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*RESEARCH ARTICLE*

# A FATAL PERIOPERATIVE STORM: SYNERGISTIC STATIN–HALOGENATED MYOTOXICITY WITH REFRACTORY HYPERKALAEMIA AND ACUTE KIDNEY INJURY — A CASE REPORT AND PROPOSED CLINICAL REASONING FRAMEWORK

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## *Abstract*

**Background:** Statins and halogenated volatile anaesthetics are independently myotoxic, but their concurrent perioperative interaction remains poorly characterised.Current guidelines recommend perioperative statin continuation in coronary patients, yet the resulting pharmacological intersection with inhalational anaesthesia has not been integrated into routine risk stratification.

**Case Presentation:** A 66-year-old woman receiving long-term high-intensity atorvastatin (40 mg/day), with poorly controlled type 2 diabetes (HbA1c 9.0%), recent inferior ST-elevation myocardial infarction with drug-eluting stent, and ischaemic cardiomyopathy (LVEF 35%), underwent uneventful 180-minute open reduction–internal fixation of an AO/OTA 13-C2 distal humeral fracture under sevoflurane in left lateral decubitus. Twenty two hours postoperatively, after an asymptomatic interval, she abruptly developed circulatory collapse, diffuse myalgia, anuria and obtundation. Investigations revealed massive rhabdomyolysis (creatine phosphokinase 31,010 IU/L), severe hyperkalaemia (8.0 mmol/L), KDIGO stage 3 acute kidney injury, severe metabolic acidosis (pH 7.20), and high-sensitivity troponin I 13,000 pg/mL with unchanged left-ventricular function. Despite intensive multimodal therapy and emergent intermittent haemodialysis, rebound hyperkalaemia and refractory cardiac arrest occurred 35 hours after surgery.

**Conclusion:** This case integrates six convergent injurious pathways — chronic high-intensity statin therapy, halogenated anaesthetic exposure, uncontrolled diabetes, advanced ischaemic cardiomyopathy, prolonged lateral decubitus and hyperkalaemia-promoting polypharmacy — to explain a fulminant fatal outcome. We propose a structured perioperative reasoning framework (the STAT OP concept) and argue for individualised consideration of total intravenous anaesthesia and continuous high cut-off renal replacement therapy in highest-risk statin-treated patients.

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## Introduction:-

Rhabdomyolysis is a syndromic disintegration of skeletal muscle that releases intracellular constituents into the systemic circulation, with creatine phosphokinase (CPK) values exceeding five times the upper limit of normal as its diagnostic biological cornerstone, and acute kidney injury (AKI), life-threatening hyperkalaemia, severe metabolic acidosis and cardiac arrest as its principal lethal complications.[1] In the perioperative setting, two pharmacological classes deserve particular attention. Statins, the cornerstone of secondary cardiovascular prevention, exert dose-dependent myotoxicity through inhibition of HMG-CoA reductase, depletion of mevalonate-derived isoprenoids that anchor small GTPases (Rab, Rho), reduction of mitochondrial coenzyme Q10 and consecutive impairment of cellular bioenergetics and sarcolemmal integrity.[2,3] Halogenated volatile anaesthetics — sevoflurane, isoflurane, desflurane — activate the type 1 ryanodine receptor (RyR1) on the sarcoplasmic reticulum, producing cytosolic $Ca^{2+}$ overload that, in genetically predisposed patients, may manifest as classical malignant hyperthermia (MH) or as atypical, subclinical, anaesthetic-induced rhabdomyolysis (AIR) without overt hyperthermia or rigidity.[4,5]

