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An Introduction to Medical Biotechnology

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CHAPTER 1

Communicable & non- Communicable diseases: Treatment of Pneumonia

Pneumonia

Pneumonia is a lung infection that can range from mild to so severe that you have to go to the hospital.

It happens when an infection causes the air sacs in your lungs (your doctor will call them alveoli) to fill with fluid or pus. That can make it hard for you to breathe in enough oxygen to reach your bloodstream. Anyone can get this lung infection. But infants younger than age 2 and people over age 65 are at higher risk. That's because their immune systems might not be strong enough to fight it. You can get pneumonia in one or both lungs. You can also have it and not know it. Doctors call this walking pneumonia. Causes include bacteria, viruses, and fungi. If your pneumonia results from bacteria or a virus, you can spread it to someone else.

Lifestyle habits, like smoking cigarettes and drinking too much alcohol, can also raise your chances of getting pneumonia.

Symptoms of Pneumonia:

Your symptoms can vary depending on what's causing your pneumonia, your age, and your overall health. They usually develop over several days.

Common pneumonia symptoms include:

- Chest pain when you breathe or cough
- Cough that produces phlegm or mucus
- Fatigue and loss of appetite
- Fever, sweating, and chills
- Nausea, vomiting, and diarrhea
- Shortness of breath

Along with these symptoms, older adults and people with weak immune systems might be confused or have changes in mental awareness, or they might have a lower-than-normal body temperature. Newborns and infants may not show any signs of infection. Or they might vomit, have a fever and a cough, and seem restless or tired.

Causes of Pneumonia:

Bacteria, viruses, or fungi can lead to pneumonia.

Top causes include:

- Flu viruses
- Cold viruses
- RSV virus (the top cause of pneumonia in babies age 1 or younger)
- Bacteria called Streptococcus pneumonia and Mycoplasma pneumoniae

Some people who are in the hospital get "ventilator-associated pneumonia" if they got the infection while using a ventilator, a machine that helps you breathe.

If you get pneumonia while you are in a hospital and aren't on a ventilator, that's called "hospital-acquired" pneumonia. But most people get "community-acquired pneumonia," which means they didn't get it in a hospital.

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Competitive questions from today topic (2 questions Minimum)-

Which of the following events occurs first in the differentiation sequence of the human B cells in the bone marrow?

A. immunoglobulin light chain rearrangement

B. immunoglobulin heavy chain rearrangement

C. surface IgD and IgM present on the B cell

D. Surface IgM present on the B cell

Exam Name DBT JRF 2015

Which of the following is an atypical signaling receptor?

A. Cytokine receptor

B. Chemokine receptor

C. T-cell receptor

D. Mannose receptor

Exam NameDBT JRF 2015

- **Suggestions to secure good marks to answer in exam-**
  - Give answer with complete labeled diagrams.
  - Explain answer with key point answers

- **Questions to check understanding level of students-**
  - What are the symptoms of pneumonia?
  - Explain Treatment of pneumonia.

CHAPTER 2

Communicable & non-Communicable diseases: Sign and Symptoms of Dysentery

Dysentery

Dysentery is a type of gastroenteritis that results in diarrhea with blood. Other symptoms may include fever, abdominal pain, and a feeling of incomplete defecation. Complications may include dehydration.

The cause of dysentery is usually Shigella, in which case it is known as shigellosis, or Entamoeba histolytica. Other causes may include certain chemicals, other bacteria, other protozoa, or parasitic worms. It may spread between people. Risk factors include contamination of food and water with feces due to poor sanitation. The underlying mechanism involves inflammation of the intestine, especially of the colon.

Efforts to prevent dysentery include hand washing and food safety measures while traveling in areas of high risk. While the condition generally resolves on its own within a week, drinking sufficient fluids such as oral rehydration solution is important. Antibiotics such as azithromycin may be used to treat cases associated with travelling in the developing world. While medications used to decrease diarrhea such as loperamide are not recommended on their own, they may be used together with antibiotics.

Shigella results in about 165 million cases of diarrhea and 1.1 million deaths a year with nearly all cases in the developing world. In areas with poor sanitation nearly half of cases of diarrhea are due to Entamoeba histolytica. Entamoeba histolytica affects millions of people and results in greater than 55,000 deaths a year. It commonly occurs in less developed areas of Central and South America, Africa, and Asia. Dysentery has been described at least since the time of Hippocrates.

Signs and symptoms:

The most common form of dysentery is bacillary dysentery, which is typically a mild sickness, causing symptoms normally consisting of mild gut pains and frequent passage of stool or diarrhea. Symptoms normally present themselves after 1–3 days, and are usually no longer present after a week. The frequency of urges to defecate, the large volume of liquid feces ejected, and the presence of blood, mucus, or pus depends on the pathogen causing the disease. Temporary lactose intolerance can occur, as well. In some caustic occasions, severe abdominal cramps, fever, shock, and delirium can all be symptoms.

In extreme cases, people may pass more than one liter of fluid per hour. More often, individuals will complain of diarrhea with blood, accompanied by abdominal pain, rectal pain and a low-grade fever. Rapid weight loss and muscle aches sometimes also accompany dysentery, while nausea and vomiting are rare. On rare occasions, the amoebic parasite will invade the body through the bloodstream and spread beyond the intestines. In such cases, it may more seriously infect other

organs such as the brain, lungs, and most commonly the liver. Mechanism Dysentery results from bacterial or parasitic infections. Viruses do not generally cause the disease. These pathogens typically reach the large intestine after entering orally, through ingestion of contaminated food or water, oral contact with contaminated objects or hands, and so on.

Each specific pathogen has its own mechanism or pathogenesis, but in general, the result is damage to the intestinal linings, leading to the inflammatory immune responses. This can cause elevated physical temperature, painful spasms of the intestinal muscles (cramping), swelling due to fluid leaking from capillaries of the intestine (edema) and further tissue damage by the body's immune cells and the chemicals, called cytokines, which are released to fight the infection. The result can be impaired nutrient absorption, excessive water and mineral loss through the stools due to breakdown of the control mechanisms in the intestinal tissue that normally remove water from the stools, and in severe cases, the entry of pathogenic organisms into the bloodstream. Anemia may also arise due to the blood loss through diarrhea.

Bacterial infections that cause bloody diarrhea are typically classified as being either invasive or toxogenic. Invasive species cause damage directly by invading into the mucosa. The toxogenic species do not invade, but cause cellular damage by secreting toxins, resulting in bloody diarrhea. This is also in contrast to toxins that cause watery diarrhea, which usually do not cause cellular damage, but rather they take over cellular machinery for a portion of life of the cell.

Some microorganisms –
For example, bacteria of the genus Shigella – secrete substances known as cytotoxins, which kill and damage intestinal tissue on contact. Shigella is thought to cause bleeding due to invasion rather than toxin, because even non-toxogenic strains can cause dysentery, but E. coli with shiga-like toxins do not invade the intestinal mucosa, and are therefore toxin dependent.

Definitions of dysentery can vary by region and by medical specialty. The U. S. Centers for Disease Control and Prevention (CDC) limits its definition to "diarrhea with visible blood". Others define the term more broadly. These differences in definition must be taken into account when defining mechanisms. For example, using the CDC definition requires that intestinal tissue be so severely damaged that blood vessels have ruptured, allowing visible quantities of blood to be lost with defecation. Other definitions require less specific damage.

