

COMA DISEASE, STAGES AND TRANSITION

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ABSTRACT:

This article discusses coma, its stages, development and the state of patients and their types during and before the coma.

KEYWORDS: Coma, clinical course, sopor, decortication, apical syndrome, deserebration.

INTRODUCTION:

The Coma (synonym - an unpleasant state) is a severe pathological condition characterized by a sharp disturbance of conscious activity. Coma is accompanied by loss of response to external influences and a sharp disruption of vital functions (cardiovascular and respiratory activity). Coma is a severe pathology of the central nervous system, the next stage of which is brain death. The Coma is not a separate disease, but it is a complication of other pathological conditions or diseases.

ETIOLOGY AND PATHOGENESIS:

The causes of coma can be divided into 5 groups.

1. Primary brain damage (cerebral factors) - insulitis, meningitis, encephalitis, epilepsy, edema, brain injuries and etc.
2. Metabolic disorders - diabetic, thyrotoxic, liver and etc.
3. Toxic factors - exogenous and endogenous intoxications, including toxic infections.
4. Disorders of gas metabolism: a) hypoxemia (decreased oxygen supply to the body from the outside) or in severe anemia, a decrease in the

supply of oxygen to the brain; b) diseases leading to respiratory or respiratory-acidic hypercapnia.

5. Disorders of water and electrolyte metabolism in the body and electrolyte deficiency is a major factor. For example, chul or other heat; long without water in places; congestion, recurrent recurrences, alimentary factors (abrupt restriction of drinking and eating) and etc.

Here, the factors that are a priority in the development of coma are highlighted separately. In fact, the etiological factors that lead to coma are inextricably linked. For example, the toxic factors that cause coma are, of course, accompanied by metabolic disorders, or these disorders lead to the formation of substances that have toxic effects on nerve tissue. Sometimes metabolic and toxic factors (endogenous toxemia) are combined. For example, thyrotoxicosis, infection, toxins, and etc. Prompt and qualified care for a patient in any coma begins with determining its etiology. Therefore, it is very important to determine the etiology of each coma.

DEGREE OF UNPLEASANT SITUATIONS:

Sopor is a mild disturbance of mood, and reactions to "taishi" effects are preserved. The patient is reminded of a sleeping man. He hears what is being said, but he does not understand what it means. But the patient feels strong effects, that is, when a needle pierces his body, he shakes his ankles, when he speaks loudly, he opens his eyes, and sometimes performs light tasks (wait for your smile, open your eyes, pull

your feet). But unable to perform slightly more complex tasks (point your nose with your left smile, lie with both feet crossed, and etc.). A number of physiological reflexes (pupil, skin and foot reflexes) are stored in the sap. Sopor may be manifested only by psychomotor disorders (for example, in acute intoxication or acute metabolic disorders).

The first-degree coma is a moderate disturbance of consciousness, in which the patient does not answer questions, does not make voluntary, purposeful actions, does not follow instructions, the response to "taishi" noises decreases sharply. On examination of the neurological status, conjunctival and corneal reflexes are called, the pupils become narrowed, their response to light is preserved or slightly reduced. Mimic reactions occur when you press an autumn apple or inject a needle into your face. Swallowing is not impaired. When a needle pierces his body, he groans and takes the ashes there. He pulls his leg out when a needle pierces his heel or Babinski is checking for symptoms. Muscle tone decreases, foot reflexes increase, skin reflexes decrease. Babinski's symptom is called, Breathing is preserved. Control of pelvic functions is impaired.

The second-degree coma is a severe disorder of consciousness in which the response to tachyarrhythmias is sharply reduced. Corneal and conjunctival reflexes and the response of the interrogator to light are sharply reduced. Depending on the etiology of the coma; miosis, anisocoria or mild mydriasis are observed. Reactions to painful effects are sharply reduced: facial expressions are slower when you press the autumn apples or pierce the face with a needle. When a needle is inserted into the body, the response decreases sharply and protective reflexes appear. Swallowing is impaired, however; if water droplets fall into the respiratory tract, a

coughing reaction occurs. Respiratory disorders: Kussmaul or Cheyne-Stokes breathing is observed. Any voluntary movements are lost, the patient lies passively as he leans to the sides. Foot reflexes and muscle tone vary depending on the etiology of the coma. Babinski and Boisha pathological reflexes appear. Skin reflexes are present. Pelvic functions are severely impaired.

The third-degree coma is a deep-level coma in which the response to all "taishi" impulses is completely lost. Corneal and conjunctival reflexes are not summoned at all, the reaction of the pupils to light is completely present. The pupils dilate to a state of mydriasis. Yutkin reflexes and mimic reactions disappear. Pay and skin reflexes are present. Muscle tone decreases sharply or complete atony is observed. In focal brain damage, symptoms change asymmetrically. AKB falls, respiratory rhythm is disturbed (bradypnoe or taxipnoe). Body temperature drops. At this stage of the coma, the patient has to be transferred to a ventilator.

The fourth-degree coma is tula areflexia, muscle atony, terminal stage of coma manifested by bilateral mydriasis and hypothermia. Because the coronary and respiratory systems are completely disrupted, they are artificially enriched. Bioelectrical activity is not detected on the EEG. It is very important to know the above clinical symptoms in the diagnosis of coma. The Glasgow scale is also widely used to assess coma levels.

Severe complications of the coma Decortication, desereption, apical syndrome, and chronic vegetative states are serious complications of coma. The post-coma period is death.

Decorative rigidity (decortication). The activity of the cortico-spinal pathways was disrupted due to bilateral damage to structures located deep in the large hemispheres of the

brain. As a result, both arms bend at the elbow joint and stick to the body, the toes bend into a fist, and both legs stretch out and become pronation. This condition is called decortic rigidity.

Deserebration rigidity (deserebration) is manifested by a sharp increase in the tone of the extensor muscles in the body, arms and legs, and a decrease in the tone of the flexor muscles.

As a result, the head is turned backwards, the limbs are stretched, and the secretory remains in a state of pronation. It is impossible to bend the legs and arms of the secretory. Deserebration rigidity develops due to damage to both sides of the cerebral hemispheres and the conductive pathways in the brainstem. The term esedeserebration is used because the spinal centers involved in the regulation of muscle tone are excluded from the influence of the cerebral centers.

Apical syndrome develops when neurons in large areas of the cerebral cortex are destroyed. Apical syndrome usually develops rapidly and its causes include severe insulitis, severe brain injuries, poisonings, and etc. Clinical signs: no voluntary movement, the patient does not speak, memory loss, emotional reactions, swallowing disorders, increased muscle tone. The patient may open and close his eyes, however; the movements of arm and legs will be limited. It responds to painful effects with the chaotic movement of the arms of the foot. Sleep and alertness are disrupted. Severe impairments in respiratory and cardiovascular function are not detected. Apical syndrome is a combination of total apraxia and agnosia.

Vegetative condition. This condition occurs due to strong morphofunctional interruptions between the large hemispheres of the brain and the structures of the brainstem. A patient in a coma for a long time (3-5 x weeks)

regains consciousness, in which only the functions of the cerebral cortex, autonomic functions, are impaired. The patient regains consciousness and faints (sopor). Tachycardia, arterial hypertension, tachypnea, hyperthermia (40-41 ° C), dystrophic changes in the body, bedsores develop rapidly. Therefore, this syndrome is more common in diffuse axonal injury. the range of speech, attention, memory, perception, and thinking is severely impaired. The vegetative state lasts from a few days to several months. The vegetative state is prognostically severe, and it resembles the symptoms of apical syndrome.

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