Prognosis	Very good	Poor	Good	Poor
Survival, mean	—	2 years	Stage I (19 months)	3-4 years
			Stage IV (12 years)	—
Remissions	90%	60%-80%	—	—
Duration	Usually long term	9-24 months	—	—
Cures	50%-70%	10%-30%	—	
	ALL	**AML**	**CLL**	**CML**
Age	Adults (25%)	Children (15%)	Children (rare)	Children (rare)
Prognosis	Poor	Poor	—v	—
Survival, mean	26 months	—	—	—
Remissions	50%-70%	56%-66%	—	—
Duration	10-19 months	8-12 months	—	—
Cures	20%	20%-40%	—	

Modified from Wetzler M, Byrd JC, Bloomfield CD. Acute and chronic myeloid leukemia. In Kasper DL, et al (eds). Harrison's Principles of Internal Medicine, 16th ed. New York, McGraw-Hill, 2005; Armitage JO, Longo DL. Malignancies of lymphoid cells. In Kasper DL, et al (eds). Harrison's Principles of Internal Medicine, 16th

lymphoma

- Lymphoma is cancer of the **lymphoid organs** and tissues that presents as discrete tissue masses.

- Lymphomas represent the **seventh most common malignancy worldwide** and affect 63,000 Americans each year.

- Lymphomas are classified by

1. **cell type** (B cell, T cell, plasma cell), appearance (small or large cell, cleaved or noncleaved nucleus),
2. **clinical behavior** (of low, intermediate, and high grade); higher grades have been noted to be more aggressive.

- Of more than 20 types, 3 common lymphomas
  - ✓ **(Hodgkin's disease, non-Hodgkin's lymphoma, and Burkitt's lymphoma)**
  - ✓ **plasma cell malignancy (multiple myeloma)** are considered in this section.
- These diseases are of importance to the dentist because initial signs often occur in the mouth (e.g., Waldeyer's ring) and in the head and neck region, and precautions must be taken before any dental treatment is provided.

HODGKIN'S DISEASE

DEFINITION

Hodgkin's disease is a neoplasm (uncontrolled growth) of B lymphocytes that was named for Thomas Hodgkin, the British pathologist who first described it.

This neoplasm contains a characteristic tumor cell called the Reed-Sternberg cell that represents usually <1% of the cellular infiltrate in affected tissues.