Amoebic dysentery

- **Amoebiasis**

Amoebiasis, also known as amoebic dysentery, is caused by an infection from the amoeba Entamoeba histolytica, which is found mainly in tropical areas. Proper treatment of the underlying infection of amoebic dysentery is important; insufficiently treated amoebiasis can lie dormant for years and subsequently lead to severe, potentially fatal, complications.

When amoebae inside the bowel of an infected person are ready to leave the body, they group together and form a shell that surrounds and protects them. This group of amoebae is known as a cyst, which is then passed out of the person's body in the feces and can survive outside the body. If hygiene standards are poor – for example, if the person does not dispose of the feces hygienically – then it can contaminate the surroundings, such as nearby food and water. If another person then eats or drinks food or water that has been contaminated with feces containing the cyst, that person will also become infected with the amoebae. Amoebic dysentery is particularly common in parts of the world where human feces are used as fertilizer. After entering the person's body through the mouth, the cyst travels down into the stomach. The amoebae inside the cyst are protected from the stomach's digestive acid. From the stomach, the cyst travels to the intestines, where it breaks open and releases the amoebae, causing the infection. The amoebae can burrow into the walls of the intestines and cause small abscesses and ulcers to form. The cycle then begins again.

Bacillary dysentery

- **Bacillary dysentery**

Dysentery may also be caused by shigellosis, an infection by bacteria of the genus Shigella, and is then known as bacillary dysentery (or Marlow syndrome). The term bacillary dysentery etymologically might seem to refer to any dysentery caused by any bacilliform bacteria, but its meaning is restricted by convention to Shigella dysentery.
Other bacteria

Some strains of Escherichia coli cause bloody diarrhea. The typical culprits are enter hemorrhagic Escherichia coli, of which O157:H7 is the best known.
Diagnosis

A diagnosis may be made by taking a history and doing a brief examination. Dysentery should not be confused with hematochezia, which is the passage of fresh blood through the anus, usually in or with stools.
Physical exam

The mouth, skin, and lips may appear dry due to dehydration. Lower abdominal tenderness may also be present.
Stool and blood tests

Cultures of stool samples are examined to identify the organism causing dysentery. Usually, several samples must be obtained due to the number of amoebae, which changes daily. Blood tests can be used to measure abnormalities in the levels of essential minerals and salts.

Prevention:
Efforts to prevent dysentery include hand washing and food safety measures while traveling in areas of high risk.

Vaccine:
Although there is currently no vaccine which protects against Shigella infection, several are in development. Vaccination may eventually become a part of the strategy to reduce the incidence and severity of diarrhea, particularly among children in low-resource settings. For example, Shigella is a longstanding World Health Organization (WHO) target for vaccine development, and sharp declines in age-specific diarrhea/dysentery attack rates for this pathogen indicate that natural immunity does develop following exposure; thus, vaccination to prevent this disease should be feasible. The development of vaccines against these types of infection has been hampered by technical constraints, insufficient support for coordination, and a lack of market forces for research and development. Most vaccine development efforts are taking place in the public sector or as research programs within biotechnology companies.

Treatment:
Dysentery is managed by maintaining fluids using oral rehydration therapy. If this treatment cannot be adequately maintained due to vomiting or the profuseness of diarrhea, hospital admission may be required for intravenous fluid replacement. In ideal situations, no antimicrobial therapy should be administered until microbiological microscopy and culture studies have established the specific infection involved. When laboratory services are not available, it may be necessary to administer a combination of drugs, including an amoebicidal drug to kill the parasite, and an antibiotic to treat any associated bacterial infection.

If shigellosis is suspected and it is not too severe, letting it run its course may be reasonable — usually less than a week. If the case is severe, antibiotics such as ciprofloxacin or TMP-SMX may be useful. However, many strains of Shigella are becoming resistant to common antibiotics, and effective medications are often in short supply in developing countries. If necessary, a doctor may have to reserve antibiotics for those at highest risk for death, including young children, people over 50, and anyone suffering from dehydration or malnutrition.

Amoebic dysentery is often treated with two antimicrobial drugs such as metronidazole and paromomycin or iodoquinol.

Epidemiology:
Insufficient data exists, but Shigella is estimated to have caused the death of 34,000 children under the age of five in 2013, and 40,000 deaths in people over five years of age. Amoebiasis infects over 50 million people each year, of whom 50,000 die.

History:
The seed, leaves, and bark of the kapok tree have been used in traditional medicine by indigenous peoples of the rainforest regions in the Americas, west-central Africa, and Southeast Asia in this disease. Bacillus subtilis was marketed throughout America and Europe from 1946 as an immunostimulatory aid in the treatment of gut and urinary tract diseases such as rotavirus and Shigella, but declined in popularity after the introduction of consumer antibiotics.

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CHAPTER 3

Communicable & non- Communicable diseases: Sign and Symptoms of Atherosclerosis/ Hyperlipidaemia.

Hyperlipidemia

Hyperlipidemia is abnormally elevated levels of any or all lipids or lipoproteins in the blood.

Lipids (water-insoluble molecules) are transported in a protein capsule. The size of that capsule, or lipoprotein, determines its density. The lipoprotein density and type of apolipoproteins it contains determines the fate of the particle and its influence on metabolism.

Hyperlipidemis are divided into primary and secondary subtypes. Primary hyperlipidemia is usually due to genetic causes (such as a mutation in a receptor protein), while secondary hyperlipidemia arises due to other underlying causes such as diabetes. Lipid and lipoprotein abnormalities are common in the general population and are regarded as modifiable risk factors for cardiovascular disease due to their influence on atherosclerosis. In addition, some forms may predispose to acute pancreatitis.

Relation to cardiovascular disease

Hyperlipidemia predisposes a person to atherosclerosis. Atherosclerosis is the accumulation of lipids, cholesterol, calcium, fibrous plaques within the artery walls of the heart. This accumulation narrows the blood vessel and reduces blood flow and oxygen to muscles of the heart. Complete blockage of the artery causes infarction of the myocardial cells, also known as heart attack.

Classification:

Hyperlipidemias may basically be classified as either familial (also called primary) caused by specific genetic abnormalities, or acquired (also called secondary) when resulting from another underlying disorder that leads to alterations in plasma lipid and lipoprotein metabolism. Also, hyperlipidemia may be idiopathic, that is, without a known cause.

Hyperlipidemias are also classified according to which types of lipids are elevated, that is hypercholesterolemia, hypertriglyceridemia or both in combined hyperlipidemia. Elevated levels of Lipoprotein (a) may also be classified as a form of hyperlipidemia.

Familial (primary)

Familial hyperlipidemias are classified according to the Fredrickson classification, which is based on the pattern of lipoproteins on electrophoresis or ultracentrifugation. It was later adopted by the World Health Organization (WHO). It does not directly account for HDL, and it does not distinguish among the different genes that may be partially responsible for some of these conditions.

