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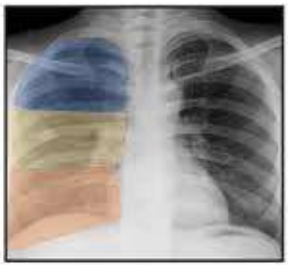



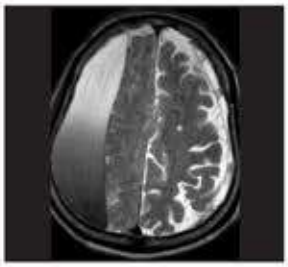





Exam Preparatory Manual for Undergraduates

Surgery

As per the Competency Based Medical Education Curriculum (NMC)

HIGHLIGHTS

- Case-based MCQs at the end of each chapter
- Online **OSCE** practice assessment
- Line diagrams, tables, and flowcharts for better understanding
- An ultimate guide for **NEET PG/NExT, MRCS and USMLE examinations.**

Editor
Gunjan S Desai

JAYPEE

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CHAPTER 7

Surgical Infections

TUBERCULAR LYMPHADENOPATHY

Causes of Generalized Lymphadenopathy

Infections	• Tuberculosis, brucellosis, syphilis, HIV, infectious mononucleosis • Toxoplasmosis
Malignancy	Lymphoma, leukemia
Autoimmune disorders	• Systemic lupus erythematosus (SLE), rheumatoid arthritis, sarcoidoses • Amyloidoses
Other	Metastasis from various malignancies, Kikuchi disease

Tubercular Cervical Lymphadenopathy

- Very common cause of cervical lymphadenopathy caused by *M. tuberculosis*.
- Children are more commonly involved. Can affect individuals from any age group.

Clinical Features

- Fever with evening rise, anorexia, weight loss, chronic cough with sputum production might precede the appearance of cervical lymph nodes.
- Initially the nodes are enlarged and discrete. This is due to lymphadenitis without periadenitis.
- Then, lymph nodes become matted due to periadenitis and adhesions.
- There is no sign of inflammation and the nodes are nontender.
- Upper jugular cervical nodes are most commonly involved.

Natural History

- If the disease progresses unhindered, then after matting of lymph nodes, caseous necrosis leads to abscess formation which is known as **cold abscess.**
- Further progression lead to tracking of this pus along the fascial layers of neck. Because of the plane of lymph nodes deep to deep cervical fascia, the pus also travels down deep to this fascial layer.
- Further increase in pus leads to increased tension and rupture of deep cervical fascia with tracking of pus in superficial fascia. This is known as **collar-stud abscess.** It shows cross-fluctuation.

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- Finally, the pus ruptures through the superficial fascia and skin and presents as **tubercular sinus or ulcer.**

Investigations

- Chest X-ray, sputum for AFB and culture, Mantoux test, ESR, total leukocyte count with differential count, hemoglobin level.
- FNAC of the lymph node and if it is inconclusive, excision or incision biopsy.
- CECT neck and chest will show the entire extent in cases with cold abscess.

Treatment

- Antitubercular medications as per the prevalent TB management guidelines.
- Local management of abscess done by antigravity aspiration if it is large or does not resolve on antitubercular treatment.
- Excision is done for enlarged lymph nodes if they do not resolve with ATT.

LUDWIG'S ANGINA

Definition

Rapidly spreading cellulitis of floor of mouth and submandibular space (submaxillary or submental and sublingual divided by mylohyoid) secondary to soft tissue infection/tonsil infection/lower premolar or molar infection (80%)/submandibular sialadenitis or injuries to the oral mucosa.

Symptoms

- Trismus and odynophagia
- Excessive salivation
- Tongue is pushed upwards and backwards due to edema of the floor of the mouth.

Extent

- Can go to parapharyngeal and retropharyngeal spaces.
- Laryngeal edema and aspiration pneumonia
- Septicemia

On Examination

- Tongue is pushed backwards.
- Woody hard feel of submandibular and submental spaces.

Organisms

- Mixed infection of aerobes and anaerobes
- Dental origin—*Streptococcus viridians* and *E. coli*

Treatment

- Systemic antibiotics
- Tracheostomy if airway is endangered.
- Incision and drainage should be delayed as long as possible, as pus is seldom found.
- Submaxillary space (superficial to mylohyoid)—transcutaneous drainage through submandibular skin fold.
- Sublingual space (deep to mylohyoid)—drained through floor of mouth.

