Effect of Type 1 Diabetes On Serum Electrolytes (Sodium and Potassium) Levels and Testosterone Hormone in Human Male Subjects

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Abstract

Aim: The aim of this study is to know the effect of type 1 diabetes on serum electrolytes (Sodium and Potassium) and its effect on testosterone (male reproductive hormone).

Methodology: The study was performed on 20 normal male subjects and 20 diabetic male subjects. 10ml of the blood samples of all subjects were collected into a properly labeled plain sample bottles. The blood samples were spun on collection and serum decanted in to fresh plain bottle. The serum was then assayed for Sodium, Potassium and Testosterone. Further statistical analysis were conducted, this was carried out using the student’s Test with the significance test.

Result: From the result, it was discovered that there was a significant increase (P<0.05) in the level of sodium ion concentration, a little or no corresponding increment in the potassium ion concentration; and a significant decrease in the level testosterone in the diabetic male subjects when compared to the normal subjects.

Conclusion: This implies that Diabetes mellitus poses a kind of risk to serum electrolytes (sodium and potassium) and testosterone.

Introduction

Diabetes mellitus, or simply diabetes, is a group of metabolic diseases in which a person has high blood sugar, either because the body does not produce enough insulin, or because cells do not respond to the insulin that is produced. There are three main types of diabetes mellitus (DM). Type 1 DM results from the body’s failure to produce insulin, and presently requires the person to inject insulin or wear an insulin pump. Type 2 DM results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency. This form was previously referred to as non-insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes". The third main form, gestational diabetes occurs when pregnant women without a previous diagnosis of diabetes develop a high blood glucose level. It may precede development of type 2 DM. In addition to the aforementioned types of diabetes, there are also additional "other specific types".[1] The "other specific types" are a collection of a few dozen individual causes.[1] The rare disease diabetes insipidus has similar symptoms as diabetes mellitus, but without disturbances in the sugar metabolism.

Diabetes mellitus type 1 (type 1 diabetes, T1DM, formerly insulin dependent or juvenile diabetes) is a form of diabetes mellitus that results from autoimmune destruction of insulin-producing beta cells of the pancreas,[2] The subsequent lack of insulin leads to increased blood and urine glucose. The classical symptoms are polyuria (frequent urination), polydipsia (increased thirst), polyphagia (increased hunger), and weight loss.[3] Incidence varies from 8 to 17 per 100,000 in Northern Europe and the U.S., with a high of about 35 per 100,000 in Scandinavia, to a low of 1 per 100,000 in Japan and China[4] and about 3.9% of Nigeria population[5]. Eventually, type 1 diabetes is fatal unless treated with insulin. Injection is the most common method of administering insulin; other methods are insulin pumps and inhaled insulin. Pancreatic islet cell transplantation is experimental, though growing.[6] Most people who develop type 1 are otherwise healthy.[7] Although the cause of type 1 diabetes is still not fully understood, it is believed to be of immunological origin. Type 1 can be distinguished from type 2 diabetes via a C-peptide assay, which measures endogenous insulin production. The classical symptoms of type 1 diabetes include: polyuria (frequent urination), polydipsia (increased thirst), polyphagia (increased hunger), fatigue, and weight loss.[3] Insulin-dependent diabetes characterized by dramatic and recurrent swings in glucose levels, often occurring for no apparent reason, is sometimes known as brittle diabetes, unstable diabetes or labile diabetes, although some experts say the "brittle diabetes" concept "has no biologic basis.
Diabetes mellitus is characterized by recurrent or persistent hyperglycemia, and is diagnosed by demonstrating any one of the following:[12]

1. Fasting plasma glucose level at or above 7.0 mmol/L (126 mg/dL).
2. Plasma glucose at or above 11.1 mmol/L (200 mg/dL) two hours after a 75 g oral glucose load as in a glucose tolerance test.
3. Symptoms of hyperglycemia and casual plasma glucose at or above 11.1 mmol/L (200 mg/dL).
4. Glycated hemoglobin (hemoglobin A1C) at or above 6.5. (This criterion was recommended by the American Diabetes Association in 2010, although it has yet to be adopted by the WHO.)[13]

