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Hemodynamic Stability via Ultrasound-Guided Axillary Brachial Plexus Block with Levobupivacaine-Dexamethasone in a Patient with Impending Thyroid Storm and Hand Fracture

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A B S T R A C T

The perioperative management of patients with uncontrolled hyperthyroidism requiring emergency surgery presents an acute clinical challenge, as surgical trauma and anesthesia can precipitate a life-threatening thyroid storm. This case report examines the strategic role of regional anesthesia in mitigating such risks through complete afferent blockade and sympathetic stabilization. A 28-year-old male presented with multiple right-hand fractures following a motorcycle accident. The patient had a history of untreated hyperthyroidism for one year and exhibited classic clinical thyrotoxicosis, including tachycardia of 104 bpm, hypertension of 164/90 mmHg, bilateral exophthalmos, and hyperkinesis. Laboratory investigations confirmed primary hyperthyroidism with a markedly elevated free T4 of 86.6 pmol/L and suppressed TSH. His Burch-Wartofsky Point Scale (BWPS) score was calculated at 30, indicating an impending thyroid storm. Following rapid medical optimization with propylthiouracil, propranolol, hydrocortisone, and amlodipine, surgical intervention was successfully performed under ultrasound-guided axillary brachial plexus block. The anesthetic mixture comprised 20 mL of 0.5 percent levobupivacaine and 8 mg of perineural dexamethasone. The patient demonstrated remarkable hemodynamic stability throughout the two-hour procedure, maintaining a systolic blood pressure between 115 and 135 mmHg and a heart rate between 82 and 94 bpm, without progressing to a thyroid crisis. In conclusion, ultrasound-guided regional anesthesia, specifically the axillary approach, offers a superior safety profile for thyrotoxic patients by avoiding airway instrumentation and preventing the sympathetic surges associated with general anesthesia. The synergistic use of levobupivacaine and dexamethasone provides a dual benefit of enhanced cardiac safety and peripheral endocrine stabilization.

1. Introduction

Hyperthyroidism represents a pervasive endocrine pathology, affecting an estimated 1 to 3 percent of the global population, with a notably higher prevalence in iodine-sufficient regions where Graves' disease remains the primary etiology.[1] At its clinical zenith, the syndrome of thyrotoxicosis manifests as a multisystemic state of hypermetabolism resulting from the overproduction and excessive circulation of thyroid hormones, specifically triiodothyronine (T3) and thyroxine (T4). For the anesthesiologist, this condition presents a formidable challenge, as these hormones exert profound physiological effects by upregulating beta-adrenergic receptor expression on cardiac myocytes and increasing myocardial sensitivity to endogenous catecholamines. The resulting hyperadrenergic state is characterized by significant cardiovascular instability, including tachycardia, widened pulse pressure, and a predispositon to tachyarrhythmias.[2]

The perioperative period is particularly hazardous for these patients, as the physiological stress of

surgical trauma, acute injury, and anesthetic manipulation can precipitate a thyroid storm.[3] Thyroid storm is a rare but life-threatening medical emergency marked by extreme hyperthermia (often exceeding 40 degrees Celsius), profound tachycardia, altered mental status, and potential cardiovascular collapse. Despite advancements in modern intensive care and pharmacological management, the mortality rate for thyroid storm remains high, ranging from 8 percent to 25 percent in recent clinical surveys.