Type I

Type I hyperlipoproteinemia exists in several forms:

Lipoprotein lipase deficiency (type Ia), due to a deficiency of lipoprotein lipase (LPL) or altered apolipoprotein C2, resulting in elevated chylomicrons, the particles that transfer fatty acids from the digestive tract to the liver

Familial apoprotein CII deficiency (type Ib), a condition caused by a lack of lipoprotein lipase activator. Chylomicronemia due to circulating inhibitor of lipoprotein lipase (type Ic)

Type I hyperlipoproteinemia usually presents in childhood with eruptive xanthomata and abdominal colic. Complications include retinal vein occlusion, acute pancreatitis, steatosis, and organomegaly, and lipemia retinalis.

Type II

Hyperlipoproteinemia type II, by far the most common form, is further classified into types IIa and IIb, depending mainly on whether elevation in the triglyceride level occurs in addition to LDL cholesterol.

Type IIa

Familial hypercholesterolemia

This may be sporadic (due to dietary factors), polygenic, or truly familial as a result of a mutation either in the LDL receptor gene on chromosome 19 (0.2% of the population) or the ApoB gene (0.2%). The familial form is characterized by tendon xanthoma, xanthelasma, and premature cardiovascular disease. The incidence of this disease is about one in 500 for heterozygotes, and one in 1,000,000 for homozygotes.

HLPIIa is a rare genetic disorder characterized by increased levels of LDL cholesterol in the blood due to the lack of uptake (no Apo B receptors) of LDL particles. This pathology, however, is the second-most common disorder of the various hyperlipoproteinemias, with individuals with a heterozygotic predisposition of one in every 500 and individuals with homozygotic predisposition of one in every million. These individuals may present with a unique set of physical characteristics such as xanthelasmas (yellow deposits of fat underneath the skin often presenting in the nasal portion of the eye), tendon and tuberous xanthomas, arcus juvenilis (the graying of the eye often characterized in older individuals), arterial bruits, claudication, and of course atherosclerosis. Laboratory findings for these individuals are significant for total serum cholesterol levels two to three times greater than normal, as well as increased LDL cholesterol, but their triglycerides and VLDL values fall in the normal ranges.

To manage persons with HLPIIa, drastic measures may need to be taken, especially if their HDL cholesterol levels are less than 30 mg/dL and their LDL levels are greater than 160 mg/dL. A proper diet for these individuals requires a decrease in total fat to less than 30% of total calories with a ratio of monounsaturated:polyunsaturated:saturated fat of 1:1:1. Cholesterol should be reduced to less than 300 mg/day, thus the avoidance of animal products and to increase fiber intake to more than 20 g/day

with 6g of soluble fiber/day. Exercise should be promoted, as it can increase HDL. The overall prognosis for these individuals is in the worst-case scenario if uncontrolled and untreated individuals may die before the age of 20, but if one seeks a prudent diet with correct medical intervention, the individual may see an increased incidence of xanthomas with each decade, and Achilles tendinitis and accelerated atherosclerosis will occur.

Type IIb

The high VLDL levels are due to overproduction of substrates, including triglycerides, acetyl-CoA, and an increase in B-100 synthesis. They may also be caused by the decreased clearance of LDL. Prevalence in the population is 10%.

Familial combined hyperlipoproteinemia (FCH)

Lysosomal acid lipase deficiency, often called (Cholesteryl ester storage disease)

Secondary combined hyperlipoproteinemia (usually in the context of metabolic syndrome, for which it is a diagnostic criterion)

Type III

This form is due to high chylomicrons and IDL (intermediate density lipoprotein). Also known as broad beta disease or dysbetalipoproteinemia, the most common cause for this form is the presence of ApoE E2/E2 genotype. It is due to cholesterol-rich VLDL (β-VLDL). Its prevalence has been estimated to be approximately 1 in 10,000.

It is associated with hypercholesterolemia (typically 8–12 mmol/L), hypertriglyceridemia (typically 5–20 mmol/L), a normal ApoB concentration, and two types of skin signs (palmar xanthomata or orange discoloration of skin creases, and tuberoeruptive xanthomata on the elbows and knees). It is characterized by the early onset of cardiovascular disease and peripheral vascular disease. Remnant hyperlipidemia occurs as a result of abnormal function of the ApoE receptor, which is normally required for clearance of chylomicron remnants and IDL from the circulation. The receptor defect causes levels of chylomicron remnants and IDL to be higher than normal in the blood stream. The receptor defect is an autosomal recessive mutation or polymorphism.

Type IV

Familial hypertriglyceridemia is an autosomal dominant condition occurring in approximately 1% of the population.

This form is due to high triglyceride level. Other lipoprotein levels are normal or increased a little.

Treatment includes diet control, fibrates and niacins. Statins are not better than fibrates when lowering triglyceride levels.

Type V

Hyperlipoproteinemia type V, also known as mixed hyperlipoproteinemia familial or mixed hyperlipidemia, is very similar to type I, but with high VLDL in addition to chylomicrons.

It is also associated with glucose intolerance and hyperuricemia.

In medicine, combined hyperlipidemia (or -aemia) (also known as "multiple-type hyperlipoproteinemia") is a commonly occurring form of hypercholesterolemia (elevated cholesterol levels) characterized by increased LDL and triglyceride concentrations, often accompanied by decreased HDL. On lipoprotein electrophoresis (a test now rarely performed) it shows as a hyperlipoproteinemia type IIB. It is the most common inherited lipid disorder, occurring in about one in 200 persons. In fact, almost one in five individuals who develop coronary heart disease before the age of 60 has this disorder. The elevated triglyceride levels (>5 mmol/l) are generally due to an increase in very low density lipoprotein (VLDL), a class of lipoprotein prone to cause atherosclerosis.

Types

Familial combined hyperlipidemia (FCH) is the familial occurrence of this disorder, probably caused by decreased LDL receptor and increased ApoB.

FCH is extremely common in people who suffer from other diseases from the metabolic syndrome ("syndrome X", incorporating diabetes mellitus type II, hypertension, central obesity and CH). Excessive free fatty acid production by various tissues leads to increased VLDL synthesis by the liver. Initially, most VLDL is converted into LDL until this mechanism is saturated, after which VLDL levels elevate.

Both conditions are treated with fibrate drugs, which act on the peroxisome proliferator-activated receptors (PPARs), specifically PPARα, to decrease free fatty acid production. Statin drugs, especially the synthetic statins (atorvastatin and rosuvastatin) can decrease LDL levels by increasing hepatic reuptake of LDL due to increased LDL-receptor expression.

Unclassified familial forms

These unclassified forms are extremely rare:

- Hyperalphalipoproteinemia
- Polygenic hypercholesterolemia

Acquired (secondary)

Acquired hyperlipidemias (also called secondary dyslipoproteinemias) often mimic primary forms of hyperlipidemia and can have similar consequences. They may result in increased risk of premature atherosclerosis or, when associated with marked hypertriglyceridemia, may lead to pancreatitis and

other complications of the chylomicronemia syndrome. The most common causes of acquired hyperlipidemia are:

Diabetes mellitus

Use of drugs such as thiazide diuretics, beta blockers, and estrogens

Other conditions leading to acquired hyperlipidemia include:

- Hypothyroidism
- Kidney failure
- Nephrotic syndrome
- Alcohol consumption
- Some rare endocrine disorders and metabolic disorders

Treatment of the underlying condition, when possible or discontinuation of the offending drugs usually leads to an improvement in the hyperlipidemia.

