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## Stress Test Performance by Preterm-Born Females in Adolescence and Adulthood

By Katarzyna Kaczmarczyk, Andrzej Magiera, Ida Wiszomirska, Katarzyna Horosz & Aleksander Ronikier

*University of Physical Education*

**Abstract-** Preterm birth often entails developmental disorders, with the circulatory and respiratory systems are particularly at risk, yet few studies have examined long-term effects. To evaluate the long-term impact of preterm birth on stress test performance in women born preterm, we administered the same stress test to the same set of preterm-born subjects, first at the age of puberty, then again in adulthood, comparing the results. The strength of the relationship between anthropometric parameters and physical fitness, as well as estimated oxygen uptake were also analyzed.

A total of 70 girls (aged  $12.2 \pm 1.5$ ) who had been born preterm ( $34.7 \pm 1.86$  weeks) were tested in 1997. Of those, after a gap of 18 years, a group of 13 as successfully re-contacted and participated in the 2015 examination as adults (then aged  $27.6 \pm 2.60$  years, born preterm at  $34.5 \pm 2.0$  weeks). Each time, an indirect HR test was performed (W150 test) while striving to maintain HR=150 bpm; the level of physical load (W) therefore depended only on physical fitness and exercise tolerance. In the group of adolescent girls, the rate of stress oxygen uptake ( $O_2$ ) and the metabolic stress response were measured by using the Jaeger ErgoOxyscreen apparatus. In the adult group, the  $VO_2$ max rate was measured.

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# Stress Test Performance by Preterm-Born Females in Adolescence and Adulthood

## Stress Test and Preterm-Born Females

Katarzyna Kaczmarczyk <sup>α</sup>, Andrzej Magiera <sup>σ</sup>, Ida Wiszomirska <sup>ρ</sup>, Katarzyna Horosz <sup>ω</sup>  
& Aleksander Ronikier <sup>¥</sup>

**Abstract-** Preterm birth often entails developmental disorders, with the circulatory and respiratory systems are particularly at risk, yet few studies have examined long-term effects. To evaluate the long-term impact of preterm birth on stress test performance in women born preterm, we administered the same stress test to the same set of preterm-born subjects, first at the age of puberty, then again in adulthood, comparing the results. The strength of the relationship between anthropometric parameters and physical fitness, as well as estimated oxygen uptake were also analyzed.

A total of 70 girls (aged 12.2±1.5) who had been born preterm (34.7±1.86 weeks) were tested in 1997. Of those, after a gap of 18 years, a group of 13 as successfully re-contacted and participated in the 2015 examination as adults (then aged 27.6±2.60 years, born preterm at 34.5±2.0 weeks). Each time, an indirect HR test was performed (W150 test) while striving to maintain HR=150 bpm; the level of physical load (W) therefore depended only on physical fitness and exercise tolerance. In the group of adolescent girls, the rate of stress oxygen uptake (O<sub>2</sub>) and the metabolic stress response were measured by using the Jaeger ErgoOxyscreen apparatus. In the adult group, the VO<sub>2</sub>max rate was measured.

In adolescence, no difference was found between the study group and the reference group. In adulthood, however, the prematurely born adults demonstrated significantly lower values of VO<sub>2</sub>max and total work performed in relation to the reference group, pointing to a decline in physical fitness in the study group.

Conclusions: 1. During puberty, prematurely born adolescents display better aerobic metabolism and higher rates of total work performed than their peers born at term. 2. There was a significant decrease in the level of physical fitness in the adult study group. 3. It is recommended to continuously monitor the level of exercise tolerance in prematurely born individuals at different stages of life.

### I. INTRODUCTION

Preterm birth, defined as birth prior to 37 completed weeks of gestation and generally associated with low birth weight, is a widespread phenomenon, accounting for 5% to 9% of all births in Europe [1], and more than 12% of all births in the United States [2], with similar high rates in Africa and southern Asia [3].

Although morbidity is still significant among very low birth weight infants, numerous technological advances, collaborative efforts between obstetricians and neonatologists, widespread use of antenatal corticosteroids, surfactant therapy, and high-frequency ventilation have all helped improve survival rates over the past two decades [4-6]. Since individuals who were born preterm are now more likely to survive into adulthood than before, a comprehensive understanding of the impact of premature birth in adulthood is therefore needed to enable earlier prevention, detection, and treatment of its long-term health sequelae. However, such information is largely lacking from the literature; most studies of the impact of premature birth have examined subjects only in childhood.

Physical fitness is always conditioned by numerous factors, and this even more so in the case of developmental disorders (such as those resulting from premature birth). For instance, respiratory and circulatory capacities depend on the maximum oxygen uptake (VO<sub>2</sub>max) and, as such, full lung maturation is of paramount importance in determining physical fitness [7]. Premature infants generally have difficulty breathing due to a reduced number of alveoli and underdeveloped pulmonary capillaries [8]. Such pathological conditions at an early age may result in diminished lung function later in life, including in terms of lung volume, ventilation homogeneity, and the mechanics of the respiratory system, which will in turn detract from physical fitness [9-12]. Alongside the respiratory system, the circulatory system also plays a significant role in the development of the child's physical fitness. Both systems are involved in the process of gas exchange through the blood. In premature babies, the circulatory system may experience a delayed closure of the ductus arteriosus and the pulmonary vascular resistance may be diminished after birth; this condition (patent ductus arteriosus or PDA) significantly impairs the respiratory function and hemodynamic conditions of premature infants.

The literature on exercise tolerance focuses mainly on preterm-born children in the early years of their lives, generally not proceeding beyond age ten. As such, it has yet to be determined whether premature birth is also associated with reduced physical fitness

Author <sup>α</sup>: Department of Physiology, Faculty of Rehabilitation, University of Physical Education in Warsaw, Poland.  
e-mail: katarzyna.kaczmarczyk@gmail.com

later in life. However, despite consistent findings concerning poor lung function, few studies have addressed exercise tolerance in low birth weight populations [13-16]. Authors have reported decreased [13,14] or relatively normal [15,16] peak  $\text{VO}_2$  values in young prematurely born children. Contrary to these findings, in another study we previously reported finding an improvement in exercise tolerance in cohorts of preterm born children in adolescence [17]. Data concerning adults who were born prematurely are scarce, as there have been few longitudinal or follow-up studies extending into adulthood. Clemm et al. [18] compared the exercise capacity of adults born at term and EP, addressing the developmental patterns from adolescence to adulthood (18 to 25 years old) and reporting that exercise capacity was slightly lower in EP-born adults; however, those values were within a normal range, positively associated with self-reported physical activity, and unrelated to neonatal factors and current airway obstruction.

The aim of the present study, therefore, was to explore the long-term effect of prematurity by examining the stress-test performance of subjects at age 12, then to re-examine a portion of the same subjects at the age of 30, using same examination setup, and to compare the results against those of a reference group. The strength of the relationship between anthropometric parameters (height and weight, BMI) and physical fitness, as well as estimated oxygen uptake were also analyzed.

## II. MATERIAL AND METHODS

### a) Participants

Individuals participating in the study group for both stages of our study were females who had been registered at the Premature Birth Clinic, Rehabilitation Department, Institute of Mother and Child in Warsaw, as having been born with low birth weight, i.e. below 2,500 g, or born preterm, i.e. prior to 37th week of pregnancy (though with normal birth weight). The total work performed by the subjects on an exercise test was evaluated twice: once in 1997 (puberty) and again in 2015 (adulthood). An additional criterion for inclusion of preterm-born individuals in both states of the study was a normal resting ECG.

In the first stage, in 1997, total of 70 preterm-born girls aged 10-14 years ( $12.22 \pm 1.52$  years) took part in the examinations (the results of which have not been previously published). The mean birth weight of among this the group was  $1,865.8 \pm 566.3$  g (min. 1,040 g, max. 2,580 g), and they were born in the  $34.5 \pm 1.92$  week (min. 32 weeks, max. 36 weeks). All of the subjects who ended up selected based on the above criteria, therefore, were all under 2,500 g birthweight and all born prior to 37th week of pregnancy. Most of the study group (85%) had been fed on formulas as infants,

whereas two had been fed on a combination of infant formula and breast milk. On the basis of their health records, none of the subjects had experienced BPD.

Eighteen years later, we attempted to reestablish contact with all of the 1997 participants by twice sending out a request letter to their previously recorded residence. Only 13 of the original participants responded and agreed to undergo reexamination in 2015. The study group participants in 2015 had a mean birth weight of  $1,864.6 \pm 533.7$  g (min. 1,040 g, max. 2,580 g) and were born at  $34.5 \pm 2.0$  weeks (min. 32, max. 36) of pregnancy.

Note that, in fact, both boys and girls had been examined in the 1997 stage of the study, for which all prematurely born and/or low-birth-weight children registered with the above-mentioned clinic who met the criteria for the study had been recruited. However, the 13 participants who ultimately agreed to participate in the follow-up stage later turned out to be exclusively female; as such, we were able to analyze and report long-term results exclusively for the female group. This sample size of 13 participants for the latter, follow-up stage, while admittedly quite small in absolute terms, still represents a considerable achievement given the long time-frame involved and the relative scarcity of such long-term data in the literature.

This study group was in each case compared against a reference group of their peers, consisting of 48 girls ( $12.4 \pm 1.52$  years) in the first stage, and of 27 women ( $28.3 \pm 2.16$  years) in the second, made up of girls and women from the province including Warsaw and its environs (the Mazowsze Voivodship), born at term, at ages corresponding to the test group. To compensate for social variables, the reference group was in each case recruited by asking the study participants (or their guardians) to invite their peers born at term and weighing more than 2,500g to take part in the study.

The basic parameters of the two groups are presented in Table 1. All participants were informed about the conditions and course of the study, and written informed consent was expressed for participation in the research (either from the legal guardians of the children or by the adult participants themselves). The study received the approval of the Ethics Committee at the Józef Piłsudski University of Physical Education in Warsaw (decision no. SKE 01-47/2012).

**Table 1:** The mean values and standard deviation for body weight, body height and BMI in 1999 and 2015

| 1998             | Reference (n=48) | Preterm (n=70) |
|------------------|------------------|----------------|
|                  | Mean±SD          | Mean±SD        |
| Body weight (kg) | 42.09±9.82       | 42.16±11.80    |
| Body height (cm) | 149.84±9.56      | 149.31±9.47    |
| 2015             | Reference (n=29) | Preterm (n=13) |
|                  | Mean±SD          | Mean±SD        |
| Body weight (kg) | 62.7±9.08        | 60.08±14.66    |
| Body height (cm) | 167.8±5.6        | 163.00±10.08   |
| BMI              | 22.23±2.48       | 22.55±4.53     |

### b) Study Methods

Two stress-test examinations were carried out at an interval of 18 years, the first stage in 1997 and the second in 2015. All measurements were performed in the early hours of the morning at a diagnostic facility of the Central Laboratory at the University of Physical Education in Warsaw. Tests were performed at least two hours after the last meal. In each case indirect HR test (W150 Test) was performed using an ERGOTEST device, based on the original W150 program [19]. Physical load values were determined by the computer using feedback (load/HR); this results in the induction of cyclometer resistance at a certain level, forcing the achievement and maintenance of physiological balance at HR=150 bpm (control level) to evaluate aerobic metabolism. The test uses a linear relationship between the frequency of the heart rate and oxygen uptake. The effort was dispensed on a bicycle ergometer Pro Med Medical Systems with a foot drive, heart rate were recorded and measured electronically using precordial electrodes (transmitter BMI- USBD1- EU- S/N 054BM/S300). One of the criteria for qualifying was a normal resting ECG. As the test was underway, for the

study group individuals (preterm-born) their ECG was monitored by a physician (Nórava S-stress, Oxford Poland).

Before undergoing the exercise test, participants were subjected to a preliminary assessment of exercise capacity using the Ruffier test, which included measuring their blood pressure and heart rate (Dura Schock Welch Allyn sphygmomanometer). Warm-up was performed on an ergometer driven by lower limbs for 10 minutes, with a load of 1W/kg of the subject's body weight. Heart rate during warm-up did not exceed HR=130 bpm. After the warm-up stage and recovery period, the proper testing began.