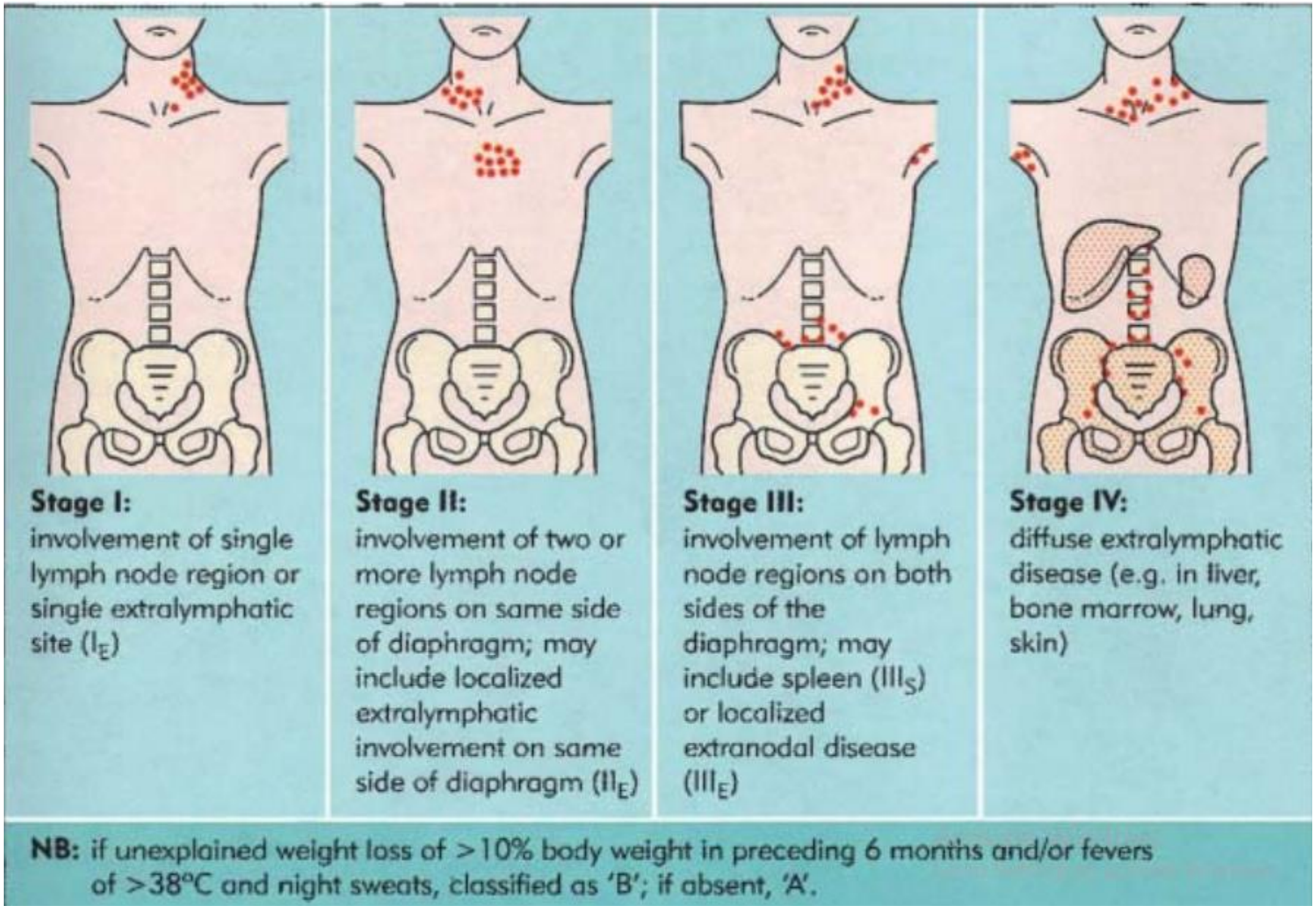
ETIOLOGY

The cause of Hodgkin's disease is unknown, but EBV is frequently present (40% of cases in the Western world) in malignant lymphocytes.

SIGNS AND SYMPTOMS

Hodgkin's disease presents most commonly as a

- painless mass or a group of firm, nontender, enlarged lymph nodes, often (i.e., in more than 50% of cases) affecting the **mediastinal nodes or the neck nodes**
- Enlarged lymph nodes in the **underarm or groin** are also common presentations.
- Fever,
- weight loss,
- night sweats occur in about one third of patients.
- Pruritus and fatigue develop and may precede the appearance of enlarging lymph nodes.
- Palpation of the lymph nodes reveals a rubbery consistency.



Stage I:
involvement of single lymph node region or single extralymphatic site (I_E)

Stage II:
involvement of two or more lymph node regions on same side of diaphragm; may include localized extralymphatic involvement on same side of diaphragm (II_E)

Stage III:
involvement of lymph node regions on both sides of the diaphragm; may include spleen (III_S) or localized extranodal disease (III_E)

Stage IV:
diffuse extralymphatic disease (e.g. in liver, bone marrow, lung, skin)

NB: if unexplained weight loss of >10% body weight in preceding 6 months and/or fevers of >38°C and night sweats, classified as 'B'; if absent, 'A'.

NON-HODGKIN'S LYMPHOMA

DEFINITION

Non-Hodgkin's lymphoma (NHL) comprises a large group of lymphoproliferative disorders **classified as of B-cell or T-cell origin**. More than 80% of these neoplasms are of B-cell origin. The World Health Organization (WHO) classification system uses immunopheno type, cytogenetics, and epidemiologic.

TYPES distinguish many types of NHL .Four major categories of NHL are described:

- ❖ precursor (immature) B-cell neoplasms,
- ❖ peripheral (mature) B-cell neoplasms,
- ❖ precursor (immature) T-cell neoplasms,
- ❖ peripheral (mature) T-cell and natural killer (NK)-cell neoplasms.

Subcategories are based on pattern of distribution (diffuse or nodular),

- ❖ cell type (lymphocytic, histiocytic, mixed),
- ❖ degree of differentiation of cells (good, moderate, poor).

Of the more than 20 types of NHL that have been identified, diffuse large B-cell and follicular lymphomas account for about 60% of cases.

NON-HODGKIN'S LYMPHOMA

SIGNS AND SYMPTOMS

NHLs may occur at any age and are often marked by
enlarged lymph nodes,
fever,
weight loss.
Different from Hodgkin's disease, which often begins with a single focus of tumor,
NHL is usually multifocal when first detected.
The most prominent sign of NHL is a painless lymph node(s) swelling of longer than 2 weeks duration.

