Case Study Scenarios

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Scenario 6

1. *The patient exhibited classic signs of Type 1 diabetes. Explain the pathophysiology of "polydipsia."*

Polydipsia is the increase in thirst as a result of high blood glucose that raises the osmolarity of the blood, thus making it more concentrated. In most cases, it is often an early symptom of Type 1 diabetes. The onset of Type 1 diabetes can explain the pathophysiology of Polydipsia. The onset of diabetes mellitus causes the blood sugar levels to get too high, increasing the concentration of blood sugar and make one feel thirsty, respective of how much water one drinks. In the case study, the patient has been experiencing unquenchable thirst, an indication of an imbalance in the osmotic concentration due to the loss of a significant amount of fluid.

2. *The patient exhibited classic signs of Type 1 diabetes. Explain the pathophysiology of "polyuria."*

The pathophysiology of polyuria can be explained by the onset of Type 1 diabetes, which causes osmotic diuresis when glucose levels are so high that glucose is excreted in the urine. Water follows the glucose concentration passively, resulting in abnormally high urine output. Notably, the individuals with Polydipsia urinate frequently and pass large volumes of urine- more than 3 liters daily compared to the normal urine output in adults of approximately 1 to 2 liters a day. In this scenario, the patient experienced frequent urination than normal, an indication of polyuria.

3. *The patient exhibited classic signs of Type 1 diabetes. Explain the pathophysiology of "poluphagia"*

The pathophysiology of polyphagia can be explained by the onset of uncontrolled Type 1 diabetes, which causes blood glucose levels to remain extremely high. As a result, the glucose from the blood cannot enter the cells due to either a lack of insulin or insulin resistance- so the body cannot convert the food eaten into energy. The lack of energy causes an increased level of hunger. Polyphagia is characterized by weight loss. In the scenario, the gets tired quickly, an indication of uncontrolled lack of energy as a result of polyphagia.

4. *The patient exhibited classic signs of Type 1 diabetes. Explain the pathophysiology of "weight loss"*

The pathophysiology of weight loss as a result of Type 1 diabetes can cause weight loss because the insufficient insulin prevents the body from obtaining glucose from the blood into the body's cells to utilize as energy. When this incidence happens, the body begins to burn fat and muscle for energy, thus leading to a reduction in the overall body weight. In the scenario, the patient lost weight despite eating more, an indication that there is insufficient insulin that prevents the body from absorbing the necessary glucose that can be broken down into energy.

5. *The patient exhibited classic signs of Type 1 diabetes. Explain the pathophysiology of "fatigues can be*

The pathophysiology of fatigue in Typ2 1 diabetes can be explained by the onset of Tape 1 diabetes, which results from the inability of the body to get enough glucose from the blood into body cells to meet the energy requirements. As a result, the individual will often experience fatigue, as noted in the patient in this scenario.

6. *How do genetic and environmental factors contribute to the development of Type 1 diabetes?*

Both environmental and genetic factors play a significant role in the development of diabetes. Environmental factors such as deficiency in vitamin-D, exposure to enteroviruses,

unhealthy diet, polluted air, and damage to immune cells trigger an autoimmune response. Obesity due to a lack of exercise is accompanied by a decrease in muscle mass and induces insulin resistance. Stress hormones, such as adrenaline and cortisol, can raise blood sugar. Genetic factors include a family history of diabetes. The risk increases with the number of affected family members due to shared genetic factors such as insulin resistance. In this scenario, the patient has a family history of diabetes. The maternal uncle had some sugar diabetes problems.

Scenario 7

1. *The hormone involved in the intermediary metabolism, exclusive of insulin can participate in the development of diabetic ketoaciosis (DKA) are epinephrine, glucagon, cortisol, growth hormone. Describe how they participate in the development of DKA.*

The hormone epinephrine participates in the development of KDA aiding the liver to convert *stored* glycogen to glucose and release it, thus raising blood sugar levels leading to KDA. Besides, glucagon has its significant physiological action at the liver to break down glycogen, which contributes to increased ketones and acidosis present in KDA. Further, cortisol promotes gluconeogenesis, which contributes to hyperglycemia noticed in KDA. Lastly, growth hormone participates in the development of KDA by enhancing the breakdown of triglyceride into free fatty acids and gluconeogenesis, which is the primary cause for the elevation in serum glucose levels in KDA. In the scenario, the blood glucose is higher than normal.

Scenario 8

1. *Explain the underlying process that lead to HHNKS or HHS*

HHNKS or HHNS is an emergency that is caused by extremely high blood sugar, often over 600 mg/gL. The condition is caused by dysregulation, which results in the process of

ketosis and the release of ketones into the blood. Ketones turn the blood acidic; a condition referred to as diabetic ketoacidosis (DKA). In the scenario, the patient complained of frequently visiting the bathroom and feels very weak. Besides, the patient stated that he could not afford the insulin, meaning that he had insufficient insulin, meaning he had Type 2 diabetes.

