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MEDICAL FACULTY

ONCOLOGY DEPARTMENT

ANAESTHESIOLOGY AND INTENSIVE CARE

Topic 1: ACID-BASE BALANCE

Topic 2: ACUTE RENAL AND HEPATIC INSUFFICIENCY

Methodical instructions for 5TH year medical student practical training

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Content

DIAGNOSTICS AND CORRECTION OF WATER-ELECTROLYTE AND ACID-BASE DISORDERS

Topic actuality.

The most different pathological conditions of patients regardless of their reason are always accompanied by severe disorders of homoeostasis. As a first line the water-electrolyte exchange and acid-base state disturbances are presents. They are inherent to the diseases of respiratory, central nervous, and secretory systems, tor the acute surgical and infectious pathology of abdomen organs, to the traumas and shocks. They also can occur as a result of the diseases, which are accompanied by the liquid losses - fever, vomiting, diarrhea, excessive sweating, or by excessive accumulation of water, including iatrogenic stimulus. Such disorders are compensated by an organism, but then can cause decompensation of organism all systems function and cell metabolism.

For the critical care patients, as a rule, compensation of water-electrolyte and acid-basic disturbances broken or ineffective. It is occurred because of continuous pathological losses, damages of water and food enteral intake and assimilate mechanisms. In such situations the parenteral correction of homoeostasis is extremely important. Therefore, a doctor using clinical and laboratory findings musts in good time diagnose and interpret the water-electrolyte and acid-base disorders. They should to know about pharmacology of fluid resuscitation agents and be able to apply them in complex intensive care.

Teaching aims.

General purpose: be able to diagnose of water-electrolyte and acid-base disorders, give the first aid and plan the tactic of patient's management.

Concrete aims	**Initial level of knowledge-abilities**
Be able:	
1. To define the main pathological states in which the water-electrolyte and acid-base disorders developed.	1. To define, which diseases can be complicated by the development of water-electrolyte and acid-base disorders. (departments of surgery, internal medicine,

	endocrinology, and infectious diseases).
2. To take the medical history, perform physical and laboratory examination for patients with water-electrolyte and acid-base disorders, to interpret the obtained results.	2. To interpret complaints, anamnesis, physical and laboratory findings for patients with disturbances of hemodynamics, breathings, digestions, and excretions (department of internal medicine propaedeutic, general surgery), to interpret the clinical and biochemical indexes of homoeostasis, obtained from laboratory examinations (department of biochemistry), changes of BP, CVP, cardiac output in patients with different pathological states (department of pathophysiology).
3. To define the preliminary diagnosis of water-electrolyte and acid-base disorders type.	3. To interpret physical and chemical patterns of isoelectric and iso-osmolarity (Medical chemistry department).

ACID-BASE BALANCE

1. Understanding acid-base balance. What's normal?

A normal range for arterial pH is 7.35 to 7.45. Acidosis is a pH less than 7.35; alkalosis is a pH greater than 7.45. Because pH is measured in terms of hydrogen (H^+) ion concentration, an increase in

H^+ ion concentration decreases pH and vice versa. Changes in H^+ ion concentration can be stabilized through several buffering systems: bicarbonate-carbonic acid, proteins, haemoglobin, and phosphates.

Acidosis, therefore, can be described as a physiologic condition caused by the body's inability to buffer excess H^+ ions. At the other end, alkalosis results from a deficiency in H^+ ion concentration. Acidaemia and alkalemia refer to the process of acidosis or alkalosis, respectively, occurring in arterial blood.

Body acids are formed as end products of cellular metabolism. Under normal physiologic conditions, a person generates 50 to 100 mEq/day of acid from metabolism of carbohydrates, proteins, and fats. In addition, the body loses base in the stool. In order to maintain acid-base homeostasis, acid production must balance the neutralization or excretion. The lungs and kidneys are the main regulators of acid-base homeostasis. The lungs release CO_2, an end product of carbonic acid (H_2CO_3). The renal tubules, with the regulation of bicarbonate (HCO_3^-), excrete other acids produced from the metabolism of proteins, carbohydrates, and fats.

2. Compensating for changes.

The body has three compensatory mechanisms to handle changes in serum pH:

* **Physiologic buffers,** consisting of a weak acid (which can easily be broken down) and its base salt or of a weak base and its acid salt. These buffers are the bicarbonate-carbonic acid buffering system, intracellular protein buffers, and phosphate buffers in the bone.

* **Pulmonary compensation,** in which changes in ventilation work to change the partial pressure of arterial carbon dioxide ($PaCO_2$) and drive the pH toward the normal range. A drop in pH, for example, results in increased ventilation to blow off excess CO_2. An increase in pH decreases ventilatory effort, which increases $PaCO_2$ and lowers the pH back toward normal.

* **Renal compensation,** which kicks in when the other mechanisms have been ineffective, generally after about 6 hours of sustained acidosis or alkalosis. While respiratory compensation occurs almost immediately, renal mechanisms can take hours to days to make a difference. In acidosis the kidneys excrete H^+ in urine and retain HCO_3^-. In alkalosis, the kidneys excrete bicarbonate and retain H^+ in the form of organic acids, resulting in near-normalization of pH. Lastly, bone may also serve as a buffer because it contains a large reservoir of bicarbonate and phosphate and can buffer a significant acute acid load. Patients who have low albumin levels and bone density due to malnutrition or chronic disease, and anaemic patients, have an ineffective buffering capability.

3. Common acid-base upsets.

Generally, if your patient has changes in acid-base homeostasis, you'd look for the cause first before intervening to normalize the pH. But because some acid-base disturbances have a limited number of causes, you can systematically eliminate some potential causes.

