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Published Online: 2026 March 31 **Case Report**

A Case Report of Suspected Anaphylactic Shock Following Sodium Thiopental Administration in a COVID-19 Patient Hospitalized in the Intensive Care Unit and Treated with Remdesivir

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Abstract

Introduction: Severe hypersensitivity reactions to sodium thiopental are rare but documented adverse events. During the COVID-19 pandemic, shortages of standard sedative agents prompted the increased use of thiopental as an alternative sedative for intubation in critically ill patients. However, data on the safety of sodium thiopental in COVID-19 patients receiving antiviral therapy, including remdesivir, remain limited.

Case Presentation: We report the case of a 93-year-old man admitted to the intensive care unit (ICU) for severe COVID-19, presenting with dyspnea, fever, and cough. Remdesivir therapy was initiated for the viral infection. On Day 3, progressive respiratory failure and decreased consciousness necessitated endotracheal intubation. Sodium thiopental (200 mg IV) was administered as an induction agent. Immediately after successful intubation, generalized angioedema and cardiac arrest occurred. Despite immediate cardiopulmonary resuscitation (CPR) and epinephrine administration, the patient died after 60 minutes of resuscitation efforts.

Conclusions: This case highlights the risk of suspected anaphylaxis after sodium thiopental administration in critically ill patients with COVID-19. Clinicians should exercise caution when selecting sedative agents for intubation in this population, taking into account potential drug interactions and hypersensitivity risk. Further investigation is required.

Keywords: COVID-19, Anaphylaxis, Thiopental, Remdesivir, Intensive Care Unit

1. Introduction

Severe hypersensitivity reactions are an infrequent but documented adverse outcome associated with thiopental (1). Experts estimate an incidence of approximately 1 per 30000 doses, with key predisposing factors including prior sensitization and female sex (2). Females have a threefold higher risk than males (2). Typical manifestations include urticaria, bronchospasm, vasodilation, and angioedema, all of which require standard therapeutic management. The precise mechanism remains debated, although some evidence supports an IgE-mediated immediate hypersensitivity pathway (3). Worldwide, the COVID-19 pandemic caused a sharp increase in the number of patients requiring mechanical ventilation and prolonged sedation in critical care facilities (4).

COVID-19, caused by SARS-CoV-2, provokes an exaggerated host immune response (5). In approximately 15% of cases, acute respiratory distress syndrome (ARDS) develops as a serious complication (6). ARDS in COVID-19 is associated with dysregulated immune activation (7), primarily driven by elevated proinflammatory cytokines, a phenomenon termed a

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cytokine storm (6). The relationship between COVID-19-associated immune dysregulation and potentially altered drug hypersensitivity responses warrants further study.

Global health systems faced immense pressure during the COVID-19 pandemic, resulting in acute shortages of standard sedative agents such as propofol and midazolam (8). Clinicians were therefore prompted to consider alternative options for sedation. Limited evidence supported the use of sodium thiopental as a substitute: Jean-Michel V and colleagues (8) suggested that thiopental was suitable for prolonged sedation in mechanically ventilated COVID-19 patients when standard agents were unavailable. Additionally, Edalatkhah et al. (9) reported comparable mortality outcomes between sodium thiopental and midazolam in intubated COVID-19 patients.

Severe hypersensitivity reactions to thiopental are rare but recognized; however, their clinical behavior in the context of COVID-19-associated immune dysregulation and concurrent novel antiviral agents such as remdesivir remains incompletely characterized. This report documents a case of suspected anaphylactic shock after sodium thiopental administration in a COVID-19 patient receiving remdesivir, with the aim of alerting clinicians to potential risks and encouraging further investigation.

2. Case Presentation

The Ethics Committee of Qazvin University of Medical Sciences approved this report (code: IR.QUMS.REC1404.365).