Tests were carried out in the presence of a physician, and were explained to the participants before they began. The period of exercise lasted 10 minutes, followed by a recovery period lasting until the participant's HR returned to pre-test level. The physical-challenge method applied (also used in the 1997) assumes the maintenance of the heart rate on control level of HR=150 bpm; load regulation is based on the individual reactions of the cardiovascular system, with the load level (W) being adjusted as necessary during the exercise and with pedal cadence ranging from 50 to 60/min. The load level therefore depended only on the subject's physical fitness and exercise tolerance.

During the exercise test the following parameters were recorded: HR, total work performed during the test, power necessary to achieve the control level (HR=150 bpm), average power for the control period (measured from the moment of attaining HR=150 bpm until the end of the test), and the time it took to achieve the control level (HR=150 bpm). At the completion of the test in the adult group the  $\text{VO}_2\text{max}$  rate was automatically calculated. In the group of adolescent girls, the rate of oxygen uptake and the metabolic stress response were measured using the Jaeger ErgoOxyscreen apparatus.



**Fig. 1:** Record of HR test (HR and power exerted)

The test was halted for in the event of any adverse cardio-respiratory reactions or signs of exercise intolerance, such as dizziness, shortness of breath, pallor, chest pain or imbalance.

#### Statistical analysis

The data was processed and all statistical calculations were made using STATISTICA 12.0 by StatSoft, according to the software instructions. The results were presented as arithmetic means  $\pm$  standard deviation. The Shapiro-Wilk test was used to evaluate the normality of distributions, and the Pearson correlation coefficient was used to measure the dependency between the parameters.

The variables were compared between the groups using a non-parametric test (the Mann-Whitney U test adjusted for continuity). The ANCOVA test (from the General Linear Model) was used to exclude a covariate (weight) in group comparisons. Because the distribution was not found to be normal, the Mann-

Whitney U test was used. The threshold for statistical significance was taken to be  $\alpha=0.05$ .

### III. RESULTS

Among the variables considered in the first study (adolescent period), no significant differences were found between the study group and the reference group in terms of the parameters indicative of exercise tolerance at the peak of oxygen uptake – i.e.  $O_2$  [L/min] at HR=150 bpm and the total work performed W [kJ]. The total work performed, however, was found to differ significantly between the study and reference groups of adults ( $p<0.001$ ). Similar differences were observed in the  $VO_2$ max rate, with the adult reference group presenting significantly higher levels of  $VO_2$ max than their peers born prematurely ( $p<0.001$ ). This indicates that adult women born prematurely do have a lower exercise tolerance.

**Table 2:** Total work (kJ) and aerobic metabolism  $VO_2$ max rates [L/kg body weight] and  $O_2$  [L/min] in the reference and preterm groups tested in 1998 and 2015

| Factor                        | Preterm group 1998 (n=70) | Reference group 1998 (n=48) | Preterm group 2015 (n=13) | Reference group 2015 (n=29) |
|-------------------------------|---------------------------|-----------------------------|---------------------------|-----------------------------|
| Work [kJ]                     | 26.94 $\pm$ 9.40          | 25.79 $\pm$ 8.74            | 46.18 $\pm$ 6.90          | 56.66 $\pm$ 11.12*          |
| $O_2$ [L/min]/                | 1.57 $\pm$ 0.40           | 1.61 $\pm$ 0.70             |                           |                             |
| $VO_2$ max [L/kg body weight] |                           |                             | 1.66 $\pm$ 0.35           | 2.30 $\pm$ 0.16             |

We considered that the total work value could be dependent on body weight, and so performed an ANCOVA analysis with covariate. The results indicated that the body weight did not affect the significant differences in the total work performed between the groups ( $F = 8.97$ ;  $p = 0.004749$ ;  $\eta = 0.1869$ ). The test power was 0.83.

**Table 3:** Correlations between total work and body weight, body height, BMI and  $VO_2$ max in adult groups

| Correlations           | Reference group 2015 (n=29) | Preterm group 2015 (n=13) |
|------------------------|-----------------------------|---------------------------|
| Total work/Body weight | 0.02                        | -0.18                     |
| Total work/Body height | 0.23                        | 0.05                      |
| Total work/BMI         | -0.10                       | 0.16                      |
| Total work/ $VO_2$ max | 0.71                        | 0.86                      |

No strong correlation (understood as  $p \geq 0.5$ ) was found between the total work and body weight, between the total work and body height, or between the total work and BMI. The correlation between the total work and  $VO_2$ max, on the other hand, at  $p \geq 0.5$ , is consistent with the exercise response of healthy individuals (the greater the work performed, the higher the  $VO_2$ max rate).

### IV. DISCUSSION

In this study, we sought to trace the long-term impact of preterm birth on physical fitness into puberty and later into adult life. Such long-term consequences of preterm birth are complex and notoriously problematic to study, particularly in maintaining the same group of subjects over a prolonged period of time. A major strength of our study, therefore, was its longitudinal design.

However, maintaining the participation of test subjects from childhood through adulthood is indeed challenging, and we were only able to recruit relatively few participants (13 out of 70, or 19%) to persuade them to take part in the follow-up stage of the study. The original participants who did not participate in the follow-up examination may have done so for any of a variety of reasons, including change of residence in the interim,

failure to receive the request letter, unwillingness or inability to participate, lack of interest, etc. Even so, given the relative scarcity of longitudinal data of this sort, we still do consider it a success to have managed to re-examine this sizeable of a share of the original group after such a time gap, and the proportion of re-participation we achieved is nevertheless comparable to those reported in most similar studies [13,15,20]. Moreover, the magnitude of the effect we found was large ( $\eta^2 > 0.18$ ), which ensured sufficient observed strength of the test ( $\text{Power} > 0.80$ ). This power analysis indicates that the sample size was sufficient to evaluate the phenomenon studied.

Most previous studies of children and adolescents, by contrast, have been cross-sectional [14-16, 21]. Vrijlandt et al. [16] found a mildly lower exercise capacity in a group of preterm individuals occurring in young adulthood, as compared to healthy, reference-group subjects. Moreover, their study group showed significantly lower anaerobic threshold than their healthy peers, and tended to have lower work efficiency. However, this might stem from less intensive participation in sporting activities, rather than impaired lung function or limited ventilation. Kriemler et al. [15] reported that children with and without respiratory abnormalities have some degree of pulmonary dysfunction at rest and following exercise, and a higher prevalence of exercise-induced bronchoconstriction with no reduction in maximal aerobic exercise performance. The higher oxygen uptake seen at a given mechanical power in the preterm group may cause early fatigability during prolonged exercise, even when aerobic performance is normal. Rogers et al. [14] reported significant differences between low birth weight adolescents and term-born reference subjects in terms of lower aerobic capacity, strength, endurance, flexibility, and activity level. This again could be the result of less intensive participation in physical activity and consequential physical limitations, such as neurodevelopment, sensory (visual and auditory deficits), respiratory and cardiovascular impairments, reduced muscle strength, coordination difficulties and social acceptance.

Our results, on the other hand, differed from those reported in these studies: in our study group, preterm-born subjects at puberty actually achieved better results of total work and peak oxygen uptake with heart rate maintained at 150 bpm than their peers born at term. These results indicate that, when it comes to submaximal exercise, the physical functions of the preterm children we studied were not impacted by developmental limitations in the first years of life. Of course, it could have been of more significance to study the exercise  $\text{VO}_{2\text{max}}$  rate in order to assess the children's physical capacity, but due to the state of health of preterm-born children and the difficulty in

motivating them to engage in this type of test, maximal exercise loads were not used.

On the other hand, in our the second, follow-up testing of part of the same preterm-born subjects in adult age, we found that, to the contrary, participants performed significantly worse in terms of exercise tolerance than their peers from the reference group. This finding suggests that the evolution of physical fitness and the consequences of prematurity need to be closely monitored into later stages of life. To our knowledge, there is only one publication that addresses the developmental trajectories of exercise tolerance from adolescence to adulthood: Clemm et al. [18]. In their study of 25-year-old adults born EP, they found that exercise tolerance was 10% lower than in a reference group born at term, albeit still within a range considered normal. A decreasing trend in exercise tolerance from 18 to 25 years of age was similar in both groups, and the  $\text{VO}_2$  peak obtained at age 18 strongly predicted the  $\text{VO}_2$  peak at age 25. Exercise tolerance was unrelated to neonatal factors and to current airway obstruction, but was positively associated with self-reported physical activity. Our results similarly show that participants in the study group performed 22% less work than the reference group. The physical load causing the same physiological response (maintaining HR at 150 bpm) was significantly lower in the case of the study group.

In comparing our results for the study group at adulthood to those obtained during at puberty, we can conclude that the anticipated deficits in their level of exercise tolerance in comparison to healthy peers nevertheless did manifest themselves in adulthood, despite having been compensated for and even improved upon during puberty (when preterm-born subjects fared 5% better than their healthy peers). Like other authors [18], we should note that this could be attributable to a failure to maintain normal levels of physical activity after puberty due to insufficient medical supervision and reduced parental control, but at the same time we cannot rule out the late consequences of abnormal morphological and functional development in childhood due to premature birth. Overall, premature birth contributes to increased risk of cognitive, sensory, neuromotor and coordination deficits, which can interfere with the development of exercise tolerance. Less intensive physical activity may also result from real or perceived limitations and weaknesses, leading to the reinforcement of negative attitudes towards exercise, and this in turn may have adverse effects on exercise tolerance [16, 22, 23].

Baraldi and Filippone [24], however, disagree with this reasoning, arguing that early lung damage may itself have long-term consequences. Chronic lung disease cannot be considered solely as a pediatric illness because it is likely to continue into adulthood and increase the risk of acquiring chronic obstructive pulmonary disease (COPD), as well as have a negative

impact on the results of pulmonary function testing. Walter et al. [25] draw similar conclusions, having found a higher risk of lung dysfunction in a group of adults born prematurely with low birth weight in relation to their peers born at term. There are few studies in the literature on the effects of cardiovascular disorders resulting from premature birth on exercise tolerance. Lewandowski et al. [26] performed an MRI on 234 individuals, and found increased left ventricular mass in young adults born prematurely, compared to the reference group. Changes in the geometry of the heart were accompanied by abnormal hemodynamic parameters. The authors argue that these disorders may consequently lead to reduced exercise tolerance, and may be associated with an increased risk of cardiac events.

In general, the long-term consequences of premature birth remain not well documented, but it is becoming clearer that its complications do not only affect individuals in childhood. As the results of our study indicate, despite the fact that subjects born prematurely had much better performance results in adolescence than their peers in the reference group, later in life their exercise tolerance decreased and the same individuals fared worse as adults than those in the reference group. Therefore, the exercise tolerance preterm-born individuals needs to be measured at different stages of life, not only during puberty. Further research in this direction may help us better understand the complex, longer-term developmental trajectories of preterm-born individuals.

## V. CONCLUSIONS

1. During adolescence, prematurely born subjects displayed better aerobic metabolism and higher rates of total work performed on a stress test than their peers born at term.
2. A subset of the same group of subjects nevertheless showed a significant decrease in the level of exercise tolerance in adulthood, as compared to their peers born at term.
3. It is recommended for the level of physical fitness in prematurely born individuals to be continually monitored at different stages of life.

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### Conflict of interest statement

The authors declare no conflicts of interest in preparing this article.

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## Convergence on the Constraints and Challenges in the Awareness, Prevention, Treatment and Control of Type 2 Diabetes and Diabetes-Related Conditions

By Dr. Chrysanthus Chukwuma Sr

*Abstract-* The global epidemiology of type 2 diabetes over time regarding the biologic, cultural, demographic, therapeutic regimen and lifestyle changes are factors which have been described with particular focus on the aetiology, complications, natural history and risks pertaining to the disorder. Expansive data depict that type 2 diabetes incidence and prevalence increase rapidly to the detriment of pecuniary measures in health services and society. Recent decades have been encumbered with tumultuous and contentious polemics marked with conflicts in research findings and budget cuts in the awareness, prevention, treatment and control of the constraints and challenges regarding diabetes and related conditions, especially in LIMCs. Our health systems are faced with adverse policy expansiveness to unavoidable or threatened accelerating global needs in health and development as well as a realizable paradigm of performing more with less. Strategies in the prevention, treatment and control of type 2 diabetes and diabetes-related conditions aim to mitigate the risk of the development of diabetes and its complications.