Start by looking at the patient's arterial blood gas analysis. Many disorders are mild and don't require treatment, and in some cases, too-hasty treatment can do more harm than the imbalance itself. Also, critically ill patients may have more than one acid-base imbalance simultaneously.

The most common acid-base derangements can be divided into four categories: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. Let's look at each and how you'd respond.

4. Metabolic acidosis.

Metabolic acidosis is an increase in the amount of absolute body acid, either from excess production of acids or excessive loss of bicarbonate, sodium, and potassium. Causes of metabolic acidosis include lactic acidosis, diabetic ketoacidosis, and loss of bicarbonate through severe diarrhoea or bicarbonate wasting through the kidneys or gastrointestinal tract.

In general, the kidneys attempt to preserve sodium by exchanging it for excreted H^+ or potassium. In the presence of an H^+ load, H^+ ions move from the extracellular fluid into the intracellular fluid. For this process to occur, potassium moves outside the cell into the extracellular fluid to maintain electroneutrality. In severe acidosis, significant overall depletion of total body potassium stores can occur despite serum hyperkalaemia. This is why I.V. potassium is given to patients with diabetic ketoacidosis early in treatment, despite the often-elevated serum potassium level. External and internal potassium balances are regulated to maintain an extracellular fluid concentration of 3.5 to 5.5 mEq/L and a total body content of about 50 mEq/kg (40 mEq/kg in females).

5. Metabolic alkalosis.

Metabolic alkalosis occurs when HCO_3^- is increased, usually as the result of excessive loss of metabolic acids. Causes of metabolic alkalosis include diuretics, secretory adenoma of the colon, emesis, hyperaldosteronism, Cushing's syndrome, and exogenous steroids.

Some causes of metabolic alkalosis respond to treatment with 0.9% sodium chloride solution. If the patient's urine chloride concentration is less than 15 mmol/L, his metabolic alkalosis is saline-responsive; urine chloride levels above 25 mmol/L indicate nonsaline-responsive metabolic alkalosis. The mechanisms resulting in saline-responsive metabolic alkalosis include GI loss, diuresis, or renal compensation from hypercapnia. Non-saline responsive metabolic alkalosis results from mineralocorticoid excess or potassium depletion.

Fluid administration is the foundation for treatment for saline-responsive metabolic alkalosis. In cases of extreme alkalosis, the patient may be given dilute hydrochloric acid. Saline-resistant alkalosis is treated by addressing the underlying aetiology.

6. Respiratory acidosis.

In respiratory acidosis, the patient's pH is less than 7.35 and his $PaCO_2$ is above 45 mm Hg (the upper limit of normal). Alveolar hypoventilation is the only mechanism that causes hypercarbia, or a

$PaCO_2$ above the upper limit of normal. The amount of alveolar ventilation necessary to maintain normal $PaCO_2$ varies depending upon CO_2 produced.

The relationship between $PaCO_2$ and plasma HCO_3^- determines arterial pH. Generally, acute increases in $PaCO_2$ are accompanied by only minimal changes in serum HCO_3^-. However, over a period of 1 to 3 days, renal conservation of HCO_3^- results in an increase in pH.

Chronic respiratory acidosis occurs secondary to a chronic reduction in alveolar ventilation—for example, in chronic lung diseases such as chronic obstructive pulmonary disease (COPD). Acute respiratory acidosis is caused by an acute change in alveolar ventilation; respiratory depression from acute opioid ingestion is one cause. Treatment for respiratory acidosis is largely supportive, but if opioid ingestion is suspected, I.V. naloxone may be given as an antidote.

7. Respiratory alkalosis.

Common in critical care, respiratory alkalosis occurs when $PaCO_2$ is reduced, causing an increase in ph. The most common cause of respiratory alkalosis is increased alveolar ventilation, which can happen in hyperventilation, mechanical overventilation, hepatic disease, pregnancy, and septicaemia.

Determining appropriate compensatory changes in HCO_3^- is key to determining if the patient also has a concomitant metabolic disorder. In chronic respiratory alkalosis, the compensatory mechanisms result in mild reduction in plasma HCO_3^- levels to maintain a near normal or normal pH. This causes a mixed acid-base disorder, which will be discussed later.

Treatment of respiratory alkalosis is directed at discovering and correcting the underlying aetiology. For example, if a patient is hyperventilating from anxiety, have him breathe into a paper bag. In mechanically ventilated patients with mechanical overventilation, reducing the minute ventilation or tidal volume will increase $PaCO_2$ and reduce pH. Monitor the patient closely, because a rapid reduction of $PaCO_2$ in a patient with chronic respiratory alkalosis may cause acute metabolic acidosis.

8. Mixed acid-base imbalances.

When a patient has two or three acid-base imbalances simultaneously, he's said to have a mixed acid-base imbalance. Examples include:

* respiratory alkalosis or acidosis that shrouds a metabolic acidosis or alkalosis
* metabolic alkalosis or acidosis that shrouds another metabolic alkalosis acidosis.

Combined respiratory and metabolic imbalances may occur when the respiratory system compensates inappropriately for metabolic imbalances. Look at the difference between the patient's observed $PaCO_2$ and the calculated changes in $PaCO_2$, or the observed or expected change in HCO_3^-. If the observed $PaCO_2$ is higher than the calculated $PaCO_2$, the patient has respiratory acidosis with a mixed metabolic disturbance. If the observed $PaCO_2$ is lower than the calculated $PaCO_2$, the patient has respiratory alkalosis mixed with a metabolic imbalance. Generally, the $PaCO_2$ should be similar to the

two last digits of the patient's pH. For example, if the patient's pH is 7.25, you'd expect his $PaCO_2$ to be about 25 mm Hg.