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TETANUS: PROPHYLAXIS AND MANAGEMENT

- Caused by *Clostridium tetani*—gram positive, anaerobic, terminal spore-bearing.
- Produces exotoxin called tetanospasmin which on reaching the neural tissue acts on spinal cord, brain, sympathetic system and motor end plate of muscles to block normal inhibitory pathways of these nerves and lead to the characteristic muscular spasms.
- Spores reside in soil and dust and enters body through contaminated wound and injuries or chronic ulcers or dental extraction sites or in injection drug abusers through injection sites from where they germinate. This entire life cycle takes around 5–10 days in body.
- Some specific modes of transfer give the tetanus its name:
  - **Tetanus neonatorum**—infection through infected cut surface of umbilical cord.
  - **Puerperal or postabortal tetanus**—infection through unsterile uterine instrumentation.
  - **Otogenic tetanus**—infected material to clean ear can introduce infection.

Clinical Features

- **Most early symptom—**trismus (Lock jaw)
- Anxious expression of face (Risus sardonicus or sardonic grin), opisthotonos
- **Constitutional symptoms:** Elevated temperature, tachycardia, tachypnea, cyanosis.
- Rigidity of muscles of lower limb and abdomen. Gradually all the muscles develop rigidity and death is due to respiratory arrest due to laryngeal spasm.
- **Other causes of death:** Aspiration pneumonia, lung infections, or hyperpyrexia.

Management of Established Case

- Patient should be admitted to a quiet, dark and well-ventilated room.
- Care of airway and breathing.
- Sedation with diazepam/barbiturate is frequently used to avoid provocation of spasms.
- Foley catheter, feeding tube, muscle relaxation and tracheostomy, IV fluids and antibiotics as per requirement. These are needed in severe cases.
- General nursing care of the patient till he stabilizes and is off ventilator.

Specific Treatment

- Passive immunization with human anti-tetanus globulin 4,000 units or equine anti-tetanus serum 1 lac units half IV and half IM should be administered with tetanus toxoid.
- Crystalline penicillin 20 lac unit 6 hourly or 10 lac unit 4 hourly should be given.
- Wound debridement

Prophylaxis

Means: Immunization, antibiotic and wound cleansing and **debridement (most important).**

Active immunization	*Passive immunization*
• Aluminum phosphate adsorbed tetanus toxoid 0.5 mL in left deltoid is the best agent for active immunization. • 3 doses at 4 weeks interval and then booster after 1 year completes the routine requirement. Booster is given at every 4–5 years to maintain immunity. Immunization schedule is 6, 10, 14 weeks age, 18 months age, 5 years age and 10 years age.	• Equine anti-tetanus serum 1,500 units SC/IM after negative sensitivity testing. • Human anti-tetanus globulin 250–500 Units IM is a homologous antitoxin and does not require sensitivity testing. It is many times more protective than equine serum and is therefore preferred.

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Plan of Immunization After Injury

- A patient who has been completely immunized as above within past 5 years needs no tetanus immunization prophylaxis. Manage only with wound debridement.
- A patient who has been completely immunized in past with a clean nonpenetrating wound <6 hours duration needs only a single tetanus toxoid injection and wound debridement.
- A patient who has been completely immunized in 5–10 years range with a dirty, penetrating or old wound needs only a single tetanus toxoid injection and wound debridement but, if it is >10 years than he needs single shot tetanus toxoid with anti-tetanus globulin.
- A patient whose immunization history is not known needs complete tetanus immunization course if he has a clean nonpenetrating wound <6 hours duration and complete tetanus immunization course with anti-tetanus globulin for old, penetrating or dirty wounds.

GAS GANGRENE

Gas gangrene is caused by *Clostridium perfringens/Clostridium welchii* and other clostridia species such as *C. septicum, C. histolyticum*, etc., anaerobic organisms also called "flesh-eating bacteria".

Pathogenesis

- These organisms enter the body through wounds which are grossly contaminated with soil/foreign bodies which provide the low tissue resistance, low pH, anaerobic environment and high calcium due to cell lysis in the wound necessary for clostridial growth.
- The organisms are also present in stools and female genital organs and therefore wounds that come in contact with these areas and body fluids are more predisposed.
- Produce exotoxins which cause characteristic gas gangrene. These include lecithinase (alpha toxin), hyaluronidase, collagenase, other hemolytic and leukocidal toxins.
- Occlusion of end arteries supplying wound, accumulation of wound exudates and inadequate wound debridement are factors that increase risk of gas gangrene.
- Diabetes and chronic arterial occlusive diseases also predispose.

