



S.Tentishev Asian Medical Institute KANT, KYRGYZSTAN





For interprofessional communication and partnership in health care and medical education"



Dr.Aftab Sheikh INTERPROFESSIONAL DISCIPLINE DEPARTMENT

AsMI PHARMACOLOGY SOCIETY



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Respected Colleagues and dear Students

Hello everyone ,Myself Dr Aftab Sheikh, i am trying to continue series of Journal which will be continue on monthly basis. If any student or teacher want to add some article please contact me.

It's my very first time that I am working on a Journal, so requesting all of you please give me your comments and suggestions on given WhatsaApp.

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Dr.AFTAB SHEIKH Senior Lecturer Orthopedic Surgeon Inter-Professional Discipline Dept.

ILIZAROV TECHNIQUE

Gavriil Abramovich Ilizarov was a Russian physician, known for inventing the Ilizarov apparatus for lengthening limb bones and for the method of surgery

named after him, the Ilizarov surgery.

The Ilizarov method is a surgery involving an orthopedic external fixator applied to the limb to reconstruct, reshaping or increasing length of bones (usually of the limb).External fixation is a surgical treatment where rods are screwed through an incision into the affected bone and exit the body to be attached to a stabilizing structure outside of the body.Ilizarov surgery involves the use of a special device called



an 'Ilizarov apparatus or Ilizarov fixator'. It is recommended in cases that are not amenable to other reconstruction techniques.

Ilizarov Apparatus This is a set of external fixators comprising rings (2-6), rods, adjustable nuts, and Kirschner wires (a type of stabilization wire/pin used in orthopedic surgery). The device works by a principle called "distraction osteogenesis" (osteo – bone, genesis – formation). Osteogenesis means the development and formation of bones

CONCEPT OF ILIZAROV SURGERY

The procedure involves gradually pulling the bones apart (distraction). It uses the body's natural ability to generate and grow new bone between the surfaces that are pulled apart. This bony growth fills up the gap in a gradual, controlled manner with the help of the Ilizarov fixator.

Complex and compound fractures	Bone infection (Osteomyelitis)	Fracture mal-union (incorrectly healed)
Fracture non-union (not healed)	Bone misalignment or misplacement	Unequal limb length and bone deformity

INDICATIONS FOR THE SURGERY

PREPARATION FOR THE SURGERY

Before the surgery, your medical history will be evaluated to check mainly for anesthetic allergy. This may be followed by a blood test. Your surgeon will also instruct you to:

> Stop medication (if taking any) that interferes with bone healing

- > Give up smoking (if applicable) as it can slow down healing
- > Arrange someone to drive you back home after the surgery

RISKS OR COMPLICATIONS

Potential risks or complications of the procedure include:

- > Pain, bruising, bleeding, swelling
- > Infection
- Blood clotting (deep vein thrombosis)
- Nerve palsy (lack of nerve function)
- > Permanent stiffness of the joint
- > Temporary tingling sensation in the skin
- Anesthesia reaction

AFTER THE SURGERY

As you recover from Ilizarov surgery, you are advised to:

- > Continue to take pain medication as prescribed.
- > Keep the operated part elevated to ease any swelling.
- > Maintain the external fixator frame by keeping it clean and dry.
- > Perform pin care as instructed by your surgeon.
- > Avoid baths due to the risk of infection in the pin sites.

BENEFITS OF ILIZAROV SURGERY

- > Ilizarov surgery benefits you in several ways. They include:
- > Minimally invasive, customizable procedure
- > A substantially shorter period of hospital stays
- > Less bleeding, infection, and soft tissue damage
- > Good for multiple fractures or angular deformity
- > Effective for open bone injuries and infections
- > Allows necessary reposition of bone fragments
- > A rare instance of joint stiffness and contractures
- > Avoids amputation and/or joint replacement
- > Equalizes bone discrepancies of several inches
- > Faster healing time and speedy recovery
- > Allows early mobility and weight-bearing
- > Enhances stability and distributes stress evenly
- > Patient mobility throughout the treatment
- Normal regenerated bone that does not wear out
- > Effective both in mature as well as young bones
- Less disability or recurrence rate
- Reduced treatment time as well as cost

REFERENCES

1-OrthoNorCal2-Webster3-Goldenstate Orthopedic and Spine



SHUBHAM TIWARI 5th year 9th semester (2019-2024)

BREAST CANCER

Breast cancer is a disease in which cells in the breast grow out of control. It is a very aggressive disease . Severity can be check by depth of lesion

25 women in 10000 women in Asia develops breast cancer

RISK FACTOR

Increase age. Early menarche. Late menopause. Both paternal and maternal family history. HRT Nulliparous women .Sporadic is more common than familial ... P53 >BRCA1>BRCA2 SCREENING AND DIAGNOSIS

- Mammography before 40 years is screening test which can reduce morbidity and mortality by 20-25%
- Mammography after 40 years is diagnostic test for breast cancer.
- > It is recommended in every healthy women after 40 years of age .
- ▶ If family history is positive then screening test should start from 35 years of age .
- If 1st degree relative are positive for BRCA 1 MUTATION then screening should start from 25 years of age by MRI, because in younger age glandular tissue are higher so mammography should avoid
- If a pregnant woman with lump is come for screening then investigation of choice will be USG

TREATMENT

> Surgery

- Radiotherapy
- Hormonal therapy

Chemotherapy

Note- All patient don't require all the treatment morality

SURGERY - For a breast lump we have two surgery option

1-Breast conservative surgery

2-mastectomy

(BCS has higher recurrence rate) and should be avoid in multicentric, SLE, Rheumatoid arthritis, Pregnancy.

CHEMOTHERAPY- Chemotherapy - it is done in ER PR -HER2 NEU+tumours .

Chemotherapy regimes

- 1- C- cyclophosphamide 2- F- 5
 - 2- F- 5 flouro uracil
- 3- A- adriyamycine or M- methotrexate

RADIOTHERAPY - It must be done after breast conservative surgery HORMONAL THERAPY done only in ER PR+ tumors

REFERENCE

1-Sabiston Text Book Of Surgery



AREESHA REHMAN 5th year 9th semester (2019-2024)

GALLSTONE

A Comprehensive Overview

INTODUCTION

Gallstones are a widespread medical condition that affects millions of individuals worldwide. This article provides a comprehensive overview of gallstones, including their occurrence, mortality rates, causes, risk factors, development process, diagnosis, treatment options, and a concluding summary of the topic.

OCCURRENCE

Gallstones are prevalent both in developed and developing countries, with a higher incidence in Western nations. Studies indicate that around 10-15% of adults in these countries are affected by gallstones. The condition is more common in females, particularly those who are overweight or lead a sedentary lifestyle (Everhart et al., 1999).

MORTALITY

While gallstones themselves are usually not life-threatening, complications associated with them, such as acute cholecystitis or gallstone pancreatitis, can lead to severe illness and even death if left untreated. Early diagnosis and appropriate treatment are vital for reducing mortality rates related to gallstone complications (Shaffer, 2005).

CAUSES

Gallstones develop when there is an imbalance in the bile, a fluid produced by the liver to aid digestion, leading to the formation of solid particles. The two main types of gallstones are cholesterol stones and pigment stones. Cholesterol stones, the most common type, form due to an excess of cholesterol in the bile. Pigment stones, on the other hand, consist of bilirubin and are typically associated with certain medical conditions such as liver cirrhosis or hemolytic anemia (Portincasa et al., 2006).

RISK FACTORS

Several factors contribute to the development of gallstones, including obesity, rapid weight loss, a sedentary lifestyle, a diet high in fat or cholesterol, a family history of gallstones, certain medications, and specific medical conditions like diabetes and metabolic syndrome (Lammert et al., 2016).

DEVELOPMENT PROCESS

The development of gallstones involves a complex interplay of genetic, environmental, and metabolic factors. An imbalance between cholesterol secretion, bile acids, and phospholipids leads to their formation. Genetic predisposition and certain hormonal factors also play a role in the development of gallstones (Portincasa et al., 2019).

DIAGNOSIS

Diagnosing gallstones involves a combination of clinical evaluation, imaging studies, and laboratory tests. Ultrasonography is the most commonly used imaging technique for identifying gallstones. In specific cases, other imaging methods such as magnetic resonance cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography (ERCP) may be utilized. Laboratory tests may reveal elevated liver enzymes and bilirubin levels in patients with gallstone-related complications (Tse et al., 2008).

TREATMENT

The management of gallstones depends on the presence of symptoms and the risk of complications. Asymptomatic gallstones usually do not require treatment. However, symptomatic gallstones or those associated with complications may necessitate intervention. The most common treatment option is laparoscopic cholecystectomy, a surgical procedure to remove the gallbladder. In select cases, non-surgical approaches such as oral bile acid therapy or extracorporeal shock wave lithotripsy (ESWL) may be considered (Friedland & Soetikno, 2015). CONCLUSION

Gallstones are a prevalent medical condition with significant implications for patient health. Understanding their occurrence, causes, risk factors, development process, diagnosis, and treatment options is crucial for effective management. Early identification and appropriate intervention can help prevent complications and improve patient outcomes.