Additional signs and symptoms include

persistent fever of unknown cause,
weight loss,
malaise, sweating,
tender lymphadenopathy, abdominal or chest pain,
extranodal tumors.
Head, neck, and intra-abdominal manifestations occur fairly often.
Less frequently, an oral presentation (e.g., a firm swelling arising from the posterior
hard palate) may be seen

TABLE 24-5 -- Comparison of Non-Hodgkin's and Hodgkin's Lymphomas

Parameter	Non-Hodgkin's	Hodgkin's
Cellular derivation site	>80% B cell 10%-19% T cell or NK cell	B cell
Localized	Uncommon	Common
Waldeyer's ring	Commonly involved	Rarely involved
Extranodal	Common	Uncommon
Abdominal (mesenteric nodes)	Common	Uncommon
Mediastinal	Uncommon	Common
Bone marrow	Common	Uncommon
Symptoms (fever, night sweats, weight loss)	Uncommon	Common
Curability	<25%	>75%

Data from Armitage JO, Longo DL. Malignancies of lymphoid cells. In Kasper DL, et al (eds). Harrison's Principles of Internal Medicine, 16th ed. New York, McGraw-Hill, 2005.

Oral Complications and Manifestations

Patients with Hodgkin's disease or non-Hodgkin's lymphoma may present with :

- cervical lymphadenopathy
- extra nodal or intraoral tumors
- This situation is of particular concern in immunosuppressed patients and individuals with Sjögren's syndrome who are at increased risk for the development of lymphoma.
- Patients should be periodically monitored for the development of orofacial neoplasia.
- Intraoral lymphoma most commonly involves Waldeyer's ring (soft palate and oropharynx)
- the salivary glands and mandible are affected.
- Intraoral lymphomas appear as rapidly expanding (or chronic), unexplained swellings of the head and neck lymph nodes, palate, gingiva, buccal sulcus, or floor of the mouth.
- Enlargements may be painless or painful.
- The presence of these orofacial abnormalities requires prompt evaluation by biopsy via needle, incisional, or excisional techniques.

BURKITT'S LYMPHOMA

DEFINITION

Burkitt's lymphoma is an **aggressive B-cell (non-Hodgkin's)** lymphoma that was originally described by Denis Burkitt.

The tumors are composed of mature B cells that express surface immunoglobulin (Ig)M.

Etiology

- All Burkitt's lymphomas are associated with translocation of the c-myc gene (**a gene involved in cellular proliferation)** onto chromosome 8.
- All endemic tumors contain latent **EBV.**
- EBV is present in about 15% to 20% of sporadic lymphomas and in about 25% of HIV-associated tumors.

SIGNS AND SYMPTOMS

- Most Burkitt's lymphomas arise at **extranodal sites**. The **endemic form** shows a **predilection for tumors of the jaw and for involvement of select abdominal organs, particularly the kidneys, ovaries, and adrenal glands.**
- **Jaw involvement is more common in patients younger than 5 years of age than among those older than age 10 .**
- **Nonendemic** Burkett's lymphoma often presents as an abdominal mass that involves the lymph nodes of the intestine and peritoneum, **with jaw lesions being less common.**
- Tumors that enlarge as abdominal masses are accompanied by fluid buildup, pain, and, possibly, vomiting. The bone marrow is infrequently involved.

