Name Fawad alam
ID 14625
paper chemical pathology
intructor name sir adnan ahmad
program Bs Mlt 4rth sem.
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Section A

1. Two students of MLT are talking about the use of cholesterol in food one says its good to have cholesterols in food the other said use of more cholesterol is not good for our health, they both asked from his class teacher and he replied "having high triglyceride levels in your blood can make you more likely to have _____
   A. Arthritis
   B. Feet infections
   C. Heart disease
   D. Beri Beri
2. Focal segmental glomerulosclerosis is a disease that scars the_____
   A. Bowman's capsule
   B. Glomeruli
   C. Pancreases
   D. Liver
3. Minimal change disease **"MCD"** is the most common cause of nephrotic syndrome in_____
   A. Women
   B. Men
   C. Children
   D. Aged
4. The main signs or symptoms make up nephrotic syndrome is/are_____
   A. Proteinuria
   B. Hyperlipidemia
   C. Hypoalbuminemia
   D. All of the above
5. To diagnose a patient nephrotic syndrome, you should go for?
   A. Urine Test
   B. Blood Test
   C. Biopsy
   D. All of the above
6. Your class teacher gives you a history of patient such that a patient having yellow skin and body fluid that is the by-product of RBCs breakdown, Red blood cells typically survive for about 120 days before the body breaks them down, an increased breakdown of RBCs made the skin and body fluids colour yellow, this is due to_____
   A. Bilirubin
   B. Cytokines
   C. Alpha blockers
   D. Side effects of NSAIDs
7. New-borns with jaundice are carefully monitored and generally improve within _____hours.
   A. 04 to 07
   B. 10 to 12
   C. 12 to 24
   D. 48 to 72
8. All are true regarding Bilirubin Test Except?
   A. Is used to detect an increased level in the blood
   B. Determine the cause of jaundice
   C. Cannot diagnose blockage of the bile ducts.
   D. Help diagnose conditions
9. A patient of malabsorption syndrome is admitted in LRH ward and you have to test the condition, of all the possible diagnostics test the most reliable test of malabsorption is_____
   A. Stool test
   B. Blood test
   C. Berth test
   D. Imaging
10. The Urine Albumin to Creatinine Ratio (UACR) is a test that estimates how much albumin is excreted in a _____period without requiring patients to collect urine for a whole day.
    A. 12-hour
    B. 24-hour
    C.
    D. 48-hour

E. 72-hour

11. Which option are not **true** about kidney functions?
    A. Filter waste materials and toxin from the blood
    B. Production of vitamin E
    C. Red Blood Cells (Erythropoietin) formation
    D. Synthesize hormones that regulate blood pressure

12. Structural and Functional unit of the kidney is?
    A. Renal corpuscle
    B. Renal tubule
    C. Nephron
    D. All of the above

13. Normally: The pH of urine
    A. acidic
    B. alkaline
    C. varies from acidic to alkaline
    D. varies from alkaline to acidic

14. Uric acid is the end product of ……. catabolism.
    A. Thymine
    B. Pyramidin
    C. Purine
    D. Urea

15. …….is known as the good cholesterol.
    A. HDL
    B. LDL
    C. VLLDL
    D. triglycerides

Section B

Q1 What do you know about proteinuria?

Proteinuria (protein and urine) means the presence of an excess of serum proteins in the urine. The excess protein in the urine often causes the urine to become foamy, although foamy urine may also be caused by bilirubin in the urine (bilirubinuria), retrograde ejaculation, pneumaturia (air bubbles in the urine) due to a fistula, or drugs such as pyridium.

Associated Conditions :

Proteinuria may be a sign of renal (kidney) damage. Since serum proteins are readily reabsorbed from urine, the presence of excess protein indicates either an insufficiency of absorption or impaired filtration. Diabetics may suffer from damaged nephrons and develop proteinuria. The most common cause of proteinuria is diabetes.

Proteinuria may be a feature of the following conditions:

Nephrotic syndromes (i.e. intrinsic renal failure)

Toxic lesions of kidneys

Collagen vascular diseases (e.g. systemic lupus erythematosus)

Dehydration

Glomerular diseases, such as membranous glomerulonephritis, focal segmental glomerulonephritis, minimal change disease (lipoid nephrosis)

Strenuous exercise

Stress

Benign orthostatic (postural) proteinuria

Focal segmental glomerulosclerosis (FSGS)

IgA nephropathy (i.e. Berger's disease)

IgM nephropathy

Membranoproliferative glomerulonephritis

Membranous nephropathy

Minimal change disease

Sarcoidosis

Diabetes mellitus (diabetic nephropathy)

Drugs (e.g. NSAIDs, nicotine, penicillamine, lithium carbonate, gold and other heavy metals, ACE inhibitors, antibiotics, or opiates (especially heroin)

Infections (e.g. HIV, syphilis, hepatitis, poststreptococcal infection, urinary schistosomiasis)

Aminoaciduria

Hypertensive nephrosclerosis

Sickle cell disease

Multiple myeloma

Organ rejection: Kidney transplant patients may have gamma-globulins in their urine if the kidneys start to reject.