A 2021 meta-analysis of 14 studies confirmed that the SLCO1B1 c.521T>C (rs4149056) polymorphism, which reduces hepatic OATP1B1-mediated atorvastatin uptake, is robustly associated with statin-induced myopathy (pooled OR 2.9 [95% CI 1.59–5.34] for CC homozygotes; OR 4.0 [1.23–12.63] for atorvastatin users with CC genotype).[6] Concurrently, RYR1 and CACNA1S variants underlie the entire continuum from clinical MH to delayed AIR.[5,7] A perioperative pharmacological intersection between these two pathways is therefore biologically inevitable in a substantial subset of statin-treated surgical patients, but remains unaccounted for in current risk-stratification practice. The 2024 AHA/ACC perioperative guideline for noncardiac surgery recommends continuation of established statin therapy in coronary patients,[8] consistent with meta-analytic evidence of reduced myocardial infarction (OR 0.44 [0.30–0.64]) and 30-day mortality with perioperative statin continuation.[9] Yet a recent case of severe rhabdomyolysis in a statin user undergoing parathyroidectomy under general anaesthesia explicitly raised the hypothesis of a synergistic statin–anaesthetic toxicity.[10] We report a fatal postoperative rhabdomyolysis in which the convergence of chronic high-intensity statin therapy, sevoflurane-based anaesthesia and a heavy comorbidity load produced a fulminant, refractory and lethal trajectory. Beyond the descriptive interest of the case, we propose an integrative pathophysiological model and a structured perioperative reasoning framework (the **STAT-OP** concept — Statin–Anaesthetic Toxicity in the Operative Period) intended to support individualised risk stratification in routine practice.

**Case Report:-**

A 66-year-old woman (60 kg, 162 cm, body mass index 22.9 $kg/m^2$) was admitted following a domestic fall with a closed comminuted distal humeral palette fracture (AO/OTA 13-C2). Her medical history included a 15-year type 2 diabetes mellitus with poor control (glycated haemoglobin 9.0%), persistent moderate asthma, and a recent inferior ST-elevation myocardial infarction (two months earlier) treated by drug-eluting stent in the right coronary artery, complicated by ischaemic dilated cardiomyopathy (left ventricular ejection fraction [LVEF] 35%; hypokinesia of the inferior, basal-to-mid lateral and basal septal segments). Chronic medications comprised atorvastatin 40 mg/day, dual antiplatelet therapy (clopidogrel 75 mg, aspirin 75 mg), sacubitril/valsartan 49/51 mg twice daily, spironolactone 25 mg/day, furosemide 40 mg/day, bisoprolol 2.5 mg/day, empagliflozin 10 mg/day, and inhaled bronchodilators. Preanaesthetic evaluation classified her as American Society of Anesthesiologists (ASA) physical status III. Baseline laboratory values were within normal limits, including serum creatinine (56.3 µmol/L), potassium (4.3 mmol/L), and complete blood count. According to current consensus, clopidogrel was withheld for 5 days and empagliflozin for 3 days; atorvastatin and other cardiovascular drugs were continued in line with the 2024 AHA/ACC perioperative guideline.[8] Surgery was performed on the fifth day after admission.

After standard monitoring, anaesthesia was induced with intravenous fentanyl, propofol, lidocaine and rocuronium, and maintained with sevoflurane (end-tidal concentration 1.0–1.4%) in left lateral decubitus for 180 minutes. Open reduction and internal fixation by dual anatomic plating with ulnar neurolysis was performed. The intraoperative course was uneventful: stable haemodynamics, normothermia (36.4–36.8 °C), normocapnia (end-tidal $CO^2$ 32–36 mmHg), no muscular rigidity, no metabolic acidosis on intraoperative blood gas, glycaemia 1.4–1.8 g/L. Extubation was uneventful and the patient was transferred to the orthopaedic ward in stable condition. Twenty-two hours after surgery, the patient developed acute hypotension (64/32 mmHg), oligo-anuria, polypnoea, diffuse myalgia and obtundation. She was urgently transferred to the intensive care unit on norepinephrine support. Investigations revealed massive rhabdomyolysis (CPK 31,010 IU/L; lactate dehydrogenase [LDH] 1,995 IU/L), severe hyperkalaemia (potassium 8.0 mmol/L), KDIGO stage 3 AKI (creatinine 130 µmol/L), severe metabolic acidosis (pH 7.20; bicarbonate 12 mmol/L), severe hyperglycaemia (5.0 g/L), and high-sensitivity troponin I 13,000 pg/mL.Bedside transthoracic echocardiography showed unchanged LVEF (35%) without new regional wall motion abnormalities. Twelve-lead electrocardiography demonstrated peaked T waves typical of hyperkalaemia, without new ischaemic changes or stent-territory ST-segment elevation.