*Keywords:* cardiovascular diseases; dipeptidyl peptidase-4 inhibitors; insulin analogues; lignans, phytoestrogens, lifestyle changes.

*GJMR-F Classification:* NLMC Code: WD 200



CONVERGENCE ON THE CONSTRAINTS AND CHALLENGES IN THE AWARENESS, PREVENTION, TREATMENT AND CONTROL OF TYPE 2 DIABETES AND DIABETES-RELATED CONDITIONS

Strictly as per the compliance and regulations of:



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Dr. Chrysanthus Chukwuma Sr

**Abstract-** The global epidemiology of type 2 diabetes over time regarding the biologic, cultural, demographic, therapeutic regimen and lifestyle changes are factors which have been described with particular focus on the aetiology, complications, natural history and risks pertaining to the disorder. Expansive data depict that type 2 diabetes incidence and prevalence increase rapidly to the detriment of pecuniary measures in health services and society. Recent decades have been encumbered with tumultuous and contentious polemics marked with conflicts in research findings and budget cuts in the awareness, prevention, treatment and control of the constraints and challenges regarding diabetes and related conditions, especially in LIMCs. Our health systems are faced with adverse policy expansiveness to unavoidable or threatened accelerating global needs in health and development as well as a realizable paradigm of performing more with less. Strategies in the prevention, treatment and control of type 2 diabetes and diabetes-related conditions aim to mitigate the risk of the development of diabetes and its complications.

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## I. INTRODUCTION

Type 2 diabetes constitutes a chronic metabolic aberration with global increasing prevalence and untoward sequelae in vulnerable populations. It is rapidly reaching epidemic proportions in certain countries, especially the non-industrialised nations; and exacerbating the already extant healthcare burdens of poor countries. With no defined cure for diabetes, therapeutic modalities have involved dietary regimen, lifestyle modifications, overweight and obesity management, as well as pathophysiologically type 2 diabetes-related therapeutic approaches (Olokoba et al., 2012). Even with the provision of diverse treatments in industrialized countries, numerous type 2 diabetes subjects do not achieve glucose control (Higgins et al., 2016). Modalities for the prevention, treatment and control of type 2 diabetes and diabetes-related

conditions aim to mitigate the risk of the development of diabetes and its complications or the attendant sequelae in susceptible and vulnerable populations due to the urgency in the exacerbation of the disorders and global acceleration in diabetes incidence and prevalence. Diabetes is a priority issue of urgent concern; if not adequately stemmed, culminates in elevating the burdens on health systems and society.

Recent decades have been encumbered with tumultuous and contentious polemics marked with conflicts in research findings and budget cuts in the awareness, prevention, treatment and control of the constraints and challenges regarding diabetes and related conditions, especially in LIMCs. This paper tends to address the convergence in therapeutic and other measures for the prevention, treatment and control of diabetes and its complications which will aid in the reduction of the prevalence, morbidity and mortality as the resultant impact of diabetes mellitus. Type 2 diabetes prevalence has been found to exacerbate as a result of inadequate awareness, prevention, treatment, control and advocacy for a healthy lifestyle (Tiwari, 2015; Liu et al., 2016).

## II. DIETARY CARBOHYDRATES

Evidence from prospective observational research and clinical trials converge to undergird the significance of selected dietary patterns, foods and nutrients to prevent and manage type 2 diabetes. The quality of dietary carbohydrates and fats consumed is more vital than the abundance of these macromolecules as micronutrients. Inasmuch as enormous progress has been enacted in the development and implementation of evidence-based nutrition guidelines or recommendations in industrialized nations, it is pertinent to promote and enhance concerted worldwide efforts and policies to mitigate regional differences (Ley et al., 2014).

The health impacts of the use of dietary carbohydrates are of concern to everyone as regards the glycaemic index (GI), glycaemic load (GL) and glycaemic response (GR). Cognizance has been given to postprandial glycaemia in the health spectrum, with

**Author:** Centre for Future-Oriented Studies Abakaliki, Ebonyi State Nigeria. e-mails: cfos\_nigeria@yahoo.com, chrysanthus\_chukwuma@yahoo.com

GI as a reliable and predictive instrument in the classification of carbohydrate diets in this instance. Consumed foods with reduced GI and GL are necessary to prevent, control and manage diabetes and coronary disease, and may be extrapolated to obesity, while transient to moderately marked associations were detected in certain oncological disorders (Augustin et al., 2015). A consensus was reached that diets low in GI and GL ought to be contextually considered as healthy in characterization of carbohydrate foods, with fibre and whole grain content which are important for insulin resistance patients (Augustin et al., 2015). The current global epidemic of obesity and type 2 diabetes has increased simultaneously with adverse metabolic events. There is expansive evidence that the type of carbohydrate consumed is important in the development or prevention of insulin resistance, obesity and the metabolic syndrome. Due to the prevalence of overweight, obesity and insulin resistance, increased concerns for the quality and carbohydrate type consumed promulgate the perception that carbohydrate diets are liable to exacerbate rather than mitigate cardiometabolic risk, with divergent views regarding their glycaemic index and fibre levels as pertinent in the management of chronic diseases.

### III. LIPID-ASSOCIATED PARAMETERS

There is widespread evidence that the type of carbohydrate consumed is vital in the development and prevention of obesity, insulin resistance, the metabolic syndrome and diabetes-related events; with other measures to control carbohydrate-lipid interactions as they impact on diabetes and obesity (Chukwuma Sr, 2017a). Investigations of the impact of serum markers of cholesterol synthesis and absorption in type 2 diabetes incidence revealed an associated risk with the presence of type 2 diabetes, invariably ascribed to insulin sensitivity (de Mello et al., 2015). Cholesterol synthesis was related to greater incidence of type 2 diabetes, while cholesterol absorption correlated with lower incidence of type 2 diabetes, with detection of a gene-lifestyle interaction on markers of cholesterol absorption. There is a linkage of increased risk to develop insulin resistance and type 2 diabetes. In obese subjects, it is realized that adipose tissue releases elevated concentrations of non-essential fatty acids, pro-inflammatory cytokines, glycerol, hormones and risk factors which are connected in insulin resistance development. With concomitant presentation of dysfunctional pancreatic islet beta cells in insulin resistance, the resultant impact is deranged control of blood glucose abundances (Khan et al., 2007).

In this wise, a vast majority of subjects were diagnosed with hypoalbuminoproteinaemia (HA) during routine lipid profile determination (Schwab & Uusitupa, 2015; Vibhuti, 2016). This can be employed as an

independent factor in the assessment of coronary artery disease risk and further management. The fundamental objective for HA management and associated lipid aberration is the reduction or atherosclerosis risk that culminates in increased morbidity and mortality. There are numerous aetiologies for low HDL cholesterol contents, and certain of these, such as type 2 diabetes, elevated triglycerides, obesity, overweight, and deficient physical exercise are related to insulin resistance. Also, implicated in low HDL content are high carbohydrate consumption, cigarette smoking, progestational drugs, anabolic steroids and beta blockers.

In an identical trajectory, ApoA-1 has the potential to undergo oxidative alterations which decrease anti-atherogenic function of HDL; elevated methionine sulfoxide (MetO) concentrations in ApoA-1 in premature MI and type 2 diabetes patients; with concomitant increased MetO concentrations in ApoA-1 leading to HDL dysfunction (Sartore et al., 2015). Thus, ApoA1 undergoes oxidative alterations which mitigate anti-atherogenic functionality of HDL in selected young subjects with CHD, and type 2 diabetic with no significant correlation in all parameters in healthy subjects. Elevated ApoA-1 levels are predictive of CHD or CAD. Type 2 diabetes results in dyslipidaemia, such as augmented triglyceride concentrations and decreased HDL contents which are established risk factors for coronary artery disease. Results show that increased concentration of ApoA-1 are not reflective of the glycaemic status, and are independent of increase in LDL: HDL ratio suggestive of disparate metabolic pathways and the genetic association for LDL and ApoA-1 (Singla et al., 2009).

### IV. LIGNANS, PHYTOESTROGENS, CAROTENOIDS

Lignans are polyphenols food micronutrients obtainable in plants. The lignan precursors are contained in an expansive variety of plant-based foods, such as fruits, legumes, seeds, vegetables and whole grains. The flaxseeds constitute the richest dietary resource of lignin precursors. On consumption, lignin precursors undergo conversion to the enterolignans, enterodiols and enterolactone by bacteria which conventionally inhabit the intestine of humans (Lampe, 2003; Rowland et al., 2003). Lignan-rich diet constitute portion of a healthy dietary regimen, that the functionality of lignans in the prevention or mitigation of hormone-associated oncological disorders is not pellucid. Lignans constitute the major source of dietary phytoestrogen in traditional Western diets (de Kleijn et al., 2002; Valsta et al., 2003). Studies suggest that phytoestrogens have anti-diabetic activity via both estrogen-dependent and oestrogen-independent pathways, with consideration that food sources, such as soy and whole flaxseed constitute portions of total

healthy dietary regimen to prevent and manage type 2 diabetes (Talaie & Pan, 2015). Certain lignans tend to lower insulin and blood sugar concentrations; and improve the cholesterol profile and glucose control in type 2 diabetes (Pan et al., 2007).

There is ample evidence that carotenoids mitigate diabetes risk because of their anti-oxidant attributes. A study (Sluijs et al., 2015) demonstrated that elevated contents of beta-carotene and alpha-carotene in diets have relationships with decreased incidence of type 2 diabetes in the healthy population. Diets with elevated concentrations of beta-cryptoxanthin, lycopene, lutein and zeaxanthin have no relationship with type 2 diabetes risk; and the relationships between dietary carotenoids and type 2 diabetes risk are not altered due to the smoking status of the subjects.

## V. ALCOHOL CONSUMPTION

An assessment of the association between alcoholic intake and type 2 diabetes incidence suggested that moderate consumption of alcohol was related to a decreased risk of type 2 diabetes development (Marques-Vidal & Vollenweider, 2015). In the study, no protective influence was associated between alcohol ingestion, and impaired fasting glucose because there was no defined association of type 2 diabetes and the quantity of intake. The study suggested that moderate alcohol intake is not associated with reduced risk of developing superimposed type 2 diabetes and impaired fasting glucose. Other findings demonstrate that associations between alcohol drinking frequency is associated with diabetes risk; and that alcohol consumption within three to four weekdays is connected with the lowest diabetes risk, even when incorporating average weekly alcohol intake; but taking into consideration ethnic background, family history, overweight and age (Burns, 2017).

## VI. CARDIOVASCULAR CONCERNS

The lowering of glucose by anti-glycaemic drugs in the early stages of type 2 diabetes may be beneficial in patients with cardiovascular disease and cardiac failure; although, it is class-dependent rather than on the resultant impact of glucose lowering. Certain classes of antiglycaemic drugs are prone to cause or elicit cardiac failure; but this is not undergirded by enough evidence (Kappel et al., 2015). Evidence stipulates that merely early intensive risk factor control can modulate CVD morbidity and mortality in subjects presenting with type 2 diabetes. A study (Catalan et al., 2015) detected a high preclinical atherosclerosis prevalence of carotid plaque presence and burden in new-onset type 2 diabetes patients, with predilection for women. Prompt intervention is effectual to prevent CVD and possibly reverse pre-clinical atherosclerosis. Carotid intima media thickness was exacerbated in new-onset

diabetes in comparison to matched controls, with significant prevalence more elevated in new-onset diabetes. HbA1c and atherogenic dyslipidaemia partly explicate these disparities. Glycaemic control optimization by employing a basal plus insulin approach by inducing a significant decrease in HbA1c profoundly improved HRV parameters correlating with sympathetic and parasympathetic functionalities; thus suggesting stringent glycaemic control employing insulin for the improvement of cardiovascular autonomic activities in type 2 diabetes (Maadjhou et al., 2017).

