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# Some haematological variables in insulin dependent diabetes mellitus patients in Imo state Nigeria

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**Abstract**

The study was done to determine some haematological variables in insulin dependent diabetes mellitus patients in Imo State.A total of ninety seven (97) subjects were recruited for the study.Seven (7) subjects were males with IDDM and 30 males were controls while 27 females were subjects with IDDM and 40 females were the the controls.Full blood count, erythrocyte sedimentation rate ,platelet count of sufferers, and non-sufferers who served as control were determined using standard haematologic methods, t-test analysis revealed significant statistical differences in all variables ($P<0.05$), except erythrocyte sedimentation rate, platelet, lymphocytes and monocytes in male diabetics ($P>0.05$). Anaemia, leukaemia and thrombocytopenia were seen in some percentage of patients,haematologic surveillance in IDDM disease, and instituting IDDM disease management programme will assist in reducing complications that may be brought about by haematologic changes and ignorance.

**Keywords:** Insulin dependent diabetes mellitus,haematological variables,Anaemia,Leukemia,Thrombocytopenia.

## Introduction

The term diabetes mellitus describes a metabolic disorder of multiple aetiology. It is characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, action or both (WHO, 1999). It has also been defined by the World health organization (WHO) on the bases of laboratory findings; as a fasting venous plasma glucose concentration greater than 7.8 mmollL or greater than ll.lmmol/L 2 hours after a carbohydrate in take or oral ingestion of 75mg of glucose even if the fasting concentration is normal (WHO). The effects of diabetes mellitus include long-term damage, dysfunction and failure of various organs. Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision and weight loss. In its most severe forms, ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor, coma, and in the absence of effective treatment, death. Most at times, symptoms are not severe or may be absent, and consequently hyperglycemia sufficient to cause pathological and functional changes may be present for a long time before diagnosis is made.

The long term effects of diabetes mellitus include progressive development of the specific complications of retinopathy with potential blindness, nephropathy that may lead to renal failure, and or neuropathy with risk of foot ulcer, amputation, charcot joint, and features of autonomic dysfunction, including sexual dysfunction. People with diabetes are at increased risk of cardiovascular peripheral vascular and cerebrovascular disease.

Several pathogenetic processes are involved in the development of diabetes. These include

processes which destroy the beta cells of the pancreas with consequent insulin efficiency, and others that result in resistance to insulin action. "he abnormalities of carbohydrate, fat and protein metabolism .are due to deficient action of insulin on tissues resulting from insensitivity or lack of insulin (Stoffers et al., 1997).

This encompasses the majority of cases which are primarily due to pancreatic islet beta - cell destruction and are prone to keto acidosis. Type I diabetes mellitus results from autoimmune destruction of the insulin-producing cells of the pancreas. It indicates the processes of beta cells destruction that may ultimately lead to diabetes mellitus in which insulin is required for survival as it keeps the blood sugar level under control. High levels of glucose are responsible for the symptoms and complications of the disease. However, most of the insulin-producing cells are destroyed before the patient develops symptoms of the disease. It has therefore been reported that most individuals with type I diabetes may be metabolically normal before the disease clinically manifests, but the process of beta cell destruction can be detected.

Type I is usually characterized by the presence of anti-gultamic acid decorboxylase, Islet cell or insulin anti-bodies which identify the auto-immune processes that lead to beta cell destruction. It has equally been reported that subjects with this clinical form of diabetes, particularly non-caucasians, show no evidence of an autoimmune disorder is demonstrable and these are classified as type I idiopathic. Aetiological classification may be possible in some circumstances and not in others. Thus, the aetiological type I process can be identified and sub-categorized if appropriate antibody determination is performed. The rate of beta cell destruction of the pancreas as stated earlier is quite variable, being rapid in some individuals and slow in others. The rapidly progressive form is commonly observed in children, but also may occur in adults (Humphrey et al., 1998). The slowly progressive form generally occurs in adults and is sometimes referred to as Latent Autoimmune Diabetes in adults. Some patients particularly children and adolescent, may present with keto acidosis as the first manifestation of the disease. Others have modest fasting hyperglycaemia and or keto acidosis in the presence of steers or infection or other stress. Still others, particularly, adults, may retain residual beta cell function, sufficient to prevent keto acidosis for many years. Individuals with this form of type I diabetes often oecome dependent on insulin for survival eventually and are at •isk of keto acidosis (Willis et al., 1995). This stage of the disease, there is little or no insulin secretion as manifested by low or undetectable levels of plasma c-peptide.