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- ✓ Everhart JE, Khare M, Hill M, et al. Prevalence and
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- ✓ Portincasa P, Moschetta A, Palasciano G. Cholesterol gallstone disease. Lancet. 2006;368(9531):230-239.
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- ✓ Portincasa P, Di Ciaula A, Wang DQ. Gallstones: Past, present, and future. Eur J Gastroenterol Hepatol. 2019;31(9):1033-1044.
- ✓ Tse F, Liu L, Barkun AN, Barkun JS, Sultanian R, Tepper J, Karmali S. EUS: A metaanalysis of test performance in suspected choledocholithiasis. Gastrointest Endosc. 2008;67(2):235-244.
- ✓ Friedland S, Soetikno RM. Gastrointestinal endoscopy in 2015: Not your grandfather's scope. Gastrointest Endosc. 2015;81(1):51-58.



SAIRAM PAPISHETTY 5th year 9th Semester (2019-2024)

MYOCARDIAL INFARCTION

Myocardial infarction, commonly known as a heart attack, is a serious medical condition that occurs when blood flow to a part of the heart muscle is blocked. It is usually caused by the formation of a blood clot (thrombus) in one of the coronary arteries, which supply oxygen and nutrients to the heart.

EPIDEMIOLOGY:

Global prevalence of MI in individuals. <60 years was found. 3.8% **MORTALITY RATE :**

In a year 33% of men & women of <60 years. Had died. (Wikipedia)

Men 73% Female 27%

ETIOLOGY:

1-Most common – Atherosclerosis (buildup of Fatty plaques in Coronary artery)

2-Thrombosis: Formation of blood clot Within coronary artery.

3-Trauma: Tears or ruptures in coronary arteries

4-Anomalous Coronary arteries..

SYMPTOMS:

The typical symptoms of a heart attack include chest pain or discomfort, which may radiate to the arm, shoulder, jaw, back, or neck. Other symptoms can include shortness of breath, sweating, nausea, vomiting, lightheadedness, and anxiety.

PATHOPHYSIOLOGY:

The pathophysiology of myocardial infarction (MI) involves a series The pathophysiology of myocardial infarction (MI) involves a series of events that occur when blood flow to a part of the heart muscle is blocked and reduced oxygen to the myocardium, the heart muscle

- -Atherosclerosis
- -Plaque rupture
- -Thrombus formation
- ischemia

WHO IS ON RISK

Age(over 40)	High blood pressure	High cholesterol	Family History	Diabetes
Smoking	Gender (Male) mostly	Female after	Metabolic Syndrome	Obesity
		Menopause		

DIAGNOSIS

Physical examination Electrocardiogram Blood tests for cardiac markers (Troponin I and T) Imaging studies like angiography

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TREATMENT

ASPIRIN (powerful antiplatelet drug)
 Vasodilators
 Anti-Hyperlipidemic drugs
 Or
 AS PER PHYSICIAN RECOMMENDATION

COMPLICATIONS

Heart failure Arrythmias Cardiogenic shock Pericarditis Blood clots

PREVENTION

Healthy diet	Plenty of fruits	Vegetables whole grain	Regular exercise
Maintain BMI	Quit smoking	Maintain Healthy weight	Stay stress free
		Limit alcohol consumption	

REFERENCE

1-Mayao Clinic and Dr Inam Danish

AWEARENCE!!



ALCOHAL DRINKING can cause High Blood Pressure, Stroke, Liver disease, head & neck cancer, breast cancer, pancreatitis, liver cancer, mouth cancer .



MOHAMMED SUHAIL 5th year 9th Semester (2019-2024)

ALZHEIMER DISEASE

Alzheimer Disease is a brain disorder that get worse cover time It characterized by change in the brain. That lead to deposition of certain Protein

SING AND SYMPTOMS

People with Alzheimer's disease may:

Repeat statements and questions over and over.

Forget conversations, appointments or events.

Misplace items, often putting them in places that don't make sense.

Get lost in places they used to know well.

Eventually forget the names of family members and everyday objects.

Have trouble finding the right words for objects, expressing thoughts or taking part in conversations.

ETIOLOGY

- > Dementia is caused by damage to brain cell
- > Combination of age related changes in the Brain
- Along with generic factor
- Environmental

EPIDEMIOLOGY

World wide the global Prevalence of dementia was estimated to be 3.9% in People age per year

- \rightarrow 1.6% in Africa
- \rightarrow 4.6% in China
- \rightarrow 4.6% In north America

About 6.5 million people in the United States age 65 and older live with Alzheimer's disease. Among them, more than 70% are 75 years old and older. Of the about 55 million people worldwide with dementia, 60% to 70% are estimated to have Alzheimer's disease.

RISK FACTOR

Age	Down Syndrome	Head trauma
Family history	Air Pollution	

PREVENTION

-Change in life Style

-Exercise regularly.

-Eat a diet of fresh produce, healthy oils and foods low in saturated fat, such as a Mediterranean diet.

-Follow treatment guidelines to manage high blood pressure, diabetes and high cholesterol.

-Quite smoke

TREATMENT

-Galantamine,

-Rivastigmine, and

-Donepezil

-Cholinesterase inhibitors that are prescribed for mild to moderate Alzheimer's symptoms. These drugs may help reduce or control some cognitive and behavioral symptoms.

REFERENCE

- 1-National Library of Medicine
- 2-National Center of Biotechnology Information
- 3-www.ncbi.nlm.nih.gov

INTRESTING FACTS



- ✓ Drinking coffee can prevent depression. ...
- ✓ Chewing gum makes you more alert. ...
- ✓ Sitting at a desk can increase death risk by almost 50 per cent. ...
- ✓ If you're an optimist, it could help you live longer. ...
- ✓ Smell an apple to prevent claustrophobia.
- ✓ Beards are the fastest growing hairs on the human body. ...
- ✓ Everyone has a unique smell, except identical twins they smell the same!
- ✓ A human fetus acquires fingerprints at the age of three months. ...
- ✓ Every individual has a unique tongue print. ...
- ✓ The fastest growing nail is on the middle finger..



SIDDU SIDDHARTHA 5th year 9th Semester(2019-2024)

PANCREATITIS

Inflammation of pancreas, where enzymes can be activated prematurely Causing damage to the pancreas and Surrounding tissues.Pancreatitis Can be Acute (Sudden onset) and Chronic (Long term inflammation that can lead to permanent damage).

ETIOLOGY

-Most common is excessive alcohol consumption and gallstones

- -For acute pancreatitis
- -Toxic like alcohol and smoking
- -Iatrogenic -ERCP iatrogenic
- -Metabolic:-hypercalcemia, hypertriglyceridemia.
- -Autoimmune
- -Genetic (PRSS-1,CTRC,SPINK-1,CFTR)
- -Infection (coxsackie B) and medications like metronidazole.

-For chronic pancreatitis

- -Toxic
- -Obstruction
- -Diabetes mellitus
- -Obesity
- -History of pancreatitis

EPIDEMIOLOGY

-Acute pancreatitis affects approximately 50-80 per 100,000 people per year in united states. Largest Increases in incidence were among women aged <35 years - 7.9% per year.

Men aged 35-44 years - 5.7% Per year and 45-54 years5.3% per year.

-Acute pancreatitis was increased significantly during the Christams and New Year Weeks by 48% for alcoholic etiology.

-Chronic pancreatitis - (alcohol induced) incidence of chronic pancreatitis ranges from 5 to 12 per 100,000 people per year with Prevalence of approximately 50/100,000 persons. incidence among- Men - 12. Cases per 100,000 people per year. Women 6 cases for 100,000 people per year.

REFERENCE

Aliment pharmacology Ther (published in July 16th, 2013), Authors:- SE Roberts, A Akbari, K Thorne.

MOTALITY RATE

Mild acute pancreatitis -<1%.

Severe acute pancreatitis- 10 % to 30% and 28.7% Patients died in ICU-(SOURCE:- WIKIPEDIA).

SYMPTOMS

In Acute

Abdominal pain limited to epigastric or upper right quadrant pain

Tenderness in abdomen to touch

In Chronic

Abdominal pain can regulate to back, endocrine insufficiency and exocrine insufficiency.

Bowel movements that appear oily and extra pungent.