In addition, adiponectin is a vital adipocyte-secreted adipokine with insulin-sensitizing and antidiabetes attributes (Kadowaki et al., 2006). In contradistinction to several pro-inflammatory adipokines/cytokines secreted by adipose tissue, the plasma levels of adiponectin are reduced in obese persons and patients presenting with type 2 diabetes, hypertension and cardiovascular disease. Other than these metabolic functionalities, adiponectin impacts several protective influences against cardiovascular disorders, such as diabetic cardiomyopathy (Shibata et al., 2004), myocardial infarction (Shibata et al., 2005) and stroke amelioration (Nishimura, 2008). The protective influence of adiponectin diabetes vascular complications is partly due to its property to counteract hyperglycaemia-mediated reduction in available circulating endothelial progenitor cells which are causally connected with diabetes cardiovascular complication (Chang et al., 2010).

Extant data reveal strong positive correlation of high resting cardiac rate and risk of type 2 diabetes (Aune et al., 2015). Although, resting heart rate is predictive of cardiovascular disease risk, its association with diabetes remains inconclusive, especially in non-Western ambient (Zhang et al., 2010). An elevated resting cardiac rate was detected to be independently related to high risk of type 2 diabetes in women; and the association of high cardiac rate with increased BMI, BP or WHR measurements are connected with a significantly augmented risk. However, cardiac rate has been limited potential as a marker for the screening of patients with undiagnosed type 2 diabetes in rural areas (Li et al., 2014). Also, elevated allostatic load score, ALS is a determinant of the biologic response to stress, but its relationship with the risk for diabetes and cardiovascular disorders in the African migrant population has not been deciphered (Utumatwishima et al., 2017a). ALS measurement portends a valid cost-effective trajectory for the detection of diabetes and cardiovascular disease risk in the African population.

## VII. DIPEPTIDYL PEPTIDASE-4 INHIBITORS, INSULIN ANALOGUES, ANTIDIABETIC THERAPEUTIC AGENTS

Speculations are rife regarding the role of Dipeptidyl peptidase-4 (DPP-4) inhibitors in type 2 diabetes treatment, but DPP-4 inhibitors decrease HbA1c, albeit, to a magnitude less than sulfonylureas, with no production of weight gain or hypoglycaemic risk (Monami et al., 2010). Type 2 diabetes exhibits progressive dissipation of beta cell functionality, thereby necessitating usage of orally active DPP-4 inhibitors, such as sitagliptin and vildagliptin. DPP-4 inhibitors present certain theoretical advantages greater than extant therapies having oral antidiabetic compounds, but amenable to or compliant with selected patients (Richter et al., 2008). MACE rate is not elevated in the presence of DPP-4 inhibitors, thus undergirding the CV safety and compliance of these newfangled antidiabetic therapeutic agents. The application of TECOS suggests that high cardiac failure hospitalization resulting from saxagliptin is not related to class effect of DPP-4 inhibitors. It may be that an evening injection with NPH insulin in combination with an extant maximal therapy with metformin and sulfonylurea can be simple, effective and well-tolerated first-choice strategy by, or patients desiring oral medication (MDedge, 2004). Short-acting insulin secretagogues may be employed in fasting diabetic patients with predominantly postprandial hyperglycaemia (Bashir et al., 2015). Oral DPP-4 inhibitors constitute an alternative to sulfonylureas for diabetes patients during fasting due to their glucose-dependent mechanism of action, efficacy and tolerance, as they cause moderate HbA1c decrease, and being non-weight dependent, and have very low hypoglycaemic risk.

Incretinomimetics constitute novel type 2 diabetes drugs which increase glucose-induced insulin production. This drug class comprises two subclasses: Exogenous Glucagon-like Peptide analogues, GLP1a, such as liraglutide and the Dipeptidyl peptidase-4 inhibitors which elongate the half-life of endogenous GLP1, such as vildagliptin. These two subclasses do not exhibit significant disparities on insulin sensitivity and insulin secretion following two weeks of treatment in type 2 diabetes subjects (Well et al., 2017). Incretin-based regimen for therapeutic provisions in type 2 diabetes patients modify diverse aspects of the disorder, such as hypersecretion of glucagon, aberrant gastric evacuation, postprandial hyperglycaemic, and rarely, pancreatic beta-cell dysfunction. DPP-4 inhibitors, gliptins augment glucagon-like peptide-1 (GLP-1) provision and modulate "incretin defect" observed in type 2 diabetes patients (Godinho et al., 2015). Good glycaemic control with minimal hypoglycaemic risk or any aberrant effects have been reported, irrespective of pancreatitis reports which

have not been clearly defined. Research is aimed at the extant capability of DPP-4 inhibitors in enacting putative pancreas functionality, especially regarding the inhibition of apoptotic pathways and inducement of beta-cell proliferation. Also, other cytoprotamine impacts on certain organs/tissues which are more associated with adverse type 2 diabetes complications, such as cardiac, renal and ophthalmic perturbations have been demonstrated (Godinho et al., 2015; Dungan et al., 2017). They do not cause hypoglycaemia unless combined with therapeutic regimen that can cause such effects. The mechanism of DPP-4 inhibitors is to elevate incretin (GLP-1 and GIP) concentrations (McIntosh et al., 2005) causing the inhibition of glucagon release, leading to increased insulin secretion, with decreased gastric evacuation, and decreased blood glucose concentrations, accompanied by marginal statistically significant exacerbation of heart failure (Wu et al., 2014). There is extant warning that alogliptin, linagliptin, saxagliptin and sitagliptin as type 2 diabetes medications are liable to cause adverse, severe and disabling joint pain (USFDA, 2016). However, there are other minimal comparative utility evidence versus other therapeutic agents concerning other DPP-4 inhibitors, such as omargliptin and trelagliptin administered once-weekly (Stoimeni et al., 2017). The utilization data of glucagon-like peptide 1 (GLP1) receptor agonists and DPP-4 inhibitors in clinical practice showed that incretin prescriptions have been conducted in numerous cases extraneous to the regulatory limits; but appropriate utilization of incretins provided commensurate results and benefits as in pivotal trials (Montilla et al., 2014).

## VIII. COMBINATION THERAPY

The choice or selection and application of a glucose lowering drug depend on the severity of hyperglycaemia, hepatic and renal-related functionalities, hypoglycaemic risks, body mass index, blood glucose self-monitoring ability and cost-benefit analysis of available therapeutic regimen. Type 2 diabetes treatment modality involve a variety of prevailing therapeutics, such as sulfonylureas and nepadlimide which augment insulin secretion, troglitazone that induces increased insulin action in fat and muscle, metformin augments insulin action in fat and muscle; while miglitol and acarbose enact retarded carbohydrate absorption from food consumption, respectively (Buse, 1999). The drugs enacted for type 2 diabetes treatment pose significant side effects or adverse risks, whereas other combinational therapy of insulin and sulfonylureas decrease the daily insulin requirement (Riddle, 1996), insulin and metformin combination therapy (Golay et al., 1995), and troglitazone-insulin in combination effectually lowered insulin requirement and promoted glycaemic control (Buse et al., 1998).

The application of combination therapy is congruous for subjects presenting with type 2 diabetes because they frequently exhibit poor responses to single-drug therapeutic regimen. Metformin and troglitazone have similar and beneficial impacts on glycaemic control in type 2 diabetes patients. Metformin functions basically by reducing endogenous glucose formation, while troglitazone accelerates peripheral glucose disposal rate. Basal insulin analogues present decreased hypoglycaemic risk in comparison to NPH insulin, but hypoglycaemia persistently constitutes a major stumbling block for the achievement of recommended fasting plasma glucose targets in diabetic subjects (Russell-Jones et al., 2015; Chukwuma Sr, 2017b). Insulin degludec consistently achieved lower FPG concentrations when compared to insulin glargine. Reduced nocturnal rates established that hypoglycaemia manifested with insulin degludec, probably due to prolonged action and insulin degludec flat profile. Thus, the lower rate of nocturnal established hypoglycaemia observed with insulin degludec compared to insulin glargine culminates in a decreased mean FPG fasting plasma glucose, especially in type 2 diabetes patients. It was detected that HbA1c and fasting glucose are inadequate as screening diabetes measurement in an African migrant population (Utumatwishima et al., 2017b). With the increasing diabetes epidemic in Africa, one of the main challenges is the accurate assessment of the presenting asymptomatic persons affected. In recent decades, the OGTT is recognized as a diagnostic norm for diabetes detection, but it is expensive and time-consuming, thus necessitating an option for a single blood test, such as HbA1c and fasting plasma glucose. The elevated prevalence of both haemoglobin C trait and sickle cell trait, SCT may obscure HbA1c diagnostic value. Also, in populations of African descent, FPG functionality may be objectionable as a marker of asymptomatic diabetes. On that score, the magnitude of African diabetes may be submerged due to constraints and challenges in the provision and identification of feasible hyperglycaemic markers.

## IX. DISCUSSION AND CONCLUSION

Diabetes is a significant endocrine and metabolic phenomenon and disorder associated with mortality and morbidity with astronomical health system and socioeconomic pecuniary embarrassment. It is pertinent to continuously implement, monitor and evaluate population-based interventions and registries which prevent diabetes, provide modalities for its early detection, utilization of lifestyle and therapeutic interventions in the prevention and/or retardation of its attendant sequelae or progression to untoward complications. A study (Chukwuma Sr, 2017c) has attempted to develop and improve the welfare and well-

being of vulnerable populations in the interactions, comorbidities or co-occurrence for other diseases with diabetes. It is imperative to compare trends in diverse countries and regions, and coordinate progress towards the global target to stem the exacerbation of diabetes prevalence by 2015 as at 2010 (WHO, 2015; NCD, 2016).

The factors which are indicted for possible population level alterations in type 2 diabetes prevalence include combinational forces in personal attributes, and environmental risk factors or gene-environment interactions (Chukwuma Sr, 2014), the detection effect, the evolutionary process of diabetes and global changes (Thibault, 2016). Also, it is important to engage in a healthy lifestyle among adults with type 1 diabetes for quality control of cardiometabolic risk factors, such as body mass index, body composition (IDXA), blood pressure, glycated haemoglobin, lipids, waist circumference, with insulin resistance as estimated glucose disposal rate (Schwab & Uusitupa, 2015).

The awareness, prevention, treatment and control of diabetes need to be integrated with those of other non-communicable diseases. We need to reduce obesity by augmenting physical activity, intake of vegetables, legumes, fruits, cereals, whole grains, particularly of high fibre presence, and decreased intake of fats. Effective and efficient intervention strategies focused on the specific needs of the population are required to stem the increase of diabetes prevalence and incidence. Information systems for baseline data evaluation, monitoring, evaluation and implementation are needed for screening and metabolic control of patients to improve diabetes care. This paper, therefore, proffers the consensus for actions to prevent, treat and control diabetes, diabetes-associated conditions with attendant sequelae, and including determination of lifestyle changes. These strategies are clear and congruously accepted as constraints and challenges in the convergence of the prevention, treatment and control of type 2 diabetes in the clinical setting and in the real world.

In the current environment, the values to ensure equitable and justifiable utilization of scarce resources in preventing, abating and controlling the epidemic of diabetes and related disorders have become increasingly important. Researchers and several well-meaning institutions have been delivering rigorous unbiased evidence to advance rights, enhance quality and improve lives which will aid governments and agencies for the improvement of programmes and policies for the assurance of the global convergence on the constraints and challenges in the awareness, prevention, treatment and control of type 2 diabetes and related conditions.

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### Vitamin B12 Encephalopathy- A Case Series

By Sunil Kumar Agarwalla & Nasreen Ali

*M.K.C.G Medical College*

**Abstract-** Vitamin B12 deficiency manifests as triad of anaemia, gastrointestinal abnormalities and neurological abnormalities. The children with vitamin B12 deficiency are often misdiagnosed as it mimics autism spectrum disorders, colics and gastroenteritis. Its deficiency in children can cause poor weight gain, developmental regression, mental changes, abnormal movements, encephalopathy or may leads to long term neurological sequelae. The existence of vitamin B12 deficiency neuropathy was recognised in 1958. Seizures are rare but are seen especially in infants and there are only a few reports regarding the relationship between infantile spasm and vitamin B12 deficiency.

