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Mental disorder as an example of sequelae of cerebral hypoxia-ischemia in case of acute blood loss in forensic medicine

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Abstract
Defining the full range of injuries and their causes is one of the most important issues in forensic medicine. Particularly difficult and insufficiently developed is the assessment of injuries manifested in a mental disorder. Forensic diagnostics in this case requires knowledge of the mechanism of injury, the peculiarities of the formation of injuries, and their correlation with neuropathological disorders.
Presented is the case in which a complex of processes in response to the primary injury and pathological processes attached to the injury led to structural and functional insufficiency of the brain (encephalopathy), the manifestation of which was an organic mental disorder. Its analysis contributes to the understanding of the process of decoding the information contained in the objects of expertise, assessing the elements of the cause-and-effect relationship and making scientifically based expert conclusions.

Keywords: Mental disorder, cause-and-effect relationship, forensic medicine

Introduction
Forensic assessment of injuries, the sign of which is a mental disorder, belongs to an insufficiently studied area, has a number of gaps and difficulties. Some of them are related to the very category of mental disorders subject to forensic evaluation, since they occupy a related position between neurology, psychiatry and neuropsychology. For example, the term "psychoorganic syndrome" is used in psychiatry and "encephalopathy" in neurology in relation to cerebral organic pathology that has clinically significant manifestations. The issues of etiopathogenetic specificity and dynamically differentiated systematics of these neuropsychiatric disorders have not been solved [6]. Their understanding remains ambiguous due to the lack of development of psychomorphological correlations, the correlation of exogenous and endogenous factors, and the imperfection of nosography [5]
Others in general overlap with a poorly conceptualized, non-standardized process for establishing causal relationship, especially in cases of its complex and multifactorial scenario [7]. In a number of cases, the establishment of such connections requires taking into account a variety of factors-complexes that appear in the course of the interrelated processes of damage and adaptation in response to the injury and inevitably arise during the provision of medical care for the correction of premortal pathology, the elimination of its main cause.
The main significance in this group of circumstances is given to cerebral hypoxia, which can be observed in a variety of pathological conditions: Shock, poisoning with various gases, drugs, myasthenia, persistent status epilepticus, mechanical asphyxia, various acute disorders of the circulatory system, and respiratory regulation disorders [16]. Gray matter structures are most vulnerable to hypoxic damage due to their need for oxygen and glucose to supply a large number of synapses [10].
However, it has been shown that hypoxia itself rarely causes significant brain damage, and the impairment of neuronal function will be temporary unless it is profound and long-lasting [8]. The most damaging effects of oxygen disruption occur when it is suddenly disrupted due to global cerebral hypoperfusion (Ischemia) with subsequent restoration of blood flow. This situation fully develops during cardiac arrest and subsequent resuscitation, but it also includes cases of successful treatment of a number of diseases

(Restoration of blood flow during embolectomy, surgeries in which artificial circulation is used, compartment syndrome, etc.) If ischemia lasts more than 40 minutes [12]. Brain damage with mental and neurological disorders in these cases is referred to as post hypoxic, post anoxic, post cardiac arrest, post resuscitation encephalopathy and is one of the components of the complex of pathophysiological processes that occur during cardiac arrest and subsequent reperfusion reaction and are called post cardiac arrest syndrome, repurphyseal syndrome, post-resuscitation illness [1, 9, 13, 15] Mechanisms of brain damage at the biochemical, cellular, molecular levels are widely presented in the literature and show that at the stage of ischemia synaptic failure occurs (temporary, complete or partial depolarization of neuron membranes), their apoptosis activates within 72 hours after cardiac arrest, which is accompanied by a violation of intersystem and intrasystem relationships. Restoration of perfusion can cause additional (Secondary) brain damage due to oxidative stress, inflammatory reactions and micro vascular damage, uneven reperfusion (No-reflow phenomenon the phenomenon of unrestored blood flow due to cerebral vasospasm, increased blood viscosity and platelet aggregation, maintaining focal or multifocal hypo perfusion. As a result, the total amount of damage to neurons consists of the vulnerability of a number of brain formations that mature in the postnatal period, exofocal death of neurons in less vulnerable parts, as well as delayed death of neurons in some areas of the brain. Therefore, during the period of functional recovery of the central nervous system, in the early post resuscitation period, the manifestations of post-resuscitation encephalopathy are caused by the formation of pathological functional systems characterized by non-reciprocal processes of excitation and inhibition, an increased ability of the brain to spread excitation with the unrestored ability of the cerebral cortex to generate its own bio potentials. This process is manifested by the restoration of the foot, grasp, suck pathological reflexes, and the development of paroxysmal activity (Convulsive syndromes).

