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FULL PAPER

Perioperative anesthetic management on burr hole drainage of subdural hematoma during thyroid storm in a patient with down syndrome: Case report

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Subdural hematoma (SDH) is an intracranial lesion formed by the accumulation of blood in the space between arachnoid and duramater due to a tear of the vein or artery between duramater and arachnoid. SDH is caused by trauma, vascular malformations, coagulopathies, neoplasms, or in rare cases such as hyperthyroidism that can increase intracranial pressure that lead to life-threatening. A thyroid storm represents an endocrine emergency marked by thyrotoxicosis and can be triggered by trauma and severe stress conditions such as SDH. A thyroid storm will increase cerebral blood flow and correlate with an elevated cerebral blood volume, resulting in increased intracranial pressure. Therefore, careful and prompt preoperative preparation is needed to prevent patient mortality. A 33-year-old woman weighing 38 kg had a gradual loss of consciousness, vomiting, and headache and was diagnosed with subdural hematoma. The patient was examined in preparation for burrhole drainage. The patient had a known history of Down syndrome since birth, with an unknown history of hyperthyroidism. The patient exhibited thyroid storm, with a free T4 level exceeding 100 ng/dL and a Burch Wartofsky score of 50. Surgery during thyroid storm is a high-risk procedure but should be performed in emergencies with careful preparation and vigilance, especially against possible complications. The anesthesiologist's role in perioperative management becomes crucial, especially in complicated cases.

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KEYWORDS

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Introduction

Subdural hematoma (SDH) is an intracranial lesion that occurs between the dural and arachnoid membranes surrounding the brain. SDH originates from bridging veins injury, especially those close to the superior sagittal sinus [1]. Decreased consciousness in SDH that occurs before hospital admission can be categorized based on time: chronic subdural hematoma, acute subdural hematoma, and subacute subdural hematoma. A sudden increase in intracranial pressure, arterial bleeding, or acute brain edema causes signs of decreased consciousness [2]. In addition, there may be signs of neurologic abnormalities due to intracranial elevation, such as diplopia, anisocortical pupils, and sensory and motor disturbances [3,4].

A thyroid storm is an endocrine emergency characterized by thyrotoxicosis that can be assessed by several scores that combine clinical and supporting parameters such as the Burch-Wartofsky Point Scale (BWPS) (Table 1) and The Japanese Thyroid Association (JTA) (Table 2). Diagnosis of a thyroid storm can be performed by the Burch-Wartofsky Point Scale (BWPS) to distinguish uncomplicated thyrotoxicosis, impending signs, or thyroid storm has occurred, or can be present by The Japanese Thyroid Association (JTA). The BPWS scale is a scoring system used to assess the severity of symptoms multiple organ decompensation, which includes thermoregulation, tachycardia/atrial fibrillation, impaired consciousness, congestive heart failure, gastrointestinal and liver dysfunction, and other precipitating factors (Table 1) [5].

In addition, the JTA criteria can be used based on a combination of specific symptoms caused by decompensation of several organs. While the BWPS criteria one of the characteristics is the presence of impaired consciousness that contributes to the incidence of thyroid storm. However, the presence of other diseases with symptoms of fever (e.g., malignant hyperthermia and pneumonia), impaired consciousness (e.g., psychiatric disorders, cerebrovascular disease), liver abnormalities (e.g., acute liver failure and viral hepatitis), and heart failure (e.g., acute myocardial infarction) will complicate the assessment of thyroid storm. Hence, establishing whether the symptoms result from a thyroid storm or simply represent a persistent manifestation of the illness proves challenging [6]. These symptoms might have been related to thyroid storms that result from the presence of precipitating factors. Using BPWS and JTA criteria as diagnostic tools in evaluating the patient's condition will improve the accuracy in determining thyroid storm clinical diagnosis [7].

TABLE 1 Diagnosis of the Burch-Wartofsky point scale

Criteria	Points
Thermoregulatory dysfunction (temperature °C):	
37.2-37.7	5
37.8-38.3	10
38.4-38.8	15
38.9-39.3	20
39.4-39.9	25
≥ 40.0	30
Cardiovascular:	
Tachycardia (beats per minute):	
90-109	5
110-119	10
120-129	15
130-139	20
≥ 140	25
Atrial fibrillation:	
Absent	0
Present	10
Congestive heart failure:	
Absent	0
Mild	5
Moderate	10
Severe	15
Gastrointestinal-hepatic dysfunction:	