Here we report 3 cases of vitamin B12 encephalopathy who presented with seizure and neurodevelopmental delay. They were later diagnosed as a case of severe vit B12 deficiency and successfully treated with IM vit B12 resulting in good neurological outcome almost towards normal.

**Keywords:** *neurological abnormalities, autism spectrum disease, infantile spasm.*

**GJMR-F Classification:** *NLMC Code: WC 542*



*Strictly as per the compliance and regulations of:*



# Vitamin B12 Encephalopathy- A Case Series

Sunil Kumar Agarwalla<sup>α</sup> & Nasreen Ali<sup>ο</sup>

**Abstract-** Vitamin B12 deficiency manifests as triad of anaemia, gastrointestinal abnormalities and neurological abnormalities. The children with vitamin B12 deficiency are often misdiagnosed as it mimics autism spectrum disorders, colics and gastroenteritis. Its deficiency in children can cause poor weight gain, developmental regression, mental changes, abnormal movements, encephalopathy or may leads to long term neurological sequelae. The existence of vitamin B12 deficiency neuropathy was recognised in 1958. Seizures are rare but are seen especially in infants and there are only a few reports regarding the relationship between infantile spasm and vitamin B12 deficiency.

Here we report 3 cases of vitamin B12 encephalopathy who presented with seizure and neurodevelopmental delay. They were later diagnosed as a case of severe vit B12 deficiency and successfully treated with IM vit B12 resulting in good neurological outcome almost towards normal.

One of our case was presented with INFANTILE SPASM which being a rare form of seizure in a case of B12 encephalopathy.

Any child presented with encephalopathy with knuckle hyperpigmentation without any prior history of fever, loose stool, vomiting one has to do Complete blood count, comment on peripheral smear & serum B12 to rule out B12 deficiency. We want to emphasize that early diagnosis & prompt treatment can alter the disease process.

**Keywords:** neurological abnormalities, autism spectrum disease, infantile spasm.

## I. CASE REPORT

In 1<sup>st</sup> case -A 19 month old female baby admitted to paediatric department of MKCG medical college with complains of hyperpigmentation of skin for 4 months, unable to stand with support for 3 months and sudden flexion of neck, arms and thighs multiple times for 2 months. The child was born out of non-consanguinous marriage by normal vaginal delivery, the child has been continuing breast feed till now along with mixed diet from family pot. The family being vegetarian. The child was apparently normal till 15 months of age and had attained all milestones appropriate for age till 15 months of life, following which she gradually lost the ability to stand with support and sit by herself. There was no associated fever, headache and vomiting. On examination, the child was irritable. There was intermittent flexor spasm (infantile spasm) multiple times a day and there was hyperpigmentation of skin over tongue, knuckles, knee and thighs (Figure 1,2 and 3). There were no signs of meningitis, reflexes were brisk

and B/L plantar was flexor. CSF study was done to rule out meningitis, which came out to be normal. CBC showed severe anaemia and MCV was 94 fL (Figure 4). Because of macrocytic anaemia, knuckle pigmentation and neurological signs with a history of vegetarian diet, a provisional diagnosis of vitamin B12 deficiency was made. It was confirmed by doing serum B12 level, which came to be very low (<100pg/ml) (Figure 5). EEG came out to be normal. The patient was treated with IM neurobion injections daily for 7 days followed by weekly dose for 7 weeks. To control infantile spasm IV valproate started and after 72 hours oral clonazepam was added as seizure persisted. The patients cognition improved by day 3. The infantile spasm came under control from 6<sup>th</sup> day onwards and from day 8 it seized completely. The patient was successfully discharged after regaining all the developmental milestones appropriate for age (figure 6).

**Author α:** Associate Professor, Junior Resident, Department of Pediatrics, M.K.C.G Medical College, Berhampur, Ganjam, Odisha-760004, India.

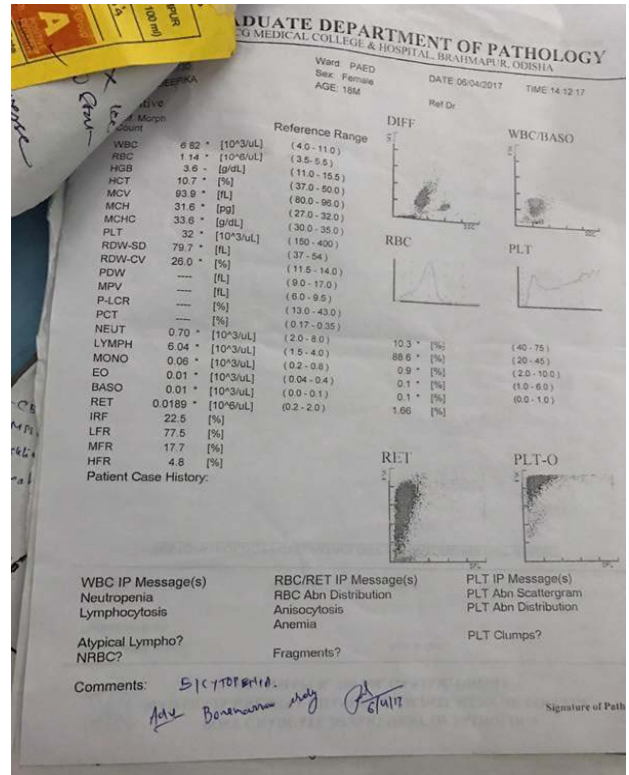


Figure 1, 2, 3: Showing pigmentation of lower limbs, hand and tongue

Figure 4: Showing CBC (low Hb and high MCV) Figure 6 showing happy child with ability to sit on its own

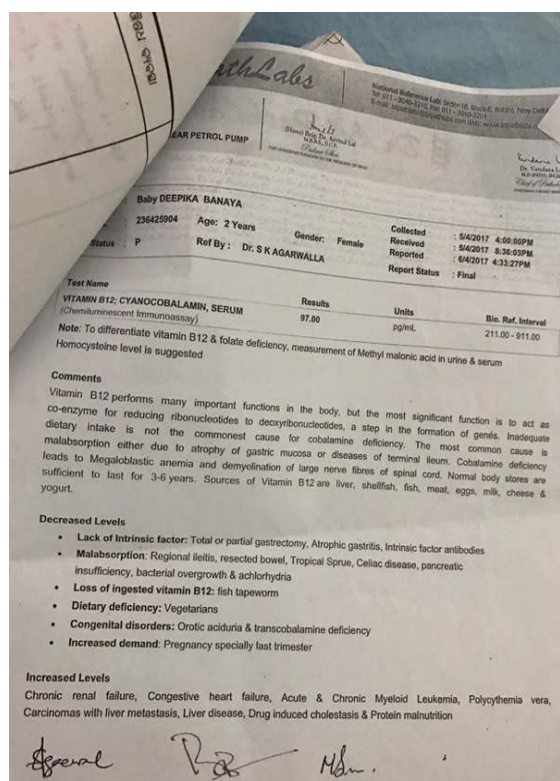


Figure 5: Showing serum B12 level

In 2<sup>nd</sup> case- A 5 yrs. old male child, product of non consanguineous marriage presented with unable to walk, stand, sit, speak with abnormal body movements and altered sensorium for last 15 days.(fig.1) There was no history of fever, convulsion, loose stool and vomiting, respiratory difficulty. There was also no history of birth asphyxia and child is neuro developmentally normal. No similar episodes in past or no sibling in the family having similar problem. On examination child was in altered sensorium, vitals were stable, anthropometric measurement was normal for the age. On head to toe examination child having knuckle and periungual hyperpigmentation of both limbs (fig.2, 3), angular stomatitis with sparse hypo pigmented brittle hair, some pallor, no cyanosis, clubbing, icterus, edema or lymphadenopathy. On CNS examination no cranial nerve deficit, hypotonia of both upper and lower limbs, power of both limbs was diminished (3/5). All superficial reflexes are normal except planter extensor, B/L Knee jerk was exaggerated and ankle jerk was diminished. Other systemic examination was within normal limits. On investigation complete blood count suggestive of megaloblastic anemia. Hb-6.7 gm%, MCV-110.8 fl. Serum electrolytes, urea, creatinine, liver function test was normal. Serum vitB12 was estimated and it was very low (73 pg/ml). CECT brain was also normal. After giving 2 wks. of daily vit B12(1mg IM) and folic acid, neurological and general well-being improved (fig 4,5). Repeat vit B12 level was 512pg/ml. Child

discharged with im vit B12 wky for 8 wk. then monthly once for 6 months along with folate therapy. The child was advised for monthly check up.



Fig. 1: Showing altered sensorium Fig. 2 & 3 Showing knuckle and periungual hyperpigmentation of both



Figure 4 & 5: Showing improved wellbeing

In the 3<sup>rd</sup> case-A 11 months female baby born out of non consanguineous marriage by normal vaginal delivery was admitted with complains of fever since last 6 days and fast breathing since last 2 days. On examination the child was febrile and pale, respiratory rate was 52/min with chest indrawing. There were B/L conducted sounds in the chest with creps. The liver was enlarged 6cm below the costal margin and spleen was just palpable. There was history of blood transfusion. Hence a provisional diagnosis of pneumonia with congenital hemolytic anaemia was made. On further inquiring about the history, it was found that the baby was apparently normal till 5 months of age, then she developed respiratory tract infection for which she was admitted in hospital for 4 days. Since then the baby had repeated respiratory tract infections. By 8 months of age there were regression of developmental milestones like ability to sit and neck control. There was one episode of generalized tonic clonic seizure. The child has been continuing breast feed till now along with mixed diet from family pot. The mother being vegetarian. There was hyperpigmentation of skin over palm, knuckles, knee and thighs (Figure 1,2 and 3). There were no signs of meningitis, reflexes were brisk and B/L plantar was flexor. CSF study was done to rule out meningitis, which

came out to be normal. CBC showed severe anaemia and MCV was 96.5 fL (Figure 4). Because of macrocytic anaemia, knuckle pigmentation and neurological signs with a history of vegetarian diet, a provisional diagnosis of vitamin B12 deficiency was made. It was confirmed by doing serum B12 level, which came to be low (145 pg/ml) (Figure 5). The patient was treated with IM neurobion injections daily for 7 days followed by weekly dose for 7 weeks. The patient was successfully discharged after regaining all the developmental milestones appropriate for age.





Vitamin B12 has a role in DNA synthesis, delayed DNA synthesis in rapidly growing hematopoietic cells may result in macrocytic anaemia. The neurological manifestation of cobalamin deficiency is may be due to homocysteine toxicity deposits in brain and infants may be predisposed due to incompletely formed blood brain barrier<sup>[5]</sup>.

Most of the initial data regarding vitamin B12 deficiency in infancy are from case studies of infants exclusively breast fed by mothers on vegetarian diet. This case reiterates the association between infantile spasm and vitamin B12 deficiency. Infantile spasm are a unique form of seizure disorder as their occurrence is mostly limited to infancy and they are refractory to conventional anticonvulsant drugs. In India, a hospital population radioassay study with a cut off of 200 pg/ml found a vitamin B12 deficiency in 0.88% of patients with border line values in 3.8%<sup>[5]</sup>. Infants born to vitamin B12 replete mothers have stores of vitamin B12 that are adequate to sustain them for first several months post partum hence vitamin B12 rarely occurs before 4 months of age<sup>[6]</sup>. The neurological complex, defined as myelosis funicularis, consists of the following symptoms:

1. Impaired perception of deep touch, pressure and vibration, loss of sense of touch, very annoying and persistent paresthesias
2. Ataxia of dorsal chord type
3. Decrease or loss of deep muscle-tendon reflexes
4. Pathological reflexes-Babinski, Rossolimo and others, also severe paresis.

### III. CONCLUSION

Encephalopathy due to vit B12 deficiency is very rare in children but any child presented with encephalopathy with knuckle hyperpigmentation without any prior history fever, loose stool, vomiting one has to do CBC, PS and serum B12 level to rule out B12 deficiency. Management with vit B12 supplementation and folic acid is mainstay of therapy. 90% patients have improvement in symptoms and rest 10% have residual moderate to severe disability following early treatment<sup>[7]</sup>. Hence early diagnosis and treatment is required.