Another acquired cause of hyperlipidemia, although not always included in this category, is postprandial hyperlipidemia, a normal increase following ingestion of food.

Screening

Serum level of Low Density Lipoproteins (LDL) cholesterol, High Density Lipoproteins (HDL) Cholesterol, and triglycerides are commonly tested in primary care setting using a lipid panel. Quantitative levels of lipoproteins and triglycerides contribute toward cardiovascular disease risk stratification via models/calculators such as Framingham Risk Score, ACC/AHA Atherosclerotic Cardiovascular Disease Risk Estimator, and/or Reynolds Risk Scores. These models/calculators may also take into account of family history (heart disease and/or high blood cholesterol), age, gender, Body-Mass-Index, medical history (diabetes, high cholesterol, heart disease), high sensitivity CRP levels, coronary artery calcium score, and ankle-brachial index. The cardiovascular stratification further determines what medical intervention may be necessary to decrease the risk of future cardiovascular disease.

Total cholesterol

The combined quantity of LDL and HDL. A total cholesterol of higher than 240mg/dL is abnormal, but medical intervention is determined by the breakdown of LDL and HDL levels.

LDL cholesterol

LDL,commonly known as "bad cholesterol", is associated with increased risk of cardiovascular disease. It is also associated with diabetes, hypertension, hypertriglyceridemia, and atherosclerosis. In a fasting lipid panel, a LDL greater than 160 mg/dL is abnormal.

HDL Cholesterol

HDL, also known as "good cholesterol", is associated with decreased risk of cardiovascular disease. It can be affected by acquired or genetic factors, including tobacco use, obesity, inactivity,

hypertriglyceridemia, diabetes, high carbohydrate diet, medication side effects (beta-blockers, androgenic steroids, corticosteroids, progestogens, thiazide diuretics, retinoic acid derivatives, oral estrogens, etc.) and genetic abnormalities (mutations ApoA-I, LCAT, ABC1).[20] Low level is defined as less than 40 mg/dL.

Triglycerides

Triglyceride level is an independent risk factor for cardiovascular disease and/or metabolic syndrome. Food intake prior to testing may cause elevated levels, up to 20%. Normal level is defined as less than 150 mg/dL. Borderline high is defined as 150 to 199 mg/dL. High level is between 200 to 499 mg/dL. Greater than 500mg/dL is defined as very high, and is associated with pancreatitis and requires medical treatment.

Screening age

Health organizations do not have a consensus on the age to begin screening for hyperlipidemia. USPSTF recommends men older than 35 and women older than 45 to be screened. NCE-ATP III recommends all adults older than 20 to be screened as it may lead potential lifestyle modification that can reduce risks of other diseases. However, screening should be done for those with known CHD or risk-equivalent conditions (e.g. Acute Coronary Syndrome, history of heart attacks, Stable or Unstable angina, Transient ischemic attacks, and Peripheral arterial disease of atherosclerotic origins, coronary or other arterial revascularization).

Screening frequency

Most screening guidelines recommend testing every 5 years. USPSTF recommends increased frequency for people with elevated risk of CHD, which may be determined using cardiovascular disease risk scores.

Management

Management of hyperlipidemia includes maintenance of a normal body weight, increased physical activity, and decreased consumption of refined carbohydrates and simple sugars. Prescription drugs may be used to treat some people having significant risk factors, such as cardiovascular disease, LDL cholesterol greater than 190 mg/dl or diabetes. Common medication therapy is a statin.

HMG-CoA reductase inhibitors

Competitive inhibitors of HMG-CoA reductase, such as lovastatin, atorvastatin, fluvastatin, pravastatin, simvastatin, rosuvastatin, and pitavastatin, inhibit the synthesis of mevalonate, a precursor molecule to cholesterol. This medication class is especially effective at decreasing elevated LDL cholesterol. Major side effects include elevated transaminases and myopathy.

Fibric acid derivatives

Fibric acid derivatives, such as gemfibrozil and fenofibrate, function by increasing the lipolysis in adipose tissue via activation of peroxisome proliferator-activated receptor-α. They decrease VLDL - very low density lipoprotein - and LDL in some people. Major side effects include rashes, GI upset, myopathy, or increased transaminases.

Niacin

Niacin, or vitamin B3 has a mechanism of action that is poorly understood, however it has been shown to decrease LDL cholesterol and triglycerides, and increase HDL cholesterol. The most common side effect is flushing secondary to skin vasodilation. This effect is mediated by prostaglandins and can be decreased by taking concurrent aspirin.

Bile acid binding resins

Bile acid binding resins, such as colestipol, cholestyramine, and colesevelam, function by binding bile acids, increasing their excretion. They are useful for decreasing LDL cholesterol. The most common side effects include bloating and diarrhea.

Sterol absorption inhibitors

Inhibitors of intestinal sterol absorption, such as ezetimibe, function by decreasing the absorption of cholesterol in the GI tract by targeting NPC1L1, a transport protein in the gastrointestinal wall. This results in decreased LDL cholesterol.

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Competitive questions from today topic (2 questions Minimum)-

- The secondary antibodies routinely used for the detection of primary antibodies in western blotting experiment are

1. anti-allotypic
2. anti-idiotypic
3. anti-isotypic
4. anti-paratypic

Exam NameCSIR NET DEC 2016

- Identify the plant species from which artemisinin, an anti-malarial drug, is extracted.

1. Artemisia maritima
2. Artemisia scoparia
3. Artemisia annua
4. Cinchona officinalis

Exam Name CSIR NET DEC 2017

CHAPTER 4

Communicable & non- Communicable diseases: Sign and Symptoms of Coronary Heart Diseases.

Coronary artery disease

Coronary artery disease (CAD), also known as coronary heart disease (CHD) or ischemic heart disease (IHD), involves the reduction of blood flow to the heart muscle due to build-up of plaque in the arteries of the heart. It is the most common of the cardiovascular diseases. Types include stable angina, unstable angina, myocardial infarction, and sudden cardiac death. A common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Occasionally it may feel like heartburn. Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and improve with rest. Shortness of breath may also occur and sometimes no symptoms are present. In many cases, the first sign is a heart attack. Other complications include heart failure or an abnormal heartbeat.

Risk factors include high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, depression, and excessive alcohol. A number of tests may help with diagnoses including: electrocardiogram, cardiac stress testing, coronary computed tomographic angiography, and coronary angiogram, among others.

Ways to reduce CAD risk include eating a healthy diet, regularly exercising, maintaining a healthy weight, and not smoking. Medications for diabetes, high cholesterol, or high blood pressure are sometimes used. There is limited evidence for screening people who are at low risk and do not have symptoms. Treatment involves the same measures as prevention. Additional medications such as antiplatelets (including aspirin), beta blockers, or nitroglycerin may be recommended. Procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) may be used in severe disease. In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improves life expectancy or decreases heart attack risk.

