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Thyroid Storm

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Introduction

Thyroid storm or thyrotoxicosis is a complex and life threatening condition with a mortality of 10 – 30% (Chiha, Samarasinghe, & Kabaker, 2015). There are several different ways that a person is able to develop thyrotoxicosis. These can be through an auto-immune process like Graves' disease, which is the most common (Min, Benjamin, & Cozma, 2014). Ideally patient's will see improvement with medical treatment within a few days, this is not always the case (Schreiber, 2017).

Treatment for thyrotoxicosis needs to be done within an intensive care unit due to the complexity and multiple systems involved. The personal should have a thorough understanding of the thyroid hormone cascade and close communication with the rest of the treatment team (Schreiber, 2017).

Signs and Symptoms

Patient's with thyrotoxicosis can present with varying degrees of symptomology. Commonly patients will have some type of "tachycardia, hyperthermia, central nervous symptom such as agitation, anxiety, delirium, or psychosis" (Conte et al., 2018, p. 1). In addition, there can be issues with nausea vomiting and diarrhea (Chiha et al., 2015).

As noted by Chiha et al., the Burch-Wartofsky Point Scale is a tool used to quantify the degree of severity or likelihood of thyrotoxicosis being involved with a patient (2015). A score the is greater that 45 means that thyrotoxicosis is likely and a score under 25 means thyrotoxicosis is unlikely (Chiha et al., 2015). Like all scoring tools the Burch-Wartofsky Point Scale should be done in the context of each patient's history and physical.

Pathophysiology

McCance and Huether efficiently broke done the production of thyroid hormone into 6 steps (2014).

1. Uniodinated thyroglobulin (a large glycoprotein) is produced by the endoplasmic reticulum of the follicular cells.
2. Tyrosine is incorporated into the thyroglobulin as it is synthesized.
3. Iodide (the inorganic form of iodine) is actively transferred (pumped) from the blood into the colloid by carrier proteins located in the outer membrane of the follicular cells. This active transport system is called the *iodide trap* and is very efficient at accumulating the trace amounts of iodide from the blood.
4. Iodide is oxidized and quickly attaches to tyrosine within the thyroglobulin molecule.
5. Coupling of iodinated tyrosine forms thyroid hormones. Triiodothyronine (T_3) is formed from coupling of monoiodotyrosine (one iodine atom and tyrosine) and diiodotyrosine (two iodine atoms and tyrosine). Tetraiodothyronine (T_4), commonly known as thyroxine, is formed from coupling of two diiodotyrosines.
6. Thyroid hormones are stored attached to thyroglobulin within the colloid until it is released into the circulation.

(McCance & Huether, 2014 p. 654)

The normal function of the thyroid has a negative feedback mechanism (McCance & Huether, 2014). As levels of free T3 and free T4 increase in the blood stream the production of thyroid releasing hormone and thyroid stimulating hormone decrease (McCance & Huether, 2014). Thyrotoxicosis develops when this negative feedback mechanize fails.

The most common disease process to cause thyrotoxicosis is Graves' disease (Schreiber, 2017). This is an autoimmune disease (Kahaly et al., 2018). Unlike other autoimmune diseases that attack and degrade the target organ, the antibodies that circulate and target the thyroid gland attach to receptors and stimulate the thyroid gland (Kahaly et al., 2018). Due to the antibodies not being produced in the hypothalamus or anterior pituitary, the negative feedback mechanism will not decrease how much the thyroid gland is stimulated.

The degree to which this autoimmune process affects and individual is dependent on the amount of antibodies in their system. If the concentration of antibodies ever exceeds the natural stimulus of the patient's thyroid stimulation hormone, symptoms will start to develop.

Significance of Pathophysiology

The significance of thyrotoxicosis comes into play when stabilizing and treating patients in the intensive care unit. An early consult to endocrinology to find the best means to inhibit the out of control thyroid hormone cascade is merited. As well as a consult to surgery if medical management is unable to suppress the thyroid gland (Uchida et al., 2015).

Inability for medical staff to effectively communicated, monitor, and recognize problems developing from thyrotoxicosis puts patients in harm's way (Schreiber, 2017).