Stages of Disease Progression

- Stage of contamination. No symptoms and signs.
- Stage of anaerobic clostridial cellulitis. No systemic symptoms.
- Spreading cellulitis and fasciitis with systemic symptoms and signs
- Clostridial myonecrosis
- Bacteremia and septicemia.

Clinical Features

- It presents as rapidly spreading infective gangrene with accumulation of gas in muscles and subcutaneous tissue and is called clostridial myonecrosis.
- Initially can present as clostridial cellulitis with edema of subcutaneous tissue and spreads rapidly to involve the muscles and subcutaneous tissue rapidly.
- Can affect the liver (foaming liver).
- Constitutional symptoms such as pyrexia, anxiety, tachycardia, vomiting and hypothermia are initial symptoms.
- Pain out of proportion to the appearance of wound, swelling and edema of the affected part, profuse, foul smelling, brown or gray color discharge called "dishwater pus" is characteristic. Finally, the wound becomes black in color marking the phase of gas gangrene.
- On examination, crepitus is almost always present and is pathognomonic.

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Investigations

- Gram stain of wound exudates reveal the organisms.
- Anaerobic cultures are positive. Nagler reaction is used to detect *C. welchii*.

Management

Treatment of a Diagnosed Case

- Wound drainage and debridement can extend from extensive debridement to even guillotine type amputation as a life-saving measure in cases of severe myonecrosis.
- Crystalline penicillin 10 lac unit 4 hourly or 20 lac unit 6 hourly
- Anti-gas gangrene serum 22,500 IU (contains 9,000 units *C. welchii*, 4,500 units *C. septicum* and 9,000 units *Cl. oedematiens*) stat and repeated at 6 hours interval for 3 consecutive doses.
- Hyperbaric oxygen therapy is also used once the wound is noninfective and healthy.

Prophylaxis

- Wound drainage and debridement to remove all devitalized tissues and foreign bodies.
- Crystalline penicillin 10 lac unit 4 hourly or 20 lac unit 6 hourly
- Anti-gas gangrene serum 22,500 IU (contains 9,000 units *C. welchii*, 4,500 units *C. septicum* and 9,000 units *Cl. oedematiens*) stat single dose is suggested but not of proven role.

NECROTIZING FASCIITIS

Important Concepts

- **Cellulitis:** Mild skin infection with erythema and inflammation of epidermis and dermis.
- **Erysipelas:** Red, tender, raised well-demarcated streaks along lymphatic caused by *Streptococcus pyogenes.*
- **Impetigo:** Caused by beta-hemolytic streptococci/*Staphylococcus aureus* leading to purulent skin lesions.
- **Necrotizing cellulitis:** Skin and subcutaneous fat infection with microvascular thrombosis and necrosis due to *Staphylococcus aureus* and beta-hemolytic streptococci.
- **Necrotizing fasciitis:** Subcutaneous tissue, fascia involvement and initial skin sparing with microvascular thrombosis and necrosis.

Definition

Rapidly progressive infection involving subcutaneous tissue and fascia and later skin that spreads along the fascial planes resulting in microvascular thrombosis, tissue necrosis and sepsis.

Predisposing Conditions

- Diabetes, obesity
- Immunosuppressed states, steroids, malnutrition

Precipitating Factors

- Blunt or penetrating trauma
- Burns
- Skin laceration, insect bite, surgery site

Pathophysiology

- Gram-positive and gram-negative aerobes, anaerobes and fungi all can cause.

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- Deep fascial planes are the primary site of infection. Bacterial infection leads to destruction of tissue along fascial planes and microvascular thrombosis.
- This results in ischemia and ischemic damage of overlying tissue due to blockage of fasciocutaneous vessels.

