SOFT TISSUE AND BONE-JOINT PATHOLOGY

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OBJECTIVES

1. Describe anatomy and cellular structure of bone, joint and soft tissue.

2. Describe mechanism and function of bone, joint and soft tissue.

3. Diagnosis of clinical pathology of bone, joint and soft tissue diseases.

All information in class and related articles can be the exam in both Lecture and Lab part.

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OUTLINE

Orthopedic pathology
  • Bone & Cartilage
  • Soft tissue & near by;
    • Synovium
    • Tendon
    • Bursa
    • Fibrous tissue
    • Muscle
BONE DISEASE

1) Developmental abnormalities in bone cells, matrix and structure
2) Fractures
3) Osteonecrosis (Avascular necrosis)
4) Infections: Osteomyelitis
5) Bone tumors
OUTLINE

JOINT DISEASE

1) Arthritis
2) Tumors

SOFT TISSUE DISEASES

1) Adipose Tumors
2) Fibrous Tumors
3) Muscle Tumors
4) Tumors of Uncertain Histogenesis
BONE FUNCTION

- **Mechanical**
  - Protection
  - Structure
  - Movement
  - Sound transduction

- **Synthetic**
  - Blood production

- **Metabolic**
  - Mineral/Growth factor/Fat storage
  - Acid-basic balance
  - Detoxicication
  - Endocrine organ - Osteocalcin
TYPES OF BONE

According to shape & functions

1. Long bone (tubular); humerus, femur, tibia
2. Short bone (cuboidal); carpal bones, tarsal bones
3. Flat bone; skull
4. Irregular bone; bones of face
5. Sesamoid bone; patella, pisiform

According to collagen forming

1. Woven bone
2. Lamellar bone
   a) Compact or cortical bone
   b) Spongy or trabecular or cancellous bone
WOVEN VS. LAMELLAR

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CELLULAR STRUCTURE

1. OSTEOPROGENITOR ("STEM")$\rightarrow$(TGF-$\beta$)

2. OSTEOBLASTS (surface of spicule), under control of calcitonin to take blood calcium and put it into bone

3. OSTEOCYTES (are osteoblasts which are now completely surrounded by bone)

4. OSTEOCLASTS (macrophage lineage), under control of Parathyroid hormone (PTH) to chew up the calcium of bone and put it into blood
OSTEOBLASTS/OSTEOCYTES

osteoprogenitor cell → osteoblast → osteocyte

http://www.histology.leeds.ac.uk
http://faculty.une.edu

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OSTEOCLASTS

OSTEOCLASTS

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http://www.pathologyoutlines.com
BONE REMODELING

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OSTEOCLAST FORMATION & FUNCTION


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Bone Disease

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BONE DISEASES:

DEVELOPMENTAL ABNORMALITIES IN BONE CELLS, MATRIX AND STRUCTURE

1) MALFORMATIONS AND DISEASES CAUSED BY DEFECTS IN NUCLEAR PROTEINS AND TRANSCRIPTION FACTORS, polydactyly, syndactyly, absence of a bone

2) DISEASES CAUSED BY DEFECTS IN HORMONES AND SIGNAL TRANSDUCTION MECHANISMS, achondroplasia, thanatophoria

3) DISEASES ASSOCIATED WITH DEFECTS IN EXTRACELLULAR STRUCTURAL PROTEINS
   - Type 1 Collagen Diseases (Osteogenesis Imperfecta)
   - Types 2, 10, and 11 Collagen Diseases

4) DISEASES ASSOCIATED WITH DEFECTS IN FOLDING AND DEGRADATION OF MACROMOLECULES
   - Mucopolysaccharidoses

5) DISEASES ASSOCIATED WITH DEFECTS IN METABOLIC PATHWAYS (ENZYMES, ION CHANNELS AND TRANSPORTERS)
   - Osteopetrosis

6) DISEASES ASSOCIATED WITH DECREASED BONE MASS
   - Osteoporosis

7) DISEASES CAUSED BY OSTEOCLAST DYSFUNCTION
   - Paget's Disease (Osteitis Deformans)

8) DISEASES ASSOCIATED WITH ABNORMAL MINERAL (Ca++) HOMEOSTASIS
   - Rickets and Osteomalacia
   - Hyperparathyroidism
   - Renal Osteodystrophy
DEFECTS IN NUCLEAR PROTEINS AND TRANSCRIPTION FACTORS