A 93-year-old man presented with progressive dyspnea, fever, and cough. Reduced oxygen saturation on admission prompted hospitalization for bilateral COVID-19 pneumonia with pleural effusion. Sepsis was considered as a contributing diagnosis but was not confirmed. He denied confusion, myalgia, headache, nausea, chest pain, and abdominal symptoms. His medical history included hypertension, coronary artery disease, and chronic obstructive pulmonary disease. On arrival, he was conscious and oriented. He was tachypneic, with dyspnea at rest. Oxygen saturation on room air was 82%, improving to 93% with supplemental oxygen. His temperature was 38.8°C, and blood pressure was 125/70 mmHg. Cardiac auscultation was unremarkable. Pulmonary examination revealed reduced breath sounds bilaterally at the bases, and bilateral lower-extremity edema was present. No cyanosis was noted. Laboratory results from admission and during hospitalization are presented in Table 1. Computed tomography (CT) of the chest demonstrated bilateral ground-glass opacities consistent with viral pneumonitis and moderate right-sided and small left-sided pleural effusions. Supplemental oxygen was delivered at 50% via a Venturi mask.

On Day 2, intravenous (IV) remdesivir (200 mg) was initiated, replacing oral favipiravir. Despite high-flow oxygen therapy, oxygen saturation continued to decline. Noninvasive mechanical ventilation was initiated, with concomitant fentanyl infusion for comfort. Echocardiography demonstrated right ventricular dysfunction, with an ejection fraction of 40%.

On Day 3, severe respiratory distress and declining consciousness prompted endotracheal intubation. Sodium thiopental (200 mg IV) and fentanyl (50 mcg IV) were administered as induction agents before laryngoscopy at approximately 08:15. Endotracheal intubation was performed successfully and confirmed by bilateral auscultation and end-tidal CO2 colorimetric detection. Within approximately 2 to 3 minutes of thiopental administration, immediately after intubation, generalized angioedema involving the face and neck was observed (Table 2).

Cardiac arrest occurred within approximately 5 minutes of thiopental administration. The initial rhythm was pulseless electrical activity (PEA), identified on continuous cardiac monitoring. Blood pressure was unrecordable, consistent with distributive/obstructive shock physiology. SpO_2 monitoring became unreliable because of peripheral vasoconstriction.

Advanced cardiopulmonary resuscitation was initiated immediately. Epinephrine 1 mg IV was administered at 3- to 5-minute intervals throughout the 60-minute resuscitation, with approximately 10 to 12 doses and an estimated total IV epinephrine dose of 10 to 12 mg. An intravenous normal saline bolus was administered for volume resuscitation. Salbutamol nebulization was administered for bronchospasm. The patient had been receiving methylprednisolone 200 mg IV every 6 hours as part of COVID-19 treatment throughout admission, and this was continued. Following the persistent absence of a palpable pulse despite IV epinephrine, concern arose regarding inadequate IV drug absorption because of distributive shock. Endotracheal epinephrine at double dose (2 mg) was administered via the endotracheal tube.

Despite these interventions, no palpable pulse was restored. Angioedema progressively extended from the face and neck to the upper extremities and trunk. Point-of-care cardiac ultrasound, performed by the attending cardiologist, confirmed the absence of cardiac mechanical activity. After 60 minutes of continuous resuscitation without return of spontaneous

Table 1. Laboratory Results on Admission and During Hospitalization

Laboratory Test	Reference Range	Day 1	Day 2	Day 3
Sodium (mmol/L)	136 - 145	138	141	135
Potassium (mmol/L)	3.6 - 5.1	4.7	4.5	3.5
Blood urea nitrogen (mg/dL)	6 - 24	126	190	185
ESR (mm/h)	< 15	15	17	17
White blood cell count (K/muL)	3.8 - 10.8	3.6	5.5	10.6
Hemoglobin (g/dL)	14 - 18	9.8	12.1	12.6
Platelet count (K/muL)	150 - 450	81	88	108
Serum creatinine (mg/dL)	0.7 - 1.3	3.9	3.2	2.8
ABG pH	7.35 - 7.45	7.30	7.28	7.14
ABG pCO_2 (mmHg)	35 - 45	42	48	55
ABG HCO_3- (mmol/L)	20 - 26	19.9	19.0	18.1
Lactate dehydrogenase (U/L)	140 - 280	643	621	634
COVID-19 PCR	Negative	Positive	-	-

