

Accelerated rigor mortis: A case letter

Sir,

A 66-year-old female patient presented to us in Baqiyatallah Hospital with colon cancer. She had undergone surgery and multiple courses of chemotherapy. The patient was suffering from severe weight loss and orthopnea for the past 3 weeks and was admitted to the hospital for this reason. She was severely cachectic and weighted about 41 kg. No abnormality was found on physical examination. Due to rapid death of the patient, no clinical tests were performed for the patient.

The vital signs on admission were heart rate – 95 beats/min, blood pressure – 129/75 mmHg, respiratory rate – 20 breaths/min, oxygen saturation – 91%, and temperature – 37.3°C.

Since admission, the patient was put under supervision of the emergency services by supportive treatments. After 3 h of continuous care and monitoring of vital signs, the patient had a sudden cardiorespiratory arrest. The patient was immediately transferred to the resuscitation room. However, while transferring within 1 or 2 min, rigor mortis and whole-body spasm occurred so that even chest compression was not possible. Despite the standard resuscitation proceedings, the patient died. The paraclinical findings, autopsy, and laboratory and pathological studies did not reveal any finding to justify the accelerated rigor mortis. Advanced metastatic cancer was determined as cause of death of the patient.

Adenosine triphosphate (ATP) is responsible for elasticity and softness of the muscles. The amount of ATP stored in the muscles is sufficient to sustain only a few seconds of contraction.^[1] After death, ATP synthesis stops but is still consumed.^[2] In the absence of ATP and in the acidic environment, actin and myosin bind together stably and form a gel.^[3] Rigor mortis starts when the ATP amount decreases to 85% of its normal level, and when this level reaches 15%, the maximum rigor occurs [Figure 1].^[1]

It has been known from the past that after death, the body muscles start relaxing, and usually after a while, the full or partial stiffness of the muscles (rigor mortis) occurs, and with the onset of putrefaction, the muscles relax again.^[4] The time of occurrence of these events may vary but can determine the approximate time of death. Furthermore, the body position in rigor mortis shows the body position at the time of death.^[5]

The initial relaxation of muscles usually lasts for 3–6 h, depending on the environmental temperature and other factors. All muscles will be involved similarly and concurrently in rigor mortis. Because of the small muscle bulk of the head and face, rigidity first appears around the eyes, mouth, jaw, and then fingers.^[3]

Rigor mortis appears on an average within 1.5–4 hours postmortem and spreads to all body muscles within 6–12 h. The full rigor mortis remains 18–36 h and will then collapse within 24–50 h in the same order that it first appeared. Rigor mortis may rarely persist for up to 6 days. This time is so variable depending on different places of living.^[1–3]

Rigor mortis occurs both in the involuntary and voluntary muscles. The iris muscle contraction will change the pupil size.^[4] The smooth muscle of seminal vesicle bags can contract. This contraction results in seminal drainage. Left ventricular myocardium contracts and its walls become thicker and can imitate left ventricular hypertrophy. Rigor mortis is a biochemical process, and its onset and duration are affected by the ambient temperature. In areas with hot weather, the body may be completely corrupted or decomposed within 24 h, in which case the appearance of rigor will not be obvious.^[4] Cold water or weather and frost can delay the formation of body rigor and make it last longer. In the extreme cold or heat, the muscles may undergo a false rigor. High temperatures will lead to the denaturation and coagulation of proteins and rigidity of the muscles. The intensity of the heat and exposure time are important in this regard.^[1]

Unlike our case in which the only reason for the accelerated rigor may be too much weight loss, in most cases, rigor mortis due to a lack of muscle mass in elderly or very thin patients may be delayed or not formed at all. Development of rigor mortis in the newborn is very fast.

Exercise or vigorous activity, electrocution, myotonic dystrophy, convulsions, and high fever before death accelerate the formation of rigor by depletion of the muscle ATP. Intoxication with stimulants such as amphetamines, cocaine, aspirin, pholcodine, and strychnine can also reduce the ATP reserves, thus accelerating the formation of rigor.^[1–4]

Hyperthermia, cerebral hemorrhage, and infection can accelerate the rigor mortis. Drowning after so much struggle in the water can cause the full establishment of rigor within 2–3 h.^[1,2]

In some cases, rigor mortis appears within a few minutes, which is said that accelerated rigor mortis, and in rare



Figure 1: Full rigor mortis 12 h postmortem

cases, it appears immediately after death which is called the cadaveric spasm. The cadaveric spasm occurs without primary loosening. This situation can be seen in deaths that have occurred after a serious physical or emotional stress.^[1]

This phenomenon often occurs only in a group of muscles such as muscles of a limb and does not involve all the muscles in the body. Cadaveric spasm has probably a neurogenic mechanism.^[3] Cadaveric spasms are common in the war, fighting, falling from a height, and plane crashes.^[1]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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