In 2015, CAD affected 110 million people and resulted in 8.9 million deaths. It makes up 15.6% of all deaths, making it the most common cause of death globally. The risk of death from CAD for a given age decreased between 1980 and 2010, especially in developed countries. The number of cases of CAD for a given age also decreased between 1990 and 2010. In the United States in 2010, about 20% of those over 65 had CAD, while it was present in 7% of those 45 to 64, and 1.3% of those 18 to 45; rates were higher among men than women of a given age.

Signs and symptoms:

Chest pain that occurs regularly with activity, after eating, or at other predictable times is termed stable angina and is associated with narrowings of the arteries of the heart.

Angina that changes in intensity, character or frequency is termed unstable. Unstable angina may precede myocardial infarction. In adults who go to the emergency department with an unclear cause of pain, about 30% have pain due to coronary artery disease.

Risk factors

Coronary artery disease has a number of well determined risk factors. These include high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, depression, family history, and excessive alcohol. About half of cases are linked to genetics. Smoking and obesity are associated with about 36% and 20% of cases, respectively. Smoking just one cigarette per day about doubles the risk of CAD. Lack of exercise has been linked to 7–12% of cases. Exposure to the herbicide Agent Orange may increase risk. Rheumatologic diseases such as rheumatoid arthritis, systemic lupus erythematosus, psoriasis, and psoriatic arthritis are independent risk factors as well.

Job stress appears to play a minor role accounting for about 3% of cases. In one study, women who were free of stress from work life saw an increase in the diameter of their blood vessels, leading to decreased progression of atherosclerosis. In contrast, women who had high levels of work-related stress experienced a decrease in the diameter of their blood vessels and significantly increased disease progression. Having a type a behavior pattern, a group of personality characteristics including time urgency, competitiveness, hostility, and impatience, is linked to an increased risk of coronary disease.
Blood fats

High blood cholesterol (specifically, serum LDL concentrations). HDL (high density lipoprotein) has a protective effect over development of coronary artery disease.
High blood triglycerides may play a role.
High levels of lipoprotein (a), a compound formed when LDL cholesterol combines with a protein known as apolipoprotein (a).

Dietary cholesterol does not appear to have a significant effect on blood cholesterol and thus recommendations about its consumption may not be needed. Saturated fat is still a concern.

Genetics
The heritability of coronary artery disease has been estimated between 40% and 60%.Genome-wide association studies have identified over 160 genetic susceptibility loci for coronary artery disease.

Other
Endometriosis in women under the age of 40.
Depression and hostility appear to be risks.
The number of categories of adverse childhood experiences (psychological, physical, or sexual abuse; violence against mother; or living with household members who were substance abusers, mentally ill, suicidal, or incarcerated) showed a graded correlation with the presence of adult diseases including coronary artery (ischemic heart) disease.

Hemostatic factors: High levels of fibrinogen and coagulation factor VII are associated with an increased risk of CAD.
Low hemoglobin.
In the Asian population, the b fibrinogen gene G-455A polymorphism was associated with the risk of CAD.
Path physiology
Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the heart's muscle cells. The heart's muscle cells may die from lack of oxygen and this is called a myocardial infarction (commonly referred to as a heart attack). It leads to damage, death, and eventual scarring of the heart muscle without regrowth of heart muscle cells. Chronic high-grade narrowing of the coronary arteries can induce transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into a dangerous heart rhythm known as ventricular fibrillation, which often leads to death.

Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery (the arteries that supply blood to the heart muscle) develops atherosclerosis. With atherosclerosis, the artery's lining becomes hardened, stiffened, and accumulates deposits of calcium, fatty lipids, and abnormal inflammatory cells – to form a plaque. Calcium phosphate (hydroxyapatite) deposits in the muscular layer of the blood vessels appear to play a significant role in stiffening the arteries and inducing the early phase of coronary arteriosclerosis. This can be seen in a so-called metastatic mechanism of calciphylaxis as it occurs in chronic kidney disease and hemodialysis (Rainer Liedtke 2008). Although these people suffer from a kidney dysfunction, almost fifty percent of them die due to coronary artery disease. Plaques can be thought of as large "pimples" that protrude into the channel of an artery, causing a partial obstruction to blood flow. People with coronary artery disease might have just one or two plaques, or might have dozens distributed throughout their coronary arteries. A more severe form is chronic total occlusion (CTO) when a coronary artery is completely obstructed for more than 3 months.

Cardiac syndrome X is chest pain (angina pectoris) and chest discomfort in people who do not show signs of blockages in the larger coronary arteries of their hearts when an angiogram (coronary angiogram) is being performed. The exact cause of cardiac syndrome X is unknown. Explanations include microvascular dysfunction or epicardial atherosclerosis. For reasons that are not well understood, women are more likely than men to have it; however, hormones and other risk factors unique to women may play a role.

Diagnosis
For symptomatic people, stress echocardiography can be used to make a diagnosis for obstructive coronary artery disease. The use of echocardiography, stress cardiac imaging, and/or advanced non-invasive imaging is not recommended on individuals who are exhibiting no symptoms and are otherwise at low risk for developing coronary disease.

The diagnosis of "Cardiac Syndrome X" – the rare coronary artery disease that is more common in women, as mentioned, is a diagnosis of exclusion. Therefore, usually the same tests are used as in any person with the suspected of having coronary artery disease:

- Baseline electrocardiography (ECG)
- Exercise ECG – Stress test
- Exercise radioisotope test (nuclear stress test, myocardial scintigraphy)
- Echocardiography (including stress echocardiography)
- Coronary angiography
- Intravascular ultrasound
- Magnetic resonance imaging (MRI)

The diagnosis of coronary disease underlying particular symptoms depends largely on the nature of the symptoms. The first investigation is an electrocardiogram (ECG/EKG), both for "stable" angina and acute coronary syndrome. An X-ray of the chest and blood tests may be performed.
Stable angina

In "stable" angina, chest pain with typical features occurring at predictable levels of exertion, various forms of cardiac stress tests may be used to induce both symptoms and detect changes by way of electrocardiography (using an ECG), echocardiography (using ultrasound of the heart) or scintigraphy (using uptake of radionuclide by the heart muscle). If part of the heart seems to receive an insufficient blood supply, coronary angiography may be used to identify stenosis of the coronary arteries and suitability for angioplasty or bypass surgery.

Stable coronary artery disease (SCAD) is also often called stable ischemic heart disease (SIHD). A 2015 monograph explains that "Regardless of the nomenclature, stable angina is the chief manifestation of SIHD or SCAD." There are U.S. and European clinical practice guidelines for SIHD/SCAD.

Prevention:
Up to 90% of cardiovascular disease may be preventable if established risk factors are avoided. Prevention involves adequate physical exercise, decreasing obesity, treating high blood pressure, eating a healthy diet, decreasing cholesterol levels, and stopping smoking. Medications and exercise are roughly equally effective. High levels of physical activity reduce the risk of coronary artery disease by about 25%.

Most guidelines recommend combining these preventive strategies. A 2015 Cochrane Review found some evidence that counselling and education in an effort to bring about behavioral change might help in high risk groups. However, there was insufficient evidence to show an effect on mortality or actual cardiovascular events.

In diabetes mellitus, there is little evidence that very tight blood sugar control improves cardiac risk although improved sugar control appears to decrease other problems such as kidney failure and blindness. The World Health Organization (WHO) recommends "low to moderate alcohol intake" to reduce risk of coronary artery disease while high intake increases the risk.