Mixed metabolic acidosis and alkalosis can be identified by calculating the anion gap. The anion gap is an approximate measure of the additional amount of acid in the body; the HCO_3^- should decrease by about an amount equalling the increase in the anion gap. If the HCO_3^- is higher than the calculated increase of the anion gap, a chief metabolic alkalosis is mixed with the metabolic acidosis. Conversely, if the HCO_3^- is lower than the increase of the anion gap, then a non-anion gap metabolic acidosis is considered to be present and is worsening the anion gap acidosis.

Table 1: Clinical Approach to Acid-Base Disorders

Following is one method that has worked well for the authors. You will need results of a blood gas and serum sodium, chloride, and bicarbonate to apply this method. This method requires methodical interpretation of laboratory results and correlation with clinical findings	
1. Look at the pH. If it is decreased below 7.35, the primary (or predominant) disturbance is acidemia; go to step 2. If the pH is increased above 7.45, the predominant disturbance is alkalemia; go to step 5. If the pH is in the normal range, but the [HCO3 –] or thePco2 is abnormal, consider a mixed acid-base disorder	5. Alkalemia. If the pH is>7.45, the alkalemia type can be determined by examining the [HCO3 –] andPco2. If the [HCO3 –] is increased, go to step 6 first; if thePco2 is decreased, go to step 7
2. Acidemia. If the pH indicates acidemia, the acidemia type can be ascertained by examining the [HCO3 –] and Pco2. If the [HCO3 –] decreased, go first to step 3; if the Pco2 is elevated, but the [HCO3 –] is normal, go to step 4.	6. Metabolic Alkalosis. If [HCO3 –] is elevated, there is a primary metabolic alkalosis. a. There is an expected ventilatory response, although it is quite varied. b. The ratio of the change upward in Pco2 (mm Hg) to the change upward in [HCO3 –] (mEq/L), each from the institutional norms, can be examined. If the ratio is much less than 0.7, there is also a respiratory alkalosis (in addition to the metabolic alkalosis). If the ratio is close to 0.7, this is likely to be a compensatory ventilatory response. If theratiois well

	above0.7, respiratory acidosis is concomitantly present.
3. Metabolic Acidosis a. If the [HCO3 –] is lower than the laboratory normal range (implying a primary metabolic acidosis), then the anion gap (AG) should be examined and, if possible, compared with a known steady-state value. b. Calculate the AG: AG = [Na+] – ([HCO3 –] + [Cl–]) • If the AG is increased compared with the known steady-state value or is >12(or above institutional threshold), then by definition, a wide AG metabolic acidosis is present, and the absolute change in the AG should be compared with the absolute change in the[HCO3 –] from normal to detect other disturbances. • If the AG is unchanged (or in the normal range), then the disturbance is non widened or normal AG metabolic acidosis, typically associated with hyperchloremia. • If the change in the AG (increase)is equal to the change in the [HCO3 –] (decrease), then the wide AG acidosis is termed pure. If the AG has risen more than the [HCO3 –] has decreased, then there is also likely to be a concomitant metabolic alkalosis. If the change in the AG is less than the change in the [HCO3 –], then a normal AG acidosis is also present. (This is a difficult concept, but two separate physiologic mechanisms resulting in increased[H+] can occur simultaneously.) c. Next examine whether the ventilatory response is as expected.	7. Respiratory Alkalosis. If thePco2 is lower than normal, there is a primary respiratory alkalosis. a. The ratio of the change in [H+] to the change in Pco2 should be examined. Subtract the institution's upper limit of normal pH from the measured pH on the blood gas; use the number of hundredths (to the right of the decimal point). Divide this number by the decrease in Pco2 in mm Hg. • Acute respiratory alkalosis has a ratio of about 0.75. If the ratio is well above 0.75, there is probably also a concomitant metabolic alkalosis to explain the greater than expected decline in [H+]. If the ratio is smaller, the condition is chronic or there may also be a metabolic acidosis component.

• The expected respiratory compensation is1:1, thePco2 decreases by1mmHg for every1 mEq/L decrease in [HCO3 –]. (1) If the measured Pco2 from the blood gas equals the expected value based on the calculated Pco2 (which is determined by the decrease in the [HCO3 –]), there is appropriate respiratory compensation. Note that the pH will not return to normal. (2) If the measured Pco2 from the blood gas is higher than the expected value based on the calculated Pco2 (which is determined by the decrease in the [HCO3 –]), there is a concomitant respiratory acidosis. With higher than expectedPco2, think respiratory acidosis. (3) If the measured Pco2 from the blood gas is lower than the calculated Pco2 (which is determined by the decrease in the [HCO3 –]), there is also a concomitant primary respiratory alkalosis.	
4. Respiratory Acidosis a. If thePco2 is elevated from normal (rather than the [HCO3 –] being decreased), the primary disturbance is respiratory acidosis. b. The next step is to determine if the respiratory acidosis is acute or chronic by examining the ratio of the downward change in pH (from normal) to the upward change in Pco2 (from normal). By determining the drop in pH, you are examining the rise in [H+]. Subtract the measured pH on the blood gas from the institution's lower limit of normal; use the number of hundredths (to the right of the	8. Mixed Acid-Base Disorder. Every arterial blood gas that shows no or minimal pH derangement should still call for examination of the Pco2, [HCO3 –], and AG, because there may well be a mixed acid-base disturbance. It is quite possible for the pH, [HCO3 –], andPco2 to be normal and yet have significant acid-base disturbances. The only evident abnormality may be the AG. Take the example of an [Na+] of 145, [Cl–] of 97, [K+] of4.5, and [HCO3 –] of 25 and a normal arterial blood gas. All the numbers look reasonably normal. However, the AG is 23, so

decimal point). Divide this number by the elevation in Pco2 in mm Hg (above normal range). • If the ratio is 0.8, it is considered acute. • If the ratio is 0.33, it is considered chronic. • If the ratio is between 0.8and0.33, it is probably an acute exacerbation of the chronic condition. An alternate explanation is that a metabolic acidosis is also present as evidenced by a decreased [HCO3 –]. • If the ratio is >0.8, there must be a metabolic acidosis as an explanation for the excess [H+]. • If the ratio is <0.33, a metabolic alkalosis must also be present	by definition, there must be a wide AG metabolic acidosis. The explanation for the normal numbers is a concomitant metabolic alkalosis.