Congenital absence of a bone (usually single): phalanx, rib, clavicle

Supernumerary digit (polydactyly)

Syndactyly

CRANIORACHISCHISIS


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ACHONDROPLASTIC “DWARF”  
(non-lethal, FGFR3 mutation)  
[Fibroblast growth factor receptor 3]  
PHENOTYPE: Short stature, rhizomelic shortening of limbs, frontal bossing, midface deficiency

THANATOPHORIC “DWARF”  
(often lethal, missense mutation in FGFR-3)  
Severe skeletal disorder in infant  
PHENOTYPE: Severe limb shortening and bowing, frontal bossing, depressed nasal bridge
DEFECTS IN EXTRACELLULAR STRUCTURAL PROTEINS

The main functions of collagen 1, 2, 3, 4: bone, cartilage, basement membrane floor (four).

**Type 1 Collagen Diseases (Osteogenesis Imperfecta)**

Mutations in genes which code for the $\alpha_1$ and $\alpha_2$ chains of COLLAGEN 1

**OSTEOGENESIS IMPERFECTA TYPES**

(“Brittle” bone disease, too little bone), *BLUE* sclerae

**Diseases Associated with Mutations of Types 2, 9, 10, and 11 Collagen**

Manifestation as CARTILAGE diseases, ranging from joint cartilage destruction to fatal sequelae
OSTEOGENESIS IMPERFECTA

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DEFECTS IN FOLDING AND DEGRADATION OF MACROMOLECULES

(GLYCOSAMINOGLYCANS)

MUCOPOLYSACCHARIDOSIS (one of MANY lysosome storage diseases)

DECREASES in ENZYMES which degrade:

• DERMATAN
• HEPARAN
• KERATAN

Chiefly CARTILAGE disorders: short, chest wall, malformed bones
MUCOPOLYSACCHARIDOSES


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DEFECTS IN METABOLIC PATHWAYS

- ENZYMES
- ION CHANNELS
- TRANSPORTERS

OSTEOPETROSIS (ROCK hard bone)

- also known as in marble bone disease and Albers-Schönberg disease
- One common one has a CARBONIC ANHYDRASE deficiency, i.e., \( \downarrow \text{acid} \)
- DECREASED osteoclast resorption
- “MARBLE” bone, increased bone, brittle, sclerotic bone

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OSTEOPETROSIS

cortex

medullary cavity

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DECREASED BONE MASS

OSTEOPOROSIS

“PEAK” bone mass is early adulthood
Normal decline, slow

Osteoporosis is accelerated bone loss

Factors:

- AGE
- Physical activity
- Estrogen withdrawal (menopause)
- Nutrition (Ca++)
- Genetics

http://health.rush.edu/
OSTEOCLAST DYSFUNCTION

PAGET’ S DISEASE

By John Kelly Coffman


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OSTEOCLAST DYSFUNCTION
PAGET’S DISEASE
(OSTEITIS DEFORMANS)

Matrix madness, Osteoblasts/cytes
gone wild

THREE PHASES:
1) Increased osteoclast resorption
2) Increased “hectic” bone formation
   (osteoblasts)
3) Osteosclerosis

ELEVATED ALKALINE-PHOSPHATASE
ELEVATED urine HYDROXYPROLINE

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Bone scan activity is DIRECTLY proportional to OSTEOBLASTIC activity.

NON-Lamellar bone

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PAGET’s DISEASE (of BONE)
85% MONOSTOTIC, WHOLE BONE
15% POLY-OSTOTIC (skull, pelvis)
“JIGSAW”, NOT LAMINAR, BONE

CLINICAL: PAIN!!!
(MICROFRACTURES)

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ABNORMAL MINERAL HOMEOSTASIS

• Ricketts (Children) and Osteomalacia (Adults)
  • VITAMIN D deficiency/dysfunction

• Hyperparathyroidism, PRIMARY
  (PTH ADENOMA)
  • ENTIRE SKELETON
  • OSTEITIS FIBROSIS CYSTICA (von Recklinghausen’s disease (of bone)
  • “BROWN” TUMOR – due to increased hemosiderin

• Hyperparathyroidism, SECONDARY (RENAL)
  (NOT AS SEVERE AS 1º)

