Sudden death of a swimmer in water caused by heterotopic intracranial ossification and anomaly of the skull base

Iznenadna smrt plivača u vodi uzrokovana heterotopičnom intrakranijalnom osifikacijom i anomalijom kostiju baze lobanje

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Abstract

Background. Expression of immersion syndrome implies sudden and unexpected death of a swimmer in water. A drowned swimmer is still a riddle for the forensic and clinical doctors. Pathophysiological mechanisms which cause immersion syndrome may be divided into vegetatively regulating and mechanical ones. Case report. We presented heterotopic intracranial ossification with the anomaly of the skull base bones development in a young man, a swimmer, drowned after entering water and where the circumstances required expertise in forensic medicine. Conclusion. Intracranial heterotopic ossification with or without a disorder in the development of skull bones may be one of the causes of immersion syndrome.

Key words: immersion; death, sudden; adolescent; autopsy; skull; osteogenesis; congenital abnormalities.

Introduction

In the last years more and more attention has been paid to the causes of swimmers getting drowned. The intriguing of this still insufficiently studied out problem, made the researchers introduce a new expression for this type of getting drowned – immersion syndrome.

Expression of immersion syndrome implies sudden and unexpected death of a swimmer in water. In the preceding years the causality of this entity was connected with thymic-lymphatic constitution and consequential possibility of sudden death in water. Thymic-lymphatic constitution as a cause of immersion syndrome has not been scientifically confirmed to the present days and therefore swimmers getting drowned still represent a riddle for the doctors involved in forensic medicine as well as for the clinical doctors.

Pathophysiological processes leading to sudden death of swimmers in water may be vegetatively regulating and mechanical. Vegetatively regulating pathophysiological processes are based on stimulative and inhibitory mechanisms inside the autonomic nervous system.

A combined action of stimulation effects of parasympathetic and inhibition of sympathetic systems results in the descent of heart rhythm and descent of contraction power of heart muscle, as well as in the descent of peripheral resistance in blood vesels. The consequence of the described changes is the descent of blood pressure, bradycardia and decrease of the ejected quantity of blood from the heart.
Naturally, other mechanisms such as direct compression to medulla oblongata, stimulation of naso-cardio-pulmonary reflexes and some local reflexes in the nose efferential fibres, *n. vagus*, by means of V cranial nerve, lead to bradycardia, loss of consciousness or instant heart stoppage and consequential immersion syndrome.

Mechanical pathophysiological processes causing the development of immersion syndrome refer to the pathological changes which, by their presence, cause the compression, first of all, to the structure of the central nervous system and the obstruction in flowing out of the cerebrospinal fluid.

During the development of the bone tissues it may come to genetic disorders which can turn out as: exostosis, heterotopic ossifications, variations of bone structure or a combination of all these. Heterotopic ossification, other than the stated reason, may appear on the places, which are exposed to trauma or as a complication of certain pathological states 8–11.

The newly formed bone formation depending on the localization and the size gives a certain symptomatology, which is the result of compression to the surrounding structures. Intracranial localization of exostosis or heterotopic ossification in closed neurotrauma or disordered intracranial hemostasis may cause a fatal biological consequence.

In our case, by internal examination, heterotopic ossification was found inside the skull cavity and the anomalies of the skull base bones. A young male swimmer, after being exposed to the sun, jumped into water and never emerged. Those anomalies of skull bones with direct exposure to sunbeams, followed by sudden entering into water, are possible causes of immersion syndrome in the presented case.

**Case report**

A dead body of an 18-year old male swimmer who after having been exposed to the sun jumped into the water and never emerged was a subject of an autopsy.

By external examination it was found out that the skin beneath the right eye, external nose and lateral sides of the neck was by dots deprived epidermis with a visible dermis that was dark red and dry.

By internal examination of the head, when lifting and separating *tentorium cerebelli*, an incorrect, asymmetrical bone structure was noticed in the form of cover closing the rear skull cavity (Figure 1).