Types

- **Type 1:** Polymicrobial
- **Type 2:** Monomicrobial gram-positive infection
- **Type 3:** Monomicrobial gram-negative infection
- **Type 4:** Fungal infection

Clinical Features

- History to identify predisposing and precipitating factors.
- More common in males and in lower half of body.
- Pain out of proportion of the physical findings is pathognomonic.
- Initially has brawny induration, tenderness, erythema and crepitus on palpation.
- Later develops bulla and vesicles with hemorrhagic discharge and "dish water pus", finally skin necrosis and gangrene.
- Systemic signs of sepsis.
- Necrotizing fasciitis of perineum is called **Fournier gangrene** and necrotizing fasciitis of anterior abdominal wall is called **Meleney's gangrene**.

Diagnosis

- Clinical findings are diagnostic in most cases and no further studies are necessary.
- If the diagnosis is equivocal, tissue biopsy (gold standard) or demonstration of gas in soft tissue by radiographic tests such as X-ray, CT or MRI can be helpful but not mandatory.
- Laboratory risk indicator for necrotizing fasciitis (LRINEC) is a tool developed for diagnosis. This includes parameters such as CRP, hemoglobin, leukocyte count, serum creatinine, sodium and glucose. Score >5 is suspicious and >7 is diagnostic.

Management

- Sepsis protocol has to be initiated.
- Urgent, complete, radical surgical debridement to bleeding margins with wound and blood cultures as well as tissue biopsy with empiric broad-spectrum antibiotics: life-saving.
- In many cases, a repeat exploration is required after 24–36 hours to confirm completeness of surgery and to excise further if required.
- Tetanus immunization, gas gangrene immunization and systemic support in ICU.
- Amputation is the last resort as a life-saving measure.
- Patients with perineal wounds may require colostomy to prevent contamination
- Once the patient stabilizes, options for wound management include vacuum-assisted closure devices (VAC), flap reconstruction or skin grafting.
- Hyperbaric oxygen therapy once the wound is noninfected, to help in healing.

CARBUNCLE

Carbuncle is caused by staphylococcal infection (*S. aureus*)—aerobic, gram-positive cocci in clusters and is an infection of subcutaneous tissue.

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Pathogenesis

- The organism penetrates deep into subcutaneous tissue and produces multiple interlinked abscesses that open separately on surface to produce a sieve-like appearance.
- They coalesce in center and produce the wound.
- This can extend peripherally and assume large sizes.

Clinical Features

- Patients are commonly diabetic males. Constitutional symptoms mark the onset.
- Common sites are nape of neck, back, dorsum of hand, shoulder, chest and abdomen.
- Begins as a painful, edematous subcutaneous swelling and progresses to a sieve-like appearance which is characteristic of carbuncle.
- The openings can finally enlarge, coalesce and form an ulcer.

Investigations

- Wound smears and culture sensitivity.
- Blood culture.
- Diabetic workup.

Treatment

- Control of diabetes.
- Wound debridement and dressing—cruciate incision and deroofing.
- Culture-specific antibiotic therapy.

MADURA FOOT

Madura foot is infectious condition affecting foot. It is also known as mycetoma.

Causes

Eumycetoma	*Actinomycetoma*
• Organism—*Madurella mycetomatis* • Disease is slowly progressive type • Treatment is antifungal therapy for prolonged periods	• Organism—*Actinomyces, Nocardia* • Disease is rapidly spreading and extensive • Treatment is amikacin + co-trimoxazole (first line) or rifampicin or amoxyclav (second line)

Entry and Route of Spread

- Inoculation from trauma site.
- From here the disease localizes in the site and further spread is via lymphatics as well as along the fascial planes.

Clinical Features

- **Triad** of noncontagious multiple sinuses, multiple painless subcutaneous masses and seropurulent discharge.
- **Site:** Foot is the disease site in most cases. Other sites are perineum, arm, leg, head and neck, thigh and hand.
- Tendons and nerves are spared till very late in the disease.
- Secondary bacterial infection can complicate the local disease.
- The disease spreads along the fascial planes so what is a small lesion superficially might be extending for large areas deeply.

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Diagnoses

- **X-ray**—areas of calcification can be seen. Also, areas of punched out lesions in bone can be seen.
- **Ultrasound**
- **MRI**—shows "dot in circle" sign.
- **Biopsy:**
  - *Type 1 mycetoma:* Multiple neutrophils surrounded by lymphocytes and mononuclear cells surrounded by fibroses.
  - *Type 2 mycetoma:* There are no neutrophils in the lesion. It only contains macrophages.
  - *Type 3 mycetoma:* It contains epithelioid granulomas with giant cells.
- **FNAC** can also be done but biopsy is more reliable.
- **Culture-sensitivity** is done to help in selection of culture-specific antibiotics.

