

“Rigor Mortis” in a Live Patient

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Abstract: Rigor mortis is conventionally a postmortem change. Its occurrence suggests that death has occurred at least a few hours ago. The authors report a case of “Rigor Mortis” in a live patient after cardiac surgery. The likely factors that may have predisposed such premortem muscle stiffening in the reported patient are, intense low cardiac output status, use of unusually high dose of inotropic and vasopressor agents and likely sepsis. Such an event may be of importance while determining the time of death in individuals such as described in the report. It may also suggest requirement of careful examination of patients with muscle stiffening prior to declaration of death. This report is being published to point out the likely controversies that might arise out of muscle stiffening, which should not always be termed rigor mortis and/ or postmortem.

Key Words: rigor mortis, live patient, vasopressors, ischemia, time of death
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Rigor mortis is a postmortem change that causes stiffening of muscles, perhaps due to coagulation of muscle proteins or a shift in the muscle’s energy containers (ATP-ADP) and all the body’s muscles are affected. Rigor mortis begins within 2 to 6 hours of death, starting with the eyelids, neck, and jaw. This sequence may be due to the difference in lactic acid levels among different muscles, which corresponds to the difference in glycogen levels and to the different types of muscle fibers. The onset of rigor mortis is more rapid if the environment is cold and if the decedent had performed hard physical work just before death. Its onset also varies with the individual’s age, sex, physical condition, and muscular build. Over exertion of muscle just prior to death may also promote the onset of early rigor mortis. The authors report a postoperative patient who manifested with symptoms akin to rigor mortis while alive and subsequently died. This report is being published to encourage discussion about the potential conflicts and medicolegal issues, when such incidents occur. Occurrence of localized rigor in an elderly individual who attempted suicide due to “centripetal collapse of glycogen and ATP supply” has been reported.¹

CASE REPORT

A 35-year-old woman, weighing 42 kg, suffering from severe mitral stenosis, severe tricuspid regurgitation, severe pulmonary hypertension, and chronic atrial fibrillation was scheduled for mitral valve replacement and maze surgery. Her history was significant for repeated episodes of heart failure, requiring hospitalization. Prior to surgery, she was hospitalized and required infusion of dopamine 5 mcg/kg/min. Physical examination revealed cold clammy peripheries. The patient was orthopneic. She continued to receive dopamine infusion and multiple doses of furosemide injection. Intraoperatively, mean arterial pressure and central venous pressure were

monitored. A decision not to insert Swan Ganz catheter was taken considering the presence of severe tricuspid regurgitation. Under general anesthesia, mitral valve replacement and maze procedure with bipolar diathermy (Cardioblate Surgical Ablation System, Medtronic, 710 Medtronic Parkway, Minneapolis, MN 55432–5604) using cardiopulmonary bypass was performed. Termination of cardiopulmonary bypass could not be achieved easily; inotropic support with intravenous infusions of dopamine 10 mcg/kg/min, adrenaline 0.05 mcg/kg/min and noradrenaline 0.05 mcg/kg/min was required to sustain a mean arterial pressure of 60 mm Hg. The patient was transferred to the cardiac surgical intensive care unit for further postoperative management. Mechanical ventilation was continued with inspired oxygen concentration of 0.8, frequency of 15 breaths/min, and tidal volume. The patient had stormy postoperative period with low cardiac output syndrome, manifested by cold clammy peripheries, urine output <0.2 mL/kg/hr despite continuing the vasopressor and inotropic medications. On the postoperative day 1, her clinical condition further deteriorated. The mean arterial pressure remained below 40 mm Hg, the urine output absent in the past 4 hours. The patient was unconscious. The arterial blood gas showed worsening acidosis. A decision to start intravenous infusions of dobutamine 5 mcg/kg/min and vasopressin 6 units/h was taken to produce a possible beneficial effect by the combined use of inodilator (dobutamine) and vasoconstrictor. The mean arterial pressure increased to 60 mm Hg. Anuria and acidosis persisted despite the increase in the mean arterial pressure. Peripheral arterial pulses were found to be absent and discoloration of the skin was noted on both the feet (Fig. 1).

