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STUDY OF CARBOHYDRATE METABOLISM IN SEVERE ACUTE MALNUTRITION AND CORRELATIONS OF WEIGHT AND HEIGHT WITH PP-SUGAR AND BMI

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ABSTRACT- Under nutrition is often an invisible and silent emergency.[2] It recognizes that hunger and malnutrition are rooted in poverty, deprivation, and under development, and that they are the result of inadequate access to the basic requirements for nutritional well-being, including safe and adequate food, care, health, education and a clean environment.[1] Present study was designed to find the efficacy of the nutritional intervention for the recovery of impaired carbohydrate metabolism and correlation of weight and height with PP-Sugar and BMI after nutritional rehabilitation.105 test and 100 control SAM children without infection, of 1 to 5 years of age and either sex were enrolled. Test group was given treatment of nutritional intervention therapy, providing 2.5 to 3gm Protein and 90-100 kcal / kg body Weight/day, for the three months.

Their blood sugar, BMI, weight and height were measured before and after the nutritional therapy. Before the nutritional intervention treatment P values for F and PP blood glucose, BMI, Weight and height were insignificant suggestive of similar baseline characteristics at enrollment. After nutritional intervention treatment P values for F and PP blood glucose, BMI, Weight and height were significant suggestive.

The r value of Pearson correlation coefficient for Sugar PP in the study group was, showing poor positive correlation with height and r value for BMI in the study group was showing poor negative correlation with height.

The r value of Pearson correlation coefficient for Sugar PP in the study group was, showing poor negative correlation with weight and r value for BMI in the study group was showing poor positive correlation with weight.

Depending on results the investigators conclude that for the speedy recovery of the impaired carbohydrate metabolism in SAM children it is the most effective food supplement.

Keywords: Glucose, Weight, Height, BMI, Nutritional intervention, SAM.

I. INTRODUCTION

Under nutrition is often an invisible and silent emergency.[1] It recognizes that hunger and malnutrition are rooted in poverty, deprivation, and under development, and that they are the result of inadequate access to the basic requirements for nutritional well-being, including safe and adequate food, care, health, education and a clean environment. [2] More than 90% of the world's stunted children live in Africa and Asia. The World Health Organization states that poor nutrition is the most important single threat to the world's health. [3] some degree of glucose intolerance mostly observed in malnutrition.

The investigators of this study have assumed that there is a considerable association between glucose intolerance, and poor β cell response to glucose load. In support of this view, it has been noticed that other workers have shown among experimental animals, which were maintained on low protein and high carbohydrate contents, has low plasma insulin levels [4] this suggestion needs reassessment of β cell function after dietary rehabilitation and supplementation. Therefore present study has focused on monitoring the glucose intolerance after providing nutritional intervention to the malnourished children Similarly Body mass index (weight in Kg./height in meters2) is the most widely used anthropometric index for assessment of nutritional status as it reflects the effect of both acute and chronic energy deficiency as well as excess. [5]

Various workers have suggested that the usefulness of currently used cut off points of BMI as indicator of various metabolic functions- especially carbohydrates, work capacity and health indices should need to be more studied[5] hence in present study, investigators have also monitored BMI and its correlations with blood sugar content were studied.

II. MATERIALS AND METHODS

A. Enrollment of Subjects

This was Open label prospective parallel group active comparator interventional study. At the four good conditioned PHC centers of town Dhadgaon, District Nandurbar, Maharashtra State, India, between the period of 2009 to 2012 the enrollment of all subjects has been conducted after getting Institutional ethics committee permission.105 test and 100 control SAM children without infection, of either sex and 1 to 5 years of age were randomly and step by step enrolled. The published random number table was used as a method of generating randomization. PHC medical officers did categorization to SAM and diagnosis to -3Z score., the treatment of study nutritional intervention therapy, providing 2.5 to 3gm Protein and 90-100 kcal /kg body Weight/day was given only to the test group, for the three months at the same time they have also received khichadi in anganwadi centers and home food in their own houses. However the control group who was not given

study nutritional intervention therapy, but has received khichadi and home food only. Before and after the therapeutic nutritional biscuits therapy their Anthropometric, and Biochemical parameters were measured. To the parents of subjects, the Patient Information Sheet was provided. At the time of enrollment for all test and control subjects Consent forms and Case Record forms were filled up. Along with signature/thumb of parents Biscuit distribution record sheet as well as follow up cards were also recorded. By Albendazole (Ankur drugs and pharmaceuticals,Solan,HP.,India) deworming of all test and control Subjects was done before the start of the project.

B. Anthropometric measurements

By using welcome classification system all enrolled 105 test and 100 control subjects were classified into kwashiorkor, marasmus, and marasmic kwashiorkor [6]. As per WHO guidelines the weight, height, BMI (by standard formula) of all enrolled study and control subjects were measured before and after the nutritional intervention treatment as follows : By stadiometer, standing height of subjects above two years was measured, while by infantometer, length of subjects below two years was measured. Similarly weight was measured by infant and regular weighing scales. On WHO growth standard charts Height