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Evaluation of Some Hematological and Biochemical Changes in Iodine-Deficient Calves

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ABSTRACT

Iodine deficiency is a significant issue affecting calves in Egypt. This study investigates the impact of iodine deficiency on various hematological and biochemical parameters. A total of 100 calves, aged between 9 months and 2 years, exhibiting clinical signs such as general weakness, dullness, reduced appetite, alopecia, and weight loss, were sourced from the New Valley Governorate. 20 healthy calves, free from external and internal parasites, were selected from a farm in the Assiut Governorate as control animals. Our findings revealed a significant reduction in urinary iodine levels, as well as in T3, T4, glucose, glucagon, insulin, total protein, albumin, globulin, Hb, and R.B.C.s levels in the affected calves compared to the control group. Conversely, there was a significant increase in levels of TSH, ALT, AST, and urea, W.B. Cs, as well as a non-significant elevation in creatinine levels in the diseased calves diagnosed with iodine deficiency compared to the control group.

INTRODUCTION

Cattle hold significant economic importance globally due to their pivotal roles in meat, milk, and leather production **(FAO 2008).** Nutrition is essential for our overall well-being and the proper functioning of all organs and systems. Both macronutrients and micronutrients are indispensable in the diet and play vital roles in nutrition. Macronutrients, which include lipids, carbohydrates, and proteins, are necessary for energy production and

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the synthesis of hormones that regulate metabolic processes **(Espinosa-Salas and Gonzalez-Arias 2024).** Micronutrients, consisting of essential vitamins and minerals, are needed in small quantities and are involved in various biochemical processes, such as gene transcription, enzymatic and hormonal reactions, cell protection, signal transduction, and both primary and secondary metabolism **(Krishnamurthy et al. 2021).**

Mineral deficiencies or imbalances are prevalent across various settings and result in significant economic losses. Research indicates that the losses attributed to trace element deficiencies are as important as those caused by infectious and parasitic diseases **(Ibrahim et al. 2017).** Iodine, in particular, is essential for the synthesis of thyroid hormones, which are crucial for regulating energy metabolism and are also vital for the reproductive and productive performance of animals (**Cardoso et al. 2021).**

The clinical image of iodine deficiency varies from weak calves, Goiter, changes in hair coat, partial or complete hair loss, increased dryness and skin wrinkling in calves, increase in the somatic cell count, hoof diseases, hoof rot and hair loss in adults, depending on the age of the animals **(Żarczyńska and Świerczyński 2023).** The thyroid gland is a crucial endocrine organ that plays a vital role in regulating physiological processes essential for health and well-being. It is responsible for the synthesis and secretion of thyroid hormones, namely thyroxine (T4) and triiodothyronine (T3), which are integral to the functioning of all systems throughout the body **(Armstrong et al. 2024).** For the critical role that iodine plays in the growth and metabolic processes of calves, alongside the significant economic repercussions stemming from its deficiency in developing livestock, this study was designed to explore and address these concerns.

This study was conducted to estimate iodine levels in the urine of the studied calves and to measure T3, T4, TSH levels, glucose, glucagon and insulin. Also, aimed for estimation of the levels of ALT, AST, Creatinine, Urea, Total protein, albumin and globulin in the serum of the studied calves and to evaluate some hematological parameters (R.B. Cs count, Hb content, PCV% and W.B. Cs count).

MATERIALS AND METHODS

1-Ethical Consideration:

This work was approved with ethical approval number 6/2025/0336 by the research ethical committee of the Faculty of Veterinary Medicine, Assiut University, Egypt.

2-Animals

This study comprised a total of 100 calves, aged between 9 months and 2 years, representing both sexes. The animals were sourced from the New Valley governorate, specifically the El-Dakhla and El-Kharga oases. The primary clinical signs observed in calves suffering from iodine deficiency included general weakness, poor body condition, weight loss, and alopecia. The calves consumed water from wells located in the El-Dakhla and El-Kharga oases. Throughout the study period, all animals were confirmed to be free of both external and internal parasites. 20 healthy calves were selected from farms in Assiut governorate based on their case history and clinical examination. They were in good body condition and free from external and internal parasites.

3- Samples

Two types of samples were collected for this study.

3.1- Blood sample

Two blood samples were collected from each calf. One with EDTA as an anticoagulant for hematological analysis and the second blood samples (7.5 ml) without anticoagulant for biochemical analysis were collected from the jugular vein of each animal in clean glass vials after clipping and disinfecting the area of the vein.