Type 1 diabetes is not currently preventable.[14] Some researchers believe it might be prevented at the latent autoimmune stage, before it starts destroying beta cells.[11] Cyclosporine A, an immunosuppressive agent, has apparently halted destruction of beta cells (on the basis of reduced insulin usage), but its nephrotoxicity and other side effects make it highly inappropriate for long-term use.[11] Anti-CD3 antibodies, including teplizumab and otelixizumab, had suggested evidence of preserving insulin production (as evidenced by sustained C-peptide production) in newly diagnosed type 1 diabetes patients.[11] A probable mechanism of this effect was believed to be preservation of regulatory T cells that suppress activation of the immune system and thereby maintain immune system homeostasis and tolerance to self-antigens.[11] The duration of the effect is still unknown, however.[11] In 2011, Phase III studies with otelixizumab and teplizumab both failed to show clinical efficacy, potentially due to an insufficient dosing schedule.[15][16] An anti-CD20 antibody, rituximab, inhibits B cells and has been shown to provoke C-peptide responses three months after diagnosis of type 1 diabetes, but long-term effects of this have not been reported.[10] Some research has suggested breastfeeding decreases the risk in later life.[17][18] Various other nutritional risk factors are being studied, but no firm evidence has been found.[19] Giving children 2000 IU of Vitamin D during their first year of life is associated with reduced risk of type 1 diabetes, though the causal relationship is obscure.[20] Children with antibodies to beta cell proteins (i.e. at early stages of an immune reaction to them) but no overt diabetes, and treated with vitamin B3 (niacin), had less than half the diabetes onset incidence in a seven-year time span than did the general population, and an even lower incidence relative to those with antibodies as above, but who received no vitamin B3.[21] Complications of poorly managed type 1 diabetes mellitus may include cardiovascular disease, diabetic neuropathy, and diabetic retinopathy, among others. However, cardiovascular disease[22] as well as neuropathy[23] may have an autoimmune basis, as well.

The consequences of diabetes are numerous, ranging from metabolic imbalance to nerve and blood vessel degeneration. One primary problem with diabetes is that the amount of glucose in the blood can offset the proportion of electrolytes. The association between blood glucose and electrolytes is a complex one and is related to a number of other factors such as age and associated conditions[24]

Testosterone is a steroid hormone from the androgen group and is found in mammals, reptiles,[25] birds,[26] and other vertebrates. In mammals, testosterone is primarily secreted in the testicles of males and the ovaries of females, although small amounts are also secreted by the adrenal glands. It is the principal male sex hormone and an anabolic steroid. In men, testosterone plays a key role in the development of male reproductive tissues such as the testis and prostate as well as promoting secondary sexual characteristics such as increased muscle, bone mass, and the growth of body hair.[27] In addition, testosterone is essential for health and well-being[28] as well as the prevention of osteoporosis.[29] On average, an adult human male body produces about 7-8 times more testosterone than an adult human female body,[30] but females are more sensitive to the
hormone.[31] Testosterone is observed in most vertebrates. Fish make a slightly different form called 11-ketotestosterone.[32] Its counterpart in insects is ecdysone.[33] In general, androgens promote protein synthesis and growth of those tissues with androgen receptors. Testosterone effects can be classified as virilizing and anabolic, though the distinction is somewhat artificial, as many of the effects can be considered both. Anabolic effects include growth of muscle mass and strength, increased bone density and strength, and stimulation of linear growth and bone maturation. Androgenic effects include maturation of the sex organs, particularly the penis and the formation of the scrotum in the fetus, and after birth (usually at puberty) a deepening of the voice, growth of the beard and axillary hair. Many of these fall into the category of male secondary sex characteristics.

Material and Methods

Recruitment of Subjects: Fouty (40) human male subjects were recruited for the purpose of this research work: twenty (20) of which were used as control after they have been screened to be diabetes free which was determined by the results of their fasting blood sugar test that falls between the normal range of (80 – 110mg/dl), while the remaining twenty (were) used as experimental subjects due to the fact that they are diabetic patients from two different hospitals – Federal Medical Centre, Abeokuta, and University College Hospital, Ibadan. The recruitment was carried out after the authorities in the two hospitals have approved the letter sent to them.

Collection of Blood Samples: The blood samples (10ml) were collected into labelled heparinized bottles and later centrifuged, giving plasma which was decanted into plain bottles and were transported in ice buckets to the department of biochemistry laboratory for analyses.

Materials Used: The materials used include; 10ml syringes, needles, 10ml heparinized bottles, test tubes, test tube racks, 10ml pipettes, micropipettes (0- 100?l) and (100-1000 ?l), cotton wool, hand gloves, distilled water, centrifuge, timer, sodium reagent, potassium reagent, centrifuging machine, spectrophotometer, randox kit for potassium, colourimeters, magnetic racks and separators, vortex mixer, 37oc water bath, reagent bottles, measuring cylinders and beakers.