Teaching content in accordance with aims.

Theoretical questions.

1.A role of water and electrolytes in an organism.

2.Concepts about osmolarity, its correction.

3.Clinical signs of dehydration and overhydration.

4.Hypertonic dehydration. Causes, clinical picture, methods of correction.

5.Isotonic dehydration. Causes, clinical picture, methods of correction.

6.Hypotonic dehydration. Causes, clinical picture, methods of correction.

7.Hypertonic overhydration. Causes, clinical picture, methods of correction.

8.Isotonic overhydration. Causes, clinical picture, methods of correction.

9.Hypotonic overhydration. Causes, clinical picture, methods of correction.

10.Causes and signs of hypo- and hypernatremia, methods of treatment.

11.Causes and signs of hypo- and hyperkaliemia, methods of correction.

12.Disorders of chlorine exchange, methods of correction.

13.Buffer systems of organism.

14.Concept about metabolic and respiratory acidosis, diagnostics, correction.

15.Concept about a metabolic and respiratory alkalosis, diagnostics, correction.

16.Description of solutions for infusion therapy.

17.Indication for parenteral nutrition. Description of preparations, rules of realization, control of efficiency.

18.Features of parenteral nutrition for children and elderly patients.

Determination and providing of initial level of knowledge-abilities.

Tasks for initial level verification.

Task 1.

A 50 years old patient was delivered to the hospital, wherein during laboratory investigation the metabolic alkalosis was educed. Which from the list below states could cause such acid-base disorder?

A. Hyperglycemic ketoacidosis state

B. Cerebral edema

C. Septic shock

D. Protracted diarrhea

E. Protracted vomiting

Task 2.

For a patient, delivered to the hospital, a stomach-ache and weakness are marked. BP is 90/60 mm Hg; Breath rate is 24 per minute. Body temperature is 37,3^{o}C. During laboratory investigation the sodium is 143 mmol/l, potassium is 3,5 mmol/l hematocrit index is 0,5 l/l. What syndrome, besides listed above, more credible could cause such blood changes?

A. Diarrhea

B. Fever

C. Shortness of breath

D. Arterial hypotension

E. Pulmonary edema

Task 3.

A 40 years old patient is delivered to the hospital. He was working in the street in a summer heat. During examination complains about thirst, uneasy, a skin and mucous membranes are dry, swallowing is labored. Body temperature is 37,4^{o}C. BP is 110/70 mm Hg. Urges to urination are not present. What need of examination for this patient to confirm of the supposed diagnosis?

A. Chest X-ray examination

B. Examination of otolaryngologist

C. Kidney and retroperitoneum ultrasound examination

D. Determination of serum sodium concentration and hematocrit index

E. Determination of potassium and calcium serum concentration

Task 4.

A patient with the dull trauma of stomach grumbles about thirst, weakness, decline of amount of the distinguished urine. Аускультативно in lungs breathing везикулярное, weak in bottom departments, dry wheezes. There is HELL of 80/40 мм.рт.ст. at an inspection, ЧСС 100 in min, T 37,1^{o}C. CVP 0 мм.рт.ст. What from the listed below physical data more exactly characterize water balance disorders which presents in this patient?

A. Dry rales in lungs

B. Dry mouth

C. Low CVP level

D. Tachycardia

E. Hyperthermia

Task 5.

A 32 years old female patient is disturbed by thirst, general weakness, oliguria. During laboratory examination there are the hemoconcentration, hypernatremia, increased plasm osmolarity. What kind of water balance disorder is present in this patient?

A. Hypertonic dehydration

B. Hypotonic dehydration

C. Isotonic dehydration

D. Hypotonic overhydration

E. Hypertonic overhydration

Task 6.

A 48 years old head trauma patient has a mixed character shortness of breath. Breath rate is 40 per minute. He is unconsciousness. During blood investigation hematocrit index is 0,47 l/l, serum sodium concentration is 150 mmol/l, 52 pH is 7,55, $paCO_2$ is 26 mm Hg., BE is + 2. What water-electrolyte or acid-base disorders is present in this patient?

A. Hyponatremia

B. Metabolic alkalosis

C. Respiratory alkalosis

D. Respiratory acidosis

E. Metabolic acidosis

Task 7.

A 52 years old patient, which suffering from ischemic heart disease, has a salt-free diet long period of time. The weakness, dizziness, palpitation and metallic foul mouth. are appeared in patient after the protracted stay in the sunlight. At examination the skin turgor is decreased, BP is 100/70 mm Hg. Urges to urination are not present. What from the listed below infusion solutions are indicated for disorders correction of this patient?

A.10% solution of glucose

B.0,0,9% solution of sodium chloride

C. Rheopolyglucinum

D. Haemodesum

E. 5% solution of glucose

Task 8.

A 20 years old female patient, delivered to the hospital in an unconscious state. During examination there are the superficial bradypnea and lips cyanosis. On the forearm there is the fresh track from an intravenous injection. In laboratory findings the $paCO_2$ is 50 mm Hg, pH is 7,28, BE is +5. What is the tactic of emergency for this patient?