RISK FACTORS

- > Alcoholism
- Family History of pancreatitis
- ➤ Gall stones
- Cystic fibrosis
- High triglycerides
- ➢ Infection
- Abdomen injury
- Smoking cigarettes
- ➢ Hypercalcemia,
- Caner of pancreas

TREATMENT

- > Intravenous (IV)- fluid hydration (fluid resuscitation).
- > Analgesics drugs are administered for pain relief and supportive therapy

PREVENTION

Stop consumption of alcohol and smoking cigarettes, maintaining a healthy lifestyle, Eating a balanced diet and low fat diet, Exercise regularly, Drinking more fluids and maintain healthy weight

CONCLUSION

Surgical management of patients with infected pancreatic necrosis would be necrosectomy with drainage and patients with sterile necrosis should be managed conservatively unless organ complications or severe clinical deterioration persist despite intensive therapy.



PRATIBHA SINGH 5th year 9th Semester (2019-2024)

FIBROIDS

Fibroids are tumors made of smooth muscle cells and fibrous connective tissue that develop in the uterus. It is estimated that 70 to 80 percent of women will develop fibroids in their lifetime

- ➢ Incidence
- ➤ 30% women
- >50 yrs 80%
- > 2.5 times of more chance if female relative has one fibroid
- ➤ Associated with chromosomal abnormality (40%)

RISK FACTORS

- > Age (older women are at higher risk than younger women)
- African American race.
- ➢ Obesity.
- Family history of uterine fibroids.
- ➢ High blood pressure.
- No history of pregnancy.
- ➢ Vitamin D deficiency.
- ➢ Food additive consumption.

CLINICAL FEATURES

> Pain

Due to contractions, compression, compaction

➢ Bleeding

Due to increase endometrial recruitment poor contractility

SIGN & SYMPTOMS

- > Heavy vaginal bleeding. Excessively heavy or prolonged menstrual bleeding.
- Pelvic discomfort.
- Pelvic pain.
- Bladder problems.
- Low back pain.

DIAGNOSIS

- ▶ USG also used for mapping of fibroids
- MRI (Best)

TREATMENT

- ➤ If Fibroid size→Small >5cm with no pain / bleeding/ infertility then No Rx required
- > If Fibroid size \rightarrow Small >5cm with pain then Rx required
- If women is young don't do hysterectomy
- > If women in elderly then definitely go for hysterectomy

- Rectal pressure.
- Discomfort or pain with sexual intercourse.

MEDICAL MANAGEMENT

- ➤ To decrease bleeding
- > To decrease size of the fibroid

.NSAIDS

- GnRH analogue
- GnRH antagonists (Cetrotide)

NEW TECHNIQUES

- Uterine artery embolization
- Uses poly vinyl alcohol particles
- Upton80% reduction in pain & bleeding

(REFERENCE - by Beckmann and Ling's)



- 1. The word "muscle" comes from the Latin term meaning "little mouse." It's said that Ancient Romans thought that's what flexed bicep muscles resembled. (*Source: natgeokids.com*)
- 2. Human teeth are just as tough as shark teeth. (Source: sciencemag.org)
- 3. Nurses make up the largest single component of hospital staff and are the primary providers of hospital patient care. (*Source: aacnnursing.org*)
- 4. The words "BSN preferred" are popping up much more frequently in classified ads for registered nurses nationwide. (In other words, even if you're registered nurse, now's the time to get your BSN.) (*Source: aacnnursing.org*)
- 5. <u>Occupational therapy</u> was founded by three men and three women in 1917—that's three years before women could vote. (*Source: napacenter.org*)
- 6. Occupational therapy techniques date back to 100 BC, when a Greek physician named Asclepiades used OT techniques to treat mental illness. *(Source: bchcares.org)*
- 7. Eleanor Roosevelt was an honored guest at the 21st Annual Meeting of the American Occupational Therapy Association. There, she paid tribute to Eleanor Clarke Slagle, one of the most influential people in the history of OT. (*Source: lewisu.edu*)
- 8. <u>Physical therapy</u> began in response to WWI. In the army, 'reconstruction aides' (aka nurses) were responsible for providing rehab to wounded soldiers using both physical and occupational therapy. *(Source: orthocarolina.com)*
- 9. PT started as a female-only profession. In fact, in 1921, the first professional physical therapy association was born: the American Women's Physical Therapeutic Association. The first president was Mary McMillan. (*Source: apta.org*)
- 10. Research has shown that colder temperatures may help reduce allergies and inflammation *and* can help you think more clearly and perform daily tasks better. (*Source: rd.com*)



SHRUTI SOVE 5th Year 9th Semester (2019-2024)

SCHIZOPHRENIA

Schizophrenia simply means spilt of mind and split of thought. Schizophrenia is a long-term mental health condition. It causes a range of different psychological symptoms.

Doctors often describe schizophrenia as a type of psychosis. This means the person may not always be able to distinguish their own thoughts and ideas from reality.

HISTORY OF SCHIZOPHRENIA

Emil kraepelin – classified psychiatric illnesses into 2 clinical types.

1.Dementia praecox (young age memory loss)

2.Manic depressive illness

Eugen bleuler – coined the term schizophrenia, which replaced dementia praecox in scientific literature.

Kurt Schneider - First Rank Symptoms

Karl kahlbaum - coied term Catatonia

4A's of BLEULER

- 1. Autism
- 2. Ambivalence
- 3. Affect Disturbances
- 4. Association Disturbances

PREVALENCE IN SPECIFIC POPULATION

- > Non twin sibling of a schizophrenia patient -8%
- > Dizygotic twin of a schizophrenia patient -12%
- Monozygotic twin of a schizophrenia patient 47%
- Child with one parents with schizophrenia -12%
- > Child with both parents with schizophrenia -40%

CAUSES OF SCHIZOPHRENIA

- Genetic Factor : Monozygotic > dizygotic
- > Deletion at chromosome 22 -velocardial syndrome / DiGeorge syndrome 30%.
- Biochemical factors: excess of Dopamine and Serotonin.
- Environmental factors
- Advanced paternal age
- Season of birth Winter and early spring
- Prenatal exposure to Influenza Virus
- Drug abuse Cannabis

SYMPTOMS

- hallucinations hearing or seeing things that do not exist outside of the mind
- delusions unusual beliefs not based on reality

- > muddled thoughts and speech based on hallucinations or delusions
- losing interest in everyday activities
- not wanting to look after yourself and your needs, such as not caring about your personal hygiene
- wanting to avoid people, including friends
- feeling disconnected from your feelings or emotions
- People with schizophrenia do not have a split personality. Schizophrenia does not usually cause someone to be violent.

DIAGNOSIS

According to DSM-V two or more of the following Symptoms should be present for 1 month.

- ➢ Delusion
- ➢ Hallucinations
- Disorganized speech (or formal thought disorder)
- Disorganized or catatonic behavior
- Negative symptoms

TREATMENT

Atypical Antipsychotics or 2nd Generation drugs :

- Serotonin Dopamine Antagonists
- Clozapine Drug of choice, only drug which decreases suicidal tendency in schizophrenia.
- Olanzapine
- Ziprasidone side effect cardiac arrhythmia & seizures
- ➢ Resperidone

REFERENCE : Short notes from Textbook of Psychiatry and from website www.nhs.uk



GUDURI HARSHAVARDHAN REDDY 5th year 9th Semester

CORONARY ARTERY DISEASE(CAD)

CAD stands for Coronary Artery Disease, which is a cardiovascular condition characterized by the narrowing or blockage of the coronary arteries. These arteries supply oxygen-rich blood to the heart muscle. CAD occurs when plaque builds up on the inner walls of these arteries, a process called atherosclerosis, causing them to become narrow and restrict blood flow to the heart.

EPIDEMIOLOGY:

The epidemiology of CAD varies across different regions and populations. Here are some key points regarding the epidemiology of CAD:

1.Prevalence: CAD is one of the most common types of heart diseases worldwide. It affects both men and women, although the incidence is generally higher in men. The prevalence of CAD tends to increase with age.

2.Global Burden: CAD is a leading cause of death globally. According to the World Health Organization (WHO), ischemic heart disease, which includes CAD, accounted for more than 8 million deaths worldwide in 2019.

3. Risk Factors: Several risk factors contribute to the development of CAD. These include smoking, high blood pressure, high cholesterol levels, diabetes, obesity, a sedentary lifestyle, family history of heart disease, and certain genetic factors.

It's important to note that the specific epidemiological data on CAD may vary over time as new research emerges, and the prevalence and impact of the disease can be influenced by various factors within different populations.

MORTALITY:

CAD is a leading cause of death worldwide. According to the World Health Organization (WHO), cardiovascular diseases, including CAD, were responsible for approximately 17.9 million deaths globally in 2019. It is important to note that mortality rates for CAD have improved over the years due to advancements in medical treatments, lifestyle modifications, and increased awareness.