• Renal Osteodystrophy = ANY bone disorder
due to chronic renal disease
PRIMARY HYPERPARATHYROIDISM

OSTEITIS FIBROSA CYSTICA

“BROWN” “TUMOR”


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II. FRACTURES

Complete/Incomplete
Closed/Open (communicating)
Communited (splintered, “greenstick”)
Displaced (NON-aligned)

PATHOGENIC, (non-traumatic, 2º to other disease, often metastases)

“STRESS” fracture

THREE PHASES

- HEMATOMA, minutes days → PDGF, TGF-β, FGF
- SOFT CALLUS (“PRO”-CALLUS), ~1 week
- HARD CALLUS (BONY CALLUS), several weeks

COMPLICATIONS

- PSEUDARTHROSIS (non-union)
- INFECTION (especially OPEN [communicating] fractures)
OSTEONECROSIS

Also called AVASCULAR necrosis
Also called ASEPTIC necrosis

CAUSE:  ISCHEMIA

• Trauma
• Steroids
• Thrombus/Embolism
• Vessel injury, e.g., radiation
• INCREASED intra-osseous pressure→vascular compression
• Venous hypertension too
OSTEONECROSIS


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OSTEONECROSIS

Normal

Ischemia

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IV. OSTEOMYELITIS (INFECTION)

• May be a complication of any systemic infection but frequently manifests as a primary solitary focus of disease.
• All types of organisms including viruses, parasites, fungi and bacteria can cause of osteomyelitis
• THE MOST COMMON;

1. PYOGENIC OSTEOMYELITIS
2. TUBERCULOUS OSTEOMYELITIS
3. SKELETAL SYPHILIS
PYOGENIC OSTEOMYELITIS

MOST caused by BACTERIA;

*E.coli*, *Klebsiella* and *Pseudomonas* are more frequently isolated from patients with genitourinary tract infections or with intravenous drug abusers.

**Mixed bacterial infections** can be seen in the setting of direct spread during surgery or open fractures.

*Salmonella* infections for unknown reasons common in sickle cell patients.

In 50% of the cases no organisms can be isolated.

**ORGANISMS MAY REACH THE BONE BY;**

- Hematogenous
- Contiguous, e.g., from a nearby joint
- Direct implantation
TUBERCULOUS OSTEOMYELITIS

Routes of entry;

- Usually **BLOOD BORNE** and originate from a focus of active visceral disease.
- **DIRECT EXTENSION** (e.g. from a pulmonary focus into a rib or from tracheobronchial nodes into adjacent vertebrae) or spread via draining lymphatics.

In patients with AIDS frequently multifocal.

**POTT DISEASE** is the involvement of spine.

Thoracic and lumber vertebrae followed by the knees and hips are the most common sites of skeletal involvement.

The infection breaks through the invertebral discs and extends into the soft tissues forming abscesses.

Syphilis *(Treponema pallidum)* and yaws *(Treponema pertenue)* both can involve bone.

The syphilitic **SABER SHIN** is produced by massive reactive periosteal bone deposition on the medial and anterior surfaces of the tibia.

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BONE DISEASES:

V. TUMORS

BONE

CARTILAGE

FIBROUS

MISCELLANEOUS TUMORS

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http://sarcoma.org
BONE TUMORS

OSTEOMA

OSTEOID OSTEOMA (nidus)

OSTEOBLASTOMA

OSTEOSARCOMA

(Osteogenic sarcoma)
OSTEOSARCOMA (OSTEEOGENIC SARCOMA)

LATE TEENS
KNEES
METAPHYSIVES
PAINFUL!!!
The most common subtype is osteosarcoma that arises in the metaphysis of long bones; is primary, solitary, intramedullary, and poorly differentiated; and produces a predominantly bony matrix.