#### Contributors

Dr. Sunil Kumar Agarwalla-revising it critically for important intellectual content.

Dr. Nasreen Ali-conception, design and drafting.

#### Conflict of Interest

There was no conflict of interest and no funds received.

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## Profile of Type 2 Diabetic Patients in Urban Slums of Mumbai

By Dr. Kedar J. Raikar, Dr. Gajanan D. Velhal & Dr. Anubha Shukla

*Seth GS Medical College*

**Abstract- Background:** The present study was carried out to study the disease profile of diabetic patients in an urban slum in Mumbai.

**Objective:** To study the patient profile among type 2 diabetic patients.

**Methodology:** Descriptive epidemiological study design was adopted. Sample size was fixed as 203.4 and samples are selected by simple random sampling technique.

**Important Finding:** Mean age of patient is 56.09 (SD=10.55) years and mean duration of disease is 5.37 (SD=4.13) years. Mean fasting and post prandial blood sugar are 171.56 (SD=52.37) and 254.71 (SD=79.60) respectively. Mean BMI is 25.62 (SD= 5.16) which is above the normal BMI while daily calorie intake is 1889.17 (SD= 588.23).

**Principal Conclusion:** Positive family history as an important risk factor. Hypertension is most common associated disease with diabetes. Ophthalmic complications are most frequent. 60% patients had diabetes past 5yrs.

**Keywords:** urban slum, hypertension, ophthalmic complications, disease profile.

**GJMR-F Classification:** NLMC Code: WD 200



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# Profile of Type 2 Diabetic Patients in Urban Slums of Mumbai

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## I. BACKGROUND

Incidence and prevalence of Diabetes Mellitus is increasing rapidly to the tune of recognizing it as modern epidemic. Contrary to our belief Diabetes is not limited only to the people of high socio economic profile but has also shown its existence across all categories of people. It emphasizes the need for periodic assessment of actions at different levels. The number of people with diabetes in India, currently around 40.9 million is expected to rise to 69.9 million by 2025 unless urgent preventive steps are taken. During the year 2004, there were an estimated 37.7 million cases of diabetes in country, of these 21.4 million in urban areas and 16.3 million in rural areas. The estimated total mortality due to diabetes was 1.09 lac; 62.5 thousand in urban areas and 46.6 thousand in rural areas. Same year 2.2 million DALYs were lost due to the disease.

*Authors α: Medical Officer, Army Medical Corps.*

*e-mails: kedarzone@gmail.com, kedarraikar1@gmail.com*

*Authors σ: Professor, Dept. of Community Medicine, Seth GS Medical College and KEM Hospital, Mumbai-400 012.*

*e-mail: vgajanan@rediffmail.com*

*Authors ρ: Medical Officer, Air Force Base, Jhalali, Bengaluru.*

*e-mail: anubha2525@gmail.com*

In 2013, according to the World Health Organization, at least 347million people worldwide suffer from diabetes, or 2.8% of the population.

The present study was undertaken to find out prevalence of Diabetes, to understand diabetes patient profile, associated diseases and complications in an urban slum area.

## II. MATERIALS AND METHODS

Study area and population-Urban slum area, Shivajinagar, Govandiwas the study area, it has population 84783 as per record of local health post.(census2011).

The population of study area consists of people migrated from different parts of India in search of job and are now engaged in small scale industries like Zari work, Bag making, Mat weaving and Carpentry etc. Most of men are self-employed and women are house wives, maid servants or vegetable vendors.

### a) Study Design

The present study adapted as descriptive epidemiological study, Community based. It was conducted during the period of February 2014 to December 2015.

### b) Sample size calculation was based on

- NFHS data 2005-06 indicates the population of more than 40 years is around 25.8%.This when applied to the study area, total Population of more than 40 years totals 21874. Assuming the prevalence of type 2 diabetes around 9.3%in urban slum (3,4), total type 2 diabetic expected were 2034.Taking 10% of this population, sample size was 203.4.The final sample of 215 were considered for study.
- Simple random sampling method (using random number table) was used for sample collection.

Descriptive statistics are used and data presented in percentages. For qualitative data analysis was done with chi square test. SPSS (16 version) was used for analysis of the data.

### III. RESULTS

*Table 1:* Socio Demographic profile (N=215, Male=71, Female=144)

| Class                |                   | Male |      | Female |      | Total |      |
|----------------------|-------------------|------|------|--------|------|-------|------|
|                      |                   | N    | (%)  | N      | (%)  | N     | (%)  |
| Age Groups           | 40-60             | 51   | 71.8 | 114    | 79.2 | 165   | 76.7 |
|                      | 61-80             | 17   | 23.9 | 27     | 18.8 | 44    | 20.5 |
|                      | >80               | 3    | 4.2  | 3      | 2.1  | 6     | 2.8  |
| Socioeconomic status | Class1            | 31   | 43.7 | 64     | 44.4 | 95    | 44.2 |
|                      | Class2            | 25   | 35.2 | 62     | 43.1 | 87    | 40.5 |
|                      | Class3            | 13   | 18.3 | 13     | 9.0  | 26    | 12.1 |
|                      | Class4            | 2    | 2.8  | 5      | 3.5  | 7     | 3.3  |
| Marital Status       | Unmarried         | 1    | 1.4  | 3      | 2.1  | 4     | 1.9  |
|                      | Married           | 60   | 84.5 | 64     | 44.4 | 124   | 57.7 |
|                      | Widow             | 10   | 14.1 | 77     | 53.5 | 87    | 40.5 |
| Type of Family       | Nuclear           | 49   | 69   | 71     | 49.3 | 120   | 55.8 |
|                      | Joint             | 7    | 9.9  | 22     | 15.3 | 29    | 13.5 |
|                      | Extended          | 15   | 21.1 | 51     | 35.4 | 66    | 30.7 |
| Education            | Illiterate        | 16   | 22.5 | 78     | 54.2 | 94    | 43.7 |
|                      | Primary           | 12   | 16.9 | 33     | 22.9 | 45    | 20.9 |
|                      | Secondary         | 25   | 35.2 | 32     | 22.2 | 57    | 26.5 |
|                      | Intermediate      | 11   | 15.5 | 0      | 0    | 11    | 5.1  |
|                      | Graduate & above  | 7    | 9.9  | 1      | 0.7  | 8     | 3.7  |
| Occupation           | Unemployed        | 4    | 5.6  | 130    | 90.3 | 134   | 62.3 |
|                      | Unskilled         | 5    | 7    | 4      | 2.8  | 9     | 4.2  |
|                      | Semiskilled       | 13   | 18.3 | 3      | 2.1  | 16    | 7.4  |
|                      | Skilled           | 16   | 22.5 | 4      | 2.8  | 20    | 9.3  |
|                      | Semi professional | 1    | 1.4  | 1      | 0.7  | 2     | 0.9  |
|                      | Professional      | 2    | 2.8  | 0      | 0    | 2     | 0.9  |
|                      | Retired           | 30   | 42.3 | 2      | 1.4  | 32    | 14.9 |
| Total                |                   | 71   | 100  | 144    | 100  | 215   | 100  |

In this study, out of the total study population of 215, 67% (144) were females and 33% (71) were males. The number of female patients were more because of absence of male patients at home due to their being at work, at the time of visit. 76.7% (165) study subjects were in the age group of 40-60 years followed by those in 61 – 80 years i.e 44 study subjects (Male- 17(23.9%), Female – 27(18.8%)). Remaining 6 study subjects

(Male – 3(4.2%), Female- 3 (2.1%)) belonged to the age group of 80 years and above.

According to modified Prasad classification 84.7% (182) of patients belonged to Socio-economic class 1 & 2.

Above table shows that 57.7% are married and 40.5% of study subjects were widows. Majority i.e 55.8% belongs to Nuclear family. Out of 215, 94 (43.7%)

patients are illiterate while only 3.7% patients are Graduate. Because study population is an urban slum population, literacy rate is poor. 62.3% of study subjects

were unemployed because most of the participants were female and were house wives.

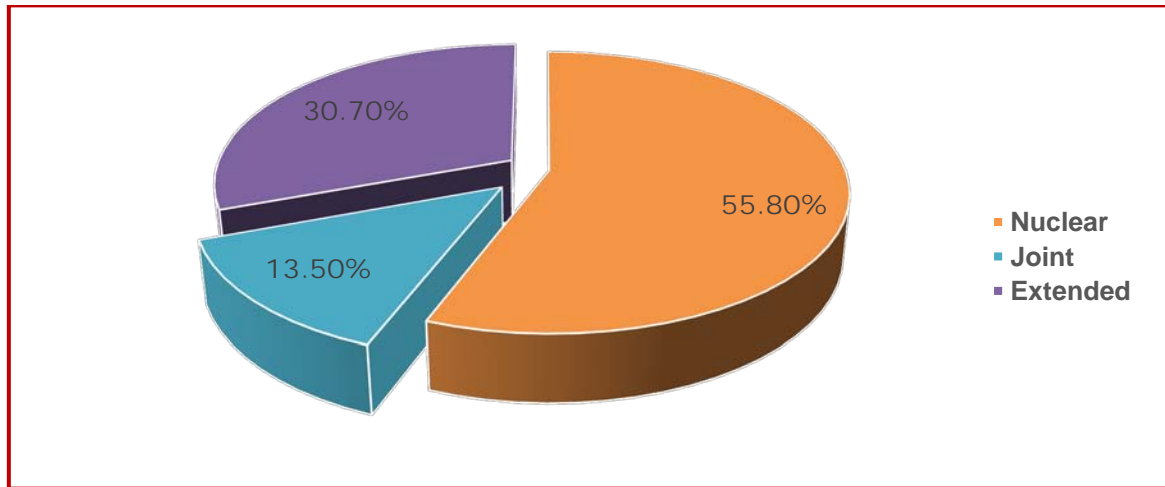


Figure 1: Distribution of study subjects according to their types of family

Table 2: Various aspects about disease (N= 215)

| Class                       |                | Male |      | Female |      | Total |      |
|-----------------------------|----------------|------|------|--------|------|-------|------|
|                             |                | N    | (%)  | N      | (%)  | N     | %    |
| F/H of Diabetes             | Present        | 20   | 28.2 | 39     | 27.1 | 59    | 27.4 |
|                             | Absent         | 51   | 71.8 | 105    | 72.9 | 156   | 72.6 |
| Associated Diseases (N=117) | HTN            | 26   | 76.5 | 65     | 78.3 | 91    | 77.8 |
|                             | IHD            | 1    | 2.9  | 4      | 4.8  | 5     | 4.3  |
|                             | HTN+IHD        | 5    | 14.7 | 11     | 13.3 | 16    | 13.7 |
|                             | Others(stroke) | 2    | 5.9  | 3      | 3.6  | 5     | 4.2  |
| Complications (N= 59)       | Ophthalmic     | 7    | 50.0 | 23     | 51.1 | 30    | 50.8 |
|                             | Renal          | 4    | 28.7 | 7      | 15.6 | 11    | 18.6 |
|                             | Foot           | 1    | 7.1  | 6      | 13.3 | 7     | 11.9 |
|                             | Heart          | 1    | 7.1  | 5      | 11.1 | 6     | 10.2 |
|                             | Others *       | 1    | 7.1  | 4      | 8.9  | 5     | 8.5  |
| Duration of Diabetes        | < 5 years      | 42   | 59.2 | 87     | 60.4 | 129   | 60   |
|                             | 5-10years      | 18   | 25.4 | 40     | 27.8 | 58    | 27.0 |
|                             | 10-15 years    | 6    | 8.4  | 14     | 9.7  | 20    | 9.3  |
|                             | > 15 years     | 5    | 7.0  | 3      | 2.1  | 8     | 3.7  |
| Total                       |                | 71   | 100  | 144    | 100  | 215   | 100  |

\* Impotency, peripheral neuropathy

Its seen from above table that 27.4% of patients have positive family history which is important risk factor for diabetes. 54.4% patients have associated diseases; most common associated disease is hypertension (77.8%). 27.4% of patients have complications of

diabetes; most common complication is ophthalmic like retinopathy and cataract (50.8%). 60% of patients have diabetes since less than 5 years and only 3.7% of patients had diabetes since more than 15 years.