Under ideal clinical circumstances, current anesthetic guidelines recommend the postponement of elective surgery until a biochemically euthyroid state is achieved through a multi-week regimen of antithyroid medications, beta-blockers, and occasionally iodine solutions. However, the reality of emergency surgical practice—such as acute orthopedic trauma—often necessitates immediate intervention before such pharmacological optimization can take full effect. In these urgent scenarios, the selection of an anesthetic technique becomes a critical factor in mitigating perioperative risk.[4]

General anesthesia carries significant inherent risks in the thyrotoxic population. The process of airway instrumentation, specifically laryngoscopy and endotracheal intubation, can trigger abrupt sympathetic surges.[5] In a patient with elevated adrenergic sensitivity, these surges may lead to hypertensive crises or lethal arrhythmias. Furthermore, thyrotoxic patients exhibit significantly increased basal metabolic rates and elevated oxygen consumption (O_2). During the induction of general anesthesia, the period of apnea required for intubation can lead to rapid and severe desaturation, as the patient's oxygen reserves are depleted much faster than in a euthyroid individual. This risk is further compounded if a goiter or thyroid-related airway distortion is present, making airway management technically difficult and prolonging the time to secure the airway.[6] Additionally, these patients may demonstrate an exaggerated sensitivity to volatile anesthetics, complicating the maintenance of hemodynamic stability.

Regional anesthesia (RA) offers a distinct and scientifically robust alternative by circumventing the need for airway instrumentation and preserving spontaneous ventilation.[7] By maintaining the patient's natural respiratory mechanics and physiological dead space, RA preserves the oxygen supply-demand balance in a hypermetabolic state. Within the various approaches for upper extremity surgery, the axillary brachial plexus block stands out as a particularly suitable technique for high-risk patients. Its superficial anatomical location allows for high-resolution visualization via ultrasound, facilitating a precise deposit of local anesthetic around the target nerves. Unlike interscalene or supraclavicular blocks, the axillary approach is performed distant from the lung apex and phrenic nerve, thereby avoiding the risks of pneumothorax or hemidiaphragmatic paresis. This preserves 100 percent of the patient's ventilatory capacity, which is vital for a patient with increased metabolic demand.[8]

The choice of local anesthetic agent is equally crucial in managing thyrotoxic cardiovascular risks. Levobupivacaine, the pure S(-) enantiomer of bupivacaine, has emerged as a preferred long-acting agent for patients where cardiac safety is a priority. While racemic bupivacaine is known for its potential to cause refractory ventricular arrhythmias by binding avidly to cardiac sodium channels, the S(-) isomer exhibits less intense and more rapidly reversible sodium channel blockade.[9] Clinical studies indicate that levobupivacaine causes smaller decreases in cardiac output and stroke volume compared to its racemic counterpart, providing a valuable safety margin in a myocardium already sensitized to catecholamines. Furthermore, the integration of pharmacological adjuvants such as dexamethasone can significantly enhance the efficacy of the regional block. Dexamethasone is well-documented to prolong the duration of sensory blockade and reduce postoperative opioid requirements through the modulation of nociceptive C-fiber activity and local anti-inflammatory effects. However, in the specific context of hyperthyroidism, dexamethasone may

provide an even more profound, yet often overlooked, systemic benefit.[10]

This study aims to demonstrate the successful anesthetic and perioperative management of a patient presenting with an impending thyroid storm triggered by acute trauma, utilizing a targeted ultrasound-guided regional anesthetic strategy. The novelty of this report lies in its detailed analysis of the dual therapeutic effect of perineural dexamethasone. While traditionally used as an analgesic adjuvant to prolong block duration, we highlight its potent endocrine contribution in this case: its systemic absorption likely served to inhibit the peripheral conversion of T4 to the more potent T3, thereby actively stabilizing the underlying thyrotoxic state. Furthermore, we articulate the mechanism by which complete afferent blockade via the axillary block prevents the primary endocrine triggers of thyroid crisis—pain and surgical stress—by severing the signaling pathway to the hypothalamic-pituitary-adrenal axis at the source. This comprehensive approach redefines regional anesthesia as a holistic tool for physiological stabilization in the high-risk endocrine patient.