A. Artificial pulmonary ventilation

B. Introduction of sodium bicarbonate

C. Infusion therapy by poly ionic solutions

D. Medicinal stimulation of breathing

E. Oxygen therapy

Task 9.

An 18 years old patient is delivered to the hospital with complaints about thirst, nausea, absence of appetite, weakness. He suffers from diabetes mellitus during last 3 years. He binds the state worsening with the insulin therapy regimen changing. Which pathological process can develop in this patient?

A. Cerebral edema

B. Total dehydration, acidosis

C. Гепатоспленомегалия

D. Acute heart failure

E. Hematuria

Task 10.

A 40 years old patient appealed to the clinic with complaints about the practically continuous vomiting, without relief, dizziness, left subcostal pain. Foregoing complaints have been developed 18 hours ago after an abundant feast. What change will be educed during patient examination?

A. Increase of diuresis rate

B. Blood pressure decreasing

C. Bradycardia

D. Peripheral edemata

E. Body temperature decreasing

Standards of right answers.

Task 1. E. Task 2. A. Task 3. D. Task 4. C. Task 5. A.

Task 6. C. Task 7. B. Task 8. A. Task 9. B. Task 10. B.

Short methodical instructions for practical study.

At the beginning of the study students pass control of initial knowledge-abilities level by means of test tasks. Students in the intensive care department examine the patients with water-electrolyte and acid-base disorders, make the plan of their investigation, interpret the indexes of ion gram, osmolarity, blood gas analysis results, got to know with the results of laboratory and tools examination in patients state dynamics, study hospital charts, determined the type of water-electrolyte and acid-base disturbances. Then students make the plan of intensive care for patients with the different variants of dehydrations and acid-base disorders, formed preparations for a parenteral nutrition, including patients of child's and elderly age. Situational tasks decide in default of such patients. Patient examination and answers of students controlled by a teacher.

In the classroom, students together with the teacher discuss the results of examination, and their mistakes. After the students pass test control. In the end worked out students get the marks of the work on study.

Suggested Literature

1. Tintinalli's Emergency Medicine A Comprehensive Study Guide, 9th edition, 2020.

2. Reichman's Emergency medicine procedures,3rd edition, 2018.

3. Anesthesiology and intensive care: textbook / F.S. Hlumcher, Yu.L. Kuchyn, S.O. Dubrov et al., 3rd edition, 2021.

4. The atlas of Emergency Medicine, Kevin J. Knoop and others,5th edition, 2020.

5. Internal Medicine: Critical Care: textbook N.M. Zhelezniakova, O.Ya. Babak, 2018.

ACUTE RENAL AND HEPATIC INSUFFICIENCY

Topic actuality.

Acute renal and hepatic insufficiency are the life-threatening rapidly progressed conditions, which complicate the course of great number of diseases. Among main causes of acute renal failure (ARF) most frequent are the shock states, inflammatory and toxic damages. Acute hepatic insufficiency (AHI) more frequent complicates the course of viral, autoimmune and toxic hepatitis. Development of these syndromes considerably worsen the course of essential disease and prognosis. Therefore, a doctor must be able to diagnose development of these states fast and in good time, be able to perform conservative therapy and choose the most rational method of efferent therapy.

Teaching aims.

General purpose: be able to diagnose acute renal and hepatic insufficiency, administer emergency, and determine the tactic of this patient's management.

Concrete aims	Initial level of knowledge-abilities
Able:	
1. To collect complaints, anamnesis, perform examination of acute renal and hepatic failure patients.	1. To interpret complaints, anamnesis, finding of examinations in patients with hemodynamics, breathing, digestion, and elimination disorders (department of internal medicine propaedeutic, general surgery).
2. To work out a plan of diagnostic investigation in patients with acute kidney and hepatic insufficiency, to interpret the obtained results.	2. To interpret the results of clinical and biochemical laboratory findings, instrumental researches (department of internal medicine propaedeutic), changes BP, CVP, and diuresis in patients with pathological conditions

	(department of pathophysiology).
3. To classify the clinical picture of acute kidney and hepatic insufficiency.	3. To interpret the homeostatic functions of liver and kidney and mechanisms of their disturbances (department of pathophysiology).
4. To form the preliminary diagnosis of acute kidney and hepatic insufficiency, administer emergency and determine the tactic of patient's management with ARF and AHI depending on the formed diagnosis.	4. To interpret the mechanism of action and indications for using of pharmacological agents during emergency in patients with acute kidney and hepatic insufficiency (department of pharmacology).
5. To explain the principles and choice of efferent therapy methods for patients with acute kidney and hepatic insufficiency.	5. To interpret the essence of dialysis method (department of biochemistry), peritoneal dialysis (department of general surgery), sorption (department of biological physics).

Kidney Failure

Your kidneys are a pair of organs located toward your lower back of the body, on either side of the spine. Your kidneys' main function is to act as a filtration system for your blood and to remove toxins from your body. The kidney transfers the toxins to the bladder, where they are later removed from the body during urination.

Kidney failure occurs when the kidneys lose the ability to sufficiently filter waste from the blood. Many factors can interfere with kidney health and function, such as toxic exposure to environmental pollutants and chemical food preservatives, certain diseases and ailments, and kidney

damage. If your kidneys cannot do their regular job, your body becomes overloaded with toxins. Left untreated, this can lead to kidney failure and can result in death.