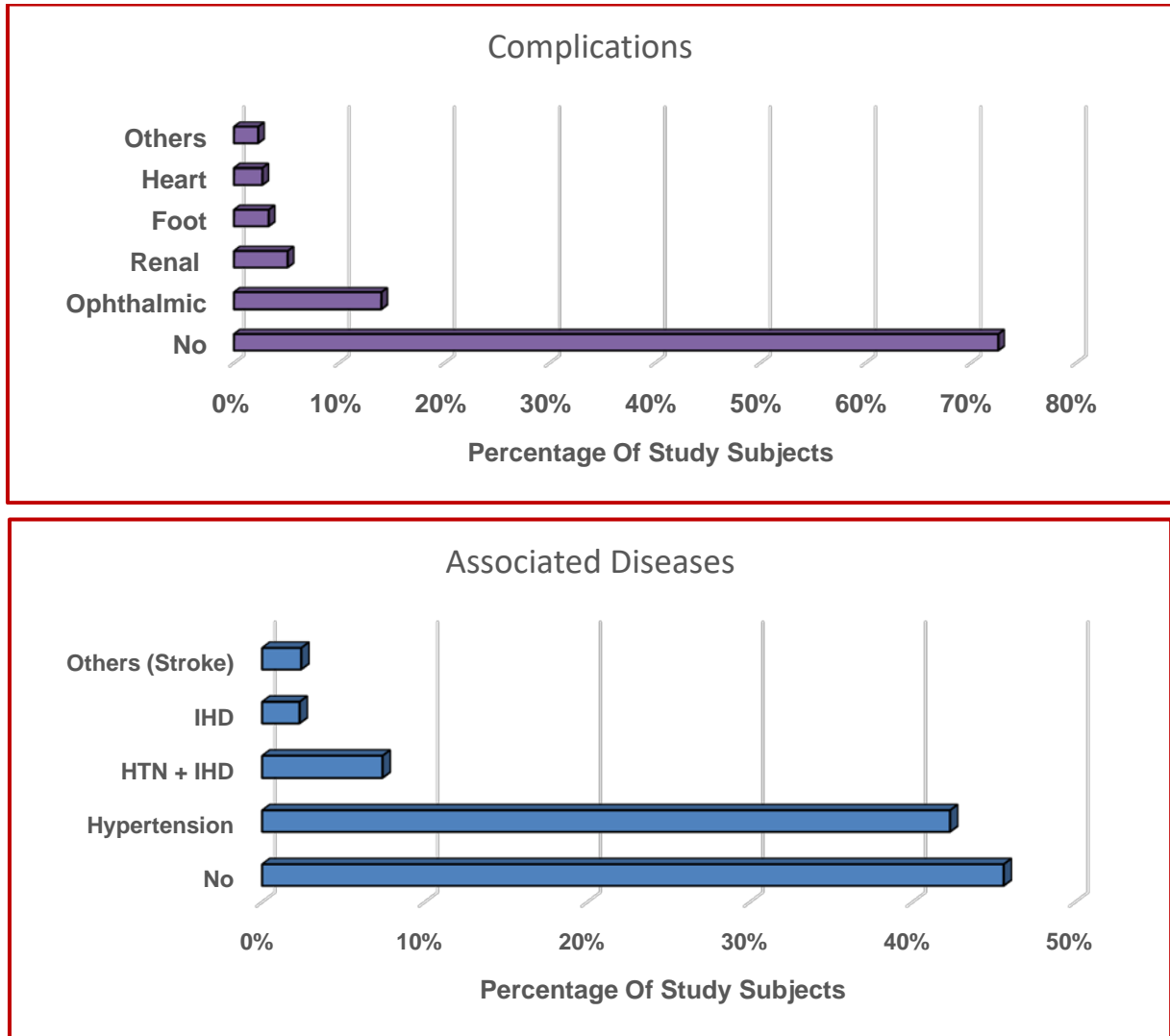


Figure 2: Distribution of Study Subjects According To Complications of Diabetes and Associated Diseases

Table 3: Risk factor profile of study subjects.(N= 215)

| Variables                | Minimum | Maximum | Mean    | Std. Deviation |
|--------------------------|---------|---------|---------|----------------|
| Age                      | 40      | 82      | 56.09   | 10.55          |
| Duration of Disease      | 2       | 20      | 5.37    | 4.13           |
| Systolic Blood Pressure  | 100     | 210     | 141.71  | 19.19          |
| Diastolic Blood Pressure | 60      | 120     | 94.08   | 11.94          |
| Fasting Blood Sugar      | 75      | 408     | 171.56  | 52.37          |
| PP Blood Sugar           | 100     | 562     | 254.71  | 79.60          |
| BMI                      | 15.60   | 40.00   | 25.62   | 5.16           |
| Calories Intake          | 1235    | 4256    | 1889.17 | 588.23         |



The above table shows that mean age of patient is 56.09 (SD=10.55) years and mean duration of disease is 5.37 (SD=4.13) years. Mean fasting and post prandial blood sugar are 171.56 (SD=52.37) and 254.71 (SD=79.60) respectively. Mean BMI is 25.62 (SD= 5.16) which is above the normal BMI while daily calorie intake is 1889.17 (SD= 588.23). This may be because of decrease calorie intake after diagnosis.

#### IV. DISCUSSION

The present community based descriptive epidemiological study, was conducted at an urban slum which is a field practice area of Department of Community Medicine. It was conducted during the period of February 2014 to December 2015, by selecting 250 diagnosed cases of type II diabetics who were identified from the community (Sample size was 203). Taking into consideration of loss to follow up 215 study subjects data was analysed.

In this study total 2123 diabetic patients were found in community, which gives the present prevalence of 9.7% in population above 40 years of age.

In this study (Table no 1) majority of patients were females this due to fact that survey was carried out during day time and majority of males of this community were engaged in their occupation such as zari work, bag making etc. Majority i.e. 76.7% of patients are in group between 40 to 60 years. Type 2 diabetes usually comes during this age group. According to modified Prasad's classification 113 (52.6%) patients belonged to socioeconomic class 2 and 3.

According to Chennai urban population study 5, the middle income group had significantly higher prevalence of type 2 diabetes compared to the low income group. Age standardised prevalence rates of Diabetes is 12.4% and 6.4% in middle income group and low income group respectively.

The study done by Singh TP, Singh AD, Singh TB from Manipur 6 reported a prevalence of 4.0% in a population aged above 45 years.

The KAP study done by Viral N. Shah et al on 238 Patients in saurashtra region Gujarat 7 shows that 61.41% of the patients were in age group between 40 to 60 years. Thus the findings of our study are in accordance with the studies done above.

The study population was mostly inhabited by Muslim population who migrated to Mumbai especially from Kerala, Andhra Pradesh Tamil Nadu. Majority i.e. 205 (75.37 %) of patients belonged to Muslim religion. Almost all i.e. 236 (86.37%) were married.

This study (Table no 1) undertaken in an urban slum population, showed poor literacy rate. Majority of the patients i.e. 94 (43.70%) were illiterate, 45 (20.90%) completed their education up to primary school and only 57 (26.50%) had completed Secondary education.

The study done by Viral N. Shah et al in Saurashtra region in Gujarat 7 also shows that 88.99% of

diabetic patients were either illiterate or had education up to secondary school.

62.3% of patients were unemployed because most of participants were female and housewives. In a study 8 it is found that diabetes is more common among women with lower education compared to higher level of education. Women who had been engaged in manual labour, had diabetes more often compared to those engaged in administrative work.

Family history of diabetes is a major risk factor for development of type 2 Diabetes. In this study (Table no 2) 59 out of 215 patients (27.40%) have positive family history.

The cross sectional study 9 carried out by D ShobhaMalini, A Sahu, et al in Behrampur, Orissa 58% of cases with Diabetes and IGT were having positive family history of Diabetes.

Similar cross sectional study 10 done by Chythra R. Rao, et al in Karnataka the positive family history of diabetes was present in 26% of the population.

In this study 54.4% of patients had associated diseases like hypertension (42.30%) and 27.4% of patients had complications. The most common complications were ophthalmic (14%) like retinopathy and cataract, nephropathy (5.1%), diabetic foot (3.3%). Females were more prone to associated diseases and complications. Associated diseases were more common in 40-60 years of age groups.

Type 2 Diabetes mellitus in association with other medical disorders like obesity and hyperlipidaemia predisposed to cardiovascular disorders. This cluster of condition is known as syndrome X 11. Diabetes is an important component of complex cardiovascular risk factors, and is responsible for acceleration and worsening of atherothrombosis. Major cardiovascular events cause, about 80% mortality in patients with type 2 diabetes patients 12, coronary artery disease, hypertension and insulin resistance. Atherosclerosis is responsible for over 80% of mortality in patients with type 2 diabetes, of which 75% due to coronary atherosclerosis and 25% is attributed to cerebrovascular or peripheral vascular disease. Over 50% of newly diagnosed type 2 diabetic patients suffer from coronary artery disease. 13

In a cohort study 14, relative risk of all diabetes related mortality in the cohort compared to general population was 2.31 in women and 1.58 in men.

Mean age of patients in this study was 56.09 (SD=10.55) years and mean duration of disease was 5.37 (SD=4.13) years (Table 3). It was observed in Bangalore Urban District Diabetes (BUD) 15 study that the mean age at diagnosis was 48.3 years to those who were aware of diabetes than 50.1 years for those not aware and 47.7 years for those with family history than 50.5 years for those without family history. Almost seven years delay in diagnosis was found between illiterate

and college educated persons and an almost three year delay between city and semi urban area.<sup>16</sup>

In this study both fasting and post prandial sugar level were high (Fasting >140, PP >200) in 70.3% and 73.5% of patients respectively. Mean blood sugar level were also found to be high (Fasting and PP blood sugar, 171.56(SD= 52.37) and 254.71 (SD=79.60) respectively. (Table 3). It means though patients had diabetes for more than five years still the blood sugar were not under control.

Obesity is an important risk factor to diabetes as it causes insulin resistance on target cells, and higher BMI is associated with high mortality in diabetes. In present study 53 % ( 114) of patients had BMI more than 25. Mean BMI was 25.62(SD=5.16) which is above the normal BMI.

## V. CONCLUSION

Risk factor profile of diabetic profile was studied and findings were mentioned in results followed by discussion.

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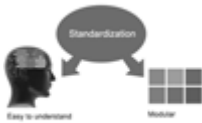


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3. Submission of Manuscripts,
4. Manuscript's Category,
5. Structure and Format of Manuscript,
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**10. Bookmarks are useful:** When you read any book or magazine, you generally use bookmarks, right! It is a good habit, which helps to not to lose your continuity. You should always use bookmarks while searching on Internet also, which will make your search easier.

**11. Revise what you wrote:** When you write anything, always read it, summarize it and then finalize it.



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**18. Pick a good study spot:** To do your research studies always try to pick a spot, which is quiet. Every spot is not for studies. Spot that suits you choose it and proceed further.

**19. Know what you know:** Always try to know, what you know by making objectives. Else, you will be confused and cannot achieve your target.

**20. Use good quality grammar:** Always use a good quality grammar and use words that will throw positive impact on evaluator. Use of good quality grammar does not mean to use tough words, that for each word the evaluator has to go through dictionary. Do not start sentence with a conjunction. Do not fragment sentences. Eliminate one-word sentences. Ignore passive voice. Do not ever use a big word when a diminutive one would suffice. Verbs have to be in agreement with their subjects. Prepositions are not expressions to finish sentences with. It is incorrect to ever divide an infinitive. Avoid clichés like the disease. Also, always shun irritating alliteration. Use language that is simple and straight forward. put together a neat summary.

**21. Arrangement of information:** Each section of the main body should start with an opening sentence and there should be a changeover at the end of the section. Give only valid and powerful arguments to your topic. You may also maintain your arguments with records.

**22. Never start in last minute:** Always start at right time and give enough time to research work. Leaving everything to the last minute will degrade your paper and spoil your work.

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**24. Never copy others' work:** Never copy others' work and give it your name because if evaluator has seen it anywhere you will be in trouble.

**25. Take proper rest and food:** No matter how many hours you spend for your research activity, if you are not taking care of your health then all your efforts will be in vain. For a quality research, study is must, and this can be done by taking proper rest and food.