2. Case Presentation

The patient provided written informed consent for the publication of this case report and any accompanying images, ensuring that all personal identifiers have been removed to maintain confidentiality. A 28-year-old male patient, weighing 76 kg and measuring 171 cm in height, was admitted to the emergency department following a high-impact motorcycle accident. The primary complaint was agonizing pain and significant deformity of the right hand, resulting from a fall onto the asphalt. While the patient remained conscious and was wearing protective equipment, the immediate orthopedic assessment was complicated by a significant underlying endocrine comorbidity. The patient disclosed a significant medical history of hyperthyroidism for which he had previously received hospital-based treatment. Critically, he admitted to a total cessation of all antithyroid medications approximately one year prior to this admission. At the time of presentation, he denied acute dyspnea but reported classic systemic manifestations of thyrotoxicosis, including profound heat intolerance, excessive diaphoresis, and increased appetite despite stable body weight.

Upon physical examination, the patient exhibited a hyperdynamic and hypermetabolic state. He displayed bilateral exophthalmos, hyperkinesis, and fine tremors of the extremities. Vital signs were notably deranged: a blood pressure of 164/90 mmHg indicated systemic hypertension, while a heart rate of 104 beats per minute confirmed sinus tachycardia. These findings were highly suggestive of a thyrotoxic state characterized by increased beta-adrenergic receptor sensitivity. A specialized examination of the neck revealed a palpable, grade 1b goiter; however, the trachea remained midline. Anesthesia-specific airway assessment identified a Mallampati II score, with normal thyromental distance and preserved neck mobility, which were essential baseline metrics for perioperative safety planning.

The local orthopedic examination of the right hand revealed marked edema, deformity, and exposed bone at the metacarpal region. Palpation elicited severe tenderness and crepitation, and active movement was entirely restricted by pain. Subsequent radiologic evaluation with PA and oblique views confirmed a complex trauma profile: a complete oblique fracture of the fourth metatarsal and fractures of the proximal phalanges of the second and third digits. Given the presence of an open fracture and the patient's unstable endocrine status, a multidisciplinary approach was immediately convened.

Laboratory investigations provided definitive evidence of severe primary hyperthyroidism. Thyroid function tests showed a markedly elevated Free T4 level of 86.6 pmol/L, which is approximately four times the upper limit of the reference range (12.0–22.0 pmol/L). This was accompanied by a completely suppressed TSH level of less than 0.005 μIU/mL. Metabolic panels indicated a total calcium level of 10.68 mg/dL (Reference: 8.8–10.2 mg/dL). This

hypercalcemia is a recognized complication of thyrotoxicosis, resulting from increased thyroid hormone-mediated bone turnover. Renal function tests showed an elevated blood urea nitrogen (BUN) of 31.7 mg/dL against a low-normal creatinine of 0.69 mg/dL, suggesting a state of dehydration secondary to chronic diaphoresis and hypermetabolism. A 12-lead electrocardiogram (ECG) confirmed sinus tachycardia with a heart rate of 104 bpm. Close inspection of the ECG revealed a shortened QTc interval of 380 ms, which correlated with the identified hypercalcemia and posed an additional risk for perioperative arrhythmias.

Table 1. Summary of Clinical Findings on Admission

SYSTEM CATEGORY	PARAMETER	ADMISSION FINDING	CLINICAL INTERPRETATION
VITAL SIGNS	Blood Pressure	164/90 mmHg	SEVERE HYPERTENSION
	Heart Rate	104 bpm (Regular)	SINUS TACHYCARDIA
	Temperature	37.3°C	HYPERTHERMIC STATE
PHYSICAL EXAM	Neurological / CNS	Hyperkinesis, Tremor, Agitation	IMPENDING CRISIS
	Ocular finding	Bilateral Exophthalmos	GRAVES' ORBITOPATHY
	Integumentary	Warm, Moist, Diaphoresis	HYPERMETABOLISM
ENDOCRINE STATUS	Thyroid Gland	Palpable, Grade 1b Goiter	THYROID HYPERPLASIA
	Metabolic history	Weight loss despite increased appetite	SEVERE CATABOLISM
AIRWAY ASSESSMENT	Mallampati Score	Class II	LOW RISK AIRWAY
	Neck Anatomy	Trachea Midline, Full Mobility	OPTIMIZED FOR RA