The differential diagnosis of acute coronary stent thrombosis was considered but judged unlikely given the absence of characteristic angina, the lack of new ischaemic ECG changes beyond hyperkalaemia signatures, the unchanged echocardiographic pattern, and the recognition that troponin elevation in massive rhabdomyolysis is non-specific (myocardial injury secondary to circulatory collapse, hyperkalaemia and acidosis — type 2 MINS rather than type 1 acute myocardial infarction).[11] After multidisciplinary discussion (anaesthesia, intensive care, cardiology, nephrology), management focused on the dominant rhabdomyolysis–AKI–hyperkalaemia syndrome while reinstating dual antiplatelet therapy. Treatment included intravenous calcium gluconate, glucose-insulin, isotonic crystalloid resuscitation calibrated to a urine output target of ≥ 1 mL/kg/h, vasopressors, and emergent intermittent haemodialysis with ultrafiltration. Sodium bicarbonate and mannitol were not used as standard, in line with current evidence.[12,13] After two hours of dialysis, partial correction was achieved (potassium 4.1 mmol/L; creatinine 90 µmol/L; CPK 27,710 IU/L). Three hours after dialysis cessation, the patient deteriorated with extreme bradycardia (32 bpm) and rebound hyperkalaemia (6.5 mmol/L), followed by refractory cardiac arrest 35 hours after the index procedure. Resuscitation was unsuccessful. The chronological sequence is summarised in Figure 1, and the proposed integrative pathophysiological model in Figure 2. Written informed consent for publication was obtained from the patient's next of kin.

## Discussion:-

This observation describes a fulminant fatal postoperative rhabdomyolysis whose temporal pattern — uneventful intraoperative course, asymptomatic 22-hour interval, abrupt biological storm, rebound hyperkalaemia despite renal replacement therapy and refractory arrest — is not explained by any single established mechanism. We argue that it represents a paradigmatic instance of synergistic statin–halogenated myotoxicity amplified by convergent comorbid stressors, and propose that this perioperative phenotype deserves explicit recognition.

**Mechanistic synthesis:-**

Statins inhibit HMG-CoA reductase, depleting mevalonate-derived isoprenoids (geranylgeranyl-PP, farnesyl-PP) required for prenylation of small GTPases (Rab, Rho, Rac), causing impaired vesicular trafficking, sarcolemmaldestabilisation, and CoQ10-dependent mitochondrial dysfunction with reduced ATP availability and increased oxidative stress.[2,3] At the genetic level, the SLCO1B1 c.521T>C polymorphism reduces hepatic OATP1B1-mediated statin uptake, increasing systemic exposure and myopathy risk roughly 3- to 4-fold for atorvastatin users carrying the CC genotype.[6] All halogenated agents activate skeletal-muscle RyR1, producing pathological sarcoplasmic $Ca^{2+}$ leak; in RYR1/CACNA1S variant carriers (1:200–1:2,000 prevalence), this leak may exceed cellular buffering capacity, manifesting as classical MH or, increasingly, as delayed atypical anaesthetic-induced rhabdomyolysis without hyperthermia or rigidity — a phenotype consistent with our patient's presentation.[4,5,7,14] The mechanistic intersection is biologically transparent: a statin-treated myocyte operates with diminished energetic reserves, weakened membrane integrity and impaired $Ca^{2+}$ handling, so that a superimposed RyR1-mediated $Ca^{2+}$ overload may overwhelm an already isoprenoid-depleted, CoQ10-deficient mitochondrial network and precipitate irreversible necrosis. A 2025 case of severe rhabdomyolysis in a statin user undergoing parathyroidectomy under general anaesthesia advanced the same hypothesis.[10]

In our patient, four additional cofactors converged: uncontrolled diabetes (hyperosmolarity, microvascular dysfunction, electrolyte and phosphate disturbance); severe ischaemic cardiomyopathy (low cardiac output reducing skeletal-muscle perfusion and metabolic reserve); prolonged left lateral decubitus (~3 h, generating direct mechanical compression and ischaemia–reperfusion injury of dependent muscle masses); and polypharmacy, with sacubitril/valsartan and spironolactone amplifying hyperkalaemia risk and surgical stress potentially impairing hepatic CYP3A4 clearance of atorvastatin. The marked troponin elevation was attributed to type 2 myocardial injury secondary to circulatory collapse, hyperkalaemia and acidosis, rather than to a primary type 1 ischaemic event. The complete six-pathway model is presented in Figure 2.