ETIOLOGY:

The etiology, or the causes, of coronary artery disease (CAD) are multifactorial and involve a combination of genetic, lifestyle, and environmental factors. Here are some key factors that contribute to the development of CAD:

1.Atherosclerosis: The primary underlying cause of CAD is atherosclerosis, which is the buildup of plaque in the coronary arteries. Plaque consists of cholesterol, fat, calcium, and other substances. Over time, plaque narrows the arteries, restricting blood flow to the heart muscle.

2. Lifestyle choices: Unhealthy lifestyle habits, such as a poor diet high in saturated and trans fats, lack of physical activity, excessive alcohol consumption, and chronic stress, can increase the risk of CAD. Adopting a healthy lifestyle, including a balanced diet, regular exercise, stress management, and avoiding tobacco and excessive alcohol, can help reduce the risk.

3. Genetic factors: Genetics plays a role in CAD susceptibility. Certain genetic variations may influence cholesterol metabolism, blood clotting factors, inflammation, and other processes related to the development of CAD. Family history of premature CAD (before age 55 in men or 65 in women) increases the risk.

4. Other medical conditions: Certain medical conditions, such as chronic kidney disease, rheumatoid arthritis, and sleep apnea, are associated with an increased risk of CAD. RISK FACTORS:

Certainly! Here are some common risk factors associated with coronary artery disease (CAD): 1.High blood pressure (hypertension): Elevated blood pressure puts strain on the arterial walls, leading to the development of CAD.

2.High cholesterol levels: High levels of LDL cholesterol (commonly known as "bad" cholesterol) can contribute to the formation of plaque in the arteries, narrowing them and restricting blood flow.

3.Smoking: Tobacco use damages the blood vessels and accelerates the development of atherosclerosis, increasing the risk of CAD.

4.Diabetes: Individuals with diabetes have an increased risk of developing CAD due to factors like high blood sugar levels and associated metabolic abnormalities.

5.Obesity: Excess body weight, particularly abdominal obesity, is associated with an increased risk of CAD.

6. Unhealthy diet: A diet high in saturated and trans fats, cholesterol, and refined carbohydrates increases the risk of CAD.

7.Family history: Having a close family member, such as a parent or sibling, with a history of CAD increases the likelihood of developing the condition.

8. Age and gender: The risk of CAD increases with age, and men tend to be at higher risk compared to premenopausal women. However, the risk for women increases after menopause.
 9. Stress: Chronic stress and certain psychological factors may contribute to the development and progression of CAD.

"It's important to note that having these risk factors doesn't necessarily mean an individual will develop CAD, but they increase the likelihood. Adopting a healthy lifestyle, managing these risk factors through medication or lifestyle changes, and regular medical check-ups are essential for preventing or managing CAD effectively."

PATHOGENESIS:

The pathogenesis of coronary artery disease (CAD) involves a complex process called atherosclerosis, which is the underlying cause of most cases. Here are the key steps in the development of CAD:

1.Endothelial injury: The inner lining of the coronary arteries, known as the endothelium, can be damaged by factors such as high blood pressure, smoking, high cholesterol levels, or inflammation. This injury triggers the development of atherosclerosis.

2.Formation of fatty streaks: In response to endothelial injury, low-density lipoprotein (LDL) cholesterol particles can penetrate the damaged endothelium and accumulate in the arterial wall. Macrophages engulf the LDL particles, leading to the formation of fatty streaks.

3.Plaque formation: Over time, the fatty streaks become more complex and evolve into plaques. Smooth muscle cells migrate to the area and release substances that promote inflammation and further plaque growth.

4.Plaque rupture and thrombosis: The plaques can become unstable and susceptible to rupture. When a plaque ruptures, it exposes the underlying tissue, triggering the formation of blood clots (thrombosis). These blood clots can partially or completely block the coronary artery, leading to reduced blood flow and potentially causing a heart attack.

5.Coronary artery narrowing and ischemia: As the plaques grow and accumulate, they can narrow the coronary arteries, reducing blood flow to the heart muscle. This reduction in blood flow results in myocardial ischemia, causing symptoms like angina (chest pain) and potentially leading to heart muscle damage if left untreated.

DIAGNOSIS:

The diagnosis of coronary artery disease (CAD) typically involves a combination of medical history evaluation, physical examination, and various diagnostic tests. Here are some common methods used for diagnosing CAD:

1.Medical history and physical examination: The healthcare provider will review your medical history, including any symptoms you may be experiencing, and conduct a physical examination to assess your overall health and risk factors for CAD.

2.Electrocardiogram (ECG/EKG): This test measures the electrical activity of the heart. An ECG can detect abnormal heart rhythms, signs of a previous heart attack, and certain patterns that may suggest CAD.

3.Exercise stress test: Also known as a treadmill test or exercise ECG, this test evaluates how your heart performs during physical activity. You will walk or run on a treadmill or pedal a stationary bike while your heart rate, blood pressure, and ECG are monitored. It helps assess blood flow to the heart and may reveal signs of CAD, such as reduced blood flow during exercise.

4.Cardiac imaging: Various imaging techniques can provide detailed pictures of the heart and its blood vessels, allowing for the assessment of CAD. These include:

•Echocardiogram: Uses ultrasound waves to create images of the heart, helping evaluate its structure and function.

•Nuclear imaging: Involves injecting a small amount of radioactive material into the bloodstream to visualize blood flow in the heart and identify areas with reduced blood supply.

•Computed tomography angiography (CTA): Uses X-ray technology to create detailed images of the heart and its blood vessels, providing information about the presence and severity of blockages.

•Magnetic resonance imaging (MRI): Produces detailed images of the heart using a powerful magnetic field and radio waves, assisting in the assessment of heart structure, blood flow, and potential CAD-related abnormalities.

5.Coronary angiography: This invasive procedure involves the insertion of a catheter into a blood vessel, usually in the groin or wrist, and injecting a contrast dye to visualize the coronary arteries. It provides detailed information about blockages and narrowing in the arteries. TREATMENT:

The treatment of coronary artery disease (CAD) aims to reduce symptoms, prevent complications, and improve overall heart health. The treatment approach depends on the severity of CAD, individual risk factors, and the presence of any associated conditions. Here are some common treatment strategies:

1.Lifestyle modifications: Healthy lifestyle changes are essential for managing CAD. These may include adopting a heart-healthy diet low in saturated fats, trans fats, and cholesterol, while emphasizing fruits, vegetables, whole grains, lean proteins, and healthy fats. Regular physical activity, such as aerobic exercise and strength training, is also important. Additionally, quitting smoking and managing stress effectively are crucial for improving heart health. 2.Medications:

•Antiplatelet medications: Drugs like aspirin or clopidogrel are prescribed to prevent blood clots and reduce the risk of heart attacks.

•Cholesterol-lowering medications: Statins are commonly prescribed to lower LDL cholesterol levels and stabilize plaques in the arteries.

•Blood pressure medications: Medications, such as ACE inhibitors, beta-blockers, or calcium channel blockers, may be prescribed to manage high blood pressure and reduce the workload on the heart.

•Nitroglycerin: It helps relieve chest pain (angina) by relaxing and widening the coronary arteries.

3.Angioplasty and stenting: In this procedure, a catheter is threaded through a blood vessel to the blocked or narrowed coronary artery. The blockage is then opened by inflating a balloon at the tip of the catheter, followed by the placement of a stent to keep the artery open and improve blood flow.

4.Coronary artery bypass grafting (CABG): CABG is a surgical procedure where a healthy blood vessel, usually taken from the leg or chest, is used to bypass the blocked or narrowed coronary artery. This allows blood to flow around the blockage and reach the heart muscle.

5.Cardiac rehabilitation: Cardiac rehabilitation programs involve supervised exercise, education, and counseling to help individuals with CAD recover from a heart attack, manage symptoms, and improve overall heart health.

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RABINA SHAW 5th Year 9th (Semester 2019-2024)

ESOPHAGEAL CANCER

Esophageal cancer is a disease in which malignant cells form in the tissue of the esophagus. **EPIDEMOLOGY**

Esophageal cancer (EsC) including squamous cell carcinoma (SCC) and adenocarcinoma is considered as a serious malignancy with respect to prognosis and a fatal outcome in the great majority of cases]. Esophageal carcinoma affects more than 450000 people worldwide and the incidence is rapidly increasing.

Esophageal cancer age-adjusted incidence of blacks was about twice that of whites (8.63/100000 vs 4.39/100000,]. SCC was more commonly diagnosed in blacks and white females, whereas adenocarcinoma was more common among white males.

MORTALITY

It is in line with the incidence rate in the world but there is no difference between men and women. Age-adjusted mortality for blacks, although showing a declining trend, was nearly twice that of whites (7.79 vs 3.96, P < 0.05)

Premalignant condition

Squamous cell carcinoma- smoking, spirit, hot beverages, dietary.

pre-malignant caustic injury, achalasia, Kelly Paterson

Cupper esophageal cancer)

adenocarcinoma-GERD / Barrett esophagus, obesity.