Absent	0
Moderate (diarrhea, abdominal pain, and nausea/vomiting)	10
Severe (jaundice)	20
Central nervous system disturbance:	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, and extreme lethargy)	20
Severe (seizure and come)	30
Precipitating event:	
Absent	0
Present	10
Total score	
Interpretation:	
≥ 45	Thyroid storm
25-44	Impending storm
< 25	Storm unlikely

Source: Karaören [8]

TABLE 2 The diagnostic criteria for thyroid storm (TS) of the Japan Thyroid Association

Prerequisite for diagnosis

Thyrotoxicosis with elevated FT3 or FT4 levels

Symptoms

1. CNS manifestations: Restlessness, delirium, mental aberration/psychosis, somnolence/lethargy, and coma (≥ 1 on the Japan Coma Scale or ≤14 on the Glasgow Coma Scale)
2. Fever: ≥ 38 ˚C
3. Tachycardia: ≥ 130 beats per minute or heart rate ≥ 130 in atrial fibrillation
4. CHF: Pulmonary edema, moist rales over more than half of the lung field, cardiogenic shock, or Class IV by the New York Heart Association or ≥ Class III in the Killip classification
5. GI/hepatic manifestations: nausea, vomiting, diarrhea, or a total bilirubin level ≥ 3.0 mg/dL

Diagnosis

Grade of TS	Combinations of features	Requirements for diagnosis
TS1	First combination	Thyrotoxicosis and at least one CNS manifestation and fever, tachycardia, CHF, or GI/hepatic manifestations
TS1	Alternate combination	Thyrotoxicosis and at least three combinations of fever, tachycardia, CHF, or GI/hepatic manifestations
TS2	First combination	Thyrotoxicosis and a combination of two of the following: fever, tachycardia, CHF, or GI/hepatic manifestations
TS2	Alternate combination	Patients who met the diagnosis of TS1 except that serum FT3 or FT4 level are not available

Notes: CHF: Congestive heart failure; CNS: Central nervous system; GI: Gastrointestinal; TS: Thyroid Storm; TS1: "Definite" TS; and TS2: "Suspected" TS.

Source: Satoh [5]

With an estimated low incidence of 1-2% in hospitals, thyroid storm demonstrates an overall mortality rate of 10-30%, signifying a mortality rate twelve times higher than that of those without thyrotoxicosis [9,10]. Underlying, untreated, or poorly controlled thyroid gland disease, when combined with significant additional stressors like surgery, infection, or trauma, can trigger a thyroid storm. This disruption in the body's compensatory mechanisms for thyroid hormone results in a life-threatening condition [11]. Management of thyroid storms is essential; in addition to normalizing thyroid hormone levels, diagnosing and rectifying the precipitating factors of the thyroid storm is equally essential (Table 3).

TABLE 3 Precipitating factors of thyroid storm

Thyroid surgery	Radioactive iodine therapy
Non-thyroid surgery	Exposure to iodine contrast
Trauma	Discontinuation of antithyroid therapy
Manipulation of the thyroid gland	Infection
Thyroiditis	Diabetic ketoacidosis
Pregnancy	Hypoglycemia
Burns	Consumption of high doses of thyroid hormones
Myocardial infarction	Thyroid cancer metastasis
Pulmonary embolism	Ovarian struma
Cerebrovascular incident	Molar pregnancy
Medications such as anesthetic drugs, salicylates, pseudoephedrine, and amiodarone	H1N1 infection
Interferon therapy	Emotional stress
Sepsis	Strenuous exercise

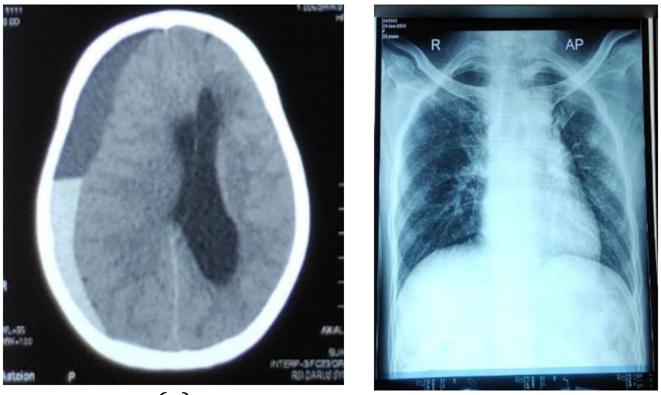
Source: Satoh [5]

SDH causes increased intracranial pressure due to increased cerebral blood volume, osmotic or vasogenic cerebral edema, elevated cerebrospinal fluid flow resistance at the arachnoid villi, and high cerebral venous pressure [12]. The thyroid storm will increase cerebral blood flow and correlate with increased cerebral blood volume, increasing intracranial pressure [13]. Conversely, the stressful condition of SDH can trigger the thyroid storm. Therefore, surgery is performed immediately along with treatment to correct the thyroid storm in surgical preparation to improve the prognosis. Thyroid storm therapy was given to prevent the worsening of the general condition and thyroid storm to lower thyroid hormone levels [14].