Makers of immune destruction, including Islet of cell autoautibodies, and/or auto antibodies to insulin and autoantbodies to glutamic acid decarboxylase are present in 85-90% of individuals with type I diabetes mellitus when fasting diabetic hyperglycaemia is initially detected (Verge et al.,1996).

The peak incidence of this type of type I diabetes occurs in childhood and adolescence but the onset may occur at any age, ranging from childhood to the ninth decade of life (Molbak et al., 1994).

There is a genetic predisposition of autoimmune destruction of beta cells, and it is also related to environmental factors that are still poorly defined. Although patients are not usually obese when they present with this type of diabetes, the presence of obesity is not incompatible with the diagnosis. These patients may also have other autoimmune disorders such as graves disease.

Type I diabetes mellitus accounts for about 10% of all diabetes, affecting approximately 1.4 million people in the U.S and 10 - 20 million globally (Rewers 1991., Libman et al, 1993).

About 40 percent of persons with type I diabetes develops the disease before 20 years of age thus making it one of the most common severe chronic diseases of childhood. In the U.S where 30,000 new cases occur each year, type I diabetes affects 1: 300 children and as many as 1: 100 adult during the life span.

Type I diabetes is the leading cause of renal disease, blindness, amputation, and a major cause of cardiovascular disease and premature death in the general population (Chase et al., 2004). The prevalence of type I diabetes in children aged less than 15 years ranges from 0.05 to 0.03% in most European and North American population (Libman et al.,1993).

In the 1990's, the number of type I diabetic patients 0-9 years of age in the United States was estimated at approximately 123,000 individuals.

However, in 2000, with the growth of this segment of the US population to over 80 million, there were approximately 160,000 children with type I diabetes. One of the most striking characteristics of type I the large geographic variability in the incidence (Karvonen et al., 2002).

For instance, Scandinwavia and the mediterrane emed of Sardinia have the highest incidence rates in the world while oriental population has the lowest rates. A child in Finland is 400 times more likely to develop diabetes than one in China. While there is a strong south-north gradient in the incidence, the geographic and ethnic variations in type I diabetes reflect the prevalence of susceptibility genes or that of causal environmental factors or both. In the general population, the prevalence of B cell autoimmunity appears to be roughly

proportional to the incidence of type I diabetes in the populations. In contrast, the prevalence of B cell autoimmunity in first degree relatives of type I diabetic persons does not differ dramatically between high and low risk countries. The incidence of B cell autoimmunity is higher in relatives younger than 5 years compared to those 5-9 years old (Adojaan et al., 1996).

The clinical picture of the disease is similar in low and high risk areas making it unlikely that the inter-population difference is due to misclassification of different types of diabetes. However, in many populations, type 2 diabetes is an increasing or already the predominant form of diabetes in children making correct diagnosis and treatment increasingly difficult. Type I diabetes incidence peaks at the ages of 2, 4-6 and 10 - 14 years, perhaps due to alterations in the pattern of infections or increase in insulin resistance. Recently published data suggest that in many populations, the highest rate of incidence is observed in the 10-4 age group, while the highest annual increase is in the 0- 4 years age children.The age distribution of onset of type I diabetes similar across geographic areas and ethnic groups. There have only been a few studies that incidence of type I diabetes in adults, mainly because of the difficulty of distinguishing type I from insulin requiring type 2 diabetes in older individuals. The incidence decreases in the third decade of life.

It has been speculated that the incidence of type I diabetes increases again in the fifth through seventh decades of life though there is no hard evidence of such increase, and it is not known whether there are etiologic differences between childhood and adult onset type I diabetes. The incidence data from early 1990 suggested a significant racial difference in type I diabetes risk in multiracial populations, although not of the same magnitudes as the geographic differences. In the US, non-Hispanic whites were about one and a half times as likely to develop type I diabetes as African Americans (Dabelea, 2005) or Hispanics (Kostraba et al., 1992). This was similar to the differences reported from Montreal, where children of British decent had about one and a half the risk of type I diabetes in children of French decent.

Recent data from such study have shown a rising trend in type I diabetes not only in non-Hispanic white population, but also among Hispanic and African American Children (Dabelea, 2005).

In fact however, the number of new type I diabetes patients of African-American origin aged 10-14 years has risen significantly during the last 10-15 years and the incidence of type I diabetes in this age group is almost equal in the white and African American population, it is still almost two fold difference between non - Hispanic white and African - American children in the incidence of type I type in young children 0 - 9 years (Rewers et al., 2004).