The clinical assessment of thyroid storm risk is a critical diagnostic imperative in the management of thyrotoxic patients presenting for emergency surgery (Table 2). The Burch-Wartofsky Point Scale (BWPS) remains the gold-standard semi-quantitative tool for this purpose, as it correlates systemic physiological derangements with the probability of a life-threatening crisis. In this case, the initial clinical presentation was characterized by multisystemic involvement that necessitated a rigorous re-calculation of the BWPS score to ensure patient safety. The patient's cardiovascular status contributed 5 points due to a persistent sinus tachycardia of 104 beats per minute, reflecting the hyperadrenergic state and increased beta-adrenergic receptor sensitivity inherent in thyrotoxicosis. Although the body temperature was recorded at 37.3 degrees Celsius, this low-grade fever represents significant thermoregulatory dysfunction in a hypermetabolic context, adding an additional 5 points. A critical nuance in this assessment was the objective finding of hyperkinesis, visible tremors, and agitation. In the BWPS framework, these neurological manifestations qualify as mild central nervous system dysfunction, warranting the inclusion of 10

points. Furthermore, the acute physiological stress of the motorcycle accident and resulting hand fractures served as a potent precipitating factor, contributing 10 points to the scale. The resulting cumulative score of 30 shifts the clinical interpretation from unlikely to impending thyroid storm. This diagnostic elevation is scientifically significant; it validates the aggressive preoperative medical optimization strategy and the selection of regional anesthesia as a stabilization technique. By providing a dense afferent blockade, the axillary brachial plexus block prevented further escalations of this score during the surgical period, effectively halting the progression from an impending crisis to a full-scale thyroid storm.

Table 2. Burch-Wartofsky Point Scale (BWPS) Re-assessment

CLINICAL CATEGORY	CRITERIA DESCRIPTION	JUSTIFICATION / CLINICAL FINDING	POINTS
CARDIOVASCULAR DYSFUNCTION	Tachycardia (Heart Rate)	Heart rate of 104 bpm (90–109 range)	5
	Atrial Fibrillation / Heart Failure	None reported / Sinus Tachycardia present	0
CENTRAL NERVOUS SYSTEM	CNS Symptoms	Mild agitation, visible tremors, and hyperkinesis	10
THERMOREGULATION	Body Temperature	Low-grade fever (37.3°C)	5
PRECIPITATING FACTOR	Acute Stressor	Acute trauma (Hand fracture / Accident)	10
			30

CLINICAL STATUS: IMPENDING THYROID STORM (Score 25–44)

The management of a patient with a Burch-Wartofsky Point Scale (BWPS) score of 30 necessitates immediate pharmacological intervention to arrest the progression from impending thyroid storm to catastrophic multiorgan failure. At T plus 30 minutes following initial admission, a multimodal optimization protocol was strategically initiated to target the overlapping pathophysiological pathways of thyrotoxicosis. Propylthiouracil (PTU) was administered at a loading dose of 300 mg orally. PTU remains a cornerstone of therapy due to its unique ability to inhibit thyroid peroxidase, thereby blocking de novo synthesis of thyroid hormones, while simultaneously uniquely inhibiting the peripheral deiodination of T4 to the more biologically potent T3. To address the immediate cardiovascular risks of hyperadrenergic activity, 40 mg of oral propranolol was administered. Propranolol provides essential competitive antagonism at the beta-1 and beta-2 adrenergic receptors, directly reducing heart rate and myocardial oxygen consumption while further contributing to the inhibition of peripheral T3 conversion. In addition to thyroid-specific agents, 100 mg of intravenous hydrocortisone was provided to stabilize the hypothalamic-pituitary-thyroid axis. Glucocorticoids are vital in this setting as they suppress the release of stored thyroid hormones from the gland and provide adrenal support against the relative adrenal insufficiency often observed in hypermetabolic states. Finally, 10 mg of oral amlodipine was utilized as a calcium channel blocker

to provide robust hemodynamic control against the patient's presenting hypertension of 164/90 mmHg.