3.2- Urine sample

Samples of urine collected from calves for estimation of iodine level (ug/l) by using of

urine catheter. The samples were sent to the laboratory immediately after they were taken.

2- Adopted methods

2.1- Biochemical and hematological analysis

2.1.1-Biochemical analysis

About 7.5 ml of blood was put in a centrifuge tube without anticoagulant and blood samples were allowed to stand for clotting for 20 minutes at 37˚C. Then samples were centrifuged at 2000-3000 rpm for 10-15 minutes. Serum aspirated by Pasteur pipette into Eppendorf tubes and stored at -20˚c until testing **(Coles 1986).** Care measures were considered to keep these samples suitable for biochemical analysis. These measures included constant freezing, protection from light, and thawing when needed **(Norbert 1986).**

Serum was used for the detection of triiodothyronine (T3) and thyroxine (T4) according to the method described by Chopra et al. (1979). Thyroid-stimulating hormone (TSH) was measured following the protocols of Caldwell et al. (1985) and Hopton and Harrop (1986), using ELISA kits supplied by Sigma (Catalog number: MDO28-96). Glucose levels were determined according to the method described by Tietz (1995). Liver enzyme activities, including AST and ALT (U/L), were measured based on the methods of Breuer (1996) and Young (1990). Kidney function tests included urea (mg/dL), measured according to Young (2001), and creatinine (mg/dL), determined following Tietz (1986). Total protein and albumin levels (g/dL) were quantified using the methods described by Tietz (1990, 1994). Glucagon levels were measured according to Okuno et al. (1993), and all of these parameters were assessed using a spectrophotometer (RoBonic Prietest ECO biochemistry analyzer). Serum globulin levels were calculated by subtracting albumin values from total protein. Insulin levels were determined by the ELISA method described by Wilson and Miles (1977).Hematological RBCs, Hb, HCT, MCV, MCH, MCHC and WBCs were determined by using the automatic cell counter Sysmex (2000 IV). Iodine concentration in the urine was measured with an ion chromatography device (ICS 3000, Dionex Corp, USA) according to Aumont and Tressol (1986)**.**

Statistical analysis

The analysis of the hematobiochemical parameters was conducted rigorously to calculate means, standard deviations, and P values. This was accomplished using an independent sample T-test and the SPSS program, following the established methods of **Sendecor and Cochran (1980).**

RESULTS

The findings of the conducted study showed a notable decline in iodine level in the urine of diseased calves (7.2 ug/l) when compared to the control, which was (83.4 ug/l) as shown in Table 1.

The results of the obtained study proved a significant decrease in T3 and T4 levels, which were (80.2 ng/dl and 1.08 ng/dl) in diseased calves when compared to control calves, which were (137.4 ng/dl and 6.4 ng/dl) while, significantly higher TSH levels were seen., which were (18.8 µ/iu/ml) in diseased calves when compared to control calves, which were (5.7 µ/ iu/ml) as demonstrated in Table 1.

The outcomes of the obtained study revealed that there was a great decrease in glucose and glucagon levels in diseased calves, which were 67.4mg/dl and 51.4 µiu/ml when compared to control ones, which were 77.2 mg/dl and 83.6 µiu/ml. there was a non-significant decrease in insulin levels in diseased calves, which were 28 mg/dl when compared to the controls, which were 29 mg/dl, as shown in Table 1.

There was a significant increase in urea level (42.2 mg/dl) and a non-significant increase in creatinine level (1.6mg/dl) in diseased calves when compared to those control ones, which were (36.2mg/dl & 1.5 mg/dl); respectively.

The ALT and AST values significantly increased in diseased calves (82.2 u/l and 107.4 u/l) compared to the controls (58.6 u/l and 83.6 u/l), respectively.

The results proved that there was a significant decrease in total protein, albumin and globulin levels in diseased calves, which were (6 g/dl, 2.9 g/dl and 3.2 g/dl) when compared to control ones, which were (7.5 g/dl, 3.4 g/dl and 4.1 g/dl); respectively, as described in Table 1.

There was a notable decline in R.B. Cs count, Hb content and PCV% in the diseased calves, which were ($2.9x10^6$/ml, 8 g/dl and 28.4%) when compared to control calves, which were ($3.5x10^6$/ml, 11 g/dl and 35%); respectively and there was a significant increase in W.B. Cs (83.6×10^3/ml) when compared to the controls (30×10^3/ml) as shown in Table 1.

