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ACCELERATED RIGOR MORTIS IN CENTRAL HYPERTERMIA: A CASE REPORT

ПРИСКОРЕНЕ ТРУПНЕ ЗАДУБІННЯ ПРИ ЦЕНТРАЛЬНІЙ ГІПЕРТЕРМІЇ: КЛІНІЧНИЙ ВИПАДОК

Krishtafor D. A.
Candidate of Medical Sciences, Assistant Professor at the Department of Anesthesiology, Intensive Care and Emergency Medicine, Faculty of Postgraduate Education Dnipro State Medical University Dnipro, Ukrainekraine

Кріштафор Д. А.
кандидат медичних наук, асистент кафедри анестезіології, інтенсивної терапії та медицини невідкладних станів факультету післядипломної освіти Дніпровський державний медичний університет м. Дніпро, Україна

Kravets O. V.
Doctor of Medical Sciences, Professor, Head of the Department of Anesthesiology, Intensive Care and Emergency Medicine, Faculty of Postgraduate Education Dnipro State Medical University Dnipro, Ukraine

Кравець О. В.
доктор медичних наук, професор, завідуюча кафедри анестезіології, інтенсивної терапії та медицини невідкладних станів факультету післядипломної освіти Дніпровський державний медичний університет м. Дніпро, Україна

Yekhalov V. V.
Candidate of Medical Sciences, Associate Professor at the Department of Anesthesiology, Intensive Care and Emergency Medicine, Faculty of Postgraduate Education Dnipro State Medical University Dnipro, Ukraine

Єхалов В. В.
кандидат медичних наук, доцент кафедри анестезіології, інтенсивної терапії та медицини невідкладних станів факультету післядипломної освіти Дніпровський державний медичний університет м. Дніпро, Україна

Rigor mortis is a stiffening, compaction and shortening of skeletal and smooth muscles, which occurs after death and after some time fixes the body in a certain position. It is one of the signs of biological death and usually begins developing in 1.5–3 hours, starting from the lower jaw. The full development of rigor mortis is observed in 12–24 hours after death [1]. But in rare cases, its development can be significantly accelerated.

The patient, a 34-year-old man, was hospitalized to the intensive care unit (ICU) with a diagnosis: explosive trauma, neck injury with right carotid artery

rupture, hemispheric ischemic stroke. The condition of the victim during his stay in the ICU was severe. Glasgow coma scale (GCS) 5. Mechanical ventilation, hectic fever, resistant to antipyretics.

On the 4th day of ICU stay, multiple organ failure developed. Coma, GCS 4. Mechanical ventilation via the tracheostomy tube. Hypotension, sympathomimetic support was initiated with norepinephrine in increasing doses. Hyperthermia 40.3 °C, resistant to antipyretics. External cooling, infusion of cold solutions, cold solutions via nasogastric tube. Anuria despite stimulation.

In the setting of increasing multiple organ failure, patient had a cardiac arrest on the background of mechanical ventilation. Resuscitation measures were immediately initiated. Cardiomonitor showed pulseless electrical activity. During cardiopulmonary resuscitation (CPR), the rhythm changed to pulseless ventricular tachycardia, and defibrillation was performed three times, after which the rhythm changed again to pulseless electrical activity. After 30 minutes of CPR the rhythm changed to asystole. After 20 minutes of asystole, according to the protocol [2], resuscitation efforts were ceased, biological death was stated. During the ascertainment of biological death, the presence of rigor mortis was noted, which was absent during the first 30 minutes of CPR and at the beginning of the asystolic rhythm. Moreover, rigor mortis was observed not only in the lower jaw, but also in the neck and the upper and lower extremities.

Discussion

Accelerated or instantaneous rigor mortis has been described in the literature before. Mesri M. et al (2017) describe a case of instantaneous rigor mortis in a 66-year-old woman with terminal colon cancer: rigor mortis fully developed within the first 2 minutes after circulatory arrest, during transferring to the resuscitation room [3].

J.H. Lee and K.Y. Jung (2012) describe two similar cases. First is 66-year-old man after a suicide attempt (glyphosate poisoning). He had a cardiac arrest at the emergency department (ED), and CPR was started immediately. Attempting intubation marked trismus with inability to open the mouth, after which the rigor spread to the upper extremities. Administration of succinylcholine had no effect. Due to the impossibility of orotracheal intubation, cricothyrotomy was performed to provide the airway, after which CPR was continued for 30 minutes. Return of spontaneous blood circulation (ROSC) was not achieved. The second described case is a 43-year-old woman after a suicide attempt via strangulation, who was found by her husband approximately after 10–15 minutes of self hanging, and CPR was started by emergency medical system unit. Estimated collapse time was 30 minutes. Pre-hospital basic life support time was 15 minutes, and CPR continued at the ED. Orotracheal intubation was attempted but was impossible because of trismus.

No stiffening in other muscles was observed. Cricothyrotomy was performed to provide the airway, and ROSC was attained in 12 minutes. The patient died on the second day [4].

R. Vock et al. (1984) describe a 24-year-old woman who suffered from tetanus attacks and was found dead at home in a state of rigor mortis with the hands position typical of a tetanic attack, indicating no period of muscle relaxation between the attack and the development of rigor mortis [5].

Severe hyperthermia seems to be a considerable factor of accelerated rigor mortis. T. Kojima et al. (1984) describe the full development of rigor mortis 1–2 hours after the death in a 25-year-old female methamphetamine abuser who died from overdose. Rectal temperature 3–4 hours after death was 38.4 °C [6]. J. R. Bellah et al. (1989) and K. L. Duncan et al. (1997) describe the accelerated development of rigor mortis in animals (cats and dogs) with malignant hyperthermia. In this case, in a cat (body temperature 41.4 °C) rigor mortis developed within 5 minutes after cardiac arrest [7]. The exact time of onset of rigor mortis in dogs was not specified, only that in 3 of 5 dogs who died of malignant hyperthermia, its development was accelerated [8].

The occurrence of rigor mortis is explained by a critical decrease in the level of adenosine triphosphate (ATP) in muscle tissue. After death, due to disruption of the integrity of myocytes, calcium ions are released from their cytoplasm. Calcium causes the binding of actin and myosin, which leads to muscle tension. In a living body, muscle relaxation after contraction happens due to the ATP-dependent movement of calcium ions back to the sarcoplasmic reticulum of myocytes, which breaks the actin-myosin bond. Due to the ATP depletion, the actin-myosin bond remains intact, and the muscles become stiff and rigid [9; 10].

Instantaneous rigor mortis is a rarely described phenomenon. It develops immediately after death and instantly in all muscle groups, and captures the lifelong posture of the victim. Sometimes the term is confused with cadaveric spasm, which occurs in a particular muscle group that was tense at the time of death (for example, a victim of electric shock may grasp the cable in their hand after death even after the power is turned off) [11]. Some authors, though, are skeptical about cadaveric spasm, as it is mostly described in suicide victims, in the circumstances where these findings could be explained by manipulation of the scene or specific body position [12].

Factors that accelerate the development of rigor mortis also include high body temperature, strenuous exercise before death, electric shock, convulsions, muscular dystrophy and intoxication by stimulants such as amphetamines, cocaine, aspirin, folcodine and strychnine. All of these conditions lead to depletion of muscle ATP [13; 14].

In our case, the patient had severe hyperthermia (40.3 °C), and defibrillation was performed during resuscitation, ie the body was exposed to

electric current. It is possible that these factors led to the accelerated development of rigor mortis.

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