Case Presentation

A 33-year-old woman weighing 38 kg experienced gradual loss of consciousness, vomiting, and headache and was diagnosed with subdural hematoma. History before admission to the hospital has decreased consciousness for 7 days, found vomiting one time and accompanied by fever. The patient was known to have a history of down syndrome since birth, with an unknown history of hyperthyroidism. On physical examination, GCS score E3V3M5, blood pressure 105/65 mmHg (MAP 80 mmHg), pulse 118 x/min, respiration 24 x/min, oxygen saturation 92-95% with simple mask 6 liters per minute, and temperature 38 ^{o}C (Figure 2).

Brain computed tomography (CT) results showed subacute SDH in the temporoparietal dextra thickness of 1.6 cm, midline shift to sinistra by 11.2 mm, early signs of non-communicating hydrocephalus (Figure 1a). Initial chest x-ray examination showed no bilateral infiltrates, prominent cor, and aortic elongation with a cardiothoracic ratio of more than 50% (Figure 1b). On arrival at the hospital, there was a clinical thyroid storm with a Burch Wartofsky score of 50 and thyroid laboratory results of free T4 (FT4) > 100 ng/dl (10.60-19.40 ng/dl) and a decreased thyroid-stimulating hormone (TSH) value of 0.006 µIU/ml (0.500-5,000 µIU/mL).



(a) (b)

FIGURE 1 Patient's head CT showed SDH at temporoparietal dextra and midline shift to the left and early signs of non-communicans hydrocephalus (a) and Chest x-ray examination showed no bilateral infiltrates, prominent cor, and aortic elongation with a cardiothoracic ratio of more than 50% (b)

Based on the head CT data, it was decided to do 2-hole burrhole drainage. While in the emergency room, thyroid storm management was carried out using thiamazole tablets 20 mg every 8 hours, propranolol tablets 10 mg every 8 hours, and methylprednisolone injection 62.5 mg daily- control of thyroid storm in the emergency room without delaying 2-hole burrhole drainage. During the intraoperative procedure, the hemodynamic condition was stable (Figure 2). After surgery, thyroid storm management continued for three days. A well-managed thyroid storm caused by SDH resulted in a decrease in pulse rate to 100×/min; laboratory results on the third day of therapy obtained free T4 3.74 ng/dl, total T3 0.55 ng/dl, and TSH 0.0060 μIU/ml. During surgery there was no massive bleeding and hemodynamics were stable (Figure 2). The patient could be extubated and then observed in the intensive care unit. During intensive care, the patient showed improvement in consciousness so that the patient could be transferred to the inpatient room.

