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ANAESTHESIOLOGY AND INTENSIVE CARE

Topic 1: ACUTE POISONINGS

Topic 2: COMATOSE STATES

Methodical instructions for 5th year medical student practical training

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Content

ACUTE POISONINGS

Topic actuality

Poisoning is a worldwide problem that consumes substantial healthcare resources and causes many premature deaths. The burden of serious poisoning is carried by the developing world1; however, poisoning related morbidity and mortality are also a significant public health concern in the developed world. Unintentional poisoning deaths in the United States are increasing, especially as a result of prescription analgesics. This increase has been ascribed to increasing prescription rates and aging of the baby-boom population. Prevention is the key to reducing unintentional poisoning deaths. Pharmacists can ensure that medications are labeled correctly, anticipate potential drug interactions, and educate patients to use medications safely. Parents have the responsibility to ensure that poisons are placed in childproof, labeled containers stored in adult only accessible nonfood storage areas to reduce pediatric exposures. Teachers and healthcare providers can provide age-appropriate education to children about the dangers of poisons. After an exposure, poison control centers staffed by highly trained individuals can provide customized advice to healthcare providers and the public. Poison control centers also participate in prevention, education, and toxico-surveillance activities.

Rapid development of industry, in particular chemical and pharmacological, in the last decades resulted in considerable expansion of domestic chemical agents assortment with potentially toxic properties. This also leaded to contamination of environment, to spreaded of self-treatment with the use of drastic medicine, and to increasing of suicidal attempts incidence and elsewhere. Therefore the quantity of the acute exogenous poisonings increases progressively, and number of such hospitalized patients is one in a thousand of population per year. Taking into account the enormous variety of poison substances, extremely important for a doctor is ability to determine character of prevailing toxic influence on an organism (neurotoxic, cardiotoxic, hepatotoxic, nephrotoxic, hemotoxic and other) and as far as possible to perform organ protective intensive therapy. Such measures include normalization of hemodynamics, hemocoagulation, breathing, and treatment of brain edema.

The rapid correction of leading syndrome in poisoning patients is often preceded to the detoxication, because its determine safety of detoxication procedures. The contact of organism with a poison results in stimulation of natural detoxication.

The role of a doctor here consists in knowledge about features of poison contact with an organism, routes of its transformation and elimination, stimulation and replacement methods of organs detoxication function.

Teaching aims.

General purpose: be able to diagnose the acute poisonings, to perform emergency support and determine the tactic of patients management with the acute poisonings by tranquilizers, barbiturates,

opiates, organophosphorous substances , ethyl and methyl alcohols, carbon monoxide, acids and alcali, by poisonous mushrooms, bites of insects and snakes.

Concrete aims	**Initial level of knowledge-abilities**
Be able:	
1. To find out complaints and anamnesis, perform the examination of patients with an acute poisoning, to interpret the disorders of vital functions and determine a leading pathological syndrome in this	1. To interpret complaints, anamnesis, examination findings for patients with hemodynamics, breathing and digestion, excretion, consciousness disturbances (department of
2. To classify the different types of the acute poisoning.	2. To interpret the syndromes of consciousness disorders and autonomic disturbances (department of neurology), cardiovascular, respiratory, hepatic-kidney insufficiency,
3. To provide emergency support for patients with the acute poisoning.	3. To execute the tube gastric or intestine lavage (Surgery department), to interpret the mechanisms of action and classification of antidotes
4. To make a plan of acute poisoning patients examination, to interpret the obtained results.	4. To interpret the results of clinical and biochemical findings, instrumental investigations (department

5. To identify the preliminary diagnosis of the acute poisoning and explain the choice of intensive care methods and define the tactic of acute poisonings patients management .	5. To interpret the mechanism of action and indications to using of pharmacological agents, for patients with acute poisonings (department of pharmacology), to interpret essence of dialysis method (department of biochemistry), peritoneal dialysis

Acute poisoning

Poisons are substances that can cause disturbances to organisms, usually by chemical reaction or other activity on the molecular scale, when a sufficient quantity is absorbed by an organism.

A toxin (Greek: τοξικόν, toxikon) is a poisonous substance produced by living cells or organisms (technically, although humans are living organisms, man-made substances created by artificial processes usually are not considered toxins by this definition).