**26. Go for seminars:** Attend seminars if the topic is relevant to your research area. Utilize all your resources.



**27. Refresh your mind after intervals:** Try to give rest to your mind by listening to soft music or by sleeping in intervals. This will also improve your memory.

**28. Make colleagues:** Always try to make colleagues. No matter how sharper or intelligent you are, if you make colleagues you can have several ideas, which will be helpful for your research.

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**30. Think and then print:** When you will go to print your paper, notice that tables are not be split, headings are not detached from their descriptions, and page sequence is maintained.

**31. Adding unnecessary information:** Do not add unnecessary information, like, I have used MS Excel to draw graph. Do not add irrelevant and inappropriate material. These all will create superfluous. Foreign terminology and phrases are not apropos. One should NEVER take a broad view. Analogy in script is like feathers on a snake. Not at all use a large word when a very small one would be sufficient. Use words properly, regardless of how others use them. Remove quotations. Puns are for kids, not grunt readers. Amplification is a billion times of inferior quality than sarcasm.

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**33. Report concluded results:** Use concluded results. From raw data, filter the results and then conclude your studies based on measurements and observations taken. Significant figures and appropriate number of decimal places should be used. Parenthetical remarks are prohibitive. Proofread carefully at final stage. In the end give outline to your arguments. Spot out perspectives of further study of this subject. Justify your conclusion by at the bottom of them with sufficient justifications and examples.

**34. After conclusion:** Once you have concluded your research, the next most important step is to present your findings. Presentation is extremely important as it is the definite medium through which your research is going to be in print to the rest of the crowd. Care should be taken to categorize your thoughts well and present them in a logical and neat manner. A good quality research paper format is essential because it serves to highlight your research paper and bring to light all necessary aspects in your research.

## INFORMAL GUIDELINES OF RESEARCH PAPER WRITING

### Key points to remember:

- Submit all work in its final form.
- Write your paper in the form, which is presented in the guidelines using the template.
- Please note the criterion for grading the final paper by peer-reviewers.

### Final Points:

A purpose of organizing a research paper is to let people to interpret your effort selectively. The journal requires the following sections, submitted in the order listed, each section to start on a new page.

The introduction will be compiled from reference matter and will reflect the design processes or outline of basis that direct you to make study. As you will carry out the process of study, the method and process section will be constructed as like that. The result segment will show related statistics in nearly sequential order and will direct the reviewers next to the similar intellectual paths throughout the data that you took to carry out your study. The discussion section will provide understanding of the data and projections as to the implication of the results. The use of good quality references all through the paper will give the effort trustworthiness by representing an alertness of prior workings.



Writing a research paper is not an easy job no matter how trouble-free the actual research or concept. Practice, excellent preparation, and controlled record keeping are the only means to make straightforward the progression.

#### **General style:**

Specific editorial column necessities for compliance of a manuscript will always take over from directions in these general guidelines.

To make a paper clear

- Adhere to recommended page limits

Mistakes to evade

- Insertion a title at the foot of a page with the subsequent text on the next page
- Separating a table/chart or figure - impound each figure/table to a single page
- Submitting a manuscript with pages out of sequence

In every sections of your document

- Use standard writing style including articles ("a", "the," etc.)
- Keep on paying attention on the research topic of the paper
- Use paragraphs to split each significant point (excluding for the abstract)
- Align the primary line of each section
- Present your points in sound order
- Use present tense to report well accepted
- Use past tense to describe specific results
- Shun familiar wording, don't address the reviewer directly, and don't use slang, slang language, or superlatives
- Shun use of extra pictures - include only those figures essential to presenting results

#### **Title Page:**

Choose a revealing title. It should be short. It should not have non-standard acronyms or abbreviations. It should not exceed two printed lines. It should include the name(s) and address (es) of all authors.



## Abstract:

The summary should be two hundred words or less. It should briefly and clearly explain the key findings reported in the manuscript-- must have precise statistics. It should not have abnormal acronyms or abbreviations. It should be logical in itself. Shun citing references at this point.

An abstract is a brief distinct paragraph summary of finished work or work in development. In a minute or less a reviewer can be taught the foundation behind the study, common approach to the problem, relevant results, and significant conclusions or new questions.

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- Reason of the study - theory, overall issue, purpose
- Fundamental goal
- To the point depiction of the research
- Consequences, including definite statistics - if the consequences are quantitative in nature, account quantitative data; results of any numerical analysis should be reported
- Significant conclusions or questions that track from the research(es)

## Approach:

- Single section, and succinct
- As an outline of job done, it is always written in past tense
- A conceptual should situate on its own, and not submit to any other part of the paper such as a form or table
- Center on shortening results - bound background information to a verdict or two, if completely necessary
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- Explain the value (significance) of the study
- Shield the model - why did you employ this particular system or method? What is its compensation? You strength remark on its appropriateness from a abstract point of vision as well as point out sensible reasons for using it.
- Present a justification. Status your particular theory (es) or aim(s), and describe the logic that led you to choose them.
- Very for a short time explain the tentative propose and how it skilled the declared objectives.

## Approach:

- Use past tense except for when referring to recognized facts. After all, the manuscript will be submitted after the entire job is done.
- Sort out your thoughts; manufacture one key point with every section. If you make the four points listed above, you will need a least of four paragraphs.



- Present surroundings information only as desirable in order hold up a situation. The reviewer does not desire to read the whole thing you know about a topic.
- Shape the theory/purpose specifically - do not take a broad view.
- As always, give awareness to spelling, simplicity and correctness of sentences and phrases.

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This part is supposed to be the easiest to carve if you have good skills. A sound written Procedures segment allows a capable scientist to replacement your results. Present precise information about your supplies. The suppliers and clarity of reagents can be helpful bits of information. Present methods in sequential order but linked methodologies can be grouped as a segment. Be concise when relating the protocols. Attempt for the least amount of information that would permit another capable scientist to spare your outcome but be cautious that vital information is integrated. The use of subheadings is suggested and ought to be synchronized with the results section. When a technique is used that has been well described in another object, mention the specific item describing a way but draw the basic principle while stating the situation. The purpose is to text all particular resources and broad procedures, so that another person may use some or all of the methods in one more study or referee the scientific value of your work. It is not to be a step by step report of the whole thing you did, nor is a methods section a set of orders.

#### **Materials:**

- Explain materials individually only if the study is so complex that it saves liberty this way.
- Embrace particular materials, and any tools or provisions that are not frequently found in laboratories.
- Do not take in frequently found.
- If use of a definite type of tools.
- Materials may be reported in a part section or else they may be recognized along with your measures.

#### **Methods:**

- Report the method (not particulars of each process that engaged the same methodology)
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- To be succinct, present methods under headings dedicated to specific dealings or groups of measures
- Simplify - details how procedures were completed not how they were exclusively performed on a particular day.
- If well known procedures were used, account the procedure by name, possibly with reference, and that's all.

#### **Approach:**

- It is embarrassed or not possible to use vigorous voice when documenting methods with no using first person, which would focus the reviewer's interest on the researcher rather than the job. As a result when script up the methods most authors use third person passive voice.
- Use standard style in this and in every other part of the paper - avoid familiar lists, and use full sentences.

#### **What to keep away from**

- Resources and methods are not a set of information.
- Skip all descriptive information and surroundings - save it for the argument.
- Leave out information that is immaterial to a third party.

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The principle of a results segment is to present and demonstrate your conclusion. Create this part a entirely objective details of the outcome, and save all understanding for the discussion.

The page length of this segment is set by the sum and types of data to be reported. Carry on to be to the point, by means of statistics and tables, if suitable, to present consequences most efficiently. You must obviously differentiate material that would usually be incorporated in a study editorial from any unprocessed data or additional appendix matter that would not be available. In fact, such matter should not be submitted at all except requested by the instructor.



## Content

- Sum up your conclusion in text and demonstrate them, if suitable, with figures and tables.
- In manuscript, explain each of your consequences, point the reader to remarks that are most appropriate.
- Present a background, such as by describing the question that was addressed by creation an exacting study.
- Explain results of control experiments and comprise remarks that are not accessible in a prescribed figure or table, if appropriate.
- Examine your data, then prepare the analyzed (transformed) data in the form of a figure (graph), table, or in manuscript form.

### What to stay away from

- Do not discuss or infer your outcome, report surroundings information, or try to explain anything.
- Not at all, take in raw data or intermediate calculations in a research manuscript.
- Do not present the similar data more than once.
- Manuscript should complement any figures or tables, not duplicate the identical information.
- Never confuse figures with tables - there is a difference.

### Approach

- As forever, use past tense when you submit to your results, and put the whole thing in a reasonable order.
- Put figures and tables, appropriately numbered, in order at the end of the report
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### Figures and tables

- If you put figures and tables at the end of the details, make certain that they are visibly distinguished from any attach appendix materials, such as raw facts
- Despite of position, each figure must be numbered one after the other and complete with subtitle
- In spite of position, each table must be titled, numbered one after the other and complete with heading
- All figure and table must be adequately complete that it could situate on its own, divide from text

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The Discussion is expected the trickiest segment to write and describe. A lot of papers submitted for journal are discarded based on problems with the Discussion. There is no head of state for how long a argument should be. Position your understanding of the outcome visibly to lead the reviewer through your conclusions, and then finish the paper with a summing up of the implication of the study. The purpose here is to offer an understanding of your results and hold up for all of your conclusions, using facts from your research and generally accepted information, if suitable. The implication of result should be visibly described. Infer your data in the conversation in suitable depth. This means that when you clarify an observable fact you must explain mechanisms that may account for the observation. If your results vary from your prospect, make clear why that may have happened. If your results agree, then explain the theory that the proof supported. It is never suitable to just state that the data approved with prospect, and let it drop at that.

- Make a decision if each premise is supported, discarded, or if you cannot make a conclusion with assurance. Do not just dismiss a study or part of a study as "uncertain."
- Research papers are not acknowledged if the work is imperfect. Draw what conclusions you can based upon the results that you have, and take care of the study as a finished work
- You may propose future guidelines, such as how the experiment might be personalized to accomplish a new idea.
- Give details all of your remarks as much as possible, focus on mechanisms.
- Make a decision if the tentative design sufficiently addressed the theory, and whether or not it was correctly restricted.
- Try to present substitute explanations if sensible alternatives be present.
- One research will not counter an overall question, so maintain the large picture in mind, where do you go next? The best studies unlock new avenues of study. What questions remain?
- Recommendations for detailed papers will offer supplementary suggestions.

### Approach:

- When you refer to information, differentiate data generated by your own studies from available information
- Submit to work done by specific persons (including you) in past tense.
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| <i>Introduction</i>           | Containing all background details with clear goal and appropriate details, flow specification, no grammar and spelling mistake, well organized sentence and paragraph, reference cited | Unclear and confusing data, appropriate format, grammar and spelling errors with unorganized matter | Out of place depth and content, hazy format                        |
| <i>Methods and Procedures</i> | Clear and to the point with well arranged paragraph, precision and accuracy of facts and figures, well organized subheads  | Difficult to comprehend with embarrassed text, too much explanation but completed                   | Incorrect and unorganized structure with hazy meaning              |
| <i>Result</i>                 | Well organized, Clear and specific, Correct units with precision, correct data, well structuring of paragraph, no grammar and spelling mistake   | Complete and embarrassed text, difficult to comprehend  | Irregular format with wrong facts and figures                      |
| <i>Discussion</i>             | Well organized, meaningful specification, sound conclusion, logical and concise explanation, highly structured paragraph reference cited   | Wordy, unclear conclusion, spurious   | Conclusion is not cited, unorganized, difficult to comprehend      |
| <i>References</i>             | Complete and correct format, well organized  | Beside the point, Incomplete  | Wrong format and structuring                                       |



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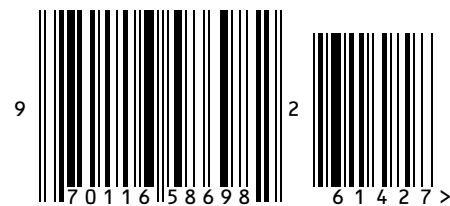
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