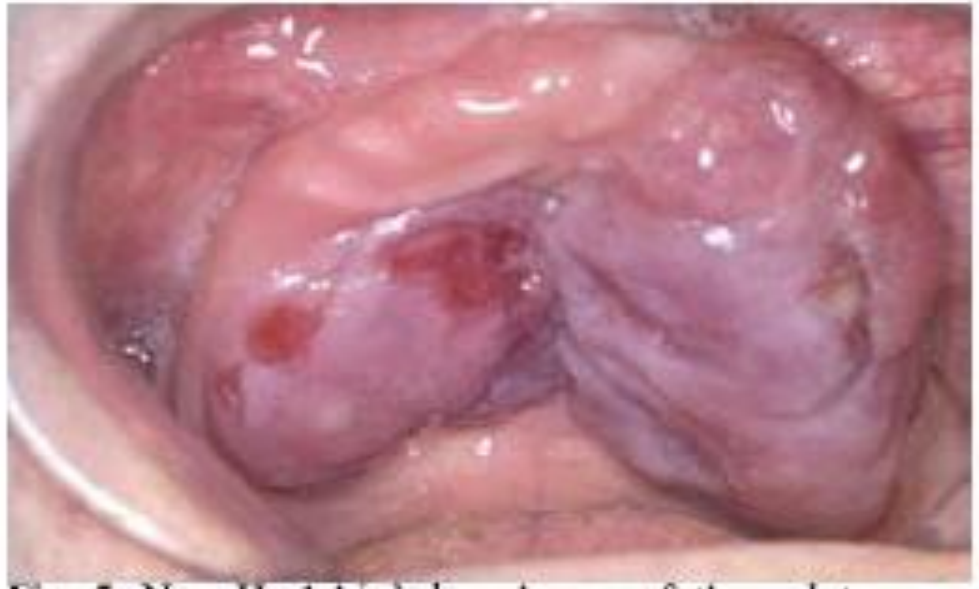
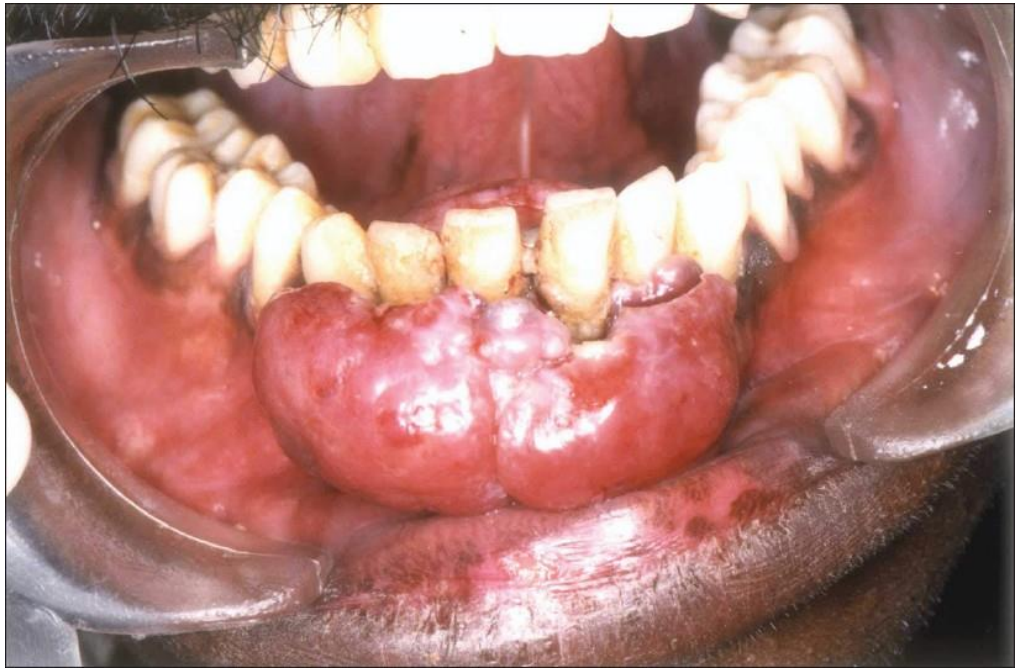
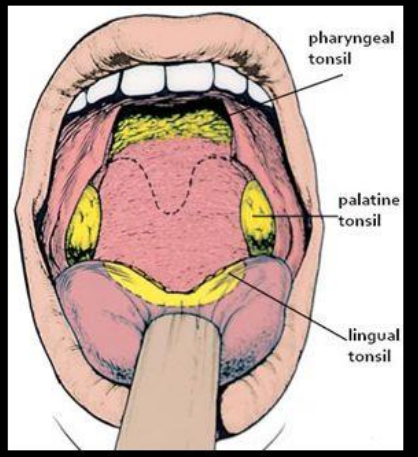


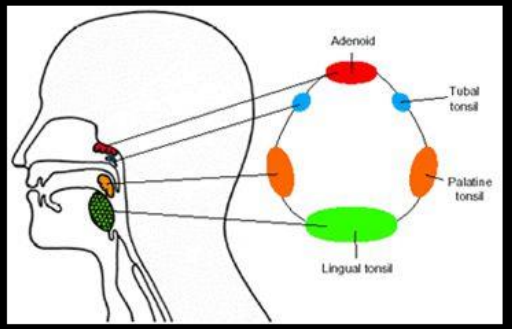
Fig. 5. Non-Hodgkin's lymphoma of the palate presenting as a slowly growing mucosal swelling. No bone involvement.