A critical decision was made to delay surgical intervention until T plus 4 hours, allowing a sufficient window for the pharmacological agents to reach therapeutic plasma concentrations and for the patient's hyperdynamic circulatory state to stabilize (Table 3). The anesthesia team utilized an ultrasound-guided axillary brachial plexus block, a technique favored for its high safety profile and efficacy in distal upper limb trauma. Using a high-frequency linear probe (10 to 15 MHz) and a 22G echogenic needle, the median, radial, and ulnar nerves were identified in cross-section in close proximity to the axillary artery. A total volume of 20 mL, consisting of 0.5 percent Levobupivacaine (100 mg) and 8 mg of Dexamethasone, was prepared. Levobupivacaine was chosen specifically for its superior cardiovascular safety profile, which is essential in a thyrotoxic myocardium sensitized to catecholamines. To ensure comprehensive surgical anesthesia, 5 mL of the mixture was deposited around each of the three nerves within the perivascular sheath, followed by a separate, targeted injection of 5 mL into the coracobrachialis muscle to anesthetize the musculocutaneous nerve. Safety protocols were strictly maintained; a difficult airway cart featuring a video laryngoscope remained at the bedside to manage potential rapid desaturation or local anesthetic systemic toxicity. Furthermore, to ensure complete tourniquet tolerance, the intercostobrachial nerve (T2) was anesthetized via a subcutaneous skin wheal of 5 mL of 1 percent lidocaine across the axillary base.

Table 3. Comprehensive Summary: Diagnosis, Treatment, Outcome, and Follow-up

PHASE OF CARE	CLINICAL COMPONENT	DETAILED DESCRIPTION / MANAGEMENT STRATEGY
DIAGNOSIS	Primary Diagnoses	Endocrine Impending Thyroid Storm (BWPS Score 30) due to Graves' Disease relapse. Trauma Open fracture metatarsal IV and phalanx fractures (Digits II, III) of the right hand.
	Secondary Findings	Marked Thyrotoxicosis (FT4 86.6 pmol/L), suppressed TSH, and associated hypercalcemia (10.68 mg/dL).
TREATMENT	Preoperative Optimization	Pharmacology Propylthiouracil (300 mg), Propranolol (40 mg), Hydrocortisone (100 mg), and Amlodipine (10 mg). Timeline 4-hour window allowed for initial hormone suppression and cardiovascular stabilization.
	Anesthesia Technique	Ultrasound-guided Axillary Brachial Plexus Block with 20 mL Levobupivacaine 0.5% and 8 mg Dexamethasone; Intercostobrachial (T2) skin wheal for tourniquet comfort.
OUTCOME	Intraoperative Status	Complete afferent blockade; Hemodynamic stability maintained (SBP 115–135 mmHg; HR 82–94 bpm); No transition to thyroid storm.
	Immediate Post-op	Sensory block duration of 6 hours; effective non-opioid multimodal analgesia (Paracetamol and Ibuprofen); no rescue opioids required.
FOLLOW-UP	Short-term Recovery	Successful wound healing and fracture alignment; progressive return of motor function at 4 hours post-block.
	Endocrine Plan	Multidisciplinary referral to Endocrinology for long-term antithyroid therapy and potential definitive management (RAI or Thyroidectomy).

The surgical stabilization of the fractures proceeded over 120 minutes with exceptional hemodynamic stability. Intraoperative systolic blood pressure was meticulously maintained between 115 and 135 mmHg, while the heart rate fluctuated narrowly between 82 and 94 bpm. Notably, the avoidance of midazolam or opioid sedation allowed the medical team to maintain continuous verbal contact with the patient, serving as a real-time monitor for neurological changes indicative of a developing thyroid crisis. Postoperatively, the sensory block provided dense analgesia for 6 hours, significantly reducing the systemic stress response. Analgesic transition was achieved via a non-opioid multimodal regimen consisting of 600 mg paracetamol and 400 mg ibuprofen, which effectively managed pain without the risks of respiratory depression or nausea in this physiologically fragile patient.