It was the organic chemist Ludwig Brieger (1849–1919) who first used the term 'toxin'.
For a toxic substance not produced by living organisms, "toxicant" is the more appropriate term, and "toxics" is an acceptable plural.

Toxins can be small molecules, peptides, or proteins that are capable of causing disease on contact with or absorption by body tissues interacting with biological macromolecules such as enzymes or cellular receptors. Toxins vary greatly in their severity, ranging from usually minor and acute (as in a bee sting) to almost immediately deadly (as in botulinum toxin).
Toxins are often distinguished from other chemical agents by their method of production - the word toxin does not specify method of delivery (compare with venom and the narrower meaning of poison – all substances that can also cause disturbances to organisms). It simply means it is a biologically produced poison. There was an ongoing terminological dispute between NATO and the Warsaw Pact over whether to call a toxin a biological or chemical agent, in which the NATO opted for chemical agent, and the Warsaw Pact for biological agent.

According to a International Committee of the Red Cross review of the Biological Weapons Convention, "Toxins are poisonous products of organisms; unlike biological agents, they are inanimate and not capable of reproducing themselves." and "Since the signing of the Convention, there have been no disputes among the parties regarding the definition of biological agents or toxins..."

According to Title 18 of the United States Code, "...the term "toxin" means the toxic material or product of plants, animals, microorganisms (including, but not limited to, bacteria, viruses, fungi,

rickettsiae or protozoa), or infectious substances, or a recombinant or synthesized molecule, whatever their origin and method of production..."

A rather informal terminology of individual toxins relate them to the anatomical location where their effects are most notable:

Hemotoxin, causes destruction of red blood cells (hemolysis)

Phototoxin, causes dangerous photosensitivity

On a broader scale, toxins may be classified as either exotoxins, being excreted by an organism, and endotoxins, that are released mainly when bacteria are lysed.

Related terms are:

- Toxoid, weakened or suppressed toxin
- Venom, toxins in the sense of use by certain types of animals
- The term "environmental toxin" is often used.

In these contexts, it can sometimes explicitly include contaminants that are man-made, which contradicts most formal definitions of the term "toxin". Because of this, when encountering the word "toxin" outside of microbiological contexts, it is important to confirm what the researcher means by the use of the term.

The toxins from food chains which maybe dangerous to human health include:

- Paralytic shellfish poisoning (PSP)
- Amnesic shellfish poisoning (ASP)
- Diarrheal shellfish poisoning (DSP)
- Neurotoxic shellfish poisoning (NSP)

Initial management for all poisonings includes ensuring adequate cardiopulmonary function and providing treatment for any symptoms such as seizures, shock, and pain.

Poisons that have been injected (e.g. from the sting of poisonous animals) can be treated by binding the affected body part with a pressure bandage and by placing the affected body part in hot water (with a temperature of 50°C). The pressure bandage makes sure the poison is not pumped troughout the body and the hot water breaks down the poison. This treatment, however, only works with poisons that are composed of protein-molecules.

If the toxin was recently ingested, absorption of the substance may be able to be decreased through gastric decontamination. This may be achieved using activated charcoal, gastric lavage, whole bowel irrigation, or nasogastric aspiration. Routine use of emetics (syrup of Ipecac), cathartics or laxatives are no longer recommended.

Activated charcoal is the treatment of choice to prevent absorption of the poison. It is usually administered when the patient is in the emergency room or by a trained emergency healthcare provider such as a paramedical or emergency doctor. However, charcoal is ineffective against metals such as sodium, potassium, and lithium, and alcohols and glycols; it is also not recommended for ingestion of corrosive chemicals such as acids and alkalis.

Whole bowel irrigation cleanses the bowel, this is achieved by giving the patient large amounts of a polyethylene glycol solution. The osmotically balanced polyethylene glycol solution is not absorbed into the body, having the effect of flushing out the entire gastrointestinal tract. Its major uses are following ingestion of sustained release drugs, toxins that are not absorbed by activated charcoal (i.e. lithium, iron), and for the removal of ingested packets of drugs (body packing/smuggling).