It was suspected that the marked increase in incidence in the African American population may be in part due to misclassification of cases actually having type 2 diabetes, as there is an epidemic of type 2 diabetes in children in the US, which largely affects African - American children over the age of 10 years and many cases of type 2 diabetes require treatment with insulin at the time of diagnosis.

Similarly to this observation, the high annual increase in the incidence of type I diabetes has been recently reported among the children of south Asian immigrants (Indian, Pakistani, Bangladesh in UK) (Dabelea, 2005). Children living in south Asia have a low incidence of type I diabetes, so it is worth to emphasize that migrants to the UK have similar overall rates of type I diabetes to the indigenous British population.

In a study on incidence of type I diabetes mellitus in Colorado i in 1978 - 88, it was observed that white had a percentage of 16.2 incidence, black 11.8, non Hispanic white 16.4, Hispanic 9.7. Among the Montreal, Canada, French had 8.2, British 15.3, Italian 10.7, Jewish 17.2 and others 13.1, Males and females have similar risk of type I diabetes with pubertial peak incident in females preceding that in ~aies by 1 -2 years. In lower risk populations, such as Japan U.S blacks, there is a female preponderance, while in high risk groups, there is a slight male excess (Karvonen et al.,1997). Type I diabetes in adulthood seems to be associated with male excess ,with a male, female ratio between 1.3 and 2.15 in most populations of European origin (Vandewelle et al., 1997) but there is considerable less information concerning type I diabetes with onset in adult life. These findings contrast with data from animal models of type I diabetes in which diabetes progression is almost twice as common in females (Gale and Gillespie, 2001) The incidence of type I diabetes varies markedly over time, both seasonally and annually. In the Northern Hemisphere, the incidence declines during the warm summer months, similarly in the southern Hemisphere, the seasonal pattern exhibits a decline during the warm months of December and January, implicating a climatic factor. This seasonal pattern appears to occur only in older children (Schoenle et al., 2001) suggesting that factors triggering diabetes may be related to school attendance. The observed seasonality does not appear to be an artifact of health care seeking or access, but the seasonal patterns differ by the HLA-DR genotype. Sometimes of pandemic proportion during 1984- 1986 appear to be superimposed on a steady secular increase in incidence.

While the increase in type I diabetes incidence has affected all age groups, several studies reported particular increase among the youngest children (Cinek et al., 2000).

A recent analysis of data on published incidence trends showed that the incidence of type I diabetes is globally

increasing by 3.0% per year, and that the incidence of type I diabetes will be 40% higher in 2010 than in 1998, (Onkamo et al., 1999).

These women are more likely to develop viremia during pregnancy resulting in congenital persistent infection of B cells and early onset of type I diabetes in the off spring. This model could explain both the increasing incidence of diabetes and the decreasing age of disease onset.

Anaemia is a common complication. It is often more severe and occurs at an earlier stage in patients with diabetic nephropathy. Numerous studies have addressed the interaction between diabetes and renal failure in its pathogenesis. The anaemia associated with nephropathy results from EPO deficiency, which seems to develop in patients with type 1 diabetes who have relatively normal levels of serum creatinine. Early EPO deficiency anaemia occurs in both types 1 and 2 diabetes with the prevalence higher in type 1 diabetes (Bosman et al., 2001). There is also a greater prevalence of EPO deficiency anaemia associated with the presence of autonomic neuropathy in diabetic patients in most studies to date, the predominant risk factor for the development of anaemia in a diabetic population has been found to be the presence of renal disease impaired renal function or albuminuria (Tomas et al., 2004). One of the most potent causes of suboptimalres resopnse to erythropoietin EPO is chronic and overt inflammation associated with an increased production of cytokines such as tumor necrosis factor interleukin 1 or interferon which might suppress erythrocyte stem cell proliferation.

## Materials and Methods

### Study area

Imo state lies in the tropical rainforest belt of South Eastern Nigeria. It is made up of 27 local Government areas and has projected population of 3.4 million people. The people enjoy two distinct seasons; the rainy season which starts in May and ends in October with annual rainfall of 222.2mm and high relative humidity of 78 percent while the dry season begins in November and ends in April with high temperature of 22.0°C. The topography of the state remains flat land around Owerri zone and some adjoining local government areas from other zones (Orlu and Okigwe). It is surrounded by neighboring states like Abia, Anambra, River, Enugu, Akwa-Ibom and Cross River. The people accommodate immigrants from all over the world. The people of Imo state are served by 28 government owned hospitals and many private ones.