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TAKE A BREAK
JOINT DISEASES

1. DEGENERATIVE ARTHRITIS (OSTEOARTHRITIS)
2. RHEUMATOID ARTHRITIS
3. ALL OTHER ARTHRITIS
   1) JUVENILE RHEUMATOID
   2) NON-INFECTIONOUS: Ankylosing Spondylitis, Reactive and Psoriasis
   3) INFECTIOUS: Supp., TB, Lyme, Viral
   4) GOUT (URATE)
   5) PSEUDOGOUT (PYROPHOSPHATE)
4. TUMORS (all are of synovium)
   1) Ganglion (Synovial Cyst), non-neoplastic
   2) Giant Cell Tumor (Pigmented VilloNodular Synovitis [PVNS]), benign
   3) Synovial Sarcoma, malignant
I. DEGENERATIVE ARTHRITIS ("OSTEO" ARTHRITIS)

Etiology/Risk Factors: Age, Trauma, Genes

Pathogenesis: Progressive EROSION of articular cartilage

Morphology: X-Ray, "eburnation" (degenerative), "joint mice" (bone fragment in the joint), osteophytes (bone spurs)

Clinical Expression: PAIN, Limitation of motion
NORMAL

capsule

cartilage

synovium

bone

OSTEOARTHRITIS

thickened capsule

cyst formation and
sclerosis in
subchondral bone

shelving ‘fibrillated’
cartilage

osteophytyic lipping

synovial hypertrophy

altered contour
of bone

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RHEUMATOID ARTHRITIS

- A chronic **systemic** inflammatory disorder
- Affect many tissues and organs—skin, blood vessels, heart, lungs, and muscles—but principally attacks the joints, producing a nonsuppurative proliferative and inflammatory synovitis that often progresses to destruction of the articular cartilage and ankylosis of the joints.
II. RHEUMATOID ARTHRITIS

Etiology/Risk Factors: Autoimmune
Pathogenesis: Progressive SYNOVITIS
Morphology: Synovial lymphocytes, macrophages, plasma cells, neutrophils, osteoclasts, pannus, hyperemia, rheumatoid nodules, vasculitis
Clinical Expression: PAIN, Limitation of motion, malaise, fatigue, rheumatoid factor IgM→IgG-Fc
The rheumatoid “nodule” shows “palisading” fibroblasts.
DIAGNOSIS

CLINICAL FEATURES (1% of population F>>M)

- MORNING STIFFNESS, MEAN AGE 45 YRS
- ARTHRITIS in MORE THAN 3 JOINT AREAS
- “TYPICAL” hand findings, metacarpophalangeal (MP) ULNAR deviation
- SYMMETRIC ARTHRITIS
- SERUM RHEUMATOID FACTOR
- “TYPICAL” X-RAY findings
ULNAR DEVIATION of MP joints is MOST consistent reliable finding. The main area of arthritis in NOT DIP or PIP, but MP
3.1) JUVENILE RHEUMATOID ARTHRITIS

- Begins BEFORE age 16, by definition
- Generally LARGER joints than RA
- Often POSITIVE ANA
ANKYLOSING SPONDYLITIS [HLA-B27] (M>>F)
(Rheumatoid spondylitis or Marie-Strumpell disease)
• a chronic inflammatory disease of the axial skeleton with variable involvement of peripheral joints and nonarticular structures.
ANGLYLOSING SPONDYLITIS

REACTIVE ARTHRITIS [HLA-B27]

- An autoimmune condition that develops in response to an infection (cross reactivity)
  - Genital infection: *Chlamydia trachomatis*
  - Gastrointestinal infection: Enteric bacteria
- REITER SYDROME (urethral & conjunctival inflammation)
- Arthritis associated with IBD

PSORIATIC ARTHRITIS [HLA-B27]

- Chronic skin condition psoriasis (โรคสะเก็ดเงิน)

HYPERSENSITIVITY VS AUTOIMMUNE DISEASE

http://missinglink.ucsf.edu/lm/immunology_module/prologue/objectives/obj09.html
HYPERSENSITIVITY

Immediate (type I) hypersensitivity
Immediate (type I) hypersensitivity is a rapid IgE and mast cell-mediated vascular and smooth muscle response that occurs in genetically susceptible people. This type of reaction results from an excessive Th-2 response; we know these responses as "allergies."

Antibody-mediated (type II) hypersensitivity
Antibody-mediated (type II) reactions result when antibodies are directed against antigens on the surface of cells or other tissue components. The deposition of the antibody can have a variety of detrimental effects, including inflammation, opsonization and phagocytosis, or functional derangements.

Immune complex-mediated (type III) hypersensitivity
Immune complex-mediated (type III) hypersensitivity results when complexes of antibodies and antigens deposit in vascular walls or other tissues and cause an inflammatory response. This type of pathology is commonly implicated in vasculitis and arthritis.