Waldeyer's Tonsillar Ring

- It is a lymphoid tissue ring located in the pharynx
- Function as a barrier to infection especially in the first few years of life
- Consists of (from superior to inferior):
  - Adenoids (pharyngeal tonsils)
  - Tubal tonsil
  - Palatine tonsil
  - Lingual tonsil

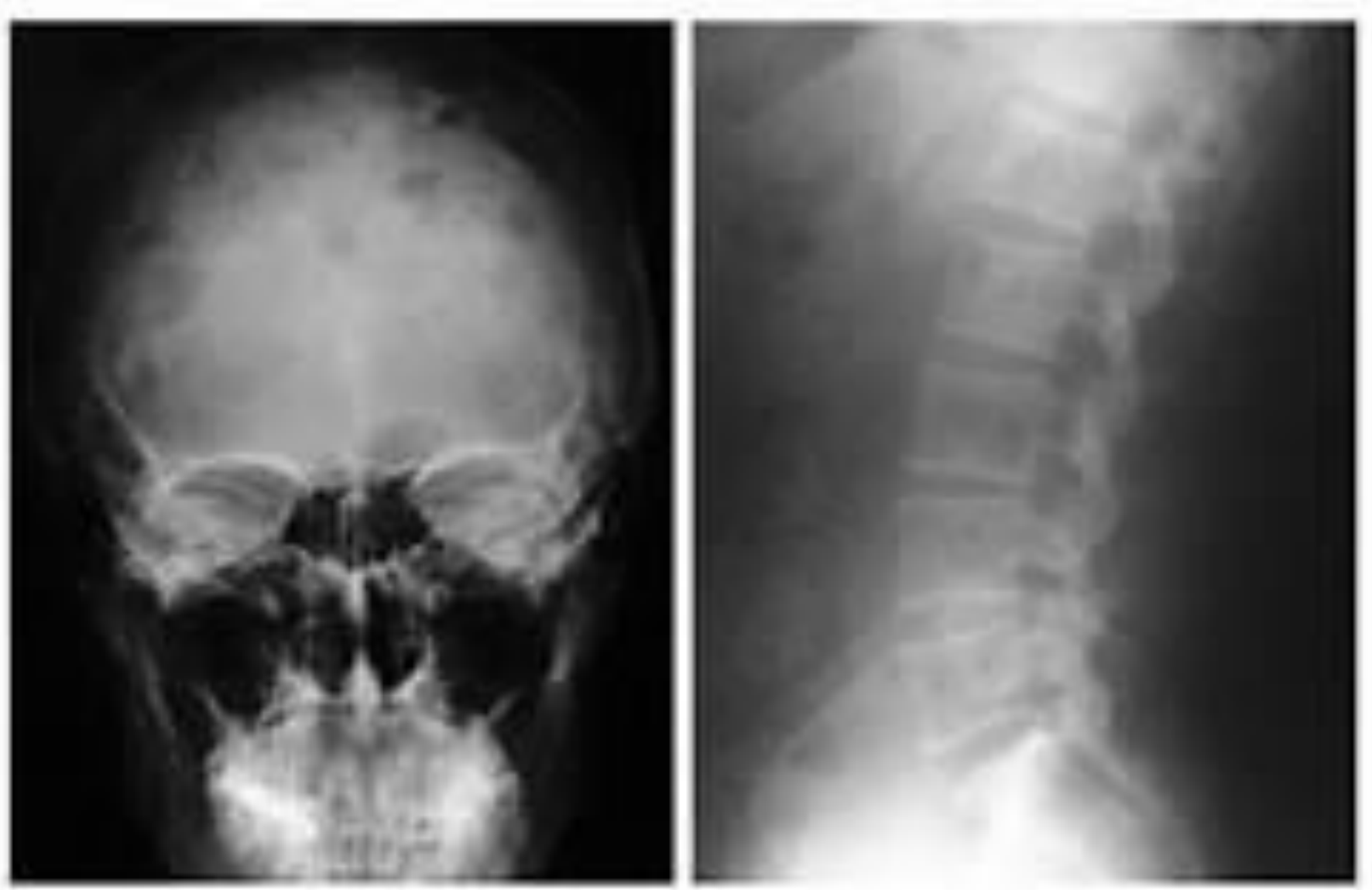




BURKITT'S LYMPHOMA

Oral Complications and Manifestations

- Endemic Burkitt's lymphoma often presents as a **rapidly expanding tumorous mass in the posterior region of the maxilla or mandible.**
- **Rapid growth pushes adjacent teeth, causing the teeth to become mobile and abnormally positioned.**
- **Pain and paresthesia** accompany the condition.
- Radiographically, the tumor produces **an osteolytic lesion with poorly demarcated margins, erosion of the cortical plate**, and soft tissue involvement.