Methods Used: The twenty samples obtained from FMC, Abeokuta and UCH, Ibadan were iced. The remaining twenty (20) were collected from healthy male subjects of Department of Biochemistry, Olabisi Onabanjo University. The blood samples from the two groups were quickly collected early in the morning before eating using 5ml syringe and taking it twice making it ten milllitre (10ml) and transferred into a labelled heparinized bottles. Blood were centrifuged at 400rev/mins for 5minutes to separate the plasma decanted into a fresh heparinized bottles. They were transported in 20oc temperature using cooler containing ice-blocks to the biochemistry laboratory from which all biochemical workers were carried out.

Procedure for sodium

Filterate preparation:

1. Test tubes were labelled: Standard, control and blank
2. 1.0ml of sodium reagent was pipetted to all test tubes
3. 50 ?l of sample were added to test tubes and distilled water to the blank
4. All test tubes were vigorously shaken and were mix continuously for 3 minutes
5. Test tubes were centrifuged at high speed (1,500) for 10 minutes.
6. 50 ?l of the supernatant was taken into another test tube
7. 1ml of the acid reagent were added to all the test tubes
8. 50 ?l of colour reagent were added to all the test tubes
9. The test tubes were mixed and the readings were taken

Colour development

1. The test tubes corresponding to the above filterate tubes were labelled.
2. 1ml of Acid reagent was pipetted to all tubes
3. 50µl of supernatant was added to respective tubes and mix.
4. 50µl of colour reagent was added to all tubes and mix
5. Zero spectrophotometer was carried with distilled water at 550nm
6. The Absorbance was read and recorded for all the tubes

Calculations
Abs. = Absorbance
S = Sample
STD = Standard
(Abs. of blank-Abs of S) X Conc. of STD = Conc. of S
(Abs of blank-Abs of STD) mEq / L mEq/ L

Procedure for Potassium

Filterate preparation:

1. The test tubes were labelled: Standard, control and blank
2. 2.0ml of potassium reagent was pipetted to all tubes
3. 20 ?l (0.01ml) of sample was added to respective tubes.
4. All the test tubes were vigorously shaken and mix continuously for 3 minutes
5. The wavelength of spectrophotometer was set to 500nm, and zero spectrophotometer with reagent blank
6. The absorbance was read and recorded for all tubes

Calculations

Abs. = Absorbance
STD = Standard
Abs. of unknown X Conc. of STD (mEq / L) Potassium conc.s
Abs. of STD mEq/ L

Determination of Testosterone using Enzyme Immunoassay design (EIA)

Tube contents

1. 100µl sample or standard
2. 100µl working suspension testosterone E1A magnetic antibody

Procedure

1. 100µl of standard or sample was pipetted into tubes 1-100 (manual 100µl pipette is used)
2. 100µl working suspension of testosterone EIA magnetic antibody was added to tubes 1-100
3. The tubes were covered and briefly mixed; the assay tubes were incubated with plastic, aluminium or other suitable film. After mixing the tubes were transferred to water-bath and was incubated for 15mins
4. The assay tubes were covered with plastic, aluminium or other suitable film. After mixing the tubes were transferred to the water-bath and incubated for 15minutes (The magnetic particles settled to the bottom of the tubes during the incubation at 37oc).

Wash Step 1

1. The assay tubes was removed from the water-bath
2. 500µl of distilled testosterone EIA was added.
3. Buffer was washed to tubes 1-100 and briefly mixed; the washed buffer was added at room temperature using repeating 500µl multi dose pipette
4. The rack of tubes was placed onto the magnetic base for 5-10 minutes. All tubes are in contact with the surface of the magnetic base. The separator and tubes were left at room temperature for 5-10minutes until all magnetic particles have sediments
5. The supernatant liquid was decanted from all tubes; the supernatant liquid was poured out of tubes by inverting the separator over sink (keeping all tubes in contact with magnetic base). The separator and tubes were left at room temperature for 5-10minutes until all magnetic particles have sediments.
6. The supernatant liquid was decanted from all tubes; the supernatant liquid was poured out of the tubes by inverting the separator over the sink (keeping all tubes in contact with the magnetic base), the inverted separator was immediately placed on absorbent paper and removed the remaining droplets in tubes by gently tapping the tubes in the separator on the paper
7. The separator was returned to an upright position
8. The rack of tubes was removed from magnetic base.

Reaction step II- Labelled antibody reaction

1. Summary of tube contents
2. 250µl diluted testosterone EIA Enzyme labelled antibody

Procedure

1. 250µl diluted testosterone EIA labelled antibody was added to tubes 1-100. After removing the rack of tubes from the magnetic base, the testosterone EIA Enzyme labelled antibody was added at room temperature using reacting 250µl multi dose pipette.
2. The tubes were covered and briefly mixed; the tubes were incubated in a water bath at 37oc for 1 hour.
3. The assay tubes were covered with plastic, aluminium or other suitable film.
4. After mixing the tubes were transferred to the water bath.