Table 1. Evaluation of the level of some haemato-biochemical parameters in iodine-deficient calves

Group Parameters	Control	Diseased
Iodine in urine (ug/l)	83.4±11	7.2±2**
T3 (ng/dl)	137.4±22	80.2±10**
T4 (ng/dl)	6.4±1.4	1.08±0.4**
TSH (µ/iu/ml)	5.7±0.9	18.8±3**
Glucose (mg/dl)	83.6±4.4	67.4±3.4**
Glucagon (µiu/ml)	77.2±5.4	51.4±2.3**
Insulin (mg/dl)	29±2.2	28±1.5
ALT (u/l)	58.6±3	82.2±7.6**
AST (u/l)	83.6±5.7	107.4±3.6**
Urea (mg/dl)	36.2±3	42.2±2.9**
Creatinine (mg/dl)	1.5±0.2	1.6±0.2
Total protein (g/dl)	7.5±0.2	6±0.4**
Albumin (g/dl)	3.4±0.2	2.9±0.1**
Globulin (g/dl)	4.1±0.2	3.2±0.3**
R.B. Cs count (10^6/ml)	3.5±0.1	2.9±0.1*
Hb. Content (g/dl)	11±0.4	8±0.6**
P. C. V%	35±1.6	28.4±2**
W.B. Cs (10^3/ml)	30±2.2	83.6±5**

*Significant at P value ≤ 0.05 **significant at P value ≤0.001

DISCUSSION

The result of the obtained study revealed a decrease in iodine levels in the urine of diseased calves relative to the control. These results agreed with **Kaur and Randhawa (2004)**, who reported that there was a significant decrease in the level of urinary iodine in deficient cows, which were (57.27 µg/L) when compared to control cows, which were (105.30 µg/L).

Urinary iodine concentration is the most accurate indicator of both the biological availability of iodine in the diet and of iodine status of an organism, as this is due to the significant correlation between iodine intake and excretion **(Żarczyńska and Świerczyński 2023).**

Inadequate dietary iodine levels and their interaction with other minerals. Furthermore, environmental factors such as iodine levels in soil and grazing pastures are the most common cause of iodine deficiency in farm animals **(Tadayonfar and Noaman 2013). Bogdanova et al. (2020)** pointed out that for calves, the primary sources of trace elements are feed and water. The diminished levels of trace elements,

including iodine, in soils and groundwater contribute to their reduced content in plant feed, ultimately resulting in deficiencies within the bodies of animals.

The current study showed a significant decrease in T3& T4 levels in diseased calves when compared to the control calves. These results agreed with those reported by **Davoodi et al. (2022)** and **Lafta et al. (2023),** who submitted that there was a significant decrease in the levels of T3 and T4 in iodine-deficient animals when compared with the control. These results may be linked to iodine deficiency in animals, which disrupts the biosynthesis of thyroid hormones **(Evglevskiy et al. 2021a).**

The low serum concentrations of thyroid hormones observed in hypothyroid calves may be attributed to endemic or dietary iodine deficiency, resulting in stunted growth and metabolic disturbances **(Kotwal et al. 2007).** In addition, a deficiency of several trace elements may affect serum thyroid hormone concentration. Copper deficiency may cause atrophy of the intestinal villi; zinc deficiency may induce anorexia. These deficiencies may impede digestive absorption, reduce feed efficiency, and lower thyroid hormone secretion **(Keles et al. 2006).**

The most useful test for assessing thyroid function is the TSH; The measurement of serum TSH is considered to have the highest sensitivity and specificity among blood tests used in the assessment of hypothyroidism. Therefore, it is advisable to utilize this test as the initial screening method **(Khalaf 2020) .**

The results of the current study proved a significant increase in the level of TSH in the diseased calves when compared to the control calves. This result agreed with **Guyot et al. (2007)**, who proved an increase in TSH in hypothyroidic calves when compared to control ones and agreed with that reported by **Kadum and Luaibi (2017)** and **Lafta et al. (2023)** proved an increase in the level of TSH in iodine-deficient animals when compared to the control. **Mostaghni and Badaee (2005)** highlighted that a deficiency of iodine within the calves' bodies prompts the pituitary gland to excessively produce thyroid-stimulating hormone (TSH). This response is the gland's attempt to stimulate the thyroid to compensate for the inadequate levels of thyroid hormones, which are vital for numerous physiological processes.