### Study population

Screening of subjects was based on their clinical presentations; hospital records oral interviews and relevant laboratory Investigations.Haematological variables determined were haemoglobin level, packed cell volume, total and differential white cell count, platelet count, erythrocyte sedimentation rate and blood picture.

### Ethical consideration

Consent were obtained from the research and ethic committee of the health institution used for the study who gave approval for the research work and informal consent obtained from patients or their relations as well as nurses and physicians in charge of the wards.

### Specimen collection

BLOOD - The subjects were made to sit comfortably. The anticubital vein of the upper arm where the blood was to be collected was sterilized with 70% alcohol soaked on a cotton wool. A rubber tourniquet was applied around the upper arm, 5.5 milliliter of blood was drawn from the vein and distributed as follows:- l.Smls of blood was delivered into a fuoride oxalate bottle, 2.5mls into ethylene diamine tetracetic acid (EDTA) bottle, 1ml into a bottle with paper clips where necessary and O.5mls was put in a dry tube and allowed to clot.

URINE - urine samples were collected on clean containers and used for urinalysis

### Screening tests:

### Sugar estimation

Principle: Glucose has the ability of reducing copper II to copper I oxide in the presence of an alkaline medium at the temperature of 100°c, when incubated for lOminuties. The Cu+SO formed reacts with arsenomolybdate to form arsenmolybdenum blue which is read in the colorimeter at a wavelength of 680nm. The procedure is divided into two stages and these include:

### Stage 1

3.7mls of isotonic solution (sodium sulphate and copper sulphate mixture) was pipetted into each tube.
O.lmls of well mixed fluoride oxalate blood was added to the solution in the tubes above using clean O.lml micropipette.
0.2ml of sodium tungstate was added to the mixture in the above tubes.
The contents were mixed properly and centrifuged at 3000 revolution per minutes for l0 minutes.

### Stage 2

Iml of supernatant from each tube above were added to another clean dry test tubes.
Iml of standard solutions from 3 standard bottles were added to test tubes.

Iml of distilled water was added to one test tube and taken as blank.
Iml of-freshly prepared solution A and B (solution A is copper sulphate while solution solution B is Alkaline tartarate) was added to the test tubes containing the supernatant standards, III and III and blank.
All the tubes were plugged with cotton wool to avoid oxidation by atmospheric oxygen which re-oxidizes copper I oxide back to copper II oxide.
They were placed in a boiling water bath and allowed to boil for 10 minutes. This is to induce reaction.
They were then allowed to cool.
One in three dilutions of arsenomolybdenum reagent in water was prepared and 3mls of this reagent were added to each of me tubes above.
They were then read colorimetrically on transfer to cuvette.

## Sugar concentration

Optical density of test x Concentration of standard
Optical density of standard

## Urinaysis

Glucose is oxidized by atmospheric oxygen in the presence of glucose oxidase to gluconic acid and hydrogen peroxide. Hydrogen peroxide in the presence of peroxidase oxidises the chromogen to shades of purple.

Procedure: The end of the regent test strip was dipped in fresh urine and removed immediately or passed briefly through urine stream.
10 seconds after wetting, the colour of test area was compared with colour chart. And the result read carefully, in good light and with strip near to colour chart.
Any colour developing after 10 seconds was ignored.
Test end turns purple within 10 second glucose present.
Test end remains cream after 10 seconds glucose absent.

## Haematological variables

## Haemoglobin estimation

The Cyanmethaemoglobin method was used. When anticoagulated blood is diluted in buffered solution of potassium ferricyanide and potassium cyanide, the potassium ferricyanide converts the haemologobin to methaemoglobin which is further converted to Cyanmethaemoglobin by the action of potassium cyanide. This produces a coloured solution whose absorbance is read in a colorimeter at a wavelength of 540nm. The optical density of the mixture is directly proportional to the concentration of the Haemoglobin present in it.

Procedure: 5mls of drabkin solution were added to 0.02ml of well mixed EDTA blood using clean haemoglobin pipette. The contents were mixed properly by inversion and allowed to stand for 10 minutes for full colour development. The mixture was then transferred into cuvette and was read at 540nm, using the Drabkin reagent as blank.