Table 2. Diagnostic Criteria for Thyroid Storm.[a]

Thermoregulatory Dysfunction: Temperature, F	Score	Cardiovascular Dysfunction: Heart Rate, bpm	Score
99–99.9	5	90–109	5
100–100.9	10	110–119	10
101–101.9	15	120–129	15
102–102.9	20	130–139	20
103–103.9	25	≥140	25
≥104	30		
Central Nervous System Dysfunction	**Score**	**Cardiovascular Dysfunction: Heart Failure**	**Score**
Absent	0	Absent	0
Mild (agitation)	10	Mild (pedal edema)	5
Moderate(delirium, psychosis, extreme lethargy)	20	Moderate (bibasilar rales)	10
Severe (seizure, coma)	30	Severe (pulmonary edema)	15
Gastrointestinal and Hepatic Dysfunction	**Score**	**Cardiovascular Dysfunction: Atrial Fibrillation**	**Score**
Absent	0	Absent	0
Moderate (diarrhea, nausea/vomiting, abdominal pain)	10	Present	10
Severe (unexplained jaundice)	20		
Precipitant History	**Score**		
Absent	0		
Present	10		

[a] Adapted from Burch and Wartofsky[2] with permission of Elsevier. A score of 45 or greater is highly suggestive of thyroid storm; a score of 25 to 44 is suggestive of impending storm, and a score below 25 is unlikely to represent thyroid storm.

(Chiha, Samarasinghe, & Kabaker, 2015, p. 134)

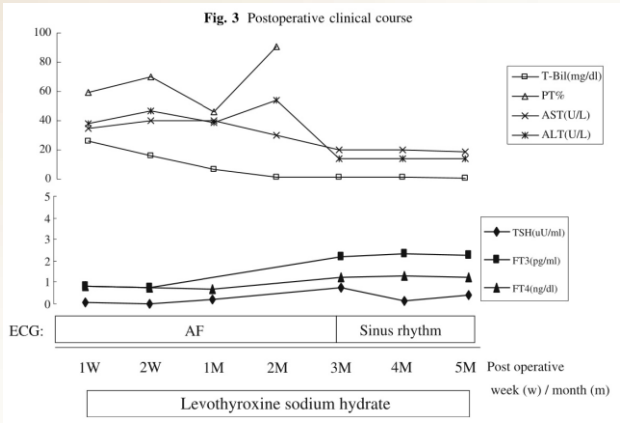
Case Study Needing a Thyroidectomy

A 37 year old patient presented to a hospital with a low thyroid stimulating hormone 0.005 μU/ml, free T3 pg/ml, and free T4 7.77 ng/ml (Uchida, Suda, & Ishiguro, 2015). This presentation is likely due to the fact that the patient was previously diagnosed with Graves' disease and stopped taking her medication 3 months prior (Uchida et al., 2015). Graves' disease is the most common cause of thyroid storm (Chiha et al., 2015).

The diagnosis of thyroid storm was given and her treatment included steroids, beta blockers, potassium iodide, methimazole, and plasma exchange (Uchida et al., 2015). This improved the hormone levels, but the patient continued to have hepatic failure (Uchida et al., 2015).

There was not enough of an improvement in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (Uchida et al., 2015). Uchida et al. postulated that the cause of the increase, despite treatment, is due to the relative hepatic hypoxia (2015). Due to the nature of the thyroid hormone cascade there is an increase in metabolic activity (McCance & Huether, 2014). The relative Hypoxia comes into play when to body is unable to provide enough oxygen for the new increase in metabolic activity. This in turn place the patient in a difficult situation of needing a procedure of a thyroidectomy that could exacerbate the initial problem that brought them in the first place.

For this patient, the present risk of sustained hepatic failure out weighted the risk of exacerbating the thyrotoxicosis and the hospital decided to go through with the thyroidectomy (Uchida et al., 2015). From the resection, the thyroid gland had large amount of swelling and an increase in the thyroid's follicular component (Uchida et al., 2015). Typically, the thyroid gland has about 2 months of hormones stored (McCance & Huether, 2014).