At this time, an increase in the airway pressure from 15 to 32 cm of H₂O was noted, the patient was “biting” on the endotracheal tube. At the author’s institute, it is not a routine practice to insert an oropharyngeal airway. In the absence of the oropharyngeal airway, it was decided to insert one, to prevent the obstruction of the endotracheal tube caused by biting of the endotracheal tube. It was not possible to open the mouth and/or insert oropharyngeal airway, because of the trismus. Unconscious patient biting the endotracheal tube is not unknown; hence, it was decided to administer a dose of neuromuscular blocking agent to facilitate mechanical ventilation. Atracurium 25 mg was intravenously administered. Despite allowing the usual onset time of about 3 minutes, the trismus persisted and the airway pressure remained high. Presuming delayed onset of action due to vasoconstriction caused by the high dose of vasopressor medications, a further 5 minutes was allowed, with no avail. The authors noted a decrease in the oxygen saturation from 95 to 85 as noted in the arterial blood gas analysis, the mean arterial pressure decreased to 40 mm Hg (probably due to air trapping). In an attempt to urgently relieve the trismus to facilitate mechanical ventilation and improving the ventilation and cardiac contraction, 50 mg of intravenous rocuronium was administered, presuming previous dose of neuromuscular blocking agent was either not delivered intravenously or a wrong drug was administered. This drug also failed to produce any muscle relaxation. Oropharyngeal airway was inserted by forcibly opening the jaw; the airway pressure immediately decreased. There was a resultant increase in mean arterial pressure. The failure of the onset of neuromuscular blockade was investigated. At this time, only the carotid, femoral, and subclavian pulses were felt. Brachial, radial, popliteal pulses could not be located by arterial

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FIGURE 1. Figure showing ischemic patches on the skin of the patient's leg.

Doppler also. At the time of performing Doppler examination of the arteries, it was noticed that the elbow and the knee joints were also stiff, mimicking rigor mortis. The joints had to be forcibly flexed to allow adequate Doppler examination of the arteries. The living status of the patient was confirmed by the presence of a palpable arterial pulse, variations in the arterial blood gas values, pupillary reaction to light, and auscultation of heart sound. The patient deteriorated over the next hour and had a cardiac arrest possibly due to low cardiac output syndrome. Satisfactory external cardiac compression could not be produced because of rigid chest wall. The patient was declared dead after 20 minutes of failed cardiopulmonary resuscitation.

DISCUSSION

The term “rigor mortis” is self explanatory—stiffening after death. The experience of the authors in the reported case suggests that “rigor” might occur in living status too. Rigor mortis manifests because of lack of blood supply to the muscles due to absence of circulation after death. Occurrence of such rigidity in the living has not been reported in the literature. Such rigidity questions the basis of the very word rigor mortis. The authors are of the opinion that the rigidity that they encountered in their patient was probably due to decrease or absence of blood supply to the muscles due to severe vasoconstriction caused by high doses of vasoconstrictors.

Moreover each of the vasoconstrictor caused its effect through various receptors, dopamine through dopaminergic receptors, nor-adrenaline through alpha receptors, and vasopressin by its direct action on the arterioles and venules. Many authors have suggested early occurrence and passing off of rigor mortis whenever the muscle glycogen stores are depleted.^{2,3} Kobayashi et al have suggested that the onset and passing off of rigor mortis in various groups of muscles depends on the glycogen and lactic acid levels.⁴ They also observed that the glycogen level at death and 1 hour after death and the lactic acid level 1 hour after death in masticatory muscles were lower than in the leg muscles. It is possible that the differences in the proportion of muscle fiber types and in glycogen level in muscles influence the postmortem change in ATP and lactic acid, which would accelerate or retard rigor mortis of the muscles.

There are 2 important ramifications to this report. First, occurrence of such rigidity is conventionally associated with the postmortem status. If experimentally our hypothesis of high doses of vasoconstrictor agent causing “cessation” of blood flow to the peripheral muscles could be proved, it might prompt a controversy that such rigidity need not always be associated with death, until proved. Unfortunately, the authors did not perform a muscle biopsy or estimation of muscle glycogen content. Second, occurrence of rigidity such as reported here, might alter the time of commencement of true rigor mortis, if the individual were to die soon after. This issue may have serious repercussions on calculation of time of death.

CONCLUSION

A case of rigor mortis in a living patient occurring possibly due to severe vasoconstriction limiting blood supply to the muscles.

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