Table 2. Patient Medications During Admission

Day Number	Patient's Medications
First	Methylprednisolone 200 mg /6h IV; Meropenem 500 mg IV; expectorant syrup 5cc /TDS Oral; Paracetamol 1 gr IV Before interferon injection; Favipiravir 600mg/BD Oral; Lactulose 10 g 30 CC stat syrup; Paracetamol 1 gr IV Before interferon injection; Vitamin B12 Oral; Glycerol 2 g Rectal; Interferon Beta 0.3 mg injection; Aspirin 80 mg Oral; Fentanyl 50 mcg / h drip IV; Famotidine 20 mg Oral; Methylprednisolone 200 mg /6h IV; Sodium Thiopental 200mg IV stat; Vitamin C 500 mg BD IV; Favipiravir 600mg/BD Oral; Fentanyl 50mcg IV stat; Budesonide 0.5 mg/QID nebulized; Vitamin C 500 mg /BD IV; epinephrine 1 mg every 3-5 minutes; Salbutamol /QID nebulized; Paracetamol 1 gr IV Before interferon injection; Meropenem 500 mg IV; Syrup: Diphenhydramine 20 ml Oral Before interferon injection; Heparin 2500 unit/TDS IV; Interferon Beta 0.3 mg injection
Second	Remdesivir 200 mg IV; Methylprednisolone 200 mg /6h IV; Heparin 2500 unit/TDS IV; Magnesium sulfate 200 mg nebulized
Third	Furosemide 40 mg/24h IV; Remdesivir 200 mg IV; Methylprednisolone 200 mg /6h IV; Ferrous sulfate 325 mg and folic acid 800 mcg Oral; Methylprednisolone 200 mg /6h IV; Meropenem 500 mg IV; Paracetamol 1 gr IV Before interferon injection; Favipiravir 600mg/BD Oral; Lactulose 10 g 30 CC stat syrup; Paracetamol 1 gr IV Before interferon injection; Vitamin B12 Oral; Glycerol 2 g Rectal; Interferon Beta 0.3 mg injection; Aspirin 80 mg Oral; Fentanyl 50 mcg / h drip IV; Famotidine 20 mg Oral; Methylprednisolone 200 mg /6h IV; Sodium Thiopental 200mg IV stat; Vitamin C 500 mg /BD IV; epinephrine 1 mg every 3-5 minutes; Salbutamol /QID nebules; Salbutamol /QID nebulized

circulation, resuscitation was terminated and death was pronounced. Generalized angioedema persisted throughout resuscitation and at the time of death.

Serum tryptase measurement was not obtained. The acute, rapidly fatal nature of the event precluded timely sample collection. Postmortem tryptase measurement was not pursued because of institutional constraints. This represents a significant limitation to diagnostic certainty in this case.

A structured chronological timeline of clinical events from admission to death is presented in Table 3.

3. Discussion

3.1. Observed Clinical Sequence

This case documents the clinical course of a 93-year-old male with severe COVID-19 who developed generalized angioedema and cardiac arrest within minutes of receiving sodium thiopental as a preintubation induction agent. Resuscitation efforts over 60 minutes were unsuccessful. The temporal association between thiopental administration and reaction onset is the primary basis for the clinical diagnosis.