Cell-mediated (type IV) hypersensitivity
Cell-mediated (type IV) hypersensitivity results from an inappropriate or excessive immune reaction that is mediated by a specific subsets of CD4+ helper T cells (Th-1 and Th-17 cells) or by CD8+ cytotoxic T cells. These reactions are the basis for diseases such as Crohn's disease and multiple sclerosis.

http://missinglink.ucsf.edu/lm/immunology_module/prologue/objectives/obj09.html

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3.3) **INFECTIOUS ARTHRITIS**

(A broader term)

- **SEPTIC/SUPPURATIVE ARTHRITIS** – Bacterial but viral, mycobacterial, and fungal arthritis occur occasionally.

- *Borrelia burgdorferi* can cause infectious arthritis, but is not associated with suppurative arthritis.
3.3) **INFECTIOUS ARTHRITIS**

Micro-organisms must reach the synovial membrane of a joint. This can happen in any of the following ways:

- Dissemination of pathogens via the blood, from abscesses or wound infections, or from an unknown focus
- Dissemination from an acute osteomyelitic focus
- Dissemination from adjacent soft tissue infection
- Entry via penetrating trauma
- Entry via iatrogenic means
3.3) INFECTIOUS ARTHRITIS

**BACTERIA**
- Staphylococcus aureus
- Streptococci
- Haemophilus influenzae
- Neisseria gonorrhoea
- Escherichia coli
- M. Tuberculosis
- Salmonella spp.
- Brucella spp.

**VIRAL**
- Parvovirus B19
- Rubella
- Hepatitis C
GOUT

Endpoint of HYPERURICEMIA from ANY cause resulting in JOINT deposition of monosodium urate crystals (TOPHI)

PATHOLOGY

- Acute arthritis
- Chronic tophaceous arthritis
- Tophi
- Gouty nephropathy

10% of population has hyperuricemia (>7 mg/dl), but only 1/20 of these has gout
Tophi: Note the mass containing a chalky substance and having an irregular outer surface.
A TOPHUS is a GRANULOMATOUS response to monosodium urate crystals.

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GOUTY NEPHROPATHY


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HYPERURICEMIA → GOUT

**Age** of the individual and duration of the hyperuricemia are factors. Gout rarely appears before 20-30 years of hyperuricemia. M >> F

**Genetic predisposition** is another factor. In addition to the well-defined X-linked abnormalities of HGPRT, primary gout follows multifactorial inheritance and runs in families.

**Heavy alcohol** consumption predisposes to attacks of gouty arthritis.

**Obesity** increases the risk of asymptomatic gout.

Certain **drugs (e.g., thiazides)** predispose to the development of gout.

**Lead toxicity** increases the tendency to develop gout.
PSEUDO-GOUT

Gout: Monosodium Urate

Pseudo-GOUT: Calcium Pyrophosphate

PSEUDOGOUT is also called CHONDROCALCINOSIS, or CPPD (Calcium Phosphate Deposition Disease)

IDIOPATHIC, HEREDITARY, SECONDARY

• Secondary joint damage, hyperparathyroidism, hemochromatosis, hypomagnesemia, hypothyroidism, ochronosis, and diabetes

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Gout and pseudogout are the common crystal-induced arthropathies. **Gout** is caused by monosodium urate monohydrate crystals. **Pseudogout** is caused by calcium pyrophosphate crystals.

Biurate has the OPPOSITE polarization pattern of pyrophosphate. In GOUT, as seen here, the yellow needles are vertical and the blue needles are horizontal, under polarization. IN PSEUDOGOUT, the OPPOSITE is seen.

Polarized Light Microscopy
http://www.microscopyu.com

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IV. JOINT TUMORS

BENIGN

• GANGLION (SYNOVIAL CYST)
• GIANT CELL TUMOR of TENDON SHEATH or Pigmented Villonodular Synovitis (PVNS)

MALIGNANT

• SYNOVIAL SARCOMA
4.1) **GANGLION** - Overuse of computer
4.2) GIANT CELL TUMOR OF TENDON SHEATH
(Giant-cell synovioma - benign tumor of synovial cells)
JOINT Tumors:

4.2) PIGMENTED VILLONODULAR SYNOVITIS

A joint disease characterized by inflammation and overgrowth of the joint lining (*hemosiderin-laden macrophages*)
4.3) SYNOVIAL SARCOMA