The haemogloin values obtained were read from a standard haemoglobin calibration curve. Haemoglobin concentration (g / dl)= A of blood sample x Cone. Of Std.x D.F

A of Std. . 1000
A = Absorbance
Std. = Standard
D.F = Dilution Factor.

## Haematocrit estimation

When anticoagulated whole blood is centrifuged in a capiliary tube, there is packing of the red cells and the space occupied by the packed red cells is measured and expressed as a percentage of whole blood volume.
Procedure:
1. A plain, non-graduated capillary tube of about 7cm in length and 1 mm bore diameter was filled with a properly mixed EDTA blood by capillary action. Two capillary tubes were filled for each sample up to about 3/4 from the upper end.
2. The filled end of each tube was properly sealed using a plastic seal. The tubes were placed in the radial grooves of the microhaematocrit centrifuge, spun for 5 minutes at 12,000 revolution per minutes (rpm).
3. The value of the PCV was obtained using the microhaematocrit reader.

## Total leucocyte counts:

Standard method of total leucocyte count was employed,cells were lysed when whole blood were diluted in Turks Solution of 1% glacial acetic acid. The white cells remain intact their nuclei stained deep violet.
Procedure: To 0.38ml of Turk's solution in a glass test tube, 0.02ml of EDTA anticoagulated blood was added.
The mixture was thoroughly mixed by rotation for about 1 minute.
The chamber was cleaned and the cover slip firmly applied.
The chamber was charged and was allowed to settle for one minute for proper settling of the cells.
Leucocytes count was done in the four corners of the counting chamber using the lower power (10 x eye piece lens) and 16mm objective lens. The value obtained was multiplied by 50 to obtain the total leucocytes count (1st principle).

## Differential white cell count

These were done by visual examination of blood film on a slide by the spread technique (Cheesbrough, 2004).

Procedure: Plain microhaematocrit tubes were used to drop well mixed EDTA blood onto the surface of clean glass slides at about 1cm from the end.
Placing the slides on a smooth surface thin film of about 2cm long was made using the spreader.
Film staining:
Procedure: Prepared thin films were flooded with Leishman's stain for 2 minutes.
Equal volume of buffered distilled water (PH 6.8) was added and mixed by gentle rocking. The diluted stain was allowed to stand for 10 minutes.
The films were washed with buffered solution until the appearance of a Salmon pink colour of red cells were observed.
Films were drained and dried in the air at room temperature, the back of the slide were cleaned and then set for examination.
The stained blood films were inspected microscopically using, x20, X 40, objectives for general survey and oil immersion objective (x 100). The cells were counted in a strip running the whole length of the film. The films were inspected using the longitudinally until all the cells were counted.

### Erythrocyte sedimentation rate (ESR)

The Westergreen method was used as described by (Cheebrough, 2004).
When anticoagulated blood is left to stand undisturbed for 1 hour the red cells sediment gradually to the bottom of the tube leaving a clear plasma on top and the distance occupied by the supernatant plasma is determined.

Procedure: The blood samples were mixed thoroughly and drawn up into a westergreen tube to a 200mm mark using teat.The blood samples were mixed in the proportion of0.4mls of trisodium citrate to l.0mls of EDTA blood.
The tubes were allowed to stand vertically undisturbed for 60 minutes.The height of the clear plasma above the upper limit of the column of sedimented cells were read to the nearest Imm. The result expressed as mm/hour.

### Platelet count:

Red cells were lysed when blood was diluted in 1°/0 ammonium oxalate solution. The platelets remain intact.
Procedure: 0.38mls of ammonium oxalate solution was mixed with 0.02mls of EDTA blood. The counting chamber was charged as in leucocyte. The four corner center square and one at the center were counted after 20 minutes of allowing the cells to settle in a wet petri dish.
Calculation = cells counted x 20 xlO6
0.2x0.1 20 = dilution factor
0.2 = area of the chamber
0.1 dept of the chamber.

### Data analysis

The data generated were analyzed statistically, using prevalence rate and student t-test to ascertain prevalence of each of the diseases and the interaction of sex and age as well as level of significant between the sufferers and non sufferers.