Gastric lavage, commonly known as a stomach pump, is the insertion of a tube into the stomach, followed by administration of water or saline down the tube. The liquid is then removed along with the contents of the stomach. Lavage has been used for many years as a common treatment for poisoned patients. However, a recent review of the procedure in poisonings suggests no benefit. It is still sometimes used if it can be done within 1 hour of ingestion and the exposure is potentially life threatening.

Nasogastric aspiration involves the placement of a tube via the nose down into the stomach, the stomach contents are then removed via suction. This procedure is mainly used for liquid ingestions where activated charcoal is ineffective, e.g. ethylene glycol poisoning.

Emesis (i.e. induced by ipecac) is no longer recommended in poisoning situations, because vomiting is ineffective at removing poisons.

Cathartics were postulated to decrease absorption by increasing the expulsion of the poison from the gastrointestinal tract. There are two types of cathartics used in poisoned patients; saline cathartics (sodium sulfate, magnesium citrate, magnesium sulfate) and saccharide cathartics (sorbitol). They do not appear to improve patient outcome and are no longer recommended.

Table 1: common toxins and antidote.

Poison/Drug	**Antidote**
Paracetamol (acetaminophen)	N-acetylcysteine
vitamin K anticoagulants, e.g. warfarin	vitamin K
Opioids	naloxone

Beta-Blockers (Propranolol, Sotalol)	Calcium Gluconate and/or Glucagon
Isoniazid	Pyridoxine
Iron (and other heavy metals)	desferrioxamine, Deferasirox or Deferiprone
benzodiazepines	flumazenil
Methanol	ethanol or fomepizole, and folinic acid
Cyanide	amyl nitrite, sodium nitrite and sodium thiosulfate
Organophosphates	Atropine and Pralidoxime
Magnesium	Calcium Gluconate
Atropine	Physostigmine
Thallium	Prussian blue
Hydrofluoric acid	Calcium Gluconate
Calcium Channel Blockers (Verapamil, Diltiazem)	Calcium Gluconate
Anticholinergics	Cholinergics (and vice-versa)

In some situations elimination of the poison can be enhanced using diuresis, hemodialysis, hemoperfusion, hyperbaric medicine, peritoneal dialysis, exchange transfusion or chelation. However, this may actually worsen the poisoning in some cases, so it should always be verified based on what substances are involved.

Resuscitation

Resuscitation is the first priority in any poisoned patient. After resuscitation, a structured risk assessment is used to identify patients who may benefit from an antidote, decontamination, or enhanced elimination techniques. Most patients only require provision of good supportive care during a period of observation in an appropriate environment. Treatment of cardiac arrest in poisoned patients follows Advanced Cardiac Life Support guidelines with the addition of interventions potentially beneficial in toxin-induced cardiac arrest.Prolonged resuscitation is generally indicated, as patients are often young with minimal preexisting organ dysfunction. Utilization of extracorporeal cardiac and respiratory assist devices until organ toxicity resolves may be lifesaving. Stabilization of airway,

breathing, and circulation represents initial priorities. Compromised airway patency or reduced respiratory drive may lead to inadequate ventilation; provision of a mechanical airway and assisted ventilation is vital in these circumstances. IV crystalloid bolus (10 to 20 mL/kg) is first-line treatment of hypotension. Since most patients without toxin-induced fluid loss are generally not fluid depleted, avoid administration of excess fluid. Persisting hypotension despite an adequate volume infusion may respond to a specific antidote. Otherwise, cautious administration of an inotropic agent is indicated. Inotrope choice is guided by knowledge of the toxin's toxicodynamic properties and assessment of circulatory status (e.g., cardiac pump failure vs. vasodilatory shock). Extracorporeal membrane oxygenation should be considered for cases of cardiovascular failure refractory to other treatment modalities.

Table 2: Examination of the Poisoned Patient.

Organ System	Examination	Example of Finding (Possible Significance
General	Mental state and dress Signs of injury Odors Nutritional state Vital signs	Unkempt (psychiatric illness). Scalp hematoma (intracranial injury). Malnourished (IV drug use, human immunodeficiency virus infection). Smell of bitter almonds (cyanide toxicity).
CNS	Conscious state. Pupil size and reactivity Eye movements. Cerebellar function/gait.	Miosis (opioids, organophosphates, phenothiazines, clonidine intoxication). Nystagmus/ataxia (anticonvulsant and ethanol toxicity).
Cardiovascular	Heart rate/blood pressure Cardiac auscultation.	Murmur (endocarditis/IV drug use).