Diet:
Diet and heart disease

A diet high in fruits and vegetables decreases the risk of cardiovascular disease and death. Vegetarians have a lower risk of heart disease, possibly due to their greater consumption of fruits and vegetables. Evidence also suggests that the Mediterranean diet and a high fiber diet lower the risk.

The consumption of Trans fat (commonly found in hydrogenated products such as margarine) has been shown to cause a precursor to atherosclerosis and increase the risk of coronary artery disease.

Evidence does not support a beneficial role for omega-3 fatty acid supplementation in preventing cardiovascular disease (including myocardial infarction and sudden cardiac death). There is tentative evidence that intake of menaquinone (Vitamin K2), but not phylloquinone (Vitamin K1), may reduce the risk of CAD mortality.

Secondary prevention
Secondary prevention is preventing further sequelae of already established disease. Effective lifestyle changes include:

- Weight control
- Smoking cessation
- Avoiding the consumption of Trans fats (in partially hydrogenated oils)
- Decreasing psychosocial stress
- Exercise

Aerobic exercise, like walking, jogging, or swimming, can reduce the risk of mortality from coronary artery disease. Aerobic exercise can help decrease blood pressure and the amount of blood cholesterol (LDL) over time. It also increases HDL cholesterol which is considered "good cholesterol".

Although exercise is beneficial, it is unclear whether doctors should spend time counseling patients to exercise. The U.S. Preventive Services Task Force found "insufficient evidence" to recommend that doctors counsel patients on exercise but "it did not review the evidence for the effectiveness of physical activity to reduce chronic disease, morbidity and mortality", only the effectiveness of counseling itself. The American Heart Association, based on a non-systematic review, recommends that doctors counsel patients on exercise.

Psychological symptoms are common in people with CHD, and while many psychological treatments may be offered following cardiac events, there is no evidence that they change mortality, the risk of revascularization procedures, or the rate of non-fatal myocardial infarction.
Treatment
There are a number of treatment options for coronary artery disease:

- Lifestyle changes
- Medical treatment – drugs (e.g., cholesterol lowering medications, beta-blockers, nitroglycerin, calcium channel blockers, etc.);
- Coronary interventions as angioplasty and coronary stent;
- Coronary artery bypass grafting (CABG)

Medications:

- Statins, which reduce cholesterol, reduce the risk of coronary artery disease
- Nitroglycerin
- Calcium channel blockers and/or beta-blockers
- Antiplatelet drugs such as aspirin

It is recommended that blood pressure typically be reduced to less than 140/90 mmHg. The diastolic blood pressure however should not be lower than 60 mmHg. Beta blockers are recommended first line for this use.

Aspirin:

In those with no previous history of heart disease, aspirin decreases the risk of a myocardial infarction but does not change the overall risk of death. It is thus only recommended in adults who are at increased risk for coronary artery disease where increased risk is defined as "men older than 90 years of age, postmenopausal women, and younger persons with risk factors for coronary artery disease (for example, hypertension, diabetes, or smoking) who are at increased risk for heart disease and may wish to consider aspirin therapy". More specifically, high-risk persons are "those with a 5-year risk ≥ 3%".

Anti-platelet therapy

Clopidogrel plus aspirin (dual anti-platelet therapy) reduces cardiovascular events more than aspirin alone in those with a STEMI. In others at high risk but not having an acute event, the evidence is weak. Specifically, its use does not change the risk of death in this group. In those who have had a stent, more than 12 months of clopidogrel plus aspirin does not affect the risk of death.

Surgery

Revascularization for acute coronary syndrome has a mortality benefit. Percutaneous revascularization for stable ischaemic heart disease does not appear to have benefits over medical therapy alone. In those with disease in more than one artery, coronary artery bypass grafts appear better than percutaneous coronary interventions. Newer "anaortic" or no-touch off-pump coronary artery revascularization techniques have shown reduced postoperative stroke rates comparable to percutaneous coronary intervention. Hybrid coronary revascularization has also been shown to be a safe and feasible procedure that may offer some advantages over conventional CABG though it is more expensive.

Epidemiology

As of 2010, CAD was the leading cause of death globally resulting in over 7 million deaths. This increased from 5.2 million deaths from CAD worldwide in 1990. It may affect individuals at any age but becomes dramatically more common at progressively older ages, with approximately a tripling with each decade of life. Males are affected more often than females.

It is estimated that 60% of the world's cardiovascular disease burden will occur in the South Asian subcontinent despite only accounting for 20% of the world's population. This may be secondary to a combination of genetic predisposition and environmental factors. Organizations such as the Indian Heart Association are working with the World Heart Federation to raise awareness about this issue.

Coronary artery disease is the leading cause of death for both men and women and accounts for approximately 600,000 deaths in the United States every year. According to present trends in the United States, half of healthy 40-year-old men will develop CAD in the future, and one in three healthy 40-year-old women. It is the most common reason for death of men and women over 20 years of age in the United States.

Society and culture

Names

Other terms sometimes used for this condition are "hardening of the arteries" and "narrowing of the arteries". In Latin it is known as morbus ischaemicus cordis (MIC).

Support groups

The Infarct Combat Project (ICP) is an international nonprofit organization founded in 1998 which tries to decrease ischemic heart diseases through education and research.

Industry influence on research

In 2016 research into the archives of the Sugar Association, the trade association for the sugar industry in the US, had sponsored an influential literature review published in 1965 in the New England Journal of Medicine that downplayed early findings about the role of a diet heavy in sugar in the development of CAD and emphasized the role of fat; that review influenced decades of research funding and guidance on healthy eating.

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Competitive questions from today topic (2 questions Minimum)-

HIV binds to the following molecule

- CD4
- CD8
- MHC I
- MHC II

Exam NameCU SAT Biotech 2012

Catalytic antibodies are called

- Abzymes
- Ribozymes
- Lysozymes
- Novozymes

Exam NameCU SAT Biotech 2012

CHAPTER 5

Communicable & non- Communicable diseases: Sign and Symptoms of Hypertension and Diabetes.

Hypertension

Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the blood pressure in the arteries is persistently elevated. High blood pressure typically does not cause symptoms. Long-term high blood pressure, however, is a major risk factor for coronary artery disease, stroke, heart failure, atrial fibrillation, peripheral arterial disease, vision loss, chronic kidney disease, and dementia.

High blood pressure is classified as primary (essential) hypertension or secondary hypertension. About 90–95% of cases are primary, defined as high blood pressure due to nonspecific lifestyle and genetic factors. Lifestyle factors that increase the risk include excess salt in the diet, excess body weight, smoking, and alcohol use. The remaining 5–10% of cases are categorized as secondary high blood pressure, defined as high blood pressure due to an identifiable cause, such as chronic kidney disease, narrowing of the kidney arteries, an endocrine disorder, or the use of birth control pills.

Blood pressure is expressed by two measurements, the systolic and diastolic pressures, which are the maximum and minimum pressures, respectively. For most adults, normal blood pressure at rest is within the range of 100–130 millimeters mercury (mmHg) systolic and 60–80 mmHg diastolic. For most adults, high blood pressure is present if the resting blood pressure is persistently at or above 130/80 or 140/90 mmHg. Different numbers apply to children. Ambulatory blood pressure monitoring over a 24-hour period appears more accurate than office-based blood pressure measurement.