MULTIPLE MYELOMA

DEFINITION

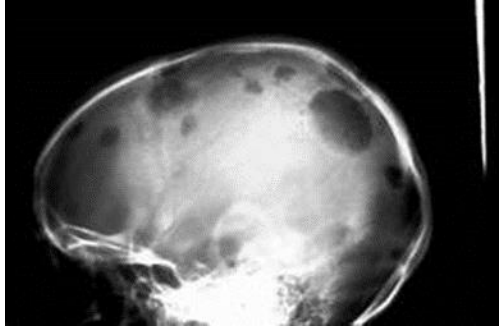
Multiple myeloma (MM) is a lymphoproliferative disorder that results from overproduction of cloned **malignant plasma cells** that results in multiple tumorous masses scattered throughout the skeletal system. Malignant plasma cells secrete monoclonal immunoglobulins and various cytokines.

ETIOLOGY

The etiology of MM is

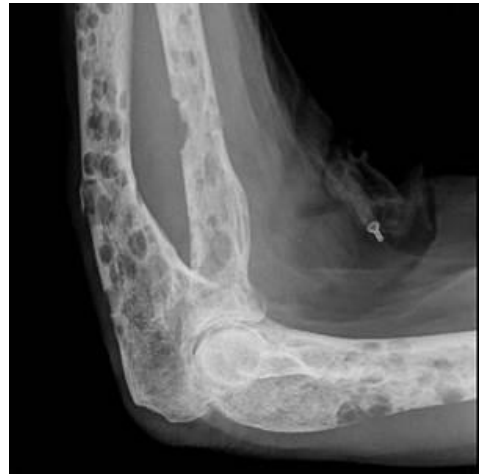
- **unknown** but involves uncontrolled division of a clonal cell that produces daughter cells of the same genetic makeup.
- **Chromosomal translocations** that frequently involve the immunoglobulin heavy chain locus (IgH; 14q32) are common. The translocated gene is placed under transcriptional control of potent IgH enhancers, thus resulting in their overexpression.
- **Various cytokines** (interleukin [IL]-1a and RANKL [receptor activator for nuclear factor k B ligand]) are also overproduced.
- Production of IL-6 by neoplastic plasma cells and normal stromal cells aids in the proliferation of tumor cells.
- Additional cytokines act as osteoclast-activating factors that stimulate osteoclasts to resorb bone.