1. What Causes Kidney Failure?

People who are most at risk for kidney failure suffer from one or more of the following causes:

- **Loss of Blood Flow to the Kidneys**

A sudden loss of blood flow to the kidneys may prompt kidney failure. Some diseases and conditions that cause loss of blood flow to the kidneys include heart attacks, heart disease, cirrhosis of the liver, dehydration, severe burns, cholesterol deposits, allergic reactions, and infections. Additionally, blood pressure and anti-inflammatory medications can limit blood flow.

- **Damage to the Kidneys**

Some diseases and conditions that may lead to kidney failure include:

- blood clots in or around the kidneys
- infections
- an overload of toxins from heavy metals
- drugs and alcohol
- vasculitis (inflammation of blood vessels)
- lupus (an autoimmune disease that can cause inflammation of many body organs)
- glomerulonephritis (inflammation of the small blood vessels of the kidneys)
- haemolytic uremic syndrome (syndrome characterized by a breakdown –haemolysis- of red blood cells following a bacterial infection, usually of the intestines)
- multiple myeloma (cancer of the plasma cells in the bone marrow
- scleroderma (an autoimmune disease that affects skin,
- thrombotic thrombocytopenic purpura (disorder of the blood clotting system that causes clots in small vessels of the body)
- chemotherapy drugs (medications that treat cancer and some immune diseases)
- dyes used in some imaging tests
- zoledronic acid used to treat elevated calcium levels
- certain antibiotics

- **Urine Elimination Problems**

When your body cannot eliminate urine, toxins build up and overload the kidneys. Some cancers can block the urine passageways. These include prostate, colon, cervical, and bladder. Other conditions can interfere with urination, such as kidney stones, an enlarged prostate, blood clots within the urinary tract, and damage to the nerves that control the bladder.

2. Five Types of Kidney Failure

There are five different types of kidney failure:

Acute Prerenal Kidney Failure

This is caused by insufficient blood flow to the kidneys. Without enough blood flow, the kidneys cannot filter toxins from the blood. This type is usually curable by resolving the cause of inadequate blood flow.

Acute Intrinsic Kidney Failure

This can be caused by direct trauma to the kidneys, such as physical impact, accidents, toxin overload, or ischemia (a lack of oxygen to the kidneys). Severe bleeding, shock, renal blood vessel obstruction, or glomerulonephritis (inflammation of the tiny filters in your kidneys) can all cause ischemia.

Chronic Pre-Renal Kidney Failure

When low blood flow to the kidneys is not treated and the condition remains for an extended period of time, chronic pre-renal kidney failure can occur. The kidneys begin to shrink and lose the ability to function.

Chronic Intrinsic Kidney Failure

Damage to the kidneys over an extended period due to intrinsic kidney disease can develop into chronic intrinsic kidney failure.

Chronic Post-Renal Kidney Failure

This happens when a long-term blockage of the urinary tract prevents urinary waste elimination, which causes pressure and eventual kidney damage.

3. The Symptoms of Kidney Failure

Many symptoms may imply kidney failure. Sometimes no symptoms are present, but usually someone with kidney failure will see a few signs of the disease. Possible symptoms include:

- a low amount of urination
- swelling of the legs, ankles, and feet from retention of fluids caused by the failure of the kidneys to eliminate water waste
- unexplained shortness of breath, drowsiness or fatigue, nausea, confusion, pain or pressure in the chest, seizures, and sometimes a coma

4. How is Kidney Failure Diagnosed?

There are several tests that can be used to diagnose kidney failure. These include:

- **Urinalysis**

A urine sample is tested for any abnormalities. Your doctor may perform a urinary sediment examination, which will measure the amount of red and white blood cells, look for high levels of bacteria, and look for high numbers of cellular casts.

- **Urine Measurements**

Measuring urine output is one of the simplest tests to help diagnose kidney failure. For example, low urinary output may suggest that kidney disease is due to a urinary blockage, which can be caused by multiple illnesses or injuries.

- **Blood Samples**

Samples of your blood are taken to measure substances that are filtered by your kidney, such as urea and creatinine. A rapid rise in their levels may mean kidney failure.

- **Imaging**

Imaging tests, such as ultrasounds, MRIs, and computerized tomography (CT) scans, allow a visual of the kidneys themselves, along with the urinary tract. This allows your doctor to visually look for blockages or abnormalities in your kidneys.

- **Kidney Tissue Sample**

Tissue samples are examined for abnormal deposits, scarring, or infectious organisms. A kidney biopsy is used to collect the tissue sample. A biopsy is a simple procedure that is performed while you are awake. A local anaesthetic is used to eliminate any discomfort. The sample is collected with a biopsy needle inserted through your skin and down into the kidney. X-ray or ultrasound equipment is used to locate the kidneys and assist the physician in guiding the needle.

5. Treatment for Kidney Failure

Dialysis

Dialysis performs the function of the kidneys. Depending on the type of dialysis, you may be connected to a large machine, or a portable catheter bag**.** A low-potassium, low-salt diet is often prescribed along with dialysis. It is important to note that dialysis does not cure kidney failure. However, it will extend your life.

Kidney Transplant

Another treatment is a kidney transplant. There is usually a long wait to receive a donor kidney that is compatible with your body. The advantages to a transplant are that the new kidney will work perfectly, and dialysis is no longer required. The disadvantage is that immunosuppressive drugs must be taken after the surgery. These drugs have their own side effects, some of which are serious. Also, there is a risk that the transplant surgery may fail, and may even result in death.

6. Can Kidney Failure be Prevented?

There are steps you can take to reduce your risk:

Limit Excessive Use of Toxic Substances.

Follow the directions for over-the-counter medications. Too-high doses (even of common drugs like aspirin) can create high toxin levels in a short amount of time, which can overload your kidneys. Whenever possible, you should limit your exposure to chemicals, such as household cleaners, tobacco, pesticides, and other toxic products.