PATHOGENICITY

Exophytic = more bleeding Endophytic= more pain Infiltrative= obstructive, dysphagia #Microscopic upper and middle 3rd - squamous cell carcinoma CMC type of ca overall) Lower 3rd Adenocarcinoma.

INVESTIGATIONS

1-Upper endoscopy- biopsy lesion
2-Chromoendoscopy- lugol's iodine
3-Barium swallow
TREATMENT
- Depend on staging: surgery for operable:T1/T2, No

For T3,N1/N2: multimodal treatment.

1-if operable, then approach-

Lower esophageal tumor - single left thoracic abdominal incision.

Ivor Lewis.

2-Extent of resection:

>10cm proximal and >5cm distal margin.

3-Reconstruction:

Conduit - gastric pull up (based on RGE)

Post operative chemotherapy - platinum based.

PREVENTION

Depending on the type of esophagitis you have, you may lessen symptoms or stop recurring problems by following these steps:

- 1. Do not eat foods that may increase reflux. ...
- 2. Use good pill-taking habits. ...
- 3. Lose weight. ...
- 4. If you smoke, quit. ...
- 5. Try not to stoop or bend, especially soon after eating.
- 6. Do not lie down after eating.

AWEARENCE!!

SMOKING CAN CAUSE

cancer, heart disease, stroke, lung diseases, diabetes, and chronic obstructive pulmonary disease (COPD), which includes emphysema and chronic bronchitis. Smoking also increases risk for tuberculosis, certain eye diseases, and problems of the immune system, including rheumatoid arthritis.



MUHAMMAD AHMAD AMMAR 5th year 9th Semester (2019-2024)

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a prevalent and progressive lung disease that poses a significant burden on global health. This article provides a comprehensive overview of COPD, including its epidemiology, mortality rates, etiology, risk factors, pathogenesis, diagnosis, and treatment options. By understanding these aspects, individuals can recognize the importance of prevention, early detection, and appropriate management of COPD to improve quality of life and reduce mortality rates.

EPIDEMIOLOGY

COPD affects millions of people worldwide, with a substantial impact on morbidity and mortality. According to the World Health Organization (WHO), an estimated 384 million people suffer from COPD globally . The prevalence of COPD varies across different regions, influenced by factors such as smoking rates, air pollution, and occupational exposures. [1]

MORTALITY

COPD is a leading cause of death worldwide. WHO reports that approximately 3 million people die from COPD each year, accounting for 6% of all global deaths. Mortality rates are higher in low- and middle-income countries, reflecting disparities in healthcare access, tobacco control measures, and environmental conditions.[2]

ETIOLOGY

Cigarette smoking is the primary cause of COPD, accounting for about 80-90% of cases. Prolonged exposure to cigarette smoke leads to chronic inflammation and structural changes in the airways and lungs. Other risk factors include long-term exposure to environmental pollutants (such as biomass fuel smoke and occupational dust), genetic predisposition, respiratory infections, and aging [3].

RISK FACTORS

Several risk factors contribute to the development and progression of COPD. Smoking is the most significant risk factor, but non-smokers can also develop COPD due to exposure to environmental pollutants. Occupational exposures, such as dust, chemicals, and fumes, increase the risk, especially in industries like mining, construction, and manufacturing. Genetic factors, including alpha-1 antitrypsin deficiency, can also predispose individuals to COPD [3].

PATHOGENESIS

The pathogenesis of COPD involves chronic inflammation, oxidative stress, and tissue damage in the lungs. Inhalation of irritants, particularly cigarette smoke, triggers an inflammatory response, leading to the recruitment of immune cells and the release of proteases. Over time, this inflammatory process damages the airways and lung tissue, resulting in airflow limitation, mucus production, and structural changes [4].

DIAGNOSIS

Accurate diagnosis of COPD is crucial for appropriate management. Healthcare professionals employ various diagnostic tools, including spirometry, to assess lung function and measure airflow limitation. Spirometry helps determine the severity of COPD and assists in staging the disease according to the GOLD system. Additionally, a comprehensive evaluation includes patient history, physical examination, imaging studies, and assessment of symptoms [4]. **TREATMENT**

While there is no cure for COPD, treatment aims to alleviate symptoms, improve lung function, prevent exacerbations, and enhance overall quality of life. The treatment plan often includes a combination of pharmacological interventions and non-pharmacological measures. Bronchodilators, such as beta-agonists and anticholinergics, help relax the airway muscles and improve airflow. Inhaled corticosteroids may be prescribed in combination with bronchodilators to reduce inflammation. Pulmonary rehabilitation programs, vaccination against respiratory infections, and oxygen therapy are also integral components of COPD management [5].

CONCLUSION

COPD is a widespread and life-threatening lung disease that requires attention and action at individual, community, and global levels. Understanding the epidemiology, etiology , risk factors, pathogenesis, diagnosis, and treatment options can aid in the prevention and effective management of COPD. By implementing preventive measures, raising awareness, and ensuring access to appropriate healthcare, we can reduce the burden of COPD and improve the lives of individuals affected by this condition.

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NEHA YADAV 5th year 9th Semester (2019-2024)

GASTRITIS

WHAT IS GASTRITIS?

Gastritis refers to the inflammation of the stomach lining, a condition that can cause discomfort and various digestive symptoms. It can be caused by several factors, including bacterial infections, excessive use of nonsteroidal anti-inflammatory drugs (NSAIDs), excessive alcohol consumption, stress, and autoimmune disorders. In this article, we will explore the types of gastritis, how it is diagnosed, and the precautions individuals can take to manage and prevent this condition.

TYPES OF GASTRITIS

Acute Gastritis:

Acute gastritis occurs suddenly and is usually a short-term condition. It is often caused by irritants such as alcohol, NSAIDs, bacterial infections (most commonly Helicobacter pylori), or severe stress. Symptoms may include abdominal pain, nausea, vomiting, and loss of appetite. With appropriate treatment and the removal of the irritant, acute gastritis typically resolves within a few days to weeks.

Chronic Gastritis:

Chronic gastritis is a long-term inflammation of the stomach lining. It can result from prolonged use of NSAIDs, H. pylori infection, autoimmune disorders, or other underlying medical conditions. Symptoms may be milder or even absent in some cases. Over time, chronic gastritis can lead to stomach ulcers, intestinal bleeding, and an increased risk of stomach cancer. **DIAGNOSIS OF GASTRITIS**

diagnose gastritis, a healthcare professional may perform the following tests: Medical History and Physical Examination:

The doctor will ask about your symptoms, medical history, and any medications you are currently taking. A physical examination may also be conducted to check for signs of gastritis. Endoscopy:

This procedure involves inserting a thin, flexible tube with a camera (endoscope) through the mouth into the stomach. It allows the doctor to visualize the stomach lining, take tissue samples for biopsy, and identify any abnormalities.

Blood Tests:

Blood tests can help identify H. pylori infection, anemia, and other underlying conditions associated with gastritis.

Stool Tests:

Stool tests may be conducted to detect the presence of H. pylori bacteria or signs of gastrointestinal bleeding.

PRECAUTIONS FOR MANAGING GASTRITIS

To manage and prevent gastritis, consider the following precautions:

Avoid Triggers:

Identify and avoid substances that can irritate the stomach lining, such as alcohol, spicy foods, caffeine, and NSAIDs. It's important to follow a healthy, balanced diet.

Quit Smoking:

Smoking can worsen gastritis and delay healing. If you smoke, quitting is highly recommended. Manage Stress:

Chronic stress can contribute to gastritis. Engage in stress-reducing activities like exercise, meditation, and relaxation techniques.

Take Medications as Prescribed:

If prescribed medications, such as proton pump inhibitors or antibiotics for H. pylori, take them as directed by your healthcare provider.

Follow a Healthy Lifestyle:

Maintain a healthy weight, exercise regularly, and get enough sleep. These lifestyle factors can support overall digestive health.

CONCLUSION

Gastritis, whether acute or chronic, can cause discomfort and affect your quality of life. Understanding the types, diagnosis procedures, and taking necessary precautions can help manage and prevent gastritis. If you experience persistent symptoms or have concerns, consult a healthcare professional for proper evaluation and guidance. With the right approach, gastritis can be effectively managed, allowing you to enjoy better digestive health.

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AWEARENCE!!

Just S'ag To Fruits & Vegetables

A diet rich in vegetables and fruits can lower blood pressure, reduce the risk of heart disease and stroke, prevent some types of cancer, lower risk of eye and digestive problems, and have a positive effect upon blood sugar, which can help keep appetite in check.



JAIPAL SINGH RAJPUT 5th year 9th Semester (2019-2024)

COMPLEX REGIONAL PAIN SYNDROME(CRPS)

CRPS, also known as reflex sympathetic dystrophy (RSD), is a chronic and often debilitating pain condition that primarily affects the extremities, typically an arm or a leg.