Respiratory	Oxygen saturation. Respiratory rate. Chest auscultation.	Fever/crepitations/hypoxia (aspiration pneumonia). Bronchorrhea/crepitations/hypoxia (organophosphate toxicity).
GI	Oropharynx. Abdomen. Bladder.	Urinary retention (anticholinergic toxicity). Oral cavity burns (corrosive ingestion). Hypersalivation (cholinergic toxidrome).
Peripheral nervous	Reflexes Tone Fasciculations Tremor Clonus	Tremor/fasciculations (lithium toxicity). "Lead pipe" rigidity (neuromuscular malignant syndrome). Clonus/hyperreflexia (serotonin toxicity).
Dermal/peripheral	Bruising Cyanosis Flushing Dry/moist skin Injection sites Bullae	Bruising (coagulopathy, trauma, coma). Flushing/warm, dry skin (anticholinergic toxicity). Warm, moist skin (sympathomimetic toxicity). Bullae (prolonged coma, barbiturates).

Further treatment.

In the majority of poisonings the mainstay of management is providing supportive care for the patient, i.e. treating the symptoms rather than the poison.

Teaching content in accordance with aims.

Theoretical questions.

1. Concept about the acute poisoning. Classification of poisonings. 2. General principles of acute poisoning intensive care.
3. Basic principles of the forced diuresis.
4. Extracorporeal methods of detoxication, indications and contraindication, means, technology.
5. Principles of antidote therapy.
6. Pathogenesis, clinical picture, intensive care of ethyl and methyl alcohol poisoning.
7. Pathogenesis, clinical picture, intensive care of tranquilizers, barbiturates, and opiates poisonings.
8. Pathogenesis, clinical picture, intensive care of organophosphorus substance poisoning.
9. Pathogenesis, clinical picture, intensive care erapy of carbon monoxide poisoning .
10. Pathogenesis, clinical picture, intensive care of mushrooms poisoning .
11. Pathogenesis, clinical picture, intensive care of acids and lyes poisoning.
12. Emergency features of the insects and animals bites.

Determination and providing of initial level of knowledge-abilities.

Tasks for initial level verification.

Task 1.

In 18 years old patient after 18 hours of home-made saltfish intake the weakness, propensity to the fainting, paropsis and deglutitive problem are appeared. A liquid stool was 4 times for this period, vomiting was not. Breath rate is 20 per minute, BP is 140/90 mm Hg, HR is 62 beat per minute. A botulism is proposed as diagnosis . What from the listed below is allow to suppose about the possibility of the respiratory failure development?

A.Propensity to the fainting

B.Breath rate

C.Bradycardia

D.Paropsis and deglutitive problem

E.Blood pressure

Task 2.

Miosis, shortness of breath, blood pressure decreasing, spastic abdominal pain and diarrhea are appeared in a patient. What type of syndrome is present in this patient?

A. N-cholinomimetic

B. M-cholinomimetic

C. α-adrenergic

D. β-adrenergic

E. Anticholinergic

Task 3.

To the 22 years old patient, which was found without consciousness in the den of drug addicts, naloxone was injected. What mechanism of antidote action is used in this case?

A. Physical interaction of poison and antidote

B. Chemical interaction of poison and antidote

C. Competition for receptors

D. Competition for the ways of metabolism

E. Stimulation of competition physiological effects

Task 4.

The shortness of breath, pallor of skin covers, lips cyanosis and acrocyanosis was increased in patient which suffering from peritonitis. What sign in such situation will specify of the necessity to primary correction of blood circulation, but not breathings?

A. Increase of heart rate

B. Decreasing of blood pressure

C. Decreasing of pulmonary artery pressure

D. Increase of alveolo-arterial difference on oxygen

E. Increase of alveolo-arterial difference on oxygen

Task 5.

A 55 years old patient admitted to intensive care department on the subject of leptospirosis, which complicated by acute hepatorenal insufficiency. The brain edema was developed, and ultrafiltration performing is planned in this patient. What does the method of ultrafiltration implies?