General

- Usually a deep seated mass present for years around large joints (80% in knee and ankle) in young adults (age 20-40), 60% male; only 10% actually involve the joint
- Represent 10% of adult soft-tissue tumors
- 5 year survival is 50-70%; 10 year survival 40%; recurs locally, 10-15% metastasize to lung and pleura, bone, regional nodes
- Rarely radiation associated (Mod Pathol 2002;15:998)
- Minute (< 1 cm) tumors of hands and feet: 2/3 female, median age 29 years, 2/3 monophasic, 40% have microcalcifications; EMA+, keratin+; have clinically favorable course if completely excised (Am J Surg Pathol 2006;30:721)

Prognostic factors

- High histologic grade (based on MIB1 index and necrosis)
- SYT-SSX1 vs. SYT-SSX2 gene fusion
SYNOVIAL SARCOMA

4.3) SYNOVIAL SARCOMA

- A 28-year-old male patient
- Swelling in the region of angle of mandible on right side since 1 year
- Patient also complained of occasional pus discharge intraorally and tingling sensation on the lower lip, as well as loss of appetite and weight loss.
- **History:** Surgical procedure on the right side 3 years back for ameloblastoma (benign tumor of odontogenic epithelium or ameloblasts). A peanut-sized swelling 1 year back. The swelling grew to the present size of 4 x 3 cm over a period of 1 year.
- Lymph node measuring 26 x 24 mm.
- Intraorally there were no significant findings. The teeth and the mandible on the affected side were missing and no draining sinuses were detected intraorally.

Epithelial cells had large, round, vesicular nuclei and pale staining cytoplasm.

Some of the epithelial cells formed glandular structures that included a central area of cystic degeneration.

In some areas, epithelial cells were seen lining cyst-like spaces in abundant connective tissue stroma.
SYNOVIAL SARCOMA

Immunohistochemical analysis

- Positivity for pancytokeratin in epithelial cells and spindle cells, and vimentin was positive for only the fibrous stroma
- A weak reactivity was observed with epithelial membrane antigen and carcinoembryonic antigen. A final diagnosis of biphasic synovial sarcoma was made because the spindle cells and epithelial cells were both positive for pancytokeratin.

SOFT TISSUE DISEASES

1) Adipose Tumors
2) Fibrous Tumors
3) Muscle Tumors
4) Tumors of Uncertain Histogenesis
SOFT TISSUE TUMORS

FAT
FIBROUS TISSUE
FIBROHISTIOCYTIC
SKELETAL MUSCLE
SMOOTH MUSCLE
VASCULAR
PERIPHERAL NERVE

 UNCERTAIN: SYNOVIAL SARCOMA, ALVEOLAR SOFT
PART SARCOMA, EPITHELIOLID SARCOMA
CAUSES OF SOFT TISSUE TUMORS
MOSTLY UNKNOWN

RADIATION association
CHEMICAL BURN association
THERMAL BURN association
TRAUMA association

VIRUS association (HHV-8 for Kaposi’s sarcoma-associated herpesvirus (KSHV))

GENETICS

Parts of many SYNDROMES
MANY TRANSLOCATIONS

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SOFT TISSUE TUMORS

ALL “SPINDLY”

Deep (desmoid) vs. Superficial (skin)

Importance of counting MITOSES

Importance of STAGING

Importance of IMMUNOPEROXIDASE

Importance of CONSULTATION
FAT

LIPOMA
LIPOSARCOMA

NORMAL FAT
LIPOMA, encapsulated
LIPOSARCOMA, often retroperitoneal


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FIBROUS TISSUE

NODULAR FASCIITIS (pseudosarcomatous)

FIBROMATOSES (plantar, palmar, penile)

FIBROSARCOMA
MYOSITIS OSSIFICANS
BENIGN FIBROUS TISSUE PROLIFERATION
PLUS OSSEOUS “METAPLASIA”

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FIBROHISTIOCYTIC
FIBROUS HISTIOCYTOMA
DERMATOFIBROSARCOMA PROTUBERANS
MALIGNANT FIBROUS HISTIOCYTOMA

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SKELETAL MUSCLE

RHABDOMYOMA

RHABDOMYOSARCOMA

SMOOTH MUSCLE
LEIOMYOMA
LEIOMYOSARCOMA

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LEIOMYOMA

Images by Department of Pathobiology, Faculty of Science, Mahidol University

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QUESTIONS?