Manage Kidney Problems as Recommended by a Doctor.

Many kidney or urinary tract conditions lead to kidney failure when not managed properly. Follow your doctor's guidance, always take prescribed medicine as directed, and adopt healthy diet guidelines.

HEPATIC FAILURE

Liver failure is the final common pathway for several types of liver disease. Progression to liver failure is varied and depends largely on comorbid entities, such as human immunodeficiency virus/acquired immunodeficiency syndrome, diabetes, obesity, continued injection drug use, and alcohol intake. Patients who develop acute liver failure have an extremely poor prognosis, with survival rates of < 30%. Among the most common of the entities that present as acute liver failure in the United States are acetaminophen overdose (46%), indeterminate causes (14%), other drugs (11%), hepatitis B virus (7%), and autoimmune hepatitis.

The clinical hallmarks of acute liver failure are hepatic encephalopathy, hepatorenal syndrome, and coagulopathy. The electrolyte imbalances seen in chronic liver disease can become extreme. Cerebral edema and intracranial hypertension are the most ominous complications. The catabolic nature of liver failure leads to negative nitrogen balance and immunodeficiency. Other clinical findings in acute liver failure include hypotension, hypoglycemia, and relative adrenal insufficiency. ED evaluation should include assessment for sepsis. Liver failure requires critical care in the ED and consultation with a liver transplant center. Recognizing fulminant hepatic failure is critical because transfer to a transplant center may be the most appropriate disposition.

transplant center may be the most appropriate disposition.

Treatment of fulminant liver failure in the ED involves care of the patient's respiratory status, blood pressure, and encephalopathy; correction of electrolyte derangements; identification of cerebral edema or intracranial hemorrhage; attention to active bleeding; and careful disposition, ensuring that the patient will be assessed for liver transplant in a timely fashion. Patients with respiratory failure due to ascites, effusions, or decreased alertness require intubation. Bilevel positive airway pressure is not typically an option in these cases because patients are too somnolent and at risk for aspiration. Blood pressure at this stage of liver disease is typically low due to malnutrition, bleeding, vomiting, diarrhea, and third spacing of fluid. Treat with a judicious initial fluid challenge; normal saline is the recommended resuscitation fluid. Norepinephrine is the initial vasopressor of choice for volume-refractory hypotension in hepatic failure, with the addition of vasopressin when escalating doses of norepinephrine are required. Take care when using vasopressin in severely encephalopathic patients, due to potential risk of increased cerebral vasodilatation and intracranial hypertension. The American

Association for the Study of Liver Diseases also recommends a trial of hydrocortisone in cases of persistent hypotension.

In fulminant liver failure, cerebral edema and intracranial hemorrhage should rise to the top of the differential for altered mental status. Patients with grade III or IV hepatic encephalopathy and concomitant renal failure, critically elevated ammonia levels, or hypotension are at high risk for cerebral edema; in this case, prophylactic hypertonic saline may be considered with a goal sodium level of 145 to 155 mEq/L. For patients with confirmed increased intracranial pressure, mannitol 0.5 to 1 gram/kg is recommended.

In the case of intracranial hemorrhage, treat coagulopathy. Consult neurosurgery or refer to a facility with neurosurgical services if the patient could benefit from hematoma evacuation or intracranial pressure monitoring.

Hepatorenal Syndrome

Hepatorenal syndrome is a complication of cirrhosis that often accompanies SBP (spontaneous bacterial peritonitis). It is defined as acute renal failure in a patient with histologically normal kidneys in the presence of pre-existing chronic or acute hepatic failure. The cause is not well understood. There are two types of hepatorenal syndrome. Type 1 is more serious and is identified by progressive oliguria and doubling of serum creatinine over a 2-week period.

Type 2 is represented by a gradual impairment in renal function that may or may not advance beyond moderate dysfunction. The discovery of abrupt renal failure in a cirrhotic patient that cannot be attributed to any other cause is a marker of extreme morbidity. Median survival for type 1 hepatorenal syndrome without medical treatment is 2 weeks.

Teaching content in accordance with aims.

Theoretical questions.

1. Etiology, pathogenesis of acute renal failure (ARF).
2. Differential diagnostics prerenal, renal and postrenal oliguria, anuria.
3. The stages of ARF clinical course.
4. Laboratory diagnostics and basic principles of ARF treatment.
5. Uremic coma, principles of intensive care.
6. Indications to hemodialysis.
7. Calculation of liquid daily requirements for ARF patients.
8. Causes of acute hepatic insufficiency.
9. Clinical picture and laboratory diagnostics of acute hepatic insufficiency.
10. Basic principles of the acute hepatic insufficiency treatment.
11. Hepatic coma, principles of intensive therapy.

Determination and providing of initial level of knowledge-abilities.

Tasks for initial level verification

Task 1.

On the third day of the onset of icteric period of hepatitis B such patient symptoms as indisposition, weakness, icterus is increased. The sizes of a patient liver are diminished. What symptom yet will be present for this patient?

A. Increased laxation

B. Local clonic cramps

C. Positive peritoneal signs

D. Hemorrhagic syndrome

E. Positive Pasternatsky symptom

Task 2.

A 50 years old female patient protractedly ingest an indomethacin for rheumatic polyarthritis. Then marked appearance of nausea, headache, weakness, edemata on extremities. In the last day urine output is 300 ml. Currently she disturbed about shortness of breath. Laboratory findings: erythrocytes are 3,0/L, serum urea is 26 mmol/L, potassium is 5,7 mmol/L. Which syndrome is an anchorman for this patient?

A. Acute hypertensive encephalopathy

B. Anemia

C. Overhydration

D. Chronic indomethacin intoxication

E. Hyper azotemia

Task 3.