Scenario 9

1. *How would you differentiate Cushing's disease from Cushing's syndrome?*

Cushing's syndrome is caused by any condition that causes the adrenal gland to produce excess cortisol regardless of the cause. Cushing's syndrome is characterized by facial and torso obesity, high blood, muscle weakness, irritability, excess hair growth, as noticed in the patient in this scenario. On the other hand, Cushing's disease is a specific form of Cushing's syndrome that can be differentiated from Cushing's syndrome when a pituitary tumor secretes excessive ACTH.

Scenario 10

1. *What is the pathogenesis of primary hyperaldosteronism?*

Primary hyperaldosteronism refers to a rennin-independent increase in the secretion of aldosterone. The condition occurs the adrenal gland secretes excessive aldosterone hormone that results in the increases in sodium reabsorption and loss of potassium and hydrogen ions. The imbalance causes the body to hold too much water, increasing the blood volume and blood pressure. In this scenario, the patient had elevated blood pressure, hypokalemia, and hypervolemia, which can explain the increase in blood volume and pressure.

Scenario 11

1. *What is the basic underlying pathophysiology of Type II diabetes?*

The basic underlying pathophysiology is characterized by peripheral insulin resistance, impaired regulation of hepatic glucose production, and a decrease in β-cell function, consequently leading to β -cell failure. Impaired insulin secretion leads to a reduction in glucose responsiveness. Impaired glucose tolerant is induced by a decrease in glucose responsiveness in the early phase of insulin secretion. Impaired insulin secretion is progressive and involves glucose toxicity and lipo-toxicity, which, when not treated, cause a reduction in pancreatic β-cell, which affects the long –term control of blood glucose, leading to Type II diabetes in the long-run. The patient then begins to show the symptoms of polyuria, polyphagia, polydipsia, and weight loss, as in the case of the patient in this scenario.

Scenario 12

1. *What causes diabetes insipidus (DI)?*

Diabetes insipidus is caused by problems with a chemical known as vasopressin (AVP), which is also referred to as antidiuretic hormone (ADH). Any damage to the pituitary gland or hypothalamus from surgery, tumor, head injury, or even any other disease leads to diabetes insipidus by the impact the normal production, storage, and release of ADH as noted in this scenario.

Scenario 13

1. *Explain how negative feedback loop controls thyroid levels.*

Negative feedback loop controls thyroid levels by increasing thyroid hormones above a particular threshold, thus making the TRH-secreting neurons in the hypothalamus to be inhibited and stopped from secretion. This way, the concentration of the hormone in the blood is maintained. When this process fails, one can develop Hyperthyroidism, a condition characterized by too much production of thyroxine hormone, which accelerates the body's metabolism and

causing unintentional weight loss and irregular heartbeat, as noted in the patient in this scenario. The patient experienced racing heartbeat accompanied by perspiration, hyperactivity, and palpitation, an indication of a lack of hormonal control.

Scenario 14

1. *How did the patient develop thyroid storm? What were the patient factors that lead to the development of thyroid?*

Thyroid storm develops in patients with hyperthyroidism but are not receiving recommended treatment. The condition is characterized by excessive overproduction of the two hormones produced by the thyroid gland. In this scenario, the patient has hyperthyroidism, and she did not take the prescribed antithyroid medication and also refused thyroidectomy. The patient factors that lead to the development of thyroid include manifestations of symptoms such as racing heartbeat, perspiration, hyperactivity, and severe palpitation, and heat intolerance.

Scenario 15

1. *What causes hypothyroidism?*

Hypothyroidism occurs when the body fails to produce enough thyroid hormones, which help the body to regulate and utilize energy. The common cause of hypothyroidism is an unregulated autoimmune response, which happens when the body confuses the normal, healthy cells for living cells. In case the autoimmune is not regulated, the body's immune system can attack healthy tissues, causing severe health issues like hyperactivity, as witnessed by the patient bursting into tears without any reason, decreased appetite, sleep problems, and depression.

Scenario 16

1. *What causes myxedema coma?*

Myxedema coma often occurs as a result of long-standing, undiagnosed or untreated hypothyroidism and is often precipitated by a systemic disease. Myxedema coma can result from any cause of untreated hypothyroidism, with the most common being chronic autoimmune thyroiditis. In this case scenario, the patient was diagnosed with hypothyroidism, which had not been treated, thus leading to myxedema coma.

Scenario 17

1. *What is Pheochromocytoma and how does it cause the classic symptoms the patient presented?*

Pheochromocytoma is a tumor in the adrenal gland tissue that results in the release of too much epinephrine and norepinephrine hormones that regulate heart rate, metabolism, and blood pressure. Pheochromocytoma causes the classic symptoms the patient presented through the overproduction of the epinephrine and norepinephrine hormones that increase the blood pressure, which causes severe headache and palpitation.

1. *What are the treatment goals for managing pheochromocytoma?*

The treatment goals for managing pheochromocytoma include the removal of the tumor and control of blood pressure. The primary treatment is surgery to remove the tumor. However, before the surgery, one is given specific blood pressure medications that block the actions of the high-adrenaline hormones to reduce the risk of developing severe high blood pressure during surgery.