INTRODUCTION

In the late 19th century, physicians such as Silas Weir Mitchell, a neurologist, described a condition known as causalgia, characterized by severe, burning pain and changes in skin color and temperature.

The term "reflex sympathetic dystrophy" (RSD) was introduced by J.S. Evans in 1946. A compelling disease in the field of traumatology is complex regional pain syndrome (CRPS). It is characterized by intense and disproportionate pain, changes in skin color and temperature, swelling, and alterations in the functioning of the affected limb. CRPS can have a profound impact on an individual's quality of life, causing physical, psychological, and functional impairment.

CRPS typically occurs following an injury or trauma, although in some cases it may develop spontaneously without any identifiable trigger. The condition is believed to result from a complex interaction between the peripheral and central nervous systems, with abnormal pain signaling and dysfunction of the sympathetic nervous system playing a significant role.

TYPES OF CRPS

(CRPS) can be classified into two main types based on the presence or absence of a known nerve injury.

CRPS Type I (formerly known as Reflex Sympathetic Dystrophy or RSD):

CRPS Type I occurs when there is no identifiable nerve injury or direct damage to a specific nerve.

CRPS Type II (formerly known as Causalgia):

CRPS Type II is characterized by the presence of a documented nerve injury or lesion.

CASUES AND RISK FACTORS

Injury or Trauma:

CRPS often occurs following an injury or trauma, such as fractures, sprains, burns, surgery, or even minor injuries.

Nerve Dysfunction:

Dysfunction of the peripheral nerves or the central nervous system (spinal cord and brain) Inflammatory Response.

Abnormal Sympathetic Nervous System Activity.

Genetic Predisposition

Psychological Factors:

Emotional and psychological factors, such as stress, anxiety, and depression, may play a role in the development and progression of CRPS.

Gender and Age:CRPS is more common in females than males, with a female-to-male ratio ranging from 2:1 to 4:1.

SYMPTOMS

 \rightarrow The pain is often described as burning, stabbing, or throbbing.

 \rightarrow Changes in skin color and temperature: The affected limb may appear red or purple and feel warmer or cooler than the surrounding areas.

 \rightarrow Changes in skin texture: The skin may become shiny, thin, or have a glossy appearance.

 \rightarrow Swelling and edema

 \rightarrow Sensory abnormalities: Hypersensitivity to touch, increased sensitivity to temperature changes.

 \rightarrow Motor dysfunction: Weakness, decreased range of motion, and muscle spasms or tremors may occur.

 \rightarrow Autonomic dysfunction: Altered sweating patterns, changes in hair and nail growth, and abnormal blood flow regulation.

DIAGNOSIS

Diagnosing CRPS can be challenging as there are no definitive tests to confirm the condition. Diagnosis is primarily based on clinical examination, medical history, and the fulfillment of diagnostic criteria, such as the Budapest criteria. Exclusion of other potential causes of similar symptoms is also important.

TREATMENT

The treatment of CRPS aims to alleviate pain, improve function, and enhance quality of life. It typically involves a multidisciplinary approach, including:

Medications: Pain medications, such as analgesics, anti-inflammatory drugs, and neuropathic pain medications, may be prescribed.

Physical therapy: Exercises, physical modalities, and rehabilitation techniques help improve mobility, reduce pain, and prevent muscle atrophy.

Sympathetic nerve blocks: These injections target the sympathetic nervous system to alleviate pain and improve blood flow.

Psychological support: Counseling, cognitive-behavioral therapy, and relaxation techniques may be beneficial in managing the emotional and psychological impact of CRPS.

REFERENCE: NATRAJAN'S Textbook of Orthopedics and Traumatology 8/e Edition



ABHYAM GANGWAR 5th Year 9th Semester (2019-2024)

PRE-ECLAMPSIA

Pre-eclampsia is a multisystem disorder characterized by development of hypertension to the extent of 140/90 mm Hg or more with proteinuria after the 20th week in a previously normotensive and non-proteinuria woman.

EPIDEMIOLOGY and INCIDENCE RATE

Pre-eclampsia affects 2 to 8% of all pregnancies and remains a leading cause of maternal and perinatal morbidity and mortality.

The incidence of pre-eclampsia in hospital practice varies widely from 5 to 15%.

Young and nulliparous women are particularly vulnerable, whereas older women are at greater risk for chronic hypertension with superimposed preeclampsia . The incidence of preeclampsia is also influenced by race, ethnicity and genetic predisposition. In addition, black women carry higher risk for associated severe adverse outcomes

RISK FACTORS FOR PRE-ECLAMPSIA

- ✓ Primigravida: Young or elderly (first time exposure to chorionic villi)
- ✓ Family history: Hypertension, pre-eclampsia
- ✓ Placental abnormalities: Hyperplacentosis: Excessive exposure to chorionic villi,placental ischemia.
- ✓ Obesity: BMI >35 kg/M2, Insulin resistance
- ✓ Pre-existing vascular disease
- ✓ New paternity.
- \checkmark Thrombophilias

Multifetal gestation, Metabolic syndrome and Homocysteinemia are also among risk factors **ETIOPATHOLOGICAL FACTORS FOR PRE-ECLAMPSIA**

- ✓ Failure of trophoblast invasion (abnormal placentation)
- ✓ Vascular endothelial damage
- ✓ Inflammatory mediators (cytokines)
- ✓ Immunological intolerance between maternal and fetal tissues
- ✓ Coagulation abnormalities
- ✓ Increased oxygen free radicals
- ✓ Genetic predisposition (polygenic disorder)
- ✓ Dietary deficiency or excess

PATHOGENESIS

In a normal pregnancy vascular remodeling occurs but here abnormal vascular remodeling and abnormal placentation takes place which are responsible for following pathological stages

Stage I—Placental Syndrome

Due to abnormal vascular remodeling taking place spiral artery fails to develop into large tortous vascular channels and remains narrow thus leading to Placental hypoperfusion and placental ischemia

Stage II—Maternal syndrome

Inflammatory changes are believed to be a continuation of the placental syndrome. In response to ischemia or other inciting causes, placental factors are released and initiate a series of events like Increased SFLT-1 and decreased VEGf and placental growth factor along with release of Inflammatory mediators ,cytokines,IL,TNF alpha etc

All these events OF ENDOTHELIAL cell Activation leads to following changes in mother

• Significant Hemodynamic changes -

M/C and first hematological manifestation is thrombocytopenia

Also hemoconcentration noted which is manifested as \uparrow Hb and \uparrow hematocrit levels.

• Kidney Changes

RBF(renal blood flow) and GFR decrease in preeclampsia. Acute renal failure is a rare complication of preeclampsia but preeclampsia is the M/C cause of obstetric ARF(acute renal failure). ARF in preeclampsia is due to Acute Tubular necrosis.

• M/C visual symptom in preeclampsia is scotoma. Blindness here noticed is mostly due to vasospasm.

Mostly blindness is reversible and corrects automatically after delivery. On clinical examination the M/C ophthalmoscopic finding in a patient with severe preeclampsia is an increase in veingasto-artery ratio and segmental vasospasm.

CLASSIFICATION OF PRE-ECLAMPSIA

Pre-eclampsia can Further be Divided into

	Mild pre-eclampsia	Severe Pre-eclampsia
B/P	>/=140/90 mm of Hg	>/= 160/110 mm Hg
	but	
	=160/110 mm Hg</th <th></th>	
Proteinuria	>300 mg in 24 hour	>5 g in 24 hour urine
	urine	
Symptoms:		
Visual symptoms	Absent	Present
Oliguria	Absent	Present
Epigastric pain	Absent	Present
Headache	Absent	Present
HELLP syndrome	Absent	Present
Hemolysis	Absent	Present
Serum transaminase	Absent	Present
Low platelet count	Absent	Present
Uric acid levels	Normal	Elevated
Blood urea nitrogen	Normal	Elevated
IUGR	Not seen	Seen

MANAGEMENT OF PRE-ECLAMPSIA

Since pregnancy induced hypertension are raised BP conditions due to placental pathology (incomplete trophoblastic invasion) and thus their definitive treatment would be always Termination of pregnancy .

Antihypertensive of choice for pregnancy-induced hyper-tension (Preeclampsia/Gestational hypertension) is Labetalol

Now depending upon patient severity we manage pre-eclampsia

For mild pre-eclampsia

- Definitive management- (as discussed earlier will always be) Termination of pregnancy that is done at 37 completed weeks of pregnancy
- Antihypertensives- Role is doubtful here. No proven efficacy. Generally bed rest and some diet restrictions are done.

For Severe preeclampsia

- 1st step in management is seizure prophylaxis-MgSO4
- Antihypertensives should be given to decrease BP in a controlled manner without compromising the uteroplacental perfusion.