## Results

Table 1:Haematological values in Male Diabetics as compared with non-Diabetic Males

| | | | | | TEST |
|---|---|---|---|---|---|
| | DIABETIC MALES | | NON - DIABETIC | | STATISTICS |
| | | | | | Ct TEST) |
| Parameters | Mean±SD | | Mean± SD | | Sig. level P |
| Haemoglobin | 10.3 | 1.7" | 14.3 | 0.7 | P<0.05 |
| PCV- % | 31.2 | 5.2 | 42.2 | 2.1 | P<0.05 |
| WBC x 109/1 | 3.9 | 0.9 | 5.7 | 1.1 | P>0.05 |
| Platelet x 1Q9/1 | 171.8 | 31.2 | 217. | 51.7 | P>0.05 |
| ESR per hour | 16.2 | 9.0 | 5.4 | 1.0 | P>0.05 |
| Neutrophil % | 57.1 | 7.7 | 46.2 | 5.2 | P<0.05 |
| Lymphocvtes % | 40.4 | 5.7 | 50.7 | 3.9 | P>0.05 |
| Eosinophil % | 1.5 | 1.3 | 2.0 | 1.3 | P>0.05 |
| Moncytes % | 0.8 | 0.8 | 1.0 | 1.0 | P>0.05 |
| Basophil % | — | — | — | _. | — |

Table 2: Haematologicaf values in female Diabetics as compared with non-Diabetic females

| | DIABETIC FEMALE | | NOW - DIABETIC | | TEST STATISTICS (t TEST) |
|---|---|---|---|---|---|
| Parameters<br>Haemoglobin<br>PCV-%<br>WBC x 109/1<br>Platelet x 109/1 | Mean<br>10.1<br>29.8<br>8.0<br>201.2 | ±SD<br>±0.6<br>±1.8<br>±0.9<br>±37.9 | Mearn<br>11.7<br>35.3<br>4.9<br>212.5 | ±SD<br>±0.7<br>±2.5<br>±0.9<br>±28.1 | Sig. level<br>P <0.05<br>P<0.05<br>P>0.05<br>P>0.05 |
| ESR per hour | 43.5 | ±9.1 | 13.1 | ±5.1 | P<0.05 |
| Neutrophil % | 57.1 | ±10.9 | 58.6 | ±7.3 | P>0.05 |
| Lymphocytes % | 37.6 | ±8.2 | 38.6 | ±6.8 | P>0.05 |
| Eosinophil % | 1.3 | ±1.0 | 1.5 | ±0.9 | P>0.05 |
| Monocytes % | 1.3 | ±0.5 | 1-2 | ±0.5 | P>0.05 |
| Basophil % | | | — | — | — |

SD Standard Deviation

## Discussion

Table 1 showed no signifcant change (p>0.05) except haemoglobin and PCV which showed significantdecrease (P<0.05) both in male and female insulin dependent diabetes meliitus patients compared to the controls.This may be because their kidneys were affected and the synthesis and release of erythropoietin to stimulate erythropoiesis.Table 2 showed significant decrease (P<0.05) in haemoglobin and PCV in diabetic females compared to the controls and other variable showed no significant change(p>0.05)

The same observation was made by Tomas et al. (2002) in their work on anaemia in type 1 diabetics Bosman et al. (2002) in their work on anaemia in diabetics, documented normocytic normochromic anaemia. These observations have confirmed anaemia as a risk factor in type 1 diabetes as documented by Tomas et al. (2002).

Causes of anaemia or stage of on set, but were not established but previous researchers suggested symptomatic neurophathy, causing efferent sympathetic deneravation of the kidney and loss of appropriate erythropoietin production, damage to the renal interstitum systemic inflammation and inhibition of release as the possible cause (Astor et al.,2002).

This finding is very important because diabetic patients with anaemia are more likely to develop macrovascular, peripheral and cerebrovascular diseases (Bosman et al.,2002). It was also documented that diabetic patients with anaemia were more likely to have proliferative retinopathy. The elevation in erythrocyte sedimentation rate observed only in female diabetics could be due to the severity of anaemia which was observed more in females, but the significant statistical difference in lymphocyte observed only in male diabetics could not be explained, but could be suggestive of infection which is very common in the diabetics.

## Conclusion

Insulin dependent diabetes mellitus and systemic lupus erythematosus seemed low in prevalence, but their presence in the state has been established. It was equally observed at the time of screening the high level of people suffering from type 2 diabetes. This calls for urgent intervention.

This finding is vital to researchers who may want to find out if there are male hormones that could be administered to the females at certain age to stop the development of autoimmune disease without much effect. It is equally interesting to note the adverse this disease had on haematologic variables determined. This is very important findings as it will help in the management of patients and in averting complications which may result from anaemia, leucopenia and thrombocytopenia.

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