Treatment

- Antibiotics or antifungals as written above.
- Surgical debridement under general or spinal regional anesthesia. Use of local anesthesia is contraindicated.

ANATOMY OF PALMAR SPACES (FIG. 7.1)

- Arrangement of the fascia and fascial septa in the hand.
- They may become infected and distended with pus.
- The three palmar septa divide the palmar space into thenar and midpalmar spaces.

Midpalmar and Thenar Spaces

- **Triangular spaces** of which base lies distally and the apex is directed proximally
- **Thenar space:** Lies between the lateral and intermediate palmar septa.
- **Midpalmar space:** Lies between the intermediate and medial palmar septa.

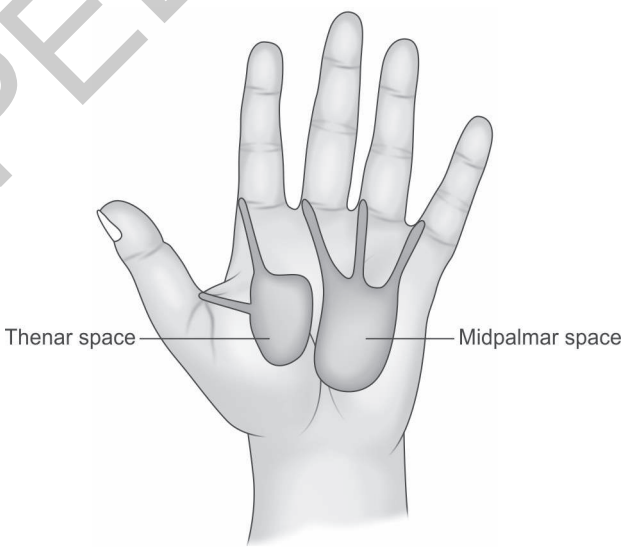

Fig. 1: Anatomy of palmar spaces.

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Boundaries of Thenar Space

- **Medially:** Intermediate palmar septum.
- **Laterally:** Lateral palmar septum.
- **Anteriorly:** Lateral part of palmar aponeurosis, and flexor tendons to index finger.
- **Posteriorly:** Adductor pollicis, transverse head.

Boundaries of Midpalmar Space

- **Medially:** Medial palmar septum.
- **Laterally:** Intermediate palmar septum.
- **Anteriorly:** Medial part of palmar aponeurosis, and flexor tendons to medial three fingers.
- **Posteriorly:** Fascia covering the medial three metacarpal bones and intervening interosseous muscles.

Extent of Spaces

- Proximally, midpalmar and thenar spaces extend up to distal margin of flexor retinaculum.
- Distally, thenar space extends up to the proximal transverse crease of the palm, and the midpalmar space extends up to the distal transverse crease.

Communications of Spaces

- The spaces are normally closed at the proximal end.
- Midpalmar space may communicate with forearm space through carpal tunnel.
- The two spaces of the palm quite frequently communicate with each other.

Contents of the Thenar and Midpalmar Spaces

- Normally filled mainly with loose connective tissue.
- They are closely related to lumbrical muscles. Thenar space contains first lumbrical muscle, while midpalmar space contains 2nd, 3rd and 4th lumbrical muscles.

Clinical Correlations

- Infection of midpalmar space may result from tenosynovitis of middle and ring fingers or from a web infection which spread proximally through lumbrical canals.
- When this happens the normal concavity of the palm is obliterated.
- Space can be drained by an incision in 3rd or 4th web depending on pus point.

ACUTE PARONYCHIA AND FELON

Definition

- Acute paronychium is the infection of the nail fold whereas Felon or Whitlow is terminal pulp space infection.
- Acute paronychium is the most common hand infection whereas Felon is the second most common infection.

Etiology

- Small pricks and abrasions and cuts in hand especially in people involved with household work, manual laborers, gardeners is the commonest etiological factor.
- Unsterile manicure instruments can also result in paronychium.
- *Staphylococcus aureus* is the most common organism.

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Clinical Features

Paronychia

- Painful swollen nail folds is the most common presentation.
- Tenderness, redness and swelling of nail fold which increases on pressing nail.
- Suppuration can extend deep to nail and reach up to base of nail.