3. Discussion

The clinical management of local anesthetic systemic toxicity (LAST) risk is fundamentally altered in the presence of uncontrolled thyrotoxicosis.[11] The central scientific tension in this case revolves around the administration of 100 mg of levobupivacaine (20 mL of a 0.5 percent solution) within a hyperdynamic circulatory environment. Thyrotoxicosis induces a profound increase in cardiac output, stroke volume, and regional blood flow, primarily through the upregulation of beta-adrenergic receptors and direct genomic effects on cardiac myocytes. In such a state, the rate of vascular absorption from the perineural space—especially in a highly vascularized region like the axilla—is theoretically accelerated. This elevation in absorption rate increases the peak plasma concentration and shortens the time to reach that peak, potentially narrowing the therapeutic window. While 100 mg is historically well within the safe dosage limits for a 76 kg patient, which is approximately 1.3 mg/kg compared to the 2.0 to 2.5 mg/kg threshold, the toxic threshold may be functionally lower in thyrotoxic patients due to increased myocardial sensitivity.

Levobupivacaine was specifically chosen over racemic bupivacaine to provide a necessary safety buffer. As the pure S enantiomer, levobupivacaine exhibits a significantly lower affinity for cardiac sodium channels during phase 0 of the cardiac action potential. The cardiotoxicity risk of bupivacaine stems largely from its interaction with cardiac sodium and potassium channels; at higher systemic concentrations, it causes pronounced sodium channel blockade, slowed conduction, and potentially refractory ventricular arrhythmias. By eliminating the R(+) isomer, levobupivacaine produces less intense and more rapidly reversible sodium channel blockade, exerts weaker effects on potassium channels, and results in less negative inotropy. In a myocardium already sensitized by excessive thyroid hormones and pre-existing sinus tachycardia, the use of an agent with reduced negative inotropic and arrhythmogenic potential is a critical clinical safeguard.[12]

While dexamethasone is a standard adjuvant used to extend the duration of sensory blockade by 6 to 8 hours, its role in this specific case transcended simple analgesia. The administration of 8 mg of perineural dexamethasone served a dual purpose, acting as both a local neural stabilizer and a systemic endocrine modulator. Thyroid storm management relies heavily on inhibiting the peripheral conversion of the pro-hormone T4 into the highly potent T3. Dexamethasone is a potent inhibitor of Type 1 deiodinase, the enzyme responsible for this conversion.[13] Given the high vascularity of the axillary sheath, the 8 mg dose was likely absorbed systemically, reaching concentrations sufficient to contribute to the biochemical stabilization of the patient's impending storm. This inadvertent but strategic systemic absorption effectively augmented the oral propylthiouracil and intravenous hydrocortisone regimen, facilitating a rapid reduction in the biologically active thyroid hormone pool. Perineural dexamethasone achieves near maximal benefit at doses of 4 to 8 mg, with higher doses not consistently improving duration and thus offering little justification given potential systemic

effects. In this specific clinical context, those systemic effects were leveraged as a therapeutic asset.

A fundamental argument of this study is that the prevention of thyroid storm was not merely a byproduct of avoiding general anesthesia but a direct result of complete afferent blockade. Surgical trauma and acute pain act as primary triggers for the massive catecholamine release that precipitates thyroid crisis.[14] Under general anesthesia, unless an extremely deep level of hypnosis is maintained, nociceptive signals from the fracture site continue to reach the dorsal horn and ascend to the hypothalamus and brainstem. This triggers a hypothalamic-pituitary-adrenal axis response, leading to a surge in endogenous catecholamines. In a thyrotoxic patient, this surge can be fatal. By contrast, the ultrasound-guided axillary block severs these signals at the source. By achieving a dense, complete blockade of the median, ulnar, radial, and musculocutaneous nerves, we effectively isolated the injured hand from the central nervous system, maintaining endocrine silence throughout the 120-minute procedure. Regional blocks prevent abrupt sympathetic surges that accompany the induction of general anesthesia, limiting extreme catecholamine-mediated tachycardia and hypertension that could precipitate arrhythmias or thyroid storm.