Subsequently, encephalopathy is associated with the dynamics of restoration of reciprocal relationships between its formations, the volume and localization of neuronal damage [14, 17]. The pattern of mental disorders of post-resuscitation encephalopathy cited in the literature includes memory impairment, insomnia and visual deficits and neurodynamic disorders as a result of disconnection of the formations of the frontal-subcortical circle [4]. Probably, the list of manifestations of post-resuscitation encephalopathy could be clarified if there were studies in the field, but in any In this case, we are talking about the presence of an organic basis and a violation of higher mental functions that allow cognition of external reality to interact with it, which is the concept of cognitive disorders and is classified in the ICD system as a mental disorder. Obviously, any aspect of the injury can be subject to forensic evaluation, presenting difficulties in understanding and assessing causal relationships, especially if its outcome is a mental disorder.

We present a case that contains all these features and demonstrates a complex chain of events that led to the occurrence of damage, the manifestations of which are classified in the ICD as a mental disorder (neurocognitive).

Case Report

During the attempted murder of D by stab wound to the anterior surface of the chest on the left, penetrating into the pleural cavity and pericardial cavity with damage to the left ventricle of the heart and the 5th segment of the left lung, with hemopneumothorax, stab wound to the anterior surface of the abdomen on the left, penetrating into the abdominal and left pleural cavity (Thoracoabdominal) with damage to the diaphragm, jejunum, mesentery of the colon in the area of the splenic flexure, with hemoperitoneum occurred: Decompensated reversible hemorrhagic shock; post-resuscitation illness. Posthypoxic encephalopathy. Cerebral insufficiency. Post-traumatic pericarditis. Chronic heart failure.

After 3 months, an outpatient psychological-psychiatric forensic examination was prescribed, based on the results of which a conclusion was issued on the presence of a non-psychotic disorder caused by brain damage (According to ICD-10 F 06.82), associated with trauma, but not reaching the level of depriving him of the ability to correctly perceive events and facts relevant to the case and testify about them.

D. was admitted to the surgical department in a serious condition: Body temperature 36.6 C, clear consciousness, the patient's state is active, the color of the skin and visible mucous membranes is normal, there is no pastiness or edema, chest-type breathing, respiratory rate 23 per minute, no shortness of breath, chest is painful on palpation in the wound area, percussion sound tympanitis on the left, during lung auscultation the weakening of vesicular breathing, no wheezing. Heart sounds are rhythmic, heart rate 102 per minute, blood pressure 100/70 mmHg, pulse in the peripheral arteries of the feet is satisfactory. Local status: There are multiple wounds (3) of the chest on the left, abdomen, and right thigh.

Primary surgical treatment was performed, thoracotomy on the left in the 5th intercostal space. In the pleural cavity there was up to 500 ml of blood with clots; during the inspection, hemorrhage of the pericardial tissue was revealed, in the basal sections there was a wound to the pericardium, and blood in the pericardium. Pericardiotomy over the phrenic nerve. Up to 50 ml of blood in the pericardium. After opening the pericardium, jet bleeding from the heart and digital pressure were noted. The audit revealed 2 wounds of the left ventricular myocardium between the left descending coronary artery and the first diagonal artery. The myocardial wounds were sutured; after suturing, unsTable hemodynamics, ventricular fibrillation, cardiac arrest, and cessation of blood circulation were noted. Resuscitation measures with direct cardiac massage for 10 minutes were successful, blood circulation was restored. The suturing of the pericardium with rare sutures was performed, counter-opening in the basal parts of the cardial sac was completed.

Further inspection of the chest cavity revealed a perforating wound of the 5th segment of the left lung, which was sutured with 3/0 Vicryl thread continuously. 2 drains were installed in the back and front surfaces of the lungs. The wound was sutured in layers, a gauze pad was left in the posteroinferior corner of the wound. Stitches were made on the skin

Primary surgical treatment of the wound on the right thigh was performed, the wound was up to 1 cm, the canal from left to right, from bottom to top and ending in the body of the ilium on the left, no evidence of penetrating into the abdominal cavity. The wound was sanitized with hydrogen peroxide, a gauze swab with peroxide was put in place, and