Felon

The pulp of finger is painful and swollen. Examination reveals tenderness, redness and swelling of pulp which increases on pressing.

Complications

Acute Paronychium

- Chronic paronychium
- Paronychium can extend into pulp space and result in felon

Felon

- Suppurative flexor tenosynovitis
- Septic arthritis of distal interphalangeal joint
- Osteomyelitis of terminal phalanx

Treatment

Acute Paronychium

- Conservative management with antibiotics and analgesics in mild cases.
- Elevation of nail fold and release of pus or nail excision and pus drainage.
- Bilateral cuticular incision and pus drainage.

Felon

- Conservative management with antibiotics and analgesics in mild cases.
- Incision at the pus point and drainage of pus.
- Lateral pulp incision and drainage of pus.

HYDATID CYST AND RELATED DISEASES

Hydatid cyst is caused by *Echinococcus* genus which has following species:

- *E. granulosus* (dog tapeworm or hydatid worm)—hydatid cyst
- *E. oligarthrus* and *E. vogeli*—polycystic hydatid disease
- *E. multilocularis*—alveolar or multilocular disease.

Definitive Hosts

Dogs, wolves, jackal harbor adult worms and eggs of the parasite.

Intermediate Hosts

Man, cattle, sheep harbor the larval form of the parasite.

Transmission

- Dogs are infected by eating contaminated meat of intermediate hosts.

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- Intermediate hosts are infected through ingestion of eggs passed in dog feces through contaminated water or food or through dog handling.

Life Cycle

- Adult worms live in small intestine of definitive hosts and discharge eggs in feces.
- The larvae hatch after 6–8 hours in the small intestine of the intermediate hosts, penetrate the intestinal wall and reach liver through portal venules.
- In liver, some get filtered and stay in liver while some larvae escape and reach lungs.
- Some get filtered while others reach systemic circulation and reach other organs such as brain, kidney, bone, spleen or eye where it matures and forms cyst.

Disease

- **Liver hydatid (discussed in liver chapter)**
- **Pulmonary hydatid**—can present as cough, hemoptysis, empyema or rupture and pneumothorax, anaphylaxis, hemoptysis.
- **Cerebral hydatid** can present with new onset seizures.
- **Can also present in spleen, bones, eye, heart.**

ENTAMOEBA HISTOLYTICA AND RELATED DISEASES

Life Cycle

- The *E. histolytica* passes its life cycle in single host.
- The *E. histolytica* has two forms in life—the trophozoite and the cyst.
- The infective form is the mature cyst in feces of carriers and reaches the human host by fecal-oral transmission.
- The cyst wall is damaged by trypsin in lower ileum or cecum and this leads to excystation and liberation of a single ameba—the quadrinucleate trophozoite.
- This trophozoite divides its contents to form eight small metacystic trophozoites each of which mature into a trophozoite.
- The trophozoites thus formed lodge in the submucous glandular crypts in colon and form cysts which are passed in feces and the cycle continues.

Diseases

- **Amebic colitis and amebic ulcers:**
  - Penetration of amebic trophozoites into colonic mucosa and submucosa with help of enzymes such as histolysin and formation of transverse ulcers with ragged undermined edges and base on the muscular coat.
  - Trophozoites enter portal radicles and are carried to extraintestinal sites.
  - Can result in **rectovesical fistula or rectocutaneous fistula.**
- **Amebic liver abscess: (discussed in liver chapter)**
- **Ameboma:** Inadequately treated amebic colitis result in formation of a chronic amebic mass in the cecum which is called ameboma. It is a tumor-like lesion and consists of granulomas. It is commonly confused with malignancy and needs differentiation before labeling it as ameboma.
- **Pulmonary amebiases:**
  - **Primary pulmonary amebiases** occurs when the ameba reach lungs through the pulmonary circulation.
  - **Secondary pulmonary amebiases** occurs due to direct extension of liver abscess through the diaphragm into lungs.

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- **Splenic amebiases**
- **Cerebral amebiases**
- **Amebiases cutis**—cutaneous amebic abscess

ACTINOMYCOSIS

Etiology

- *Actinomyces israelii*: It is an anaerobic gram-positive fungus like bacterium, branching filamentous organism (Ray fungus).
- Commonly found as normal flora of the oral cavity (within gingival crevices, tonsillar crypts, periodontal pockets, dental plaques, and carious teeth), pharynx, tracheobronchial tree, gastrointestinal tract, and female urogenital tract.