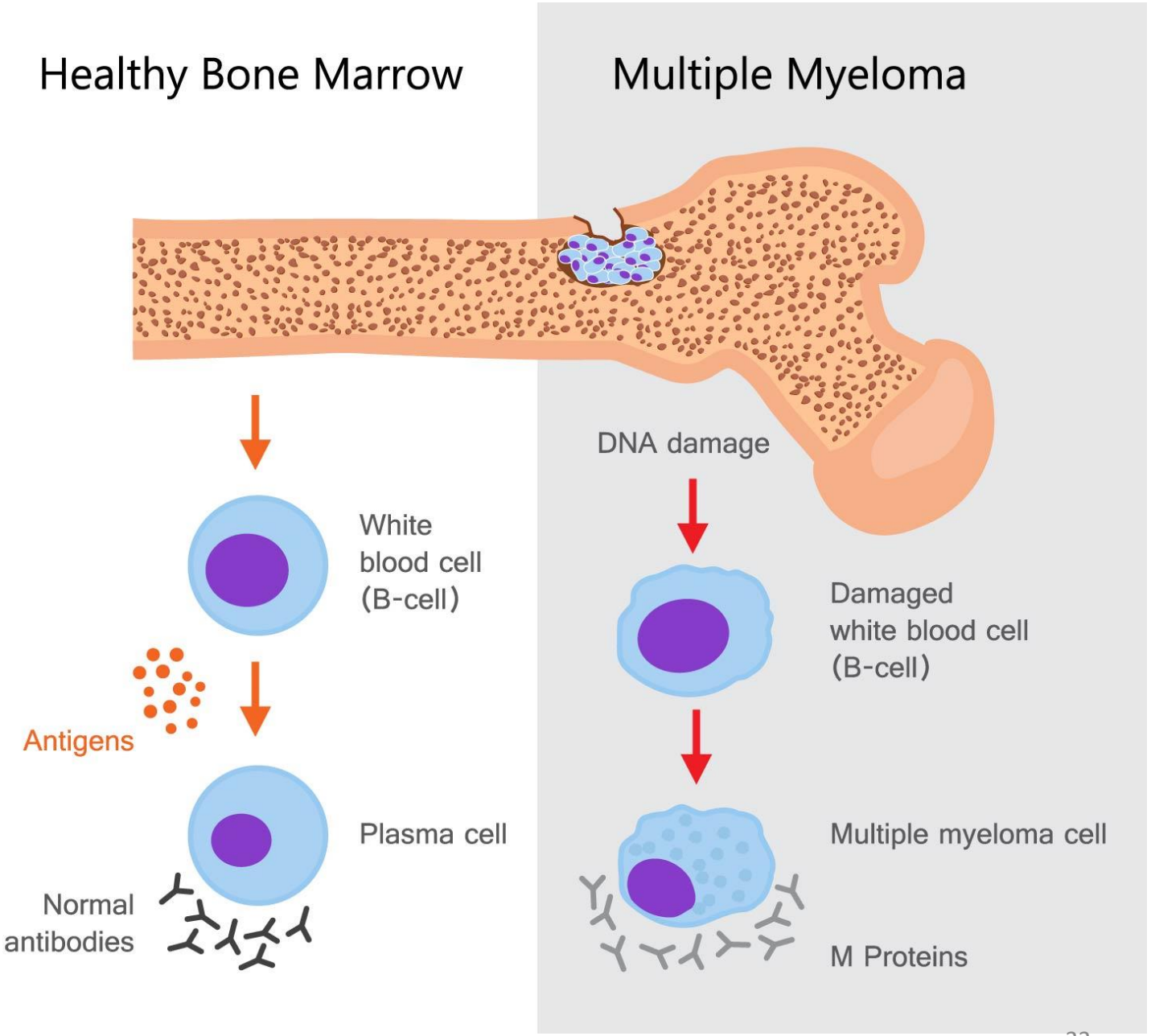
MULTIPLE MYELOMA



SIGNS AND SYMPTOMS

- The most prominent feature of MM is observed radiographically. This disease produces multiple "**punched-out" lesions** or mottled areas, which represent areas of tumor that appear in the **spine, ribs, and cortical regions of the skull.**
- **Osteolytic lesions of the jaw** occur in up to 30% of patients.
- **Amyloid deposition** is seen in various soft tissues (heart, liver, nervous system). Because of the hypogammaglobulinemia, pneumonia and pyelonephritis commonly develop.
- The most prominent symptom is persistent bone pain. The sites most commonly affected are the spine, ribs, and sternum.
- As bone marrow is replaced, anemia develops, along with associated features of
- weakness,
- weight loss,
- recurrent infection.
- Headache and peripheral neuropathy are associated with hypercalcemia.
- Tumor destruction of bone may cause pathologic fracture.




Healthy Bone Marrow
Multiple Myeloma
DNA damage
White
blood cell
(B-cell)
Damaged
white blood cell
(B-cell)
Antigens
Plasma cell
Multiple myeloma cell
Normal
antibodies
M Proteins

MULTIPLE MYELOMA

Oral Complications and Manifestations

- Patients with MM may have jaw lesions, soft tissue lesions, and soft tissue deposits of amyloid.
- Bone and soft tissue lesions often are painful.
- Dental radiographs may show "punched-out" lesions or mottled areas that represent areas of tumor. These osteolytic lesions are more common in the posterior body of the mandible and may be associated with cortical plate expansion.
- Extra medullary plasma cell tumors can occur in the oral pharynx.
- An amyloid-like protein is found sometimes in oral soft tissues (e.g., tongue) as a result of MM, and these areas may be swollen and painful.
- Biopsy and special amyloid stains can be used for diagnosis.

Treatment Planning Modifications

Dental management of patients in whom a WBC disorder is diagnosed requires consideration of the three phases of medical therapy. Planning involves :

(1) pretreatment assessment and preparation of the patient, **goal of minimizing and/or eliminating oral disease prior to the start of chemotherapy.**

(2) oral health care during medical therapy, **Patients who are undergoing chemotherapy or radiotherapy are susceptible to many oral complications, including mucositis, neutropenia, infection, excessive bleeding, and alterations in growth and development.**

(3) Post treatment management, including long-term considerations and possible remission.