Ebola hemorrhagic fever

Nail patella syndrome

Familial Mediterranean fever

HELLP Syndrome

Systemic lupus erythematosus

Rheumatoid arthritis etc.

Causes :

There are three main mechanisms to cause proteinuria:

1.Due to disease in glomerulus

2. Because of increased quantity of proteins in serum (overflow proteinuria)

3.Due to low reabsorption at proximal tubule (Fanconi syndrome)

Measurement:

Conventionally, proteinuria is diagnosed by a simple dipstick test.

Traditionally, dipstick protein tests would be quantified by measuring the total quantity of protein in a 24-hour urine sample.

There is a basal level of proteinuria that can occur below 30 mg/day which is considered non-pathology.

Values between 30–300 mg/day are termed microalbuminuria which is considered pathologic.

Urine protein lab values for microalbumin of >30 mg/day correspond to a detection level within the 'trace' to '1+' range of a urine dipstick protein assay.

Treatment :

1. Treating proteinuria mainly needs proper diagnosis of the cause. The most common cause is diabetic nephropathy; so in this case, proper glycemic control may slow the progression.

2. Medical management consists of angiotensin converting enzyme (ACE) inhibitors, which are typically first-line therapy for proteinuria.

3. In patients whose proteinuria is not controlled with ACE inhibitors, the addition of an aldosterone antagonist (i.e., spironolactone) or angiotensin receptor blocker (ARB) may further reduce protein loss.

4. Caution must be used if these agents are added to ACE inhibitor therapy due to the risk of hyperkalemia.

5. Proteinuria secondary to autoimmune disease should be treated with steroids or steroid-sparing agent plus the use of ACE inhibitors.

Q2 explain Ketonuria and phenylketonuria in detail.

Ketonuria:

Ketonuria is a medical condition in which ketone bodies are present in the urine.

It is seen in conditions in which the body produces excess ketones as an indication that it is using an alternative source of energy. It is seen during starvation or more commonly in type I diabetes mellitus. Production of ketone bodies is a normal response to a shortage of glucose, meant to provide an alternate source of fuel from fatty acids.

Causes of Ketonuria:

Metabolic abnormalities such as diabetes, renal glycosuria, or glycogen storage disease.

Dietary conditions such as starvation, fasting, high protein, or low carbohydrate diets, prolonged vomiting, and anorexia

Conditions in which metabolism is increased, such as hyperthyroidism, fever, pregnancy or lactation

In non-diabetic persons, ketonuria may occur during acute illness or severe stress. Approximately 15% of hospitalized patients may have ketonuria, even though they do not have diabetes. In a diabetic patient, ketone bodies in the urine suggest that the patient is not adequately controlled and that adjustments of medication, diet, or both should be made promptly. In the non diabetic patient, ketonuria reflects a reduced carbohydrate metabolism and an increased fat metabolism.

Pathophysiology of Ketonuria :

Ketones are metabolic end-products of fatty acid metabolism. In healthy individuals, ketones are formed in the liver and are completely metabolized so that only negligible amounts appear in the urine. However, when carbohydrates are unavailable or unable to be used as an energy source, fat becomes the predominant body fuel instead of carbohydrates and excessive amounts of ketones are formed as a metabolic byproduct. Higher levels of ketones in the urine indicate that the body is using fat as the major source of energy.

Ketone bodies that commonly appear in the urine when fats are burned for energy are acetoacetate and beta-hydroxybutyric acid. Acetone is also produced and is expired by the lungs. Normally, the urine should not contain a noticeable

concentration of ketones to give a positive reading. As with tests for glucose, acetone can be tested by a dipstick or by a lab. The results are reported as small, moderate, or large amounts of acetone. A small amount of acetone is a value under 20 mg/dl; a moderate amount is a value of 30–40 mg/dl, and a finding of 80 mg/dl or greater is reported as a large amount.

Screening for Ketonuria :

Screening for ketonuria is done frequently for acutely ill patients, presurgical patients, and pregnant women. Any diabetic patient who has elevated levels of blood and urine glucose should be tested for urinary ketones. In addition, when diabetic treatment is being switched from insulin to oral hypoglycemic agents, the patient's urine should be monitored for ketonuria. The development of ketonuria within 24 hours after insulin withdrawal usually indicates a poor response to the oral hypoglycemic agents. Diabetic patients should have their urine tested regularly for glucose and ketones, particularly when acute infection or other illness develops.