the skin along the edges was sutured with separate stitches.
Further, in the operating room, a laparotomy was performed, there was up to 100 ml of blood in the abdominal cavity, a wound of the diaphragm was revealed during the inspection, at 7 cm from the ligament of Treitz there was a wound of the antimesenteric edge of the jejunum 0.5x0.1 cm, the mesentery of the splenic flexure of the colon.
The next day the ECG showed left ventricular diastolic dysfunction against the background of tachycardia. Within 3 days after arrival at the hospital, motor restlessness was noted upon awakening, and medical sedation continued. A lower tracheostomy was applied to prolong artificial ventilation, it was carried out in all parts of the lungs. In the absence of medical sedation, D. had a series of secondary generalized tonic seizures. In response to pain stimulation, epileptic seizures begin with chewing and sucking (Opercular cortex). This was followed by a turn of the head to the left with a deviation of gaze to the left and the subsequent appearance of a tonic component in the right limbs, a short breath holding. After the attack he is in postictal state, a similar series develops again against its background. Objectively: General condition is severe, level of consciousness is confusion, BP 128/70 mmHg, pulse 93 per minute, respiration ventilator through TST, saturation 100%, temperature 36, 70 °C. Neurological status: Cranial nerves: 1, 2 pairs it is difficult to determine reliably, 3, 4, 6 pairs: Pupils are rounded, equal, pupil reaction to light is lively (Photoreactions are preserved), gaze fixes briefly, does not follow the hammer. During an attack, head and eyes deviation to the left, 5th pair-trigeminal points are painless upon palpation, 7th pair-face without gross asymmetry, 8th pair no spontaneous nystagmus, 9th, 10th pairs-D. did not let examine the oral cavity and pharynx, dysphagia due to the severity of the condition, feeding through a nasogastric tube, 11 pair-symmetrical shoulders, 12 pair-tongue in the mouth, does not come out, meningeal symptoms are negative, absent. Active movements in the limbs are present, he does not follow commands, muscle tone is reduced, equal, tendon reflexes from the arms and legs are lively, to some extent more readily on the right, pathological reflexes the left foot is rotated outward, symptoms of oral automatism-sucking, dermographism-pink, higher mental functions cannot be assessed.
MSCT of the brain was performed in both hemispheres of the cerebellum (On the right up to 21x15 mm, on the left up to 27x26.5 mm), in both occipital lobes (On the right up to 1.2x41.5 mm, on the left up to 49x36.5 mm), in the parietal lobes on both sides (On the right up to 50x32.5 mm, on the left up to 30x24 mm), in the left frontal lobe up to 11.3x10.7 mm, in the basal ganglia on the left up to 12x6 mm, hypo dense regions of irregular shape with unclear contours, density up to 23-27 HU are determined.
MSCT was repeated after 10 days, the available data on the structure of the brain showed hypoxic changes in the left parietal lobe. For 19 days D. was in the Department of Anaesthesia and Intensive care in serious condition, moderately stunned. The last time motor restlessness was noted on the 13th day after admission to the hospital; in addition, fasciculation of facial muscles, divergent strabismus, proboscis reflex and other pathological reflexes, along with paresis of the upper left limb, were noted. The neurologist diagnosed «Post hypoxic encephalopathy against the background of the hemorrhagic shock (Grade I) due to a stab wound to the heart on February 30, 2022, with the formation of multiple foci of ischemia in both hemispheres of the cerebellum, in the parietal and occipital regions on the left".
MSCT of the chest on the 12th day revealed bilateral congestive changes in the dorsal parts of the lungs. Left-sided hydrofibrinothorax. Dissolved hematoma of the left chest wall. Ultrasound of the pleural cavities 12 days after admission: Hydrothorax on the left. In subsequent control MSCT revealed: Compression atelectasis of the lower part of the left lung. Left-sided hydrothorax. Cardiomegaly. Hydro pericardium. Pericardial calcification. Mediastinal lymphadenopathy. Dissolved hematoma of the left chest wall. By the time of hospital discharge, hydrothorax had almost achieved resolution, leaving pneumofibrotic changes in the left lung and moderate mediastinal lymphadenopathy. Accumulation of heterogeneous contents in the pericardium (Lysed blood?). Calcification of the pericardium.
On the 20th day, 07/19/10, the consciousness is clear, confirmed sense of self, cognitively reduced. Transferred to the surgery department.
D. was discharged from hospital 26 days after admission in satisfactory condition; there was still a decrease in short-term memory and emotional scarcity, which the doctor regarded as a manifestation of encephalopathy the discharge summary said with elements of encephalopathy.
After being discharged from hospital, D. did not seek any medical assistance, since he believes that he neither needs it nor the registration of disability. He does not work, explaining that problems with decreased vision persist ("vision was lost" now it is recovering with a slight positive dynamics). During the day he sleeps a lot, watches TV, and leaves the house only when necessary (For investigative actions).
At the time of the psychological and psychiatric inspection (3 months after the injury), he noted weakness, increased fatigue, drowsiness during the day, and insomnia such as prolonged sleep latency. He declares a desire to recover faster in order to work. The psychiatrist noted the liability of the emotional-volitional sphere, its lack of expression, monotony, exhaustion of mental activity, torpidity of thinking, decreased productivity, absent-mindedness, decreased memory for current events, complaints of decreased vision cannot distinguish small objects, sees text poorly.
The psychologist points out his slow thinking (Requires questions repetition, has difficulties with choosing words, verbalizing his thoughts, replaces them with gestures), his low educational level (His outlook is limited to and by his everyday way of life), a slowdown in the pace of mental activity and performance. When presented with psychological tests, he assimilates instructions for tasks after several repeated presentations, does not fully retain them during work, asks again and needs to clarify them. The ability to perform mental operations is preserved at the conceptual and functional levels of simple tasks; when performing operations of analysis, synthesis, comparison, exclusion, generalization, specific and situational, insignificant properties of objects are used in more complex tests. Direct mechanical learning is moderately reduced (Learning curve 4, 4, 6, 7, 7 words). Long-term low level retention (Delayed, 2 words after an hour). Completing spatial-constructive tasks (Kohs blocks) is not possible even after a clear example and direct help from the experimenter (D. places them chaotically on the Table).