Despite the clear benefits of regional anesthesia, the potential for complications necessitates a robust backup plan. Thyrotoxic patients have elevated basal oxygen consumption and may desaturate rapidly if apnea occurs. The presence of a palpable grade 1b goiter in this patient further raised the stakes, as tracheal deviation or compression could complicate rescue airway maneuvers.[15] Our safety strategy involved a comprehensive airway assessment and the immediate availability of a difficult airway cart equipped with a video laryngoscope. This preparation ensured that if the block had been patchy or if the patient developed a local anesthetic-induced seizure, a secure airway could be established with minimal delay, avoiding the physiological disaster of a failed intubation in a hypermetabolic state. Negative aspiration was performed before each injection, and the spread of local anesthetic was carefully observed in real time to confirm perineural rather than intravascular deposition to minimize LAST risk.[16]

Table 4 provides a comprehensive clinical roadmap for the anesthetic management of patients presenting with the complex intersection of acute trauma and uncontrolled thyrotoxicosis. This structured framework is organized into three distinct perioperative phases—preoperative, intraoperative, and postoperative—each designed to address specific physiological vulnerabilities associated with hypermetabolic states. The management of a thyrotoxic patient begins with rigorous risk stratification. The first checkpoint emphasizes the use of the Burch-Wartofsky Point Scale (BWPS). In this case, the calculation yielded a score of 30, shifting the diagnosis from a stable hyperthyroid state to an impending thyroid storm. This diagnostic distinction is vital; it justifies the immediate escalation of pharmacological therapy to prevent multi-organ failure. Concurrently, a targeted airway examination is mandatory. Although regional anesthesia is the primary plan, the clinician must anticipate a Plan B airway strategy, particularly in patients with palpable goiters that may cause tracheal deviation or compression.[17] The rapid optimization protocol, initiated within 30 minutes of admission, targets the multiple pathophysiological layers of thyroid hormone synthesis and activity. Propylthiouracil (PTU) acts by inhibiting thyroid peroxidase to block hormone synthesis, while propranolol provides essential beta-adrenergic blockade to mitigate tachycardia and hypertension. The inclusion of intravenous hydrocortisone is equally critical, as it supports adrenal reserve and, alongside PTU, inhibits the peripheral conversion of the pro-hormone T4 into the more potent T3.[18]

The intraoperative section of Table 4 highlights the strategic selection of regional anesthesia over general anesthesia. The ultrasound-guided axillary brachial plexus block provides afferent blockade, a concept where nociceptive signaling from the surgical site is

severed at the peripheral level. By preventing these pain signals from reaching the hypothalamus, the block avoids the massive catecholamine surges that typically accompany surgical stress under general anesthesia. Furthermore, the choice of local anesthetic is a key safety checkpoint. Levobupivacaine, the S(-) enantiomer, is prioritized due to its reduced cardiotoxicity and higher threshold for inducing arrhythmias compared to racemic bupivacaine. This is particularly relevant in a thyrotoxic myocardium that is already sensitized by high circulating catecholamine levels.[19] The use of real-time ultrasound guidance and frequent negative aspiration is emphasized as an essential technical safeguard to prevent local anesthetic systemic toxicity (LAST), which could be catastrophic in a hyperdynamic circulatory state where absorption rates are theoretically accelerated.