The present investigation revealed a substantial drop in glucose and glucagon levels in diseased calves when compared to the control calves. The result of the decrement in glucose agreed with those mentioned by **Mostaghni et al. (2008)**, **Joshi et al. (2017), Mousa et al. (2021), Lafta et al. (2023)** and **Yevglevsky and Gostev (2021)**, who mentioned that there was a significant decrease in the level of glucose in hypothyroid calves, which was 1.2-1.4mmol/l when compared to control ones, which was 2.2-3.3mmol/l.

Decreased glucose levels in diseased calves may result from the inhibition of gluconeogenesis and glycogenolysis, as well as inadequate absorption of glucose from the gastrointestinal tract due to thyroxine deficiency. Alternatively, it may also be linked to reduced activity of hepatic glucose-6-phosphatase in cases of hypothyroidism **(Gupta et al. 2013).**

The decrement of glucagon agreed with that reported by **El-Nile et al. (2018),** who proved a decrease in the level of glucagon in calves with decreased micro and macro elements, which was 69 µIU/m when compared to control ones, which was 78 µIU/m. Glucagon is a peptide hormone produced by the pancreas that plays a crucial role in glucose metabolism and homeostasis. Its secretion occurs in response to hypoglycemia, making it the counter-regulatory hormone to insulin. The fundamental function of glucagon is the stimulation of the liver's glucose production by enhancing glycogenolysis and gluconeogenesis while inhibiting glycogen synthesis. Additionally, glucagon promotes increased lipolysis in adipose tissue **(Fosgerau et al. 2011 and Mullur et al. 2014).** Furthermore, in the case of iodine deficiency, decreased glucose levels are associated with lowered glucagon concentrations. Thyroid hormones influence pancreatic beta-cell function, which is responsible for insulin secretion and increase glucagon release by pancreatic α-

cells. In hypothyroidism, reduced thyroid hormone levels may result in decreased insulin and glucagon levels in blood **(Biondi and Cooper 2008; Nishi 2018).**

Insulin deficiency may be linked to magnesium deficiency, as magnesium plays a critical role in insulin action. It influences the activity of insulin receptors, glucose uptake in cells, and the proper functioning of pancreatic beta cells, which are essential for insulin secretion. Research has shown that a magnesium deficiency is associated with insulin resistance and impaired insulin secretion **(Akimbekov et al. 2024).**

The results of the study proved that there was a marked increase in ALT and AST levels in diseased calves when compared to the control one. These results came by those reported by **Mostaghni et al. (2008)** and **Gupta et al. (2013)**, who proved that there was a significant increase in the level of ALT and AST in hypothyroid animals when compared to the control ones.

The relationship between the thyroid gland and the liver is intricate and multifaceted. The liver contributes to the activation and bioavailability of thyroid hormones by synthesizing transport proteins such as thyroxine-binding globulin and albumin **(Piantanida et al. 2020; Ritter et al. 2020).** Additionally, the liver is the primary site for the metabolism of thyroid hormones, where T4 undergoes conversion into T3 **(El-Kabbany et al. 2012).** Thyroid hormones regulate the basal metabolic rate of hepatocytes and thus modulate the hepatic function and the liver, in turn, metabolizes the thyroid hormones and regulates their actions. Therefore, thyroid dysfunction may disturb the hepatic functions and in turn, hepatic diseases adversely affect the thyroid function **(Malik and Hodgson 2002).**

The rise in liver enzyme levels may be linked to decreased metabolic activity or heightened extracellular leakage from the damaged hepatocytes in affected calves. The altered cholesterol metabolism associated with hypothyroidism impacts the permeability of hepatic cell membranes and the biliary excretory system, ultimately resulting in elevated enzyme activity **(Singh et al. 2003).**

This study showed a significant increase in urea level and a non-significant increase in creatinine level in diseased calves with hypothyroidism when compared to the control ones. This result agreed with that reported by **Suher et al. (2005), Lippi et al. (2008)** and **Katta (2015).**

There is a well-established interaction between thyroid and kidney functions. Thyroid hormones play a crucial role in the growth and functioning of the kidneys, while the kidneys are essential for the metabolism, degradation, and excretion of thyroid hormones **(Yu et al. 2007).** Hypothyroidism can impact renal blood flow, glomerular filtration rate, water and electrolyte balance, as well as the structure of the kidneys **(Vargas et al. 2006).** The result of increased urea level in hypothyroidic calves came under that reported by **Mostaghni et al. (2008)** who proved that the increase in urea level may be due to glomerular lipidosis and mild to moderate degrees of degenerative changes in the tubular epithelium of diseased animals with hypothyroidism or lipid clogging of renal glomeruli causing progressive renal failure in diseased animals**. Basu and Mohapatra (2012)** and **Mariani and Berns (2012)** reported that in the case of hypothyroidism, there was a decrease in glomerular filtration rate and renal blood flow, which is associated with a decrement in the creatinine clearance from the blood of diseased animals.