We aimed systolic BP between the range of 140 to 155 mm of Hg and Diastolic BP between 90 to 105 mm of Hg

- Glucocorticoids-for lung maturation
- Definitive management is termination of pregnancy at 34 completed weeks but if a pregnant woman comes with severe PIH before 23 weeks then terminate pregnancy immediately Note:

We terminate the pregnancy irrespective of the weeks of gestation in case of following conditions

- Severe preeclampsia, with impending eclampsia
- HELLP syndrome
- Pulmonary edema
- Significant renal dysfunction, coagulopathy
- Abruption, Previable fetus and Fetal compromise

PREVENTION

Some Methods to Prevent Preeclampsia That Have Been

Evaluated in Randomized Trials

- Dietary manipulation: low -salt diet, calcium or fish oll supplementation
- Exercise: physical activity, stretching
- Cardiovascular drugs: diuretics, antihypertensive drugs
- Antioxidants: ascorbic acid (vitamin c), alpha-tocopherol (vitamin E) and vitamin D

• Antithrombotic drugs: low-dose aspirin, aspirin/dipyridamole, aspirin + heparin, aspirin + ketanserin

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T.S.A.M.1



MUHAMMAD SAAD KHAN 5th Year 9th Semester (2019-2024)

THYROID CANCER

COMPREHENSIVE OVERVIEW

Thyroid cancer is a growth of abnormal cells that starts in the thyroid, a small, butterfly-shaped gland at the base of neck. This gland produces hormones that regulates metabolism. Thyroid hormones also help control body temperature, blood pressure and heartrate. Thyroid cancer, a type of endocrine cancer, is generally highly treatable, with an excellent cure rate. (source: Cleveland clinic)

OCCURRENCE

Close to 53,000 Americans receive a thyroid cancer diagnosis every year. Treatments for most thyroid cancers are very successful. Still, about 2,000 people die from the disease every year. Women and people assigned female at birth (AFAB) are three times more likely to get thyroid cancer compared to men and people assigned male at birth (AMAB). The disease is commonly diagnosed in women and people AFAB in their 40s and 50s and men and people AMAB in their 60s and 70s. Even children can develop the disease.

MORTALITY

The 5-year relative survival rate for thyroid cancer in the world is 98%. The survival rates for thyroid cancer vary based on several factors. These include the stage of cancer, a person's age and general health, and how well the treatment plan works.(Source: American society of clinical oncology .,2005)

CAUSES AND RISK FACTORS

In most cases, the cause of thyroid cancer is unknown. However, certain things can increase your chances of developing the condition.

Risk factors for thyroid cancer include:

- \checkmark having a benign (non-cancerous) thyroid condition
- \checkmark having a family history of thyroid cancer (in the case of medullary thyroid cancer)
- \checkmark having a bowel condition known as familial adenomatous polyposis
- \checkmark acromegaly a rare condition where the body produces too much growth hormone
- ✓ having a previous benign (non-cancerous) breast condition
- \checkmark weight and height
- ✓ radiation exposure

(Source: NHS INFO 24 -V1.1.1.25179)

PATHOGENESIS

Driver mutations in the TSH receptor signaling pathway play an important role in the pathogenesis of toxic adenomas. Activating somatic mutations in one of two components of this signaling system—most often the gene encoding the TSH receptor itself (TSHR) and, less commonly, the α -subunit of Gs (GNAS)—allow follicular cells to secrete thyroid hormone

independent of TSH stimulation (thyroid autonomy). The result of this overabundance is symptomatic hyperthyroidism, with a "hot" thyroid nodule seen on imaging studies. Overall, somatic mutations in the TSH receptor signaling pathway are present in slightly over half of toxic adenomas. Not surprisingly, such mutations also are observed in a subset of autonomous nodules that give rise to toxic multinodular goiters. Thus, autonomy in both toxic goiter and toxic adenomas stems from the ability of follicular cells to release thyroid hormone independent of trophic factors (i.e., TSH). A minority of nonfunctioning follicular adenomas.

(Source: ROBBINS BASIC PATHOLOGY, TENTH EDITION © 2018 by Elsevier) **DIAGNOSIS**

Tests and procedures used to diagnose thyroid cancer include;

Physical exam; examine neck to feel for changes in thyroid, such as a lump (nodule) in the thyroid.

Thyroid function blood tests; Test that measure blood levels of thyroid-stimulating hormone (TSH)

Ultrasound imaging; ultrasound to create images of the lymph nodes in the neck (lymph node mapping) to look for signs of cancer.

Removing a sample of thyroid tissue; During a fine-needle aspiration biopsy, your provider inserts a long, thin needle through your skin and into the thyroid nodule. Ultrasound imaging is typically used to precisely guide the needle. Your provider uses the needle to remove some cells from the thyroid. The sample is sent to a lab for analysis.

An imaging test that uses a radioactive tracer; A radioactive iodine scan uses a radioactive form of iodine and a special camera to detect thyroid cancer cells in body.

Genetic testing; A portion of medullary thyroid cancers are caused by inherited genes that are passed from parents to children.

(Source; 1998 Mayo Foundation for Medical and Research) TREATMENT

- ✓ Doxorubicin Hydrochloride
- ✓ Cabozantinib-S-Malate
- ✓ Caprelsa (Vandetanib)

(Source: NCIinfo@nih,gov)



ABID HUSSIAN 5th Year 9th Semester (2019-2024)

VIRAL HEPATITIS

Viral hepatitis refers to inflammation of the liver caused by infection with one of several viruses, including hepatitis A, B, C, D, and E and other including EBV and CMV.Early it causes acute hepatitis but with passage of time it my progress to chronic hepatitis **Ref-(Pathoma) EPIDEMIOLOGY**

About 2.3 billion people of the world are infected with one or more of the hepatitis viruses. Viral hepatitis results in around 1.4 million deaths each year, HBV and HCV are responsible for about 90% of these fatalities, whilst the remaining 10% of fatalities are caused by other hepatitis viruses(WHO) AND NCBI)

MORTALITY RATE IN VIRAL HEPATITIS

Mortality rates in viral hepatitis vary depending on the specific type, but generally range from less than 0.5% for hepatitis A to higher rates for chronic hepatitis B and C, particularly in cases of cirrhosis and liver cancer Ref-(Wikipedia+ chat gpt)

SIGNS AND SYMPTOMS

Fatigue: People with viral hepatitis often experience extreme tiredness and lack of energy, which can significantly impact their daily activities..

Jaundice: One of the hallmark symptoms of viral hepatitis is yellowing of the skin and eyes. This occurs due to the buildup of bilirubin, a yellow pigment, in the body.

Abdominal pain: Many individuals with viral hepatitis complain of pain or discomfort in the upper right side of the abdomen. This can range from mild to severe and may be accompanied by bloating or tenderness.

Loss of appetite: Viral hepatitis can cause a significant decrease in appetite, leading to unintentional weight loss and malnutrition.

Nausea and vomiting: Many people with viral hepatitis experience feelings of nausea and may vomit, especially after eating fatty or greasy foods.

Dark urine: The urine of individuals with viral hepatitis may appear dark or tea-colored due to the presence of bilirubin.

Pale stools: On the other hand, the stools may become pale or clay-colored, indicating a disruption in the normal bile flow.

Flu-like symptoms: Some individuals with viral hepatitis may initially experience symptoms resembling the flu, such as fever, headache, muscle aches, and general malaise.

ONE OF THE HALLMARK OF VIRAL HEPATITIS IS ELEVATED (ALT AND AST)

These symptoms persist More than 6 months it can lead to chronic hepatitis Ref-Internal Medicine by Inam Danish

DIAGNOSIS OF VIRAL HEPATITIS:

To diagnose viral hepatitis, a combination of clinical evaluation, laboratory tests, and sometimes imaging studies may be used. Here are some key points you might find in the book regarding the diagnosis of viral hepatitis:

History and Physical Examination: The journal will emphasize the importance of obtaining a detailed medical history, including risk factors such as exposure to contaminated blood or body fluids, intravenous drug use, sexual practices, recent travel, and vaccination history. The physical examination may reveal signs such as jaundice (yellowing of the skin and eyes), hepatomegaly (enlarged liver), or other signs of liver dysfunction.

Laboratory Tests: The journal will discuss specific laboratory tests used to diagnose viral hepatitis. These may include:

Liver Function Tests (LFTs): LFTs assess liver function and include tests such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and bilirubin levels. Elevated ALT and AST are typically seen in acute viral hepatitis.

Serology: Serologic testing detects specific antibodies or antigens associated with different types of viral hepatitis. For example, hepatitis B surface antigen (HBsAg) and anti-hepatitis B core antibody (anti-HBc) are used to diagnose hepatitis B infection.

Polymerase Chain Reaction (PCR): PCR testing can detect and quantify viral genetic material (DNA or RNA) in the blood, providing valuable information about the presence and activity of the virus.