In conditions associated with acidosis, urinary ketones are tested to assess the severity of acidosis and to monitor treatment response. Urine ketones appear before there is any significant increase in blood ketones; therefore, urine ketone measurement is especially helpful in emergency situations. During pregnancy, early detection of ketonuria is essential because ketoacidosis is a factor associated with intrauterine death.

Test Equipments :

A wide variety of companies manufacture ketone screening strips. A strip consists of a thin piece of plastic film slightly larger than a matchstick, with a reagent pad on one end that is either dipped into a urine sample or passed through the stream while the user is voiding. The pad is allowed to react for an exact, specified amount of time (it is recommended to use a stopwatch to time this exactly and disregard any resultant colour change after the specified time); its resulting colour is then compared to a graded shade chart indicating a detection range from negative presence of ketones up to a significant quantity. It is worth noting that in severe diabetic ketoacidosis, the acidosis causes acetoacetate to accept an H+ and become betahydroxybutyrate. Since the nitroprusside reaction on dipstix detects acetoacetate but NOT betahydroxybutyrate, this can be deceptive in very acidotic patients.

Phenylketonuria :

The inherited inability to metabolize (process) the essential amino acid phenylalanine due to complete or near-complete deficiency of the enzyme phenylalanine hydroxylase. It is a Genetic disease.

Phenylketonuria is inherited in an autosomal recessive manner, as are lesser degrees of phenylalanine hydroxylase deficiency. Phenylketonuria is abbreviated and commonly referred to as PKU

Newborns are screened for phenylketonuria (PKU) by a blood test, usually with the Guthrie card bloodspot obtained from a heelprick.

Maternal Phenylketonuria :

A mother with the genetic disease phenylketonuria (PKU) whose high blood levels of phenylalanine (phe) are dangerous to a developing fetus. High phe is a teratogen. It can damage a baby before birth.

Women with PKU who are off the special PKU diet should restart back it, ideally prior to conception. During pregnancy they must be on the diet and have their blood phe levels carefully monitored. If the maternal PKU is not controlled, the baby (who does not have PKU) is at risk for congenital heart disease, growth retardation, microcephaly (abnormally small head), and mental retardation.

Treatment :

Treatment is with a special diet low in phenylalanine. The goal is to normalize the levels of phenylalanine and tyrosine in the blood to prevent brain damage. Failure of treatment results in profound irreversible mental retardation, microcephaly (an abnormally small head), epilepsy, and behavior problems. It is clear that if the diet is not followed closely, especially during childhood, some impairment is inevitable. Maternal phenylketonuria requires a diet low in phenylalanine.

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Q3 Enlist all the possible cause of Cushing syndrome:

There are several possible causes of Cushing's syndrome.

Exogenous causes:

The most common cause of Cushing's syndrome is exogenous administration of glucocorticoids prescribed by a health care practitioner to treat other diseases (called iatrogenic Cushing's syndrome).

This can be an effect of corticosteroid treatment of a variety of disorders such as asthma and rheumatoid arthritis, or in immunosuppression after an organ transplant.

Administration of synthetic ACTH is also possible, but ACTH is less often prescribed due to cost and lesser utility. Although rare, Cushing's syndrome can also be due to the use of medroxyprogesterone acetate.

In this form of Cushing's, the adrenal glands atrophy due to lack of stimulation by ACTH, since glucocorticoids downregulate production of ACTH.

Cushing syndrome in childhood usually results from use of glucocorticoid medication.

Endogenous Cushing's syndrome :

It results from some derangement of the body's own system of secreting cortisol. Normally, ACTH is released from the pituitary gland when necessary to stimulate the release of cortisol from the adrenal glands.

In pituitary Cushing's, a benign pituitary adenoma secretes ACTH. This is also known as Cushing's disease and is responsible for 70% of endogenous Cushing's syndrome.

In adrenal Cushing's, excess cortisol is produced by adrenal gland tumors, hyperplastic adrenal glands, or adrenal glands with nodular adrenal hyperplasia.

Tumors outside the normal pituitary-adrenal system can produce ACTH (occasionally with CRH) that affects the adrenal glands. This etiology is called ectopic or paraneoplastic Cushing's disease and is seen in diseases like small cell lung cancer.

Finally, rare cases of CRH-secreting tumors (without ACTH secretion) have been reported, which stimulates pituitary ACTH production.

Pseudo-Cushing's syndrome :

Elevated levels of total cortisol can also be due to estrogen found in oral contraceptive pills that contain a mixture of estrogen and progesterone, leading to Pseudo-Cushing's syndrome.

Estrogen can cause an increase of cortisol-binding globulin and thereby cause the total cortisol level to be elevated. However, the total free cortisol, which is the active hormone in the body, as measured by a 24 hour urine collection for urinary free cortisol, is normal.