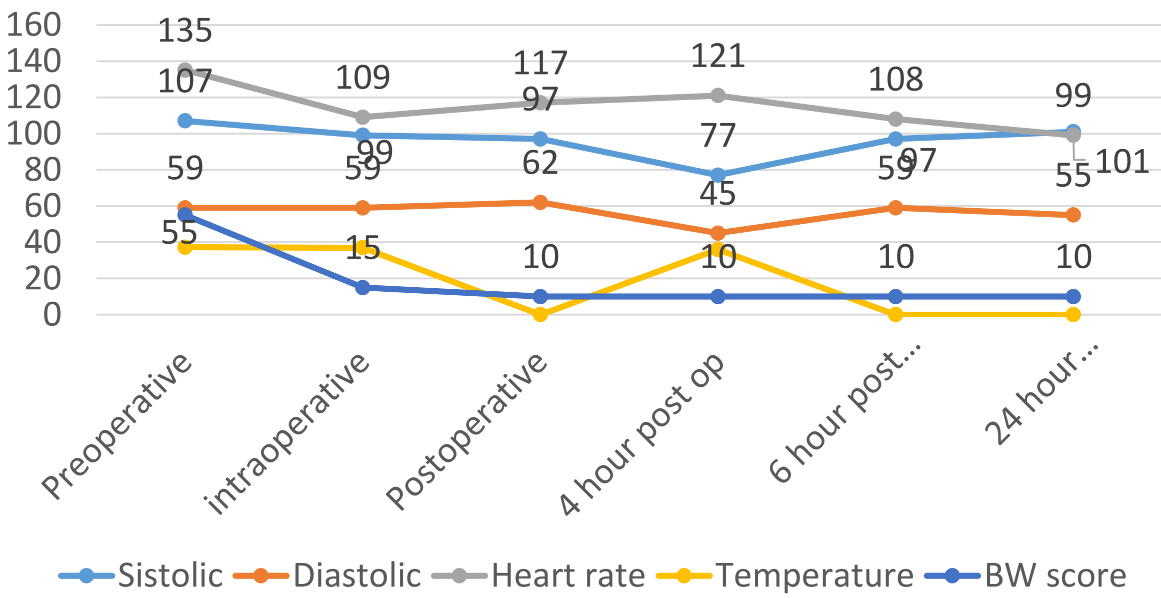


FIGURE 2 Hemodynamics from perioperative to postoperative

Discussion

A subdural hematoma is an accumulation of blood in the subdural cavity (between the dura mater and arachnoid). This hemorrhage often results from a rupture of the connecting vein between the cerebral cortex and the venous sinus, where the vein enters [15]. Still, it can also result from laceration of arterial blood vessels on the surface of the brain. Subdural hematoma is most common on the lateral surface of the hemisphere and partly in the temporal region, corresponding to the distribution of bridging veins [16]. Bridging veins are venous vessels that run on the brain surface towards the superior sagittal sinus in the midline. Subdural hemorrhage also covers the entire hemispheric surface of the brain, and damage to the underlying brain is severe. A subdural hematoma can occur spontaneously or be caused by a procedure. Mortality and morbidity rates can be high, even with the best care [17].

Supportive examinations are performed with head CT to evaluate intracranial hematoma progression, residual foreign bodies presence, and the extent of cerebral edema. Small subdural hematoma may blend into the skull bone image and only be visible by adjusting the CT window width. Midline shift will be seen in moderate or large volume subdural hematoma [18]. If there is no midline shift, a contralateral mass should be suspected, and if the midline shift is severe, underlying cerebral edema should be inferred. Subdural hematoma is rarely located in the posterior fossa as the cerebellum is relatively immobile and thus protective of the bridging veins [19].

Thyroid storm, as a complication of hyperthyroidism has a high mortality risk. Thyroid storms can occur due to precipitating factors such as infection, sepsis, trauma, cerebrovascular events, or radioactive iodine therapy (Table 1). Clinical examination, along with thyroid function results indicating low TSH (<0.01 mU/L), elevated FT4 and/or FT3, and manifestations of other organ disorders, forms the basis for diagnosing thyroid storm. Symptoms include gastrointestinal or hepatic complaints, tachycardia, congestive heart failure, fever, and central nervous system manifestations [7].

In the absence of specific clinical or laboratory findings, precise diagnosis of thyroid storm is essential. Early recognition of this clinical condition is vital, facilitating prompt and targeted treatment for assessing early organ dysfunction [10,20].

The thyroid storm, in this case, occurred due to stress that could be caused by SDH, where there was an increase in intracranial pressure [21,22]. Therefore, it is necessary to immediately perform 2-hole burr hole drainage without waiting for the euthyroid condition while still providing therapy for the thyroid storm to improve prognosis [23,24]. Thyroid storm therapy aims to prevent the worsening of the condition and control thyroid levels. This therapy is given while preparing for surgery and perioperative observation is carried out to reassess thyroid function to normal [14].

Treatments include thionamide, which blocks further hormone production; iodine preparations to prevent the transport of pre-formed hormones into circulation; beta-blockers to reduce the sympathetic effects of hormones on the body; steroids for adrenal insufficiency, addressing the autoimmune component and reducing the conversion of T4 to T3 [25,26,27]. Other modalities, including bile acid sequestrants and iodinated radiocontrast agents, have also been reported to be of use [25,27,28].

Conclusion

Patients with SDH who present to the hospital with thyroid storm due to multiple precipitating factors should be examined and managed promptly. Surgery during thyroid storm is a high-risk procedure but should be performed in emergency cases with careful preparation and vigilance, especially against possible complications and good postoperative care. The anesthesiologist's role in perioperative management is vital, especially in complicated cases. This result will lead to better patient outcomes.

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The authors have stated their absence of any conflicts of interest regarding this study.

Authors' contributions

All authors contributed to data analysis, article preparation, and paper revision and have collectively assumed responsibility for all aspects of this work.

Conflict of Interest

The authors have stated their absence of any conflicts of interest regarding this study.

Ethical consideration

The authors bear the responsibility for thoroughly investigating and appropriately resolving any questions pertaining to the accuracy or integrity of any section of the work.

Data availability

The article contains all the necessary data to support the results; no supplementary source data is needed.

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