The bone formation was inserted between the upper edges of temporal bone pyramids and *squama* of the occipital bone. The central part of the bone structure which corresponds to the *cerebellum vermis* was fulfilled by a three-sided prismatic cavity full of blood, on which the front and the rear base could be clearly distinguished, as well as the lower, upper-left and upper-right side. Two larger and one smaller opening could be noticed on the lower part, over which the communication with the central bone cavity was established and through which the connecting veins were passing.

By the skull base examination, a significant decrease of *foramen jugulare* was found out on the fissure-like left side.

Through this opening passed the sigmoid sinus, but of a small calibre, with nervous stem (Figure 2).

![Fig. 1 – Bone structure in the form of “cover” in the part of the rear skull cavity](image1)

![Fig. 2 – Left jugular opening (tip of tweezers inserted into the opening)](image2)
On the right side foramen jugulare was wide and through it passed the sigmoid sinus of the larger calibre than the left one (Figure 3).

Occipital sinus was present on both sides, divided into the right and the left marginal sinus around the occipital opening where, macroscopically, its connection with the plexus venosus vertebralis could be noticed.

Macroscopically, the edema of brain was also noticed, with expanded and flattened convolutions, narrowed lines and narrowed lateral brain lobes.

The lungs were increased and of a grey pink colour, filled pleuropulmonary space, and with its front, round edges covered the pericardium. The traces of ribs were clearly outlined on the surface of lungs. When cutted, the lung tissue was of an average humidity and contained a sufficient quantity of foam flowing out only when pressed or rubbed with a knife.

By checking digestive organs it was found out that the submersion liquid in the stomach was mixed with sand.

By checking other internal organs, no visible pathological changes were found out.

Discussion

From the presented case it is clear that the presence of heterotopic ossification is not only important in studying antropological features, but in forensic practice, as well. The riddle for the clinical doctors is unexpected death of young and healthy people who got drowned and the explanation for such phenomenon was searched in reflexive mechanisms. Attempts to determine the cause of immersion syndrome morphologically on autopsy remained without any success to the present days. However, the various pathological states in neurocraunium, under certain conditions, may give a reply to the question: What does sudden death of a swimmer who got drowned originate from?

Per analogiam, a finding of heterotopic ossification inside the skull cavity and knowing that the person before entering into water was exposed to the sun, as well as a finding on the internal organs basically refers a what contributed to the development of immersion syndrome.

In the presented case, exposure of the head to direct sunbeams caused consecutive edema of brain, which made pressure to certain vital centers, consequential dizziness and immersion syndrome after entering into water. The stated mechanism of brain edema creation is very frequent and must not always end by getting drowned. However, the presence of heterotopic intracranial ossification which may and may not be associated with other skull bone development anomalies makes conditional upon a disorder of brain hemostasis which is developed much faster than in case of absence of any intracranial pathological formation or variation.

In the presented case, the decrease in the left foramen jugulare made conditional upon the increase in the right one and flowing out of one part of cerebrospinal fluid through the occipital sinus into the plexus venosus vertebralis internus. This way of maintaining brain hemostasis is inadequate compensatory mechanism with rapid increase in intracranial pressure. In this way a sudden loss of consciousness in water can be explained, as well as the cause of immersion syndrome.

Naturally, a crucial question for doctors facing this pathology is if it is necessary to remove intracranial heterotopic ossification by surgery in case this one shows no symptomaology. According to current doctrinarian attitudes, if heterotopic ossification does not make compression onto the surrounding structures of vital importance, it should not be “touched”. Such patients are recommended to be less exposed to direct sunbeams and to avoid sudden entering into water.

Fig. 3 – Wide expanded right jugular opening (tip of tweezers inserted into the opening)

Conclusion

In the presented case, heterotopic intracranial ossification was found with the anomalies of skull base bones in the form of a decreased foramen jugulare on the left side. Due to the direct exposure to sunbeams and sudden entering into water it came to a fatal biological consequence in the form of immersion syndrome that, most probably, was connected with intracranial heterotopic ossification and anomaly of skull bone development.

Intracranial heterotopic ossification with or without a disorder in the development of skull bones could be one of the causes of immersion syndrome.

REFERENCES


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