Imaging Studies: The journal may mention imaging studies like ultrasound or computed tomography (CT) scan of the abdomen. These imaging modalities can help evaluate the liver for structural changes, such as cirrhosis or hepatocellular carcinoma, which may occur as a result of chronic viral hepatitis.

Additional Tests: Depending on the clinical scenario, the book may discuss additional tests such as liver biopsy, which can provide detailed information about the extent of liver damage and the presence of fibrosis or inflammation Ref-USLME STEP 1

TREATMENT OF VIRAL HEPATITIS:

Interferons (IFNs): These are naturally occurring proteins that help boost the immune response against viruses. Interferon-alpha is used for the treatment of chronic hepatitis B and C.

Ribavirin: This antiviral drug is primarily used in combination with peginterferon-alpha for the treatment of chronic hepatitis C. It may also be used in some cases of respiratory syncytial virus (RSV) infection.

Sofosbuvir: It is a direct-acting antiviral (DAA) agent used for the treatment of chronic hepatitis C. Sofosbuvir is often used in combination with other DAAs, such as ledipasvir or velpatasvir. Simeprevir: This is another DAA used specifically for the treatment of chronic hepatitis C. It is often used in combination with other antiviral agents.

Daclatasvir: It is a DAA that is used in combination with other antiviral medications for the treatment of chronic hepatitis C.

Ledipasvir: This DAA is used in combination with sofosbuvir for the treatment of chronic hepatitis C. The combination of ledipasvir and sofosbuvir has shown high efficacy against certain genotypes of the hepatitis C virus.

Velpatasvir: It is a pan-genotypic DAA used in combination with sofosbuvir for the treatment of chronic hepatitis C.

It is important to note that the field of viral hepatitis treatment is constantly evolving, and new drugs may become available over time. Additionally, specific treatment regimens may vary based on the viral genotype, the presence of cirrhosis, and other individual patient factors. It is always recommended to refer to the most up-to-date guidelines and consult with a healthcare professional for accurate and current information on the treatment of viral hepatitis.

COMPLICATIONS OF VIRL HEPATITIS

Chronic Hepatitis: Viral hepatitis infections, particularly hepatitis B and C, can progress to a chronic state, leading to ongoing liver inflammation and damage. Chronic hepatitis can eventually result in complications such as cirrhosis (scarring of the liver), liver failure, and hepatocellular carcinoma (liver cancer).

Cirrhosis: Prolonged liver inflammation can cause fibrosis (scarring) of the liver tissue, leading to cirrhosis. Cirrhosis can impair liver function, resulting in portal hypertension (high blood pressure in the portal vein), ascites (accumulation of fluid in the abdomen), hepatic encephalopathy (brain dysfunction due to liver failure), and other complications.

Liver Failure: Advanced stages of viral hepatitis can lead to liver failure, wherein the liver loses its ability to perform essential functions. Liver failure can be life-threatening and may necessitate liver transplantation.

Hepatocellular Carcinoma (HCC): Chronic infection with hepatitis B or C viruses increases the risk of developing hepatocellular carcinoma, which is the most common form of liver cancer. **PREVENTION OF VIRAL HEPATITIS**

Vaccination: Vaccination is available for hepatitis A and hepatitis B. Hepatitis A vaccine is recommended for individuals at high risk, such as healthcare workers, travelers to endemic areas, and men who have sex with men. Hepatitis B vaccine is part of routine childhood immunization and is also recommended for high-risk adults.

Universal Precautions: Healthcare workers should follow standard precautions, including hand hygiene, wearing gloves, masks, and protective clothing, and safely handling and disposing of medical sharps to prevent the transmission of hepatitis viruses.

Safe Injection Practices: Proper sterilization techniques and the use of disposable needles and syringes should be followed to prevent transmission of hepatitis viruses.

Blood and Organ Screening: Screening donated blood, organs, and tissues for hepatitis B and C viruses reduces the risk of transmission through transfusion or transplantation.

Safe Sexual Practices: Practicing safe sex, such as using condoms and limiting sexual partners, can reduce the risk of acquiring hepatitis B and C through sexual contact.

Injection Drug Use Safety: Avoiding the use of illicit drugs or sharing needles and other drug paraphernalia can prevent the transmission of hepatitis B and C viruses.



IRSA YASEEN 5th Year 9th Semester (2019-2024)

ENDOCARDITIS

COMPREHENSIVE OVERVIEW

Bacterial endocarditis refers to infection of the endocardial surface of the heart. It usually involves heart valves, but it can occur on the endocardium or intracardiac devices. OCCURRENCE

In developed countries, the incidence of endocarditis ranges from 2.6 to 7 cases per 100,000 population per year. The median age of patients with endocarditis is 58 years. **SYMPTOMS**

Common symptoms of endocarditis include:

Aching joints and muscles	Night sweats
Chest pain when you breathe	Shortness of breath
Fatigue	Swelling in the feet, legs or belly
Flu-like symptoms, such as fever and chills	A new or changed whooshing sound in the heart (murmur)

CAUSES

Most cases are caused by viridans streptococci, Streptococcus gallolyticus, Staphylococcus aureus, coagulase-negative staphylococci, HACEK organisms

(Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella, Kingella), and enterococci. Rarer organisms include pneumococci, Candida, gram-negative bacilli, and polymicrobial organisms **RISK FACTORS**

Artificial heart valves	Illegal IV drug use
Damaged heart valves	Poor dental health
Congenital heart defects	Long-term catheter use
Implanted heart device	Older age. Endocarditis occurs most often in adults over age 60.

Source; 1998 Mayo foundation for Medical Education

PATHOGENESIS

Endothelial injury allows for either direct infection by virulent organisms or the development of uninfected platelet-fibrin thrombus which becomes a nidus for transient bacteremia, except in the case of *S. aureus*, which can infect intact endothelium. These organisms enter the bloodstream from the skin, mucosal surfaces or previously infected sites and adhere to nonbacterial thrombus due to valvular damage or turbulent blood flow. In the absence of host defenses, this organism is allowed to proliferate forming small colonies and shed in the bloodstream. Left-sided infection is much more common than right-sided infection, except among intravenous drug users.

DIAGNOSIS

Tests used to help diagnose endocarditis include:

1 0	
Blood culture test	Chest X-ray
Complete blood count	Electrocardiogram (ECG or EKG)
Echocardiogram	Computerized tomography (CT) scan or magnetic resonance imaging (MRI)

TREATMENT

-High doses of IV antibiotics are used to treat endocarditis caused by bacteria. If you receive IV antibiotics, you'll generally spend a week or more in the hospital so that care providers can determine if the treatment is working.

-If endocarditis is caused by a fungal infection, antifungal medication is given. Some people need lifelong antifungal pills to prevent endocarditis from returning.

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AWEARENCE!!



Keep a normal temperature. Lubricate and cushion joints. Protect your spinal cord and other sensitive tissues. Get rid of wastes through urination, perspiration, and bowel movements. AND MANY MORE



AVINASH SINGH 5th Year 9th Semester (2019-2024)

BROWN-SEQUARD SYNDROME(BSS)

It is a neurological conditions characterized by lesion in the spinal cord which results in weakness or paralysis on one side of the body and loss of sensation on opposite sides

ETIOLOGY

Gun shot	Blunt trauma
Knife injury	Spinal fractures from an accident such as fall
Stab wounds	

AFFECTED TRACK & SIGNS

Dorsal Column:- Ipsilateral loss of proprioception fine Touch, vibration, 2 point discrimination below the level of lesion

Spinothalamic Tract- Contralateral loss of pain, crude touch and temperature below the level of lesion

Corticospinal Tract: -Ipsilateral motor loss below the level of lesion

At the level of Lesion: -Total loss of all sensation as no fiber enters the spinal cord

DIAGNOSIS

- \checkmark Perform a thorough physical exam.
- \checkmark Perform a neurological exam consisting of detailed motor and sensory evaluation.
- ✓ Ask detailed questions about your medical, neurological and trauma history.
- ✓ Your provider will then order certain tests, such as an MRI scan, to confirm the diagnosis or rule out other possible causes of your symptoms

TREATMENT

The treatment for Brown-Sequard syndrome (BSS) depends on the underlying cause of the spinal cord damage.

- \checkmark Preserving or improving motor and sensory function.
- ✓ Preventing secondary injuries.
- ✓ Minimizing complications.
- ✓ Surgery: Non-traumatic causes of BSS usually involve mechanical compression or herniation of your spinal cord that requires surgical treatment to alleviate the compression.
- ✓ Medication to prevent blood clots: People with spinal cord injuries have the highest rate of venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism, among people with severe trauma. Your provider will likely give you medications known as anticoagulants, such as heparin or warfarin, to try to prevent blood clots.





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