Lifestyle changes and medications can lower blood pressure and decrease the risk of health complications. Lifestyle changes include weight loss, physical exercise, decreased salt intake, reducing alcohol intake, and a healthy diet. If lifestyle changes are not sufficient then blood pressure medications are used. Up to three medications can control blood pressure in 90% of people. The treatment of moderately high arterial blood pressure (defined as >160/100 mmHg) with medications is associated with an improved life expectancy. The effect of treatment of blood pressure between 130/80 mmHg and 160/100 mmHg is less clear, with some reviews finding benefit and others finding unclear benefit. High blood pressure affects between 16 and 37% of the population globally. In 2010 hypertension was believed to have been a factor in 18% of all deaths (9.4 million globally).

Signs and symptoms:
Hypertension is rarely accompanied by symptoms, and its identification is usually through screening, or when seeking healthcare for an unrelated problem. Some people with high blood pressure report headaches (particularly at the back of the head and in the morning), as well as lightheadedness, vertigo, tinnitus (buzzing or hissing in the ears), altered vision or fainting episodes. These symptoms, however, might be related to associated anxiety rather than the high blood pressure itself.

On physical examination, hypertension may be associated with the presence of changes in the optic fundus seen by ophthalmoscopy. The severity of the changes typical of hypertensive retinopathy is graded from I to IV; grades I and II may be difficult to differentiate. The severity of the retinopathy correlates roughly with the duration or the severity of the hypertension.

Secondary hypertension
Hypertension with certain specific additional signs and symptoms may suggest secondary hypertension, i.e. hypertension due to an identifiable cause. For example, Cushing's syndrome frequently causes truncal obesity, glucose intolerance, moon face, a hump of fat behind the neck/shoulder (referred to as a buffalo hump), and purple abdominal stretch marks. Hyperthyroidism frequently causes weight loss with increased appetite, fast heart rate, bulging eyes, and tremor. Renal artery stenosis (RAS) may be associated with a localized abdominal bruit to the left or right of the midline (unilateral RAS), or in both locations (bilateral RAS). Coarctation of the aorta frequently causes a decreased blood pressure in the lower extremities relative to the arms, or delayed or absent femoral arterial pulses. Pheochromocytoma may cause abrupt ("paroxysmal") episodes of hypertension accompanied by headache, palpitations, pale appearance, and excessive sweating.

Hypertensive crisis
Severely elevated blood pressure (equal to or greater than a systolic 180 or diastolic of 110) is referred to as a hypertensive crisis. Hypertensive crisis is categorized as either hypertensive urgency or hypertensive emergency, according to the absence or presence of end organ damage, respectively.

In hypertensive urgency, there is no evidence of end organ damage resulting from the elevated blood pressure. In these cases, oral medications are used to lower the BP gradually over 24 to 48 hours.

In hypertensive emergency, there is evidence of direct damage to one or more organs. The most affected organs include the brain, kidney, heart and lungs, producing symptoms which may include confusion, drowsiness, chest pain and breathlessness. In hypertensive emergency, the blood pressure must be reduced more rapidly to stop ongoing organ damage; however, there is a lack of randomized controlled trial evidence for this approach.

Pregnancy

Hypertension occurs in approximately 8–10% of pregnancies. Two blood pressure measurements six hours apart of greater than 140/90 mm Hg are diagnostic of hypertension in pregnancy. High blood pressure in pregnancy can be classified as pre-existing hypertension, gestational hypertension, or pre-eclampsia.

Pre-eclampsia is a serious condition of the second half of pregnancy and following delivery characterised by increased blood pressure and the presence of protein in the urine. It occurs in about 5% of pregnancies and is responsible for approximately 16% of all maternal deaths globally. Pre-eclampsia also doubles the risk of death of the baby around the time of birth. Usually there are no symptoms in pre-eclampsia and it is detected by routine screening. When symptoms of pre-eclampsia occur the most common are headache, visual disturbance (often "flashing lights"), vomiting, and pain over the stomach, and swelling. Pre-eclampsia can occasionally progress to a life-threatening condition called eclampsia, which is a hypertensive emergency and has several serious complications including vision loss, brain swelling, seizures, kidney failure, pulmonary edema, and disseminated intravascular coagulation (a blood clotting disorder).

In contrast, gestational hypertension is defined as new-onset hypertension during pregnancy without protein in the urine.

Children

Failure to thrive, seizures, irritability, lack of energy, and difficulty in breathing can be associated with hypertension in newborns and young infants. In older infants and children, hypertension can cause headache, unexplained irritability, fatigue, failure to thrive, blurred vision, nosebleeds, and facial paralysis.

Causes:

Primary hypertension

Hypertension results from a complex interaction of genes and environmental factors. Numerous common genetic variants with small effects on blood pressure have been identified as well as some rare genetic variants with large effects on blood pressure. Also, genome-wide association studies (GWAS) have identified 35 genetic loci related to blood pressure; 12 of these genetic loci influencing blood pressure were newly found. Sentinel SNP for each new genetic locus identified has shown an association with DNA methylation at multiple nearby CpG sites. These sentinels SNP are located within genes related to vascular smooth muscle and renal function. DNA methylation might affect in some way linking common genetic variation to multiple phenotypes even though mechanisms underlying these associations are not understood. Single variant test performed in this study for the 35 sentinel SNP (known and new) showed that genetic variants singly or in aggregate contribute to risk of clinical phenotypes related to high blood pressure.

Blood pressure rises with aging and the risk of becoming hypertensive in later life is considerable. Several environmental factors influence blood pressure. High salt intake raises the blood pressure in salt sensitive individuals; lack of exercise, central obesity can play a role in individual cases.

The possible roles of other factors such as caffeine consumption, and vitamin D deficiency are less clear. Insulin resistance, which is common in obesity and is a component of syndrome X (or the metabolic syndrome), is also thought to contribute to hypertension. One review suggests that sugar may play an important role in hypertension and salt is just an innocent bystander.

Events in early life, such as low birth weight, maternal smoking, and lack of breastfeeding may be risk factors for adult essential hypertension, although the mechanisms linking these exposures to adult hypertension remain unclear. An increased rate of high blood urea has been found in untreated people with hypertension in comparison with people with normal blood pressure, although it is uncertain whether the former plays a causal role or is subsidiary to poor kidney function. Average blood pressure may be higher in the winter than in the summer. Periodontal disease is also associated with high blood pressure.

Secondary hypertension

Secondary hypertension results from an identifiable cause. Kidney disease is the most common secondary cause of hypertension. Hypertension can also be caused by endocrine conditions, such as Cushing's syndrome, hyperthyroidism, hypothyroidism, acromegaly, Conn's syndrome or hyperaldosteronism, renal artery stenosis (from atherosclerosis or fibromuscular dysplasia), hyperparathyroidism, and pheochromocytoma. Other causes of secondary hypertension include obesity, sleep apnea, pregnancy, coarctation of the aorta, excessive eating of liquorice, excessive drinking of alcohol, certain prescription medicines, herbal remedies, and stimulants such as cocaine and methamphetamine. Arsenic exposure through drinking water has been shown to correlate with elevated blood pressure. Depression was also linked to hypertension.