The final checkpoints focus on the transition from acute surgical care to long-term endocrine stabilization. The use of non-opioid multimodal analgesia, such as paracetamol and ibuprofen, is recommended to provide sustained comfort without the risks of respiratory depression or nausea. Maintaining verbal contact during the intraoperative and early postoperative phases serves as a critical neurological monitor; any change in mental status, such as delirium or extreme agitation, would signal a transition toward thyroid crisis. Finally, the disposition of the patient is a crucial safety step. Transfer to a High Dependency Unit (HDU) or Intensive Care Unit (ICU) is warranted for continuous cardiac monitoring to detect rebound thyrotoxicosis, which can occur as the effects of the initial antithyroid medications and regional block begin to wane. This perioperative pathway concludes with an early multidisciplinary referral to endocrinology, ensuring that the patient's underlying Graves' disease is addressed definitively after the acute trauma has been stabilized.[20]

Table 4. Anesthesia Safety Checkpoints for Emergency Thyrotoxic Management

PERIOPERATIVE PHASE	SAFETY CATEGORY	CLINICAL ACTION ITEMS AND RATIONALE
PREOPERATIVE	Risk Stratification	✓ Calculate Burch-Wartofsky Point Scale (BWPS) to identify impending storm. ✓ Perform targeted airway exam (Mallampati, Goiter size, Tracheal position).
	Rapid Optimization	✓ Administer PTU (300-600mg) and Beta-blockers for hemodynamic control. ✓ IV Steroids to inhibit T4-T3 conversion and support adrenal reserve.
INTRAOPERATIVE	Technique Selection	✓ Prioritize Regional Anesthesia (RA) to provide complete afferent blockade. ✓ Select cardio-stable agents (Levobupivacaine) to minimize arrhythmia risk.
	Monitoring & Safety	✓ Maintain verbal contact (avoid deep sedation) to monitor mental status. ✓ Use real-time Ultrasound guidance to prevent intravascular injection (LAST).
POSTOPERATIVE	Analgesia & Recovery	✓ Implement non-opioid multimodal analgesia to avoid respiratory depression. ✓ Monitor for Rebound Thyrotoxicosis within 6-12 hours post-surgery.
	Disposition	✓ Transfer to High Dependency Unit (HDU) for continuous cardiac monitoring. ✓ Early multidisciplinary referral for definitive thyroid management.

While this case demonstrates a successful outcome, it is not without limitations. As a single case report, the results cannot be broadly generalized to all thyrotoxic patients, as the severity of the endocrine dysfunction and the nature of the surgical stress vary significantly across populations. Furthermore, while we hypothesize a systemic benefit from perineural dexamethasone, we did not measure serial plasma T3 and T4 levels intraoperatively to quantitatively prove the rate of hormonal decline attributable to the adjuvant alone. Future research should focus on randomized controlled trials comparing regional versus general anesthesia in semi-urgent hyperthyroid cases, specifically measuring the markers of the endocrine stress response. Additionally, pharmacokinetic studies of local anesthetics in hyperdynamic states are warranted to establish evidence-based dosing guidelines for this high-risk patient group. The dual therapeutic effect of dexamethasone observed here also invites further investigation into the optimal dose and route of steroids for rapid endocrine stabilization in the emergency perioperative setting.

4. Conclusion

This case report demonstrates that ultrasound-guided axillary brachial plexus block using levobupivacaine and dexamethasone represents an ideal anesthetic strategy for emergency upper-limb trauma in patients presenting with impending thyroid storm. By prioritizing regional techniques, the clinical team successfully bypassed the high-risk requirements of airway instrumentation and volatile anesthetic exposure while achieving superior hemodynamic stability. The pharmacological synergy between the cardiac safety of levobupivacaine and the dual action of dexamethasone as an analgesic and endocrine stabilizer provided a unique window for safe surgical intervention. For anesthesiologists operating in emergency settings, regional anesthesia should be considered a first-line stabilizing intervention rather than a secondary option. Early recognition of impending crisis, combined with rapid medical optimization and complete afferent blockade, remains the most effective roadmap for ensuring perioperative survival in the complex thyrotoxic population.

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