**Lessons for management:-**

Three management lessons emerge. First, postoperative myalgia was masked by surgical pain until frank circulatory deterioration; in highest-risk patients, systematic biochemical surveillance (CPK, potassium, creatinine, urine output, serial ECG) during the first 24 hours may allow earlier detection. Second, regarding renal replacement, intermittent haemodialysis with standard high-flux membranes has limited capacity to clear myoglobin (17.8 kDa). Heyne et al. demonstrated up to 20-fold higher myoglobin clearances using high cut-off (HCO) membranes (median clearance 44.2 vs 3.7 mL/min for conventional HD).[15] The 2025 ReplaceRhabdo pilot randomised trial confirmed that continuous veno-venous haemodialysis with HCO dialyzers and haemoadsorption-augmented strategies offer superior myoglobin elimination versus conventional CVVH.[16] These findings support a low threshold for continuous high cut-off RRT in massive ongoing rhabdomyolysis. Third, regarding adjunctive therapies, the EAST (Eastern Association for the Surgery of Trauma) guideline conditionally recommends against bicarbonate or mannitol, with isotonic crystalloid resuscitation as the cornerstone.[12,13]

**Proposed STAT-OP framework:-**

We propose that statin-treated patients undergoing inhalational anaesthesia warrant a structured perioperative approach, the STAT-OP framework (Statin–Anaesthetic Toxicity in the Operative Period), articulated across four phases.

**Preoperative:-**

Identify the high-risk profile: high-intensity statin (atorvastatin ≥ 40 mg, rosuvastatin ≥ 20 mg), uncontrolled diabetes (HbA1c ≥ 8%), advanced heart failure (LVEF ≤ 40%), chronic kidney disease (eGFR ≤ 60 mL/min/1.73 $m^2$), polypharmacy with CYP3A4 modulators, and surgery requiring prolonged dependent positioning. Continue statin per cardiovascular guidelines but document the risk in the anaesthetic record.[8]Optimiseglycaemic control. Withhold clopidogrel 5 days and SGLT2 inhibitors 3 days. Measure baseline CPK, creatinine, electrolytes. Where pharmacogenomic testing is available, SLCO1B1 c.521T>C and RYR1/CACNA1S genotyping may further refine risk in patients with previous unexplained myalgia or family history.[6,7]

**Intraoperative:-**

In the highest-risk subset, prefer total intravenous anaesthesia (TIVA) with propofol-based maintenance over halogenated agents. TIVA avoids RyR1 activation entirely and has been associated with reduced inflammatory and oxidative-stress responses in observational and randomised work.[17,19] Avoid succinylcholine. Apply strict positioning protocols with pressure-distribution devices. Maintain normothermia, normocapnia and normovolaemia; monitor temperature, $ETCO_2$, arterial pressure and neuromuscular blockade.

**Early postoperative (0–24 h):-**

Active inquiry for myalgia, weakness and dark urine. Serial CPK at H+6, H+12 and H+24 in selected patients. Continuous ECG and electrolyte surveillance. Maintain isotonic crystalloid infusion with urine output ≥ 1 mL/kg/h.

**Established rhabdomyolysis:-**

Discontinue statin. Aggressive isotonic crystalloid resuscitation calibrated to a urine-output target of ≥ 1 mL/kg/h, while avoiding both under-resuscitation and excessive volumes that may precipitate fluid overload.[12,18,20] Treat hyperkalaemia urgently. Avoid bicarbonate and mannitol unless individualised indication exists.[12,13] Low threshold for ICU transfer and continuous high cut-off renal replacement therapy when ongoing necrosis is suspected, with consideration of haemoadsorption-augmented strategies in selected refractory cases.[15,16,18]

**Strengths and limitations:-**

The principal limitation is the single-patient nature of the observation: causality cannot be quantitatively apportioned among the convergent mechanisms. Muscle biopsy, in vitro contracture testing, RYR1/CACNA1S and SLCO1B1 genotyping, atorvastatin plasma levels and autopsy were not available. The proposed synergy and the STAT-OP framework therefore remain hypothesis-generating, and confirmation requires translational studies, pharmacovigilance signal analyses and ideally a prospective registry of perioperative rhabdomyolysis in statin users. The principal strength is the integration of mechanistic, genetic, and management dimensions into a coherent model anchored in 2020–2025 high-quality evidence.