3.2. Differential Diagnosis and Causality Assessment

Although the clinical presentation is consistent with anaphylactic shock, definitive diagnosis was limited by the absence of confirmatory allergy testing, including serum tryptase, histamine, complement studies, or postmortem evaluation. The following alternative and contributing explanations were considered: 1) pre-existing right ventricular dysfunction (ejection fraction, 40%) provided a substrate for cardiovascular decompensation under any hemodynamic stress; 2) severe hypoxemia on Day 3 independently contributed to cardiovascular instability at the time of intubation; 3) fentanyl is a recognized, although less common, trigger of nonimmunological mast cell degranulation;

Table 3. Structured Clinical Timeline from Admission to Death

Days	Time	Respiratory Status	Key Medications	Clinical Events
1	Admission	SpO_2 82% on room air; 93% with O2	Methylprednisolone, meropenem, favipiravir, fentanyl, interferon	Admitted for COVID-19 pneumonia; fluid bilateral pleural effusion; dobutamine initiated for hypotension
2	Morning	SpO_2 declining despite high-flow O2	Remdesivir initiated (200 mg IV); NIMV initiated	Non-invasive mechanical ventilation started; right ventricular dysfunction (EF 40%) on echocardiogram
3	~08:00	Severe respiratory distress; declining consciousness	Remdesivir continued; methylprednisolone	Decision made to proceed with endotracheal intubation
3	~08:15	Pre-intubation	Sodium thiopental 200 mg IV + fentanyl 50 mcg IV (induction)	Induction agents administered before laryngoscopy
3	~08:17	Intubated; SpO_2 unreliable	-	Endotracheal intubation completed successfully (confirmed by auscultation and end-tidal CO2); immediate onset of facial and neck angioedema within 2 - 3 minutes of thiopental administration
3	~08:20	Cardiac arrest	Epinephrine 1 mg IV q3 - 5 min	Pulseless electrical activity (PEA); ACLS initiated; IV normal saline bolus; salbutamol nebulization
3	~08:25	No pulse	Epinephrine 2 mg via ETT	Angioedema extending to upper extremities and trunk; IV epinephrine without response; endotracheal epinephrine attempted
3	~09:15 - 09:20	No cardiac activity	-	60 minutes of ACLS; point-of-care ultrasound confirmed absent cardiac mechanical activity; resuscitation terminated; death pronounced

however, the patient had been receiving a continuous fentanyl infusion before the event without an apparent reaction; 4) airway manipulation may cause vasovagal responses; however, this would not account for generalized, progressive angioedema; and 5) COVID-19-associated endothelial dysfunction and cytokine-mediated vascular permeability may have modulated the severity of the reaction.

Sodium thiopental is considered the most probable primary trigger for the following reasons: 1) the immediate temporal association between the thiopental bolus and angioedema onset (2 to 3 minutes); 2) the classical pattern of IgE-mediated anaphylaxis, with generalized angioedema rapidly progressing to cardiovascular collapse; 3) remdesivir had been administered for 24 hours preceding the event without any signs of immediate hypersensitivity; and 4) no prior reactions to fentanyl were observed despite its ongoing use. However, without confirmatory immunological testing, definitive attribution to thiopental cannot be established, and contributions from other agents or COVID-19-related physiology cannot be excluded.

3.3. Thiopental in COVID-19 Critical Care

Although uncommon, hypersensitivity reactions to sodium thiopental pose significant challenges in critically ill patients. Jean-Michel V and colleagues (8) published a retrospective analysis suggesting sodium thiopental as an alternative sedative agent for mechanically ventilated COVID-19 patients during periods of standard drug shortage. Their study concluded that thiopental provided adequate sedation with acceptable recovery profiles after cessation. Similarly, Edalatkhah et al. (9) reported comparable mortality outcomes between midazolam and sodium thiopental in intubated COVID-19 patients, positioning thiopental as a practical alternative during shortage-driven decisions. The selection of 200 mg sodium thiopental in this case was consistent with these published recommendations under conditions of drug shortage and an urgent need for intubation.

3.4. Hypotheses and Mechanistic Considerations

The following hypotheses cannot be confirmed on the basis of this single case report. Whether COVID-19-associated immune dysregulation, including cytokine storm, altered the threshold or severity of hypersensitivity reactions to thiopental in this patient is unknown. It has been hypothesized that cytokine-mediated upregulation of mast cell or basophil reactivity could lower the threshold for drug-induced degranulation; however, this mechanism has not been established in the clinical literature for this population (6, 7).