A 40 years old patient admitted to the hospital with complaints about nausea, right subcostal pain, sclera icteritiousness, urine darkening and excrement discoloring. In laboratory findings there are increasing of aminotransferase and bilirubin levels. What from the indexes of blood given below is it yet needed to investigate necessarily?

A. Amylase

B. Cholesterol

C. Triglycerides

D. Magnesium

E. Prothrombin index

Task 4.

An 18 years old patient admitted to the hospital with complaints about a weakness, nausea, decreasing of urine output. It is known from anamnesis, that about two week ago there was an allergic reaction to the insect bite, which accompanied by dizziness and cold sticky sweat. He did not apply for medical service, and he is treated oneself independently. What changes are possible for this patient during laboratory investigation?

A. High specific gravity of urine

B. Unchanged and lixiviated erythrocytes

C. Urinary acid salts

D. Leucocyte cylinders

E. Hemopigment cylinders

Task 5.

A 56 years old patient, which during 6 years suffers from hepatocirrhosis, admitted to the hospital in the unconscious state. At examination: icteric skin, fetor hepaticus, and nystagmus. During percussion the liver is under a costal arc. In blood analysis: total bilirubin is 520 mkmol/L. What stage of hepatic encephalopathy does take place for this patient?

A. First

B. Second

C. Third

D. Fourth

E. Fifth

Task 6.

A 20 years old patient complained of permanent nausea, vomiting, frequent liquid stool, all this occur after the use of hot dog, which purchased on the street. A weakness and dizziness joined later. At examination: patient in consciousness, inhibited. Breathing independent, breath rate is 22 per minute. Heart tones are muffled, rhythmic, BP is 80/40 mm Hg, pulse is 100 per minute. During palpation the abdomen soft, painful in the paraumbilical area. Peritoneal signs are negative. Diuresis is 0,4 ml/min. What form of oligoanuria was complicate a cardinal disease?

A. Hemodynamic

B. Parenchymatous

C. Obstructive

D. Infectious

E. Vascular

Task 7.

A 17 years old patient, injection drug addict, delivered to the hospital with complaints of nausea, skin covers icteric. On a background the conducted therapy the state became worse, an icterus increased, vomiting and hiccup appeared, a liver diminished in sizes. What complication did develop in this patient?

A. Acute hepatic insufficiency

B. Acute kidney insufficiency

C. Acute adrenal insufficiency

D. Hemolytic crisis

E. Acute cholecystitis

Task 8.

A 50 years old patient after accidental use of ethylene glycol marked the expressed pallor of skin, total edemata, a muscular weakness. Objectively: breathing is become more frequent up to 24 per minute, there are the moist rales during auscultation in lungs. BP is 160/100 mm Hg., breath rate is 88 per minute. Abdomen is soft and painless. Liver during percussion is on a 2-3 cm below than edge of costal arc. What the most credible cause of edematous syndrome does a patient have?

A. Congestive heart failure

B. Hepatocirrhosis

C. Hypothyroidism

D. Hypertensive disease

E. Acute kidney insufficiency

Task 9.

A 48 years old patient admitted to the hospital with an acute rhabdomyolysis, complicated by acute kidney insufficiency in the stage of anuria. What from the listed below methods is optimal for the overhydration elimination?

A. Stimulation of diaphoresis

B. Limitation of the drinkable mode

C. Ultra-hemofiltration

D. Gastric lavage

E. Provocative diarrhea

Task 10.

A 51 years old patient, admitted to the intensive care department after 3 days of methyl alcohol ingesting. The forced diuresis therapy is performed. After transfusion of 2 L of salt and colloid solutions his state became worse. The dormancy, shortness of breath, cough with a pink sputum are appeared. During auscultation in lungs mass of fine moist rales. BP is 180/110 mm Hg. Laboratory: urea is 40 mmol/L, creatinine is 0,7 mmol/L, potassium is 6,8 mmol/L. What will be a primary measure for this patient treatment?

A. Hypotension therapy

B. Corticosteroids, antifoaming agents

C. Intensive stimulation of diuresis

D. Correction of hyperkalemia

E. Hemodialysis with an ultrafiltration

Standards of right answers.

Task 1. D. Task 2. C. Task 3. E. Task 4. B. Task 5. D.

Task 6. A. Task 7. A. Task 8. E. Task 9. C. Task 10. E.

Short methodical pointing to work on practical employment.

At the beginning of study students pass control of initial knowledge-abilities level by means of test tasks. In the department of intensive care students examine patients with acute kidney and hepatic insufficiency, make the plan of their examination, interpret present data of laboratory and instrumental examination, study hospital charts, make a preliminary diagnosis. Then students make the plan of patient's intensive care for acute renal and hepatic insufficiency, work off the methods of prophylaxis of their development, formulate indications for extracorporeal methods of detoxication. Situational tasks decide in default of such patients. Patients examination and answers of students controlled by a teacher.

In an educational room student together with a teacher discuss the results of examination and students mistake. After its students pass test control. In the end of study reviews previous work, and students get the mark of the work on study.

Suggested Literature

1. Tintinalli's Emergency Medicine A Comprehensive Study Guide, 9th edition, 2020.

2. Reichman's Emergency medicine procedures,3rd edition, 2018.

3. Anesthesiology and intensive care: textbook / F.S. Hlumcher, Yu.L. Kuchyn, S.O. Dubrov et al., 3rd edition, 2021.

4. The atlas of Emergency Medicine, Kevin J. Knoop and others,5th edition, 2020.

5. Internal Medicine: Critical Care: textbook N.M. Zhelezniakova, O.Ya. Babak, 2018.

6. Tarascon adult emergency pocketbook, 5th edition,2017.