A 2018 review found that any alcohol increased blood pressure in males while over one or two drinks increased the risk in females.

Pathophysiology

In most people with established essential hypertension, increased resistance to blood flow (total peripheral resistance) accounts for the high pressure while cardiac output remains normal. There is evidence that some younger people with prehypertension or 'borderline hypertension' have high cardiac output, an elevated heart rate and normal peripheral resistance, termed hyperkinetic borderline hypertension. These individuals develop the typical features of established essential hypertension in later life as their cardiac output falls and peripheral resistance rises with age. Whether this pattern is typical of all people who ultimately develop hypertension is disputed. The increased peripheral resistance in established hypertension is mainly attributable to structural narrowing of small arteries and arterioles, although a reduction in the number or density of capillaries may also contribute.

It is not clear whether or not vasoconstriction of arteriolar blood vessels plays a role in hypertension. Hypertension is also associated with decreased peripheral venous compliance which may increase venous return, increase cardiac preload and, ultimately, cause diastolic dysfunction.

Pulse pressure (the difference between systolic and diastolic blood pressure) is frequently increased in older people with hypertension. This can mean that systolic pressure is abnormally high, but diastolic pressure may be normal or low, a condition termed isolated systolic hypertension. The high pulse pressure in elderly people with hypertension or isolated systolic hypertension is explained by increased arterial stiffness, which typically accompanies aging and may be exacerbated by high blood pressure.

Many mechanisms have been proposed to account for the rise in peripheral resistance in hypertension. Most evidence implicates either disturbances in the kidneys' salt and water handling (particularly abnormalities in the intrarenal renin–angiotensin system) or abnormalities of the sympathetic nervous system. These mechanisms are not mutually exclusive and it is likely that both contribute to some extent in most cases of essential hypertension. It has also been suggested that endothelial dysfunction and vascular inflammation may also contribute to increased peripheral resistance and vascular damage in hypertension. Interleukin 17 has garnered interest for its role in increasing the production of several other immune system chemical signals thought to be involved in hypertension such as tumor necrosis factor alpha, interleukin 1, interleukin 6, and interleukin 8.

Excessive sodium or insufficient potassium in the diet leads to excessive intracellular sodium, which contracts vascular smooth muscle, restricting blood flow and so increases blood pressure.

Diagnosis

Hypertension is diagnosed on the basis of a persistently high resting blood pressure. The American Heart Association recommends at least three resting measurements on at least two separate health care visits. The UK National Institute for Health and Care Excellence recommends ambulatory blood pressure monitoring to confirm the diagnosis of hypertension if a clinic blood pressure is 140/90 mmHg or higher.

Measurement technique

For an accurate diagnosis of hypertension to be made, it is essential for proper blood pressure measurement technique to be used. Improper measurement of blood pressure is common and can change the blood pressure reading by up to 10 mmHg, which can lead to misdiagnosis and misclassification of hypertension. Correct blood pressure measurement technique involves several steps. Proper blood pressure measurement requires the person whose blood pressure is being measured to sit quietly for at least five minutes which is then followed by application of a properly fitted blood pressure cuff to a bare upper arm. The person should be seated with their back supported, feet flat on the floor, and with their legs uncrossed. The person whose blood pressure is being measured should avoid talking or moving during this process. The arm being measured should be supported on a flat surface at the level of the heart. Blood pressure measurement should be done in a quiet room so the medical professional checking the blood pressure can hear the Korotkoff sounds while listening to the brachial artery with a stethoscope for accurate blood pressure measurements. The blood pressure cuff should be deflated slowly (2-3 mmHg per second) while listening for the Korotkoff sounds. The bladder should be emptied before a person's blood pressure is measured since this can increase blood pressure by up to 15/10 mmHg. Multiple blood pressure readings (at least two) spaced 1–2 minutes apart should be obtained to ensure accuracy.

Ambulatory blood pressure monitoring over 12 to 24 hours is the most accurate method to confirm the diagnosis. An exception to this is those with very high blood pressure readings especially when there is poor organ function.

With the availability of 24-hour ambulatory blood pressure monitors and home blood pressure machines, the importance of not wrongly diagnosing those who have white coat hypertension has led to a change in protocols. In the United Kingdom, current best practice is to follow up a single raised clinic reading with ambulatory measurement, or less ideally with home blood pressure monitoring over the course of 7 days. The United States Preventive Services Task Force also recommends getting measurements outside of the healthcare environment. Pseudohypertension in the elderly or noncompressibility artery syndrome may also require consideration. This condition is believed to be due to calcification of the arteries resulting in abnormally high blood pressure readings with a blood pressure cuff while intra arterial measurements of blood pressure are normal. Orthostatic hypertension is when blood pressure increases upon standing.

Prevention

Much of the disease burden of high blood pressure is experienced by people who are not labeled as hypertensive. Consequently, population strategies are required to reduce the consequences of high blood pressure and reduce the need for antihypertensive medications. Lifestyle changes are recommended to lower blood pressure, before starting medications. The 2004 British Hypertension Society guidelines proposed lifestyle changes consistent with those outlined by the US National High BP Education Program in 2002 for the primary prevention of hypertension:

- maintain normal body weight for adults (e.g. body mass index 20–25 kg/m2)
- reduce dietary sodium intake to <100 mmol/ day (<6 g of sodium chloride or <2.4 g of sodium per day)
- engage in regular aerobic physical activity such as brisk walking (≥30 min per day, most days of the week)
- limit alcohol consumption to no more than 3 units/day in men and no more than 2 units/day in women
- consume a diet rich in fruit and vegetables (e.g. at least five portions per day);

Effective lifestyle modification may lower blood pressure as much as an individual antihypertensive medication. Combinations of two or more lifestyle modifications can achieve even better results. There is considerable evidence that reducing dietary salt intake lowers blood pressure, but whether this translates into a reduction in mortality and cardiovascular disease remains uncertain. Estimated sodium intake ≥6g/day and <3g/day are both associated with high risk of death or major cardiovascular disease, but the association between high sodium intake and adverse outcomes is only observed in people with hypertension. Consequently, in the absence of results from randomized controlled trials, the wisdom of reducing levels of dietary salt intake below 3g/day has been questioned. ESC guidelines mention periodontitis is associated with poor cardiovascular health status.

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- **Competitive questions from today topic (2 questions Minimum)-**

RNA interference is mediated by both siRNA and miRNA. Which one of the following statement about them is NOT true?

- Both siRNA and miRNA are processed by DICER.
- Both siRNA and miRNA usually guide silencing of the same genetic loci from which they originate.
- miRNA is a natural molecule while siRNA is either natural or a synthetic one.
- miRNA, but not siRNA is processed by Drosha

Exam Name CSIR NET JUNE 2016

Following are some of the characteristics of MHC class I and class II molecules except one which is applicable only for MHC class I. Identify the appropriate statement.

- They are expressed constitutively an all nucleated cells.
- They are glycosylated polypeptides with domain structure.
- They are involved in presentation of antigen fragments to cells.
- They are expressed on surface membrane of B cells.

Exam Name CU SAT Biotech 2012

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