Note: The magnetic particles settled to the bottom of
the tubes during the incubation at 37°C.

Wash Step II

1. The assay tubes was removed from the water-bath
2. 500µl of distilled testosterone EIA was added. Buffer was washed to tubes 1-100 and briefly mixed; Wash buffer was added at room temperature using repeating 500µl multi dose pipette
3. The rack of tubes was placed onto the magnetic base in the magnetic separator for 5-10 minutes. All tubes are in contact with the surface of the magnetic base. The separator and tube was left at room temperature for 5-10 minutes until all magnetic particles have sediments.
4. The supernatant liquid was decanted from all tubes; the supernatant liquid was poured out of tubes by inverting the separator over sink (keeping all tubes in contact with magnetic base). The separator and tubes were left at room temperature for 5-10 minutes until all magnetic particles have sediments.
5. The supernatant liquid was decanted from all tubes; the supernatant liquid was poured out of tubes by inverting the separator over the sink (keeping all tubes in contact with the magnetic base), the inverted separator was immediately placed on absorbent paper and the remaining droplets in tubes was removed by gently tapping the tubes in the separator on the paper.
6. The separator was returned to an upright position.
7. Step ii to vi was repeated.
8. 1ml of EIA stop buffer was added to tubes 1-101 and briefly mixed.
9. NOTE: EIA stop buffer was added to tubes in the same sequence and at the same rate as that used to add substrate solution.

The rack of tubes was placed onto a magnetic base and the magnetic separation was left for at least 10 minutes, producing a clear solution for color measurement.

Results

After the test was carried out the following results were gotten on the effect of type 1 diabetes on serum electrolyte and testosterone hormone level in male human subject.

Table 1: Showing the level of Sodium and potassium in both diabetic and control human male subjects

See Illustration 1

Table 2: Showing the levels of Testosterone in both diabetic and control human male subjects

See Illustration 2

Discussion

One primary problem with diabetes is that the amount of glucose in the blood can offset the proportion of serum electrolytes. The association between blood glucose and serum electrolytes is a complex one and is related to a number of other factors such as age and associated conditions. Serum electrolyte imbalance in type 1 diabetes is primarily a result of elevated blood glucose. With hyperglycemia, the body tries to rid itself of the excess blood glucose by increasing urinary output. Increased urination produces water and electrolyte loss, which then upsets the body’s balance of electrolytes. The balance is especially disturbed between sodium and potassium. From Table 1, this increase was noticed in experimental group (236.56 ± 36.51) when compared to the control group (140.02 ± 14.63), as this shows a statistical significance in sodium ion concentration. In addition, with respect to the potassium ion, when the experimental and control
groups are compared, one would observe little or no significance difference in the concentration values. This results correspond with the work done by (Folaranmi and Adesiyan, 2004)[34]; Sudhakar et al., 2004)[24]. Moreso, taking a close look at Table 2, it is apparent that the experimental (7.02 ± 0.85) and control group (25.65 ± 4.66) results are statistically significant, with respect to testosterone. This is in line with Grinspoon et al., 2000[35].

For the sake of simplicity, these discussed results have been represented in figures; 1, 2 and 3.

Conclusion(s)

I conclude that serum electrolytes imbalance contributes to the complication observed in type 1 diabetes and it can likely lead to kidney failure, kidney stone, Hypertension and cardiac arrythmias as a result of increase in sodium level (Hypernatremia) and increase in potassium level (Hyperkalemia). Also, there is a higher probability of infertility due to testosterone deficiency,[30], and this can lead to hypogonadism and patient might not be able to impregnate the opposite sex, which is as a result of low level of testosterone hormone.

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References

5. International diabetes federation (Wednesday, October 27, 2010)


Illustrations

Illustration 1

Showing the level of Sodium and potassium in both diabetic and control human male subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of Subjects (N)</th>
<th>Sodium (meq/L)</th>
<th>Potassium (meq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>20</td>
<td>140.02 ± 14.63</td>
<td>4.25 ± 0.64</td>
</tr>
<tr>
<td>Experimental</td>
<td>20</td>
<td>236.56 ± 36.51</td>
<td>5.28 ± 0.65</td>
</tr>
</tbody>
</table>
Illustration 2

Showing the levels of Testosterone in both diabetic and control human male subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of Subjects (N)</th>
<th>Testosterone (nm/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>20</td>
<td>25.65 ± 4.66</td>
</tr>
<tr>
<td>Experimental</td>
<td>20</td>
<td>7.02 ± 0.85</td>
</tr>
</tbody>
</table>
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