Regarding a possible role for remdesivir, remdesivir has been associated with rare cases of anaphylaxis, as reported by Fukushima et al. (10). In the present case, the patient had received remdesivir for 24 hours before the event without apparent hypersensitivity manifestations. Although this argues against remdesivir as the primary acute trigger, the possibility that prior remdesivir exposure contributed to immune sensitization cannot be excluded or confirmed without specific IgE testing or skin prick testing. This represents

an important knowledge gap requiring further study (10).

3.5. Management Challenges

The resuscitation course in this patient illustrates the challenges of managing anaphylaxis in the context of critical illness. Despite immediate cardiopulmonary resuscitation and epinephrine administration, persistent generalized angioedema and refractory cardiac arrest were observed. The lack of response to IV epinephrine may reflect circulatory failure impairing drug delivery, in addition to the patient's underlying right ventricular dysfunction and COVID-19-associated endothelial injury. These findings underscore the need for preparedness protocols for managing peri-intubation anaphylaxis in this population.

3.6. Conclusions

This case report documents a fatal suspected anaphylactic reaction occurring in temporal association with sodium thiopental administration in a critically ill COVID-19 patient receiving remdesivir. The following conclusions are limited to what can be inferred from a single case report without confirmatory allergy testing: 1) a fatal allergic-type reaction occurred with a temporal association consistent with thiopental-induced anaphylaxis, although definitive attribution cannot be established without tryptase or allergy testing; 2) clinicians should exercise caution when selecting sedative agents for endotracheal intubation in critically ill COVID-19 patients, given the potential for altered immune reactivity and drug interactions that are not yet fully characterized; 3) anaphylaxis management protocols should be immediately available during peri-intubation sedation in this high-risk population; and 4) prospective data and immunological follow-up studies in surviving cases of peri-intubation reactions in COVID-19 patients are needed to characterize the mechanisms and risk factors for drug hypersensitivity in this population.

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Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

Authors' Contribution: Study concept and design: L. Y. and H. Z. conceived the study idea and designed the case report. Acquisition of data: H. Z., H. K., and A. N. collected the patient's clinical data from hospital records and the medical team. Analysis and interpretation of data: H. K. and A. N. analyzed and interpreted the clinical findings; H. Z. and M. S. analyzed the nursing monitoring data and patient outcomes; L. Y. supervised the integration of the nursing and medical perspectives. Drafting of the manuscript: H. Z. drafted the abstract, introduction, case presentation, and conclusion; H. K. prepared the discussion on anesthesia challenges and drug interactions; A. N. drafted the emergency management and resuscitation sections; M. S. prepared the tables and acknowledgments. Critical revision of the manuscript for important intellectual content: L. Y., H. Z., H. K., A. N., and M. S. critically revised the manuscript. L. Y. reviewed the nursing and ethical aspects; H. K. reviewed the anesthesiology content; A. N. reviewed the emergency care content; H. Z. and M. S. reviewed the overall coherence and compliance with ICMJE criteria. Statistical analysis: Not applicable; A. N. reviewed the descriptive summaries of laboratory findings and vital signs. Administrative, technical, and material support: H. Z. managed ethics approval and project administration; M. S. provided technical support in data organization and literature retrieval. Study supervision: L. Y. supervised the study; H. Z. supervised manuscript preparation and submission; H. K. and A. N. supervised the clinical and procedural aspects.

Conflict of Interests Statement: The authors do not declare any conflicts of interests for this study.

Data Availability: The data supporting the findings of this case report are included within the manuscript and its supplementary materials. Due to privacy and ethical considerations regarding patient confidentiality, the raw and detailed medical records (including imaging studies and laboratory reports) are not publicly available. They may, however, be made available from the corresponding author upon reasonable request and with permission from the relevant ethics committee.

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