A. Gradient of hydrostatical pressure

B. Gradient of concentration of toxic substance

C. Gradient of osmolarity

D. Combination of dialysis and osmose principles

E. Isolated diffusion of toxic substance

Task 6.

A patient accidentally have a drink of the unknown industrial-use liquid. There are continuous vomiting and dormancy after 2 hours of onset. BP is 160/90 mm Hg, minute diuresis is 0,1 ml per minute, the abdomen is mildly swollen and painful during palpation. Which syndrome of poisoning is leading for this patient?

A. Acute kidney insufficiency

B. Arterial hypertension

C. Consciousness disorders

D. Dyspeptic syndrome

E. Acute hepatic insufficiency

Task 7.

A patient is delivered to the hospital with complaints of convulsive twitches in muscles, sweating, spastic abdominal pain. It is known that he worked on a summer residence the day before. Objectively: excited, pupils narrow, equal, skin moist, clean. There are extremities fibrillar muscles twitches. BP is 140/90 mm Hg, weak filling pulse is 50 per minute. Which pathological syndrome is leading for this patient?

A. Cholinomimetic

B. β-adrenomimetic

C. adrenolytic

D. Cholinolytic

E. α-adrenomimetic

Task 8.

A victim with the acute ethyl alcohol poisoning is delivered to admission department without consciousness, there is face hyperemia, pupils are moderately dilated. Spontaneous breathing is hoarse. Breath rate is 16 per minute. BP is 150/90 mm Hg. HR is 100 beat per minute. What is it necessary to begin in this patient as emergency?

A. Tube gastric lavage

B. Infusion of 10% glucose solution

C. Open airway support

D. Forced diuresis

E. Injection of thiamine bromide

Task 9.

A 70 years old patient admitted to ICU with complaints of nausea, vomiting, headache, unsharp vision and flick flies before the eyes. He is alcohol abuse. Mental is confusion. There are dry skin and face hyperemia. Hearts tones are muffled, unrhythmic. BP is 90/50 mm Hg, HR is 96 per minute. Abdomen is soft, during palpation painful in an epigastrium and left subcostal area. Peristalsis is active. What is the mechanism of antidote action, which is necessary for this patient treatment?

A. Chemical binding in the humoral environment

B. Change of toxic substance metabolism

C. Opposite effect on receptors

D. Physical binding of poison in GI tract

E. Forming of binding as antigen-antibody

Task 10.

A 60 years old patient, which suffering from rheumatism and mitral insufficiency, ingested 20 pills of phenobarbital with a suicidal purpose. Objectively: 2 degree coma. Skin is pale, the "white spot" symptom is positive. Breathing is spontaneous, breath rate is 18 per minute, BP is 90/60 mm Hg., a pulse is unrhythmic, with weak filling and tension, HR is 100 beats per minute. Abdomen is soft, there is no reaction to palpation. Urine output slowed down. Which method of treatment is preferable for this patient?

A. Forced diuresis

B. Antidote therapy

C. Hemocarboperfusion

D. Hemodialysis

E. Peritoneal dialysis

Standards of right answers.

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

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

Short methodical instructions for practical study

The initial level of students knowledge-abilities performed at the beginning of the study, using the test tasks. Students in the ISU examine patients with the acute poisonings, distinguish the leading syndrome of the acute poisoning, perform differential diagnostics with similar symptomatology diseases, preliminary diagnose, analyse histories of illnesses with the estimation of laboratory and additional methods of inspection, make the plan of additional examination. Also students trained in the methods of gastric lavage and forced diuresis, make the plan of emergency support and treatment of patients with the different types of the acute poisonings, formulate indications for extracorporeal methods of detoxication. In default of thematic patients they are decide of situatioonal tasks . Patient examination and students answers are controlled by a teacher.

In an educational room students together with a teacher discuss the results of examination, and students mistakes. After it students pass test control. In the end of study students get the mark of their work.

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

COMATOSE STATES

Topic actuality.