## Conclusion:-

We report a fatal postoperative rhabdomyolysis emerging within 22 hours of elective orthopaedic surgery under sevoflurane-based anaesthesia in a high-risk diabetic patient on chronic high-intensity atorvastatin. The clinical pattern is most consistent with a multifactorial process in which a probable statin–halogenated synergy was amplified by uncontrolled diabetes, advanced cardiomyopathy, lateral decubitus positioning and hyperkalaemia-promoting polypharmacy. While guidelines correctly recommend perioperative statin continuation, this case highlights three actionable points: individualised myotoxic risk stratification (potentially augmented by SLCO1B1 and RYR1 pharmacogenomics), a low threshold for total intravenous anaesthesia in the highest-risk subset, and prompt deployment of continuous high cut-off renal replacement therapy when massive necrosis is suspected. The proposed STAT-OP framework provides a structured starting point for prospective evaluation, and we encourage anaesthesiologists, intensivists, cardiologists and pharmacologists to recognise and report this clinically meaningful, potentially preventable, and currently under-reported perioperative phenotype.

**Figure Legends:-**

**Figure 1.** Chronological timeline of perioperative events. From the index orthopaedic surgery (T0) through an asymptomatic interval to abrupt clinical deterioration at T+22 h, ICU admission with massive rhabdomyolysis and life-threatening hyperkalaemia, partial correction after haemodialysis, rebound hyperkalaemia at T+33 h, and refractory cardiac arrest at T+35 h. CPK = creatine phosphokinase; LVEF = left ventricular ejection fraction; HD = haemodialysis.