- **Patients who have WBC disorders and are in a state of remission can receive most indicated dental treatment.**
- **Patients who have advanced disease and a limited prognosis, as occurs in many cases of leukemia and MM, should receive emergency care only.**

Dental Management of WBC Discreasia

1. **Detection**
   a. History
   b. Examination
   c. Screening laboratory tests
      (1) White cell count
      (2) Differential white cell count
      (3) Smear for cell morphologic study
      (4) Hemoglobin or hematocrit level
      (5) Platelet count

2. **Referral**
   a. Medical diagnosis
   b. Treatment

3. **Consultation before any dental care is rendered**
   a. Current status
   b. Review of dental treatment needs
   c. Dental management plan

4. **Routine dental care**
   a. None for patient with acute symptoms
   b. Once disease is under control, patient may receive indicated dental care Scaling and surgical procedures
      (1) Platelet count on day of procedure: if normal, proceed; if <50,000, avoid invasive procedures, if possible; if <40,000, provide platelet replacement
      (2) Prophylactic antibiotic therapy to prevent postoperative infection (if severe neutropenia is present)
5. **Emergency dental care**
   a. Treatment of oral ulcers
      (1) Antibiotics
      (2) Bland mouth rinse
      (3) Antihistamine solutions
      (4) Orabase
   b. Oral candidiasis—Treat with antifungal medication
   c. Conservative management of pain and infection
      (1) Antibiotic sensitivity testing
      (2) Strong analgesics for pain

Table 3

Regimens for a Dental Procedure*

Situation	Agent	Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2 g IM or IV	50 mg/kg IM or IV
	or		
	cefazolin or ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergic to penicillins or ampicillin (oral)	Cephalexin†	2 g	50 mg/kg
	or		
	clindamycin	600 mg	20 mg/kg
	or		
	azithromycin or clarithromycin	500 mg	15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medication	Cephalexin† or ceftriaxone‡	1 g IM or IV	50 mg/kg IM or IV
	or		
	clindamycin	600 mg IM or IV	20 mg/kg IM or IV

*Single dose administered 30-60 min before procedure.

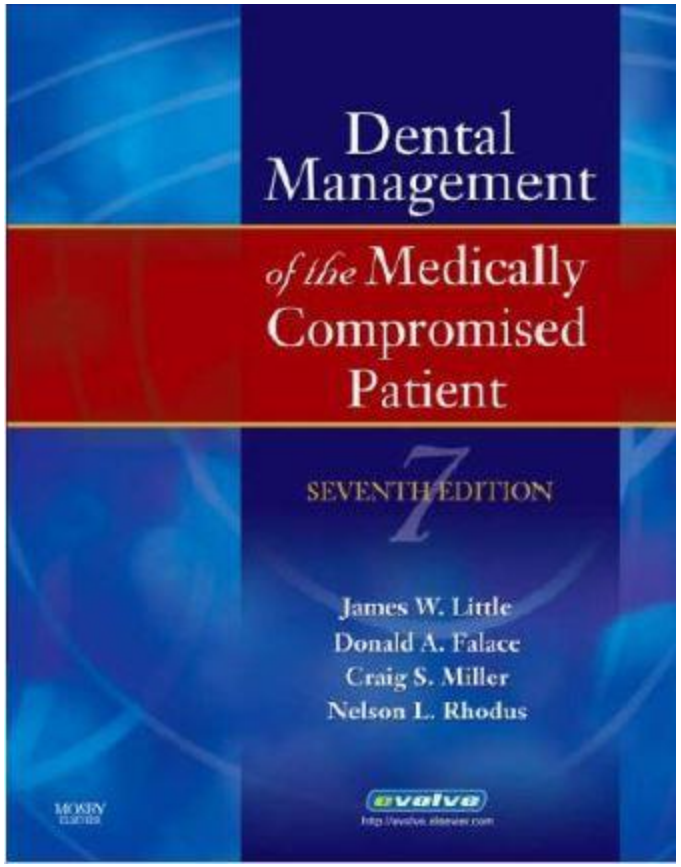
†Or other first- or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

‡Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.

IM = intramuscular; IV = intravenous.

References

https://drive.google.com/file/d/1EhhhpHhK5CiP9uQdEaT3DGI1JfK48adE/view?usp=sharing



THANK YOU