A coma is the state of the deep depression of CNS, attended with the loss of consciousness. Most often in practice there are acute disturbances of cerebral blood circulation , exotoxic comae (alcoholic, narcotic), diabetic, craniocerebral traumas. All these states are frequently complicated by cerebral edema, hyperthermal and convulsive syndrome, disorders of hemodynamics and breathing. Sometimes these states became more threatening for life, then primary disease and require specific prophylactics, diagnostic and medical measures for preventing of severe complications and even patients death. A doctor must be able not only to render the first aid but also able to examine a patient, perform differential diagnostics and determined the cause of the comatose state for comprehensive and adequate therapy.

Teaching aims.

General purpose: be able to diagnose the comatose state, render the first aid and determine the tactic of patients management for hypoglycemic, hyperglycemic, hyperosmolar, hepatic and uremic comae.

Concrete aims	**Initial level of knowledge-abilities**
Be able:	
1. To take the history, perform the objective examination of unconsciousness patients and recognize the syndrome of coma.	1. To interpret anamnesis, objective findings for unconsciousness patients (department of neurology), hemodynamics and breathing disturbances (department of
2. To render the first aid for comatose patient, including. cerebral edema, convulsive and hyperthermal syndrome as for adults and children.	2. To interpret the changes of breathing and hemodynamics duringthe генерализованного attack of cramps (department of neurology and medical genetics), mechanisms of fever

3. To make a plan of comatose patients examinations, to interpret the obtained results.	3. To interpret the clinical and biochemical laboratory findings, the results of instrumental examination of patients with CNS disorders
4.To perform differential diagnostics of the comatose state causes.	4. . To interpret and determine the symptoms of focal anddiffuse brain lesions (department of neurology), ways of carbohydrate exchange decompensation in
5. To preliminary diagnose and determine the tactic of management in the different origin comatose state patient.	5. To interpret the concepts of etiotropic, nosotropic and symptomatic therapy (department of pathophysiology), mechanism of action and indication to use of

Diabetic coma

1. Diabetic coma is a reversible form of coma found in people with diabetes mellitus. It is a medical emergency.

Three different types of diabetic coma are identified:

Severe diabetic hypoglycemia

Diabetic ketoacidosis advanced enough to result in unconsciousness from a combination of severe hyperglycemia, dehydration and shock, and exhaustion

Hyperosmolar nonketotic coma in which extreme hyperglycemia and dehydration alone are sufficient to cause unconsciousness.

In most medical contexts, the term diabetic coma refers to the diagnostical dilemma posed when a physician is confronted with an unconscious patient about whom nothing is known except that they have diabetes. An example might be a physician working in an emergency department who receives an unconscious patient wearing a medical identification tag saying DIABETIC. Paramedics may be called to rescue an unconscious person by friends who identify them as diabetic. Brief descriptions of the three major conditions are followed by a discussion of the diagnostic process used to distinguish among them, as well as a few other conditions which must be considered.

An estimated 2 to 15 percent of diabetics will suffer from at least one episode of diabetic coma in their lifetimes as a result of severe hypoglycemia.

2. Types:

Severe hypoglycemia

People with type 1 diabetes mellitus who must take insulin in full replacement doses are most vulnerable to episodes of hypoglycemia. It is usually mild enough to reverse by eating or drinking carbohydrates, but blood glucose occasionally can fall fast enough and low enough to produce unconsciousness before hypoglycemia can be recognized and reversed. Hypoglycemia can be severe enough to cause unconsciousness during sleep. Predisposing factors can include eating less than usual or prolonged exercise earlier in the day. Some people with diabetes can lose their ability to recognize the symptoms of early hypoglycemia.

Unconsciousness due to hypoglycemia can occur within 20 minutes to an hour after early symptoms and is not usually preceded by other illness or symptoms. Twitching or convulsions may occur. A person unconscious from hypoglycemia is usually pale, has a rapid heart beat, and is soaked in sweat: all signs of the adrenaline response to hypoglycemia. The individual is not usually dehydrated and breathing is normal or shallow. Their blood sugar level, measured by a glucose meter or laboratory measurement at the time of discovery, is usually low but not always severely, and in some cases may have already risen from the nadir that triggered the unconsciousness.

Unconsciousness due to hypoglycemia is treated by raising the blood glucose with intravenous glucose or injected glucagon.