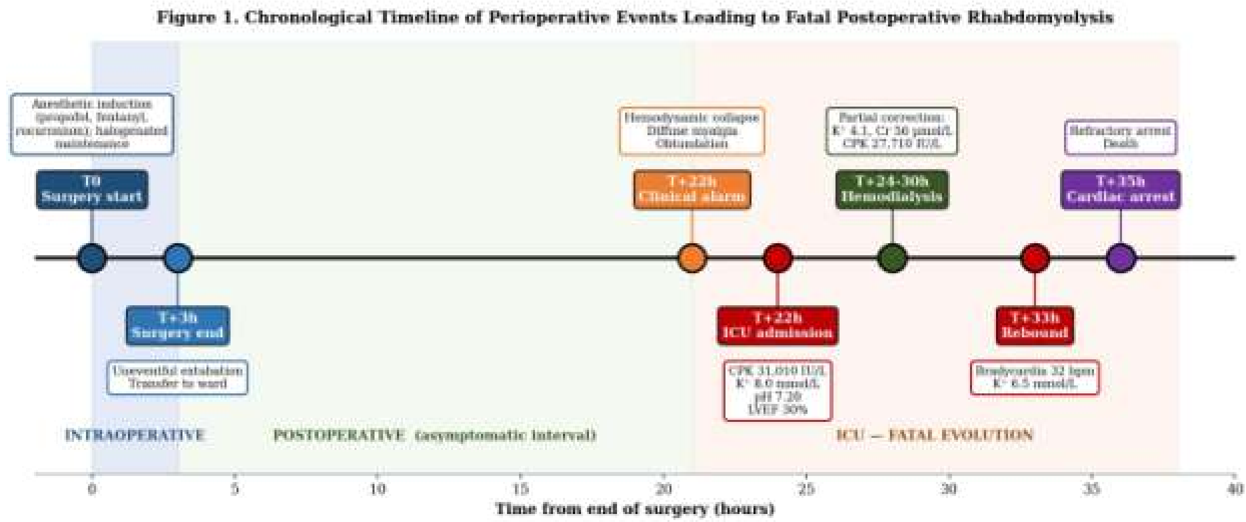
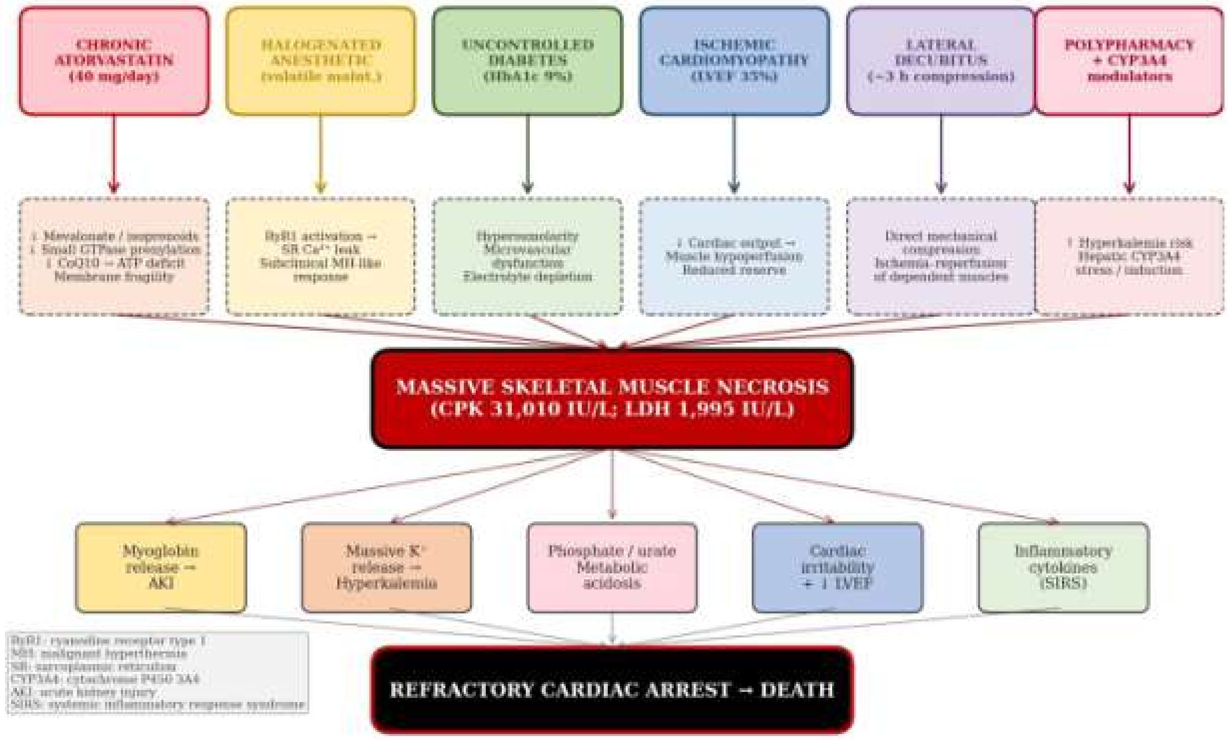



**Figure 2.** Proposed integrative pathophysiological model. Six convergent injurious pathways acting on skeletal muscle generate massive necrosis (CPK 31,010 IU/L), triggering myoglobinuric AKI, life-threatening hyperkalaemia, metabolic acidosis, cardiac irritability and systemic inflammation, culminating in refractory cardiac arrest. RyR1 = ryanodine receptor type 1; CYP3A4 = cytochrome P450 3A4; AKI = acute kidney injury; SIRS = systemic inflammatory response syndrome; SR = sarcoplasmic reticulum; MH = malignant hyperthermia.
[Figures 1 and 2 are submitted as separate high-resolution JPEG files, in accordance with the IJAR submission requirements: > 1800 × 1200 px, < 4 MB.]

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### Ethical Considerations and Consent:-
Written informed consent for the publication of this anonymised case report and accompanying data was obtained from the patient's next of kin in accordance with the principles of the Declaration of Helsinki (revised 2013). Institutional review board approval was waived in accordance with local regulations governing single anonymised case reports. The signed consent form is archived under the authors' responsibility and is available upon request from the journal editorial office. This case report was prepared in accordance with the CARE (CAseREport) reporting guideline.

### Conflicts of Interest:-
The authors declare that no conflict of interest, financial or otherwise, exists in relation to this work.

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### Use of Artificial Intelligence Tools:-
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