Pathogenesis

Organism enters through deeper plane of the tissue, causes subacute inflammation with induration and nodule formation. Eventually discharging sinus forms at the surface. Pus collected in a swab or sterile tube will show sulfur granules.

Clinical Presentation

- **Cervicofacial disease** (most common site):
  - Occurs in setting of poor dental hygiene, recent dental surgery, or trauma
  - Painful soft tissue swelling commonly seen at the angle of the mandible
  - Fever, chills, and weight loss, trismus
  - Soft tissue facial infection with sinus tract or fistula formation
- **Thoracic disease:**
  - Can involve the lungs, pleura, mediastinum, or chest wall.
  - Presumed secondary to aspiration of *Actinomyces* organisms in patients with poor oral hygiene.
  - Fever, cough, weight loss, and pleuritic chest pains are common symptoms.
  - Signs of pneumonia or pleural effusion may be present.
  - With extension beyond the lungs to mediastinal structures and the chest wall, signs and symptoms of pericarditis, empyema, chest wall sinus drainage.
  - Tracheoesophageal fistula can occur
- **Abdominal disease:**
  - Occurs most commonly after appendectomy, perforated bowel, diverticulitis, or surgery to the gastrointestinal tract.
  - Lesions develop most commonly in the ileocecal valve, causing abdominal pain, fever, weight loss, and a palpable mass.
  - Extension may occur to the liver, causing jaundice and abscess formation.
  - Sinus tracts to the abdominal wall can occur.
- **Pelvic disease:** Commonly occurs by extension from abdominal disease of the ileocecal valve to the right adnexa (80% of cases) and causes endometritis.

Differential Diagnosis

- **Cervicofacial disease:** Odontogenic abscesses, brachial cleft cyst.
- **Pulmonary disease:** Tuberculosis, nocardiosis, botryomycosis, chromomycosis, fungal disease of the lung

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- **Intestinal disease:** Intestinal tuberculosis, ameboma, colon cancer, inflammatory bowel disease.
- **Pelvic disease:** Pelvic inflammatory disease, Crohn's disease
- **CNS disease:** Brain abscess, brain tumors, toxoplasmosis, intracranial hematoma

Investigation

- Isolating "sulfur granules" from tissue specimens or draining sinuses confirms the diagnosis of actinomycosis.
- Sulfur granules are nests of *Actinomyces* species. Sulfur granules may be macroscopic or microscopic. Sulfur granules are crushed and stained for identification of *Actinomyces* organisms and may take up to 3 weeks to grow in culture media.

Treatment

Nonpharmacologic Therapy

- Incision and drainage of abscesses
- Excision of sinus tract

Treatment for Acute Phase

- Penicillin 10–20 lac units per day in 4 divided doses for 4–6 weeks.
- In penicillin—allergic patients, erythromycin, tetracycline, clindamycin, or cephalosporins are reasonable alternatives
- Chloramphenicol 50–60 mg/kg/day can be used for CNS actinomycosis.

Treatment for Chronic Phase

Following 4–6 week IV penicillin, oral penicillin V 500 mg PO 4 times a day for 6–12 months. Longer period of treatment is needed to eradicate the disease.

SAMPLE QUESTIONS

1. **Which of the following is the first-line management for gas gangrene?**
   A. Systemic antibiotics
   B. Hyperbaric oxygen therapy
   C. Surgical debridement
   D. Wound care and dressings
2. **Which of the following is a distinguishing feature between felon and paronychia?**
   A. Location of infection
   B. Age group affected
   C. Type of pathogen involved
   D. Presence of fever
3. **Which of the following organisms is a common causative agent of carbuncles?**
   A. *Streptococcus pyogenes*
   B. *Staphylococcus aureus*
   C. *Escherichia coli*
   D. *Pseudomonas aeruginosa*
4. **A 35-year-old male presented with a 3-day history of bloody diarrhea and abdominal cramps after coming from an adventure trip of 7–10 days. On physical examination, he had a temperature of 38.3°C, and his abdomen was tender on palpation. A stool sample was collected and sent for laboratory analysis. Which of the following is the most likely causative agent of the patient's symptoms?**
   A. *Salmonella enterica*
   B. *Shigella flexneri*
   C. *Escherichia coli*
   D. *Entamoeba histolytica*
   E. *Giardia lamblia*

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