Advanced diabetic ketoacidosis

Diabetic ketoacidosis (DKA), if it progresses and worsens without treatment, can eventually cause unconsciousness, from a combination of severe hyperglycemia, dehydration and shock, and exhaustion. Coma only occurs at an advanced stage, usually after 36 hours or more of worsening vomiting and hyperventilation.

In the early to middle stages of ketoacidosis, patients are typically flushed and breathing rapidly and deeply, but visible dehydration, pallor from diminished perfusion, shallower breathing, and tachycardia are often present when coma is reached. However these features are variable and not always as described.

If the patient is known to have diabetes, the diagnosis of DKA is usually suspected from the appearance and a history of 1–2 days of vomiting. The diagnosis is confirmed when the usual blood chemistries in the emergency department reveal hyperglycemia and severe metabolic acidosis.

Treatment of DKA consists of isotonic fluids to rapidly stabilize the circulation, continued intravenous saline with potassium and other electrolytes to replace deficits, insulin to reverse the ketoacidosis, and careful monitoring for complications.

Nonketotic hyperosmolar coma

Nonketotic hyperosmolar coma usually develops more insidiously than DKA because the principal symptom is lethargy progressing to obtundation, rather than vomiting and an obvious illness. Extreme hyperglycemia is accompanied by dehydration due to inadequate fluid intake. Coma from NKHC occurs most often in patients who develop type 2 or steroid diabetes and have an impaired ability to recognize thirst and drink. It is classically a nursing home condition but can occur in all ages.

The diagnosis is usually discovered when a chemistry screen performed because of obtundation reveals extreme hyperglycemia (often above 1800 mg/dl (100 mM)) and dehydration. The treatment consists of insulin and gradual rehydration with intravenous fluids.

3. Identifying the cause

Diabetic coma was a more significant diagnostic problem before the late 1970s, when glucose meters and rapid blood chemistry analyzers became universally available in hospitals. In modern medical practice, it rarely takes more than a few questions, a quick look, and a glucose meter to determine the cause of unconsciousness in a patient with diabetes. Laboratory confirmation can usually be obtained in half an hour or less. Other conditions that can cause unconsciousness in a person with diabetes are stroke, uremic encephalopathy, alcohol, drug overdose, head injury, or seizure.

Fortunately, most episodes of diabetic hypoglycemia, DKA, and extreme hyperosmolarity do not reach unconsciousness before a family member or caretaker seeks medical help.

4. Treatment

Treatment depends upon the underlying cause:

Ketoacidotic diabetic coma: intravenous fluids, insulin and administration of potassium and sodium.

Hyperosmolar diabetic coma: plenty of intravenous fluids, insulin, potassium and sodium given as soon as possible.

Hypoglycaemic diabetic coma: administration of the hormone glucagon to reverse the effects of insulin, or glucose given intravenously.

Teaching content in accordance with aims.

Theoretical questions.

1. Types of consciousness disturbances, estimation of consciousness level.
2. Intensive care principles for different origin of comatose states.
3. Pathogenesis, clinical picture and intensive care for hypoglycemic coma patients.
4. Pathogenesis, clinical picture and intensive care for hyperglycemic coma patients.
5. Pathogenesis, clinical picture and intensive care for hyperosmolar coma patients.
6. Intensive therapy of cerebral edema, convulsive and hyperthermal syndrome for adults and children

Determination and providing of knowledge-abilitiesinitial level

Tasks for verification of initial level.

Task 1.

The 32 years old patient, sitting on a bench, suddenly lost consciousness fell down. What more credible pathological process did determine rapid development of this patient comatose state?

A. Acute exogenous intoxication

B. Tubercular meningoencephalitis

C. Brain tumor

D. Kidney insufficiency

E. Diabetic ketoacidosis

Task 2.

A patient was examined by the emergency doctor, the temperature of body is educed 38,5oC. Duration of disease is 2-3 hours. How is it possible to describe such temperature for a patient?

A. Fever

B. Hypothermia

C. Hyperthermia

D. Hyperpyrexia

E. Subfebrile fever

Task 3.

On the CT-scan of the patients head focus of hemorrhage was found in the right internal capsule area. What movement disorder can be found in this patient during neurological examination?