Despite the lack of data on the premorbid level of functioning, but taking into account his previously performed professional activities as a driver, which required significant strain on the functions of attention and memory, it becomes obvious that they have decreased. The identified contour of the pattern of cognitive impairments fully corresponds to the predominant topical localization of the main morphofunctional disorders (MSCT data) in the parietal region, the specificity of which is given by a disorder of spatial orientation, constructional apraxia.

Discussion

The presented case makes it possible to clarify the mechanism of brain damage resulting in neurocognitive impairment. At the first stage, after the onset of bleeding and a decrease in circulating blood volume (CBV), hypo perfusion and hypoxia of the brain occurred, which resulted in loss of consciousness (Observed collapse). Further, as the volume of blood decreased and hemorrhagic shock developed along with the development of centralization of blood circulation to maintain perfusion of the heart and brain, consciousness restored (On admission, consciousness was clear, the state was active, he made complaints, reported information about what happened). There were no signs of debilitating mechanisms of compensating for blood loss (Pallor, sweating, cold extremities, increased diastolic blood pressure to 90–100 mm Hg, etc.) at the time of admission. From that time on, medical care began in accordance with the treatment protocols for hemorrhagic shock and the resulting injuries. Therefore, the short-term hypoxic effect of hypo perfusion due to a sharp decrease in blood volume could not lead to irreversible impairment of neuronal function

The second significant event was cardiac arrest during suturing of a cardiac wound. Considering the stability of hemodynamics during the operation, sufficient cardiac output, and sinus rhythm, cardiac arrest was reflexive in nature due to forced traction of the myocardium. Reperfusion is considered the main pathological stage of hypoxic-ischemic encephalopathy, which is an integral part of post-resuscitation disease

Hypoxic-ischemic encephalopathy has been reported to have either a favorable outcome with recovery following mild to moderate hypoxia of short duration or an adverse outcome following severe injury, including coma or persistent vegetative state, dementia, extrapyramidal syndrome, and cognitive deficits [11]. Some examples of successful treatment of post-resuscitation illness with complete restoration of motor, sensory, cognitive functions within a year or with minimal neurological impairment are given [2]. This case shows that in the early period hypoxic-ischemic encephalopathy resembles the dynamics of severe traumatic brain injury, and subsequently is determined by the volume and localization of diffuse focal hypoxic damage, as well as a global disruption of neurotransmitter relationships and receptor interactions, which do not allow the complete restoration of integrative brain activity and manifesting itself in cognitive and emotional disorders of a neurodynamic nature.

Also noteworthy is the insufficiency of organizing the earliest rehabilitation care of such patients. D., after discharge from the hospital, had obvious signs of not only cognitive impairment, inadequate awareness of his condition, but also other, probably neurological consequences, which required detailed research and rehabilitation treatment. Thus, there were complaints of decreased vision, but no ophthalmological examination was performed either in the hospital or after the discharge. For example, the development of cortical blindness and other types of visual impairment due to damage to the visual pathways are associated with post hypoxic encephalopathy [3].

Conclusion

Thus, the presented case demonstrates that hypoxic-ischemic brain damage can lead to transient or persistent morphofunctional failure of higher mental functions. The course of post-resuscitation ischemia-hypoxia corresponds to the data available in the literature. The identified complex of neurological, neuropsychological, mental and behavioral disorders is the basis for assessing health disorders, i.e. another aspect of the forensic assessment of damage. At the same time, the complex of processes in response to the primary injury and the pathological processes attached to the injury could not only have an independent forensic assessment, but also become components of a cause-and-effect relationship. Detailed and systematic studies are necessary to identify the distinctive and dynamic characteristics of mental disorders that are a manifestation of damage, to develop a universally applicable methodology for assessing the cause-and-effect relationship for different types of cases of varying degrees of complexity in forensic medical examination reports.

Conflict of interest

The authors have no conflict of interest related to this article.

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