(Uchida et al., 2015, p. 113)

As noted in Figure 3, after the thyroidectomy there was a gradual improvement in the patient's AST and ALT levels. Also due to the removal of the thyroid gland, the patient needed to begin taking Levothyroxine as a substitute for the natural function of the Thyroid gland. At the 3 months mark the patient made significant progress in both laboratory values and a return to sinus cardiac rhythm. Strong follow up with this patient is encouraged due to the previous noncompliance that precipitated this event.

Nursing Care

In the treatment of thyrotoxicosis there are several aspects of the hormonal cascade that need to be monitored and kept in check. Predominantly the cardiovascular system, serum glucose, thermoregulation, and mental status are key areas of assessment (Schreiber, 2017).

Looking at the cardiovascular system, the thyroid hormone cascade increases the heart's activity (McCance & Huether, 2014). This is done by the increase in activity of the electrolyte pumps in the sarcolemma (McCance & Huether, 2014). This puts a heavy burden on the heart and many tachyarrhythmias are expected.

Within the hyper metabolic state of thyrotoxicosis, hyperglycemia is more likely than hypoglycemia (Schreiber, 2017). Considering that both are possible depending on the available stores of glucose and the high metabolic function of the body, frequent checking of the patient serum glucose is needed.

When the body is in a high metabolic state hyperthermia becomes a concern. As side from medication interventions, ice packs and cooling blankets should be use to help further control a patient's temperature (Schreiber, 2017).

Lastly, the patient's overall mental state should be closely monitored (Schreiber, 2017). This is a good overall evaluation of how well each aspect is being regulated. If the patient's mental status sharply declines, one of the aforementioned areas is likely in need of attention.

Medications

In addition to monitoring these systems, there are several medications that a nurse should expect to be ordered for a patient in thyrotoxicosis. These may include thionamides, anti-adrenergics, glucocorticoids, iodine compounds, and antipyretics (Schreiber, 2017).

The prescribing provider will likely pick between Propylthiouracil, Methiazole, Carbimazole as their choice of thionamide (Schreiber, 2017; Ross, 2019). This will help to prevent the natural process of the thyroid cascade and limit the severity of the thyrotoxicosis. As an example, Propylthiouracil inhibits deiodinase D1 which is dominant in a hyperthyroid state (Chiha et al., 2015).

Due to increased level of free T3, there will be an increase activity of the cardiovascular system and tachyarrhythmias (McCance & Huether, 2014). In light of the tachyarrhythmias, Metoprolol or Propranolol will likely be ordered (Nakashima, Kenzaka, Okayama, & Kajii, 2014; Chiha et al., 2015). These anti-adrenergics will protect the heart from overworking itself and developing heart failure (Kahaly et al., 2018).

The use of glucocorticoids is an additional inhibition of the thyroid hormone cascade. The steroids effect inhibits the change of free T4 to free T3 (Chiha et al., 2015).

Iodine compounds are slightly counterintuitive and need to be administered with caution. Iodine is a critical component within the thyroid hormone cascade (Vitti, 2019). Too much or too little Iodine can cause problems with the function of the thyroid (Vitti, 2019). Due to this, potassium iodine is given with the expectation of being another aspect of inhibition to the thyroid hormone cascade (Schreiber, 2017). It most be given by at least an hour after the thionamide is given (Ross, 2019). If this delay dose not happen, the iodine could be used for making more thyroid hormone (Ross, 2019).

Lastly, Acetaminophen is a great choice as an antipyretic (Schreiber, 2017). Due to the nature of thyrotoxicosis causing multiorgan failure, liver functions should be monitored with acetaminophen's use (Schreiber, 2017).

Conclusion

Patient's come into the hospital of a number of reasons. Some of them are as simple as a sprained ankle or as complex as adult respiratory distress syndrome. Thyrotoxicosis is clearly as complex as adult respiratory distress syndrome and need skilled staff to manage and treat in the intensive care unit.

Staff need to be aware of the thyroid hormone cascade and the common treatment for thyrotoxicosis (Schreiber, 2017). This way patient will be given the best chance possible given the dire circumstances.

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