This study proved that there was a significant decrease in total protein, albumin and globulin levels in diseased calves when compared to control one. This result was consistent with that reported by **Yevglevsky and Gostev (2021),** who mentioned that there was a significant decrease in the level of total protein in hypothyroidic calves when compared to the control calves. This reduction in protein parameters could be attributed to a decrease in the basal metabolic rate due to hypothyroidism or hepatic and renal impairment associated with hypothyroidism **(Sokkar et al. 2000).** Additionally, **Barsham et al. (2005)** reported that the major effect of T3 and T4 is to stimu-

late protein synthesis and promote a positive nitrogen balance. Consequently, hypothyroidism is often associated with deficiencies in total protein, albumin and globulin levels.

Hematological analysis of the blood revealed a significant decrease in total erythrocytic count, Hb, and PCV, while there was an elevation in the levels of the total leukocytic count in diseased calves when compared to control calves. Similar findings were detected by **Sokkar et al. (2000)** and **El-Nile et al. (2018).**

The reduction in blood parameters may be owing to the direct impact of thyroid hormones on these parameters, as they stimulate the precursors of erythrocytes and indirectly enhance the production of erythropoietin **(Szczepanek-Parulska and Hernik 2017).** Additionally, a decrease in thyroxine levels is associated with a lowered basal metabolic rate and reduced oxygen consumption by erythrocytes, which affects and shortens their normal lifespan, leading to fragility and hemolysis **(Sokkar et al. 2000).**

Insufficient iodine levels can hinder the body's ability to efficiently transport and deliver oxygen to tissues, disrupting cellular respiration and energy production. As a consequence, this impairment can ultimately contribute to the development of anemia, characterized by reduced hemoglobin levels and diminished oxygen-carrying capacity **(Zimmermann et al. 2003).**

Thyroid hormones play a crucial role in stimulating erythrocyte precursors by enhancing the production and secretion of erythropoietin, as well as promoting the proliferation of erythroid progenitors. Consequently, in cases of hypothyroidism, there is a notable decrease in the levels of red blood cells **(Szczepanek-Parulska and Hernik 2017).** Anemia is attributed not only to nutritional deficiencies but also to a reduction in thyroid hormones, which leads to decreased stimulation of erythrocyte precursors in the bone marrow. This results in a diminished oxygen supply to various tissues and a reduction in erythropoietin levels **(Ashraf 2011 and Schindhelm 2013).** Approximately 25% to 30% of hypothyroid calves experience anemia due to erythropoietin deficiency **(Cunningham 2002).**

Another factor contributing to anemia may be attributed to a significant positive correlation between the serum levels of zinc and iron and hemoglobin concentration. Since zinc and iron reduction impair the synthesis of Hb, as in the case of Fe-deficiency anemia **(Nhien et al. 2006),** the decline in the RBC count is often linked to selenium deficiency, as selenium performs a vital role in synthesizing the cell membrane of RBCs **(Radostits et al. 2007).** The increase in total leukocytic count in iodine-deficient calves may be attributed to reduced animal appetite and malnutrition **(Malik et al. 2013).**

In hypothyroidism, T-cell function and humoral immunity are reduced. This decline in local immune response makes the skin more vulnerable to infections. As a result, the skin becomes more susceptible to secondary bacterial, fungal, or parasitic infections, which can lead to an elevation in total leukocyte count **(Roopali et al. 2020**).

CONCLUSION

Iodine deficiency is a critical problem affecting the calves' productivity in Egypt. It was associated with the decrement of glucose, glucagon, insulin, R.B. Cs, total protein, albumin and globulin levels in diseased calves when compared to control one while, there was a significant increase in ALT, AST, W.B. Cs, urea level and non-significant increase in creatinine level in diseased calves with hypothyroidism when compared to those control ones.

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