A. Right-side hemiparesis

B. Left-side hemiparesis

C. Alternating hemiparesis

D. Overhead paraparesis

E. Bottom paraparesis

Task 4.

A 53 years old patient, delivered to the hospital in the unconscious state. A diabetic ketoacidotic coma is suspected. Wat is the basic nosotropic mechanism of diabetic ketoacidosis development in this patient?

A.Progressed hyperkalemia

B. Metabolic alkalosis

C. Excess of lactic acid

D. Hypovolemia, activating of lipolysis

E. Respiratory insufficiency

Task 5.

A 20 years old patient, suffering from diabetes mellitus during 4 years, delivered to the hospital with a comatose state. After an examination the diagnosis of diabetic ketoacidotic coma is supposed. What is etiotropic therapy in this case?

A. Insulin therapy

B. Infusion of 5% solution of glucose

C. Infusion of 4% solution of sodium bicarbonate

D. Oxygen therapy

E. Ingection of large doses of vitamins

Task 6.

A patient does not react on the turned speech and pain. There is otal ophthalmoplegia, it is low muscles tone, and tendon reflexes are languid, Cheyne-Stokes respiration appeared. Wtat is the coma degree for this patient?

A. Mild degree (coma I)

B. Middle degree (coma II)

C. Deep coma (coma III)

D. Terminal coma (coma IV)

E. –

Task 7

A 30 years old patient, resident in an own house, during two days was disturbed for cough, permanent headache and weakness, which are increased progressively. Next day in the morning relatives were not able to awaken a patient and call the ambulance. Patient after intravenous injection of 20 ml of 40% glucose solution and ascorbic acid was delivered to the hospital. What comatose state, as in this patient, does have the gradual beginning?

A. Viral meningitis

B. Hypoglycemic coma

C. Epileptic coma

D. Carbon monoxide poisoning

E. Narcoma

Task 8.

The patient of 52 years old was delivered to the hospital from a street in the unconscious state. At examination consciousness at the level of lump 2 items. Of mouth the smell of alcohol is audible.

On the right parietal area a haematoma is educed, there is anisocoria. What examination does need to be performed in this patient?

A. Electroencephalography

B. Computer tomography of head

C. Determination of blood hematocrit

D. Determinations of alcohol in urine

E. Spinal puncture

Task 9.

A 59 years old patient was delivered to the hospital with relatives complaints of unconscious state. According to relatives, within a week grumbled about ear pain, irregularly intake an analgin. At examination consciousness is at the deep sopor level, rigidity of cervical muscles is marked. Body temperature is 37,8oC. What examination does need to be performed for this patient?

A. Maxillary sinus puncture

B. Electroencephalography

C. Determination of blood coagulation time

D. Determinations of alcohol in urine

E. Spinal puncture

Task 10.

A patient is 29 years, to being in a clinic on an occasion эпистатуса, conduct anticonvulsant therapy. For 2 hours 20 mgs of сибазона are entered, 6 g of оксибутирата sodium, 20 мл of 25% solution of sulfate of magnesium. Attacks became more short and rare, but did not pass fully. What is needed to prevent ventilation disorders in this patient?

A. Inhalation of oxygen

B. Inhalation of carbonic acid

C. Pulmonary mechanical ventilation

D. Periodic bemegride injection

E. Periodic flyumazenil injection

Standards of right answers.

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

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

Short methodical pointing to work on practical employment.

At the beginning of study students pass control of initial level of knowledge-abilities by means of test tasks. Students in the department of intensive care examine patients with the comatose states, determine the level of the comatose state, work off the methods of first aid for patients with cerebral edema, convulsive and hyperthermal syndrome, including. for children. Then students make the plan of patients examination, analyse histories of illnesses with the estimation of laboratory and additional methods of investigation, perform differential diagnostics and preliminary diagnose of the comatose state cause. After it students determine the plan of medical measures for patients with hypoglycemic, hyperglycemic, hyperosmolar, hepatic, uremic comae. Situatioonal tasks decide in default of thematic patients. Patient examination and answers of students controlled by a teacher.

In an educational room students together with a teacher discuss the results of examination and students mistakes. After it students pass test control. In the end of the study students get the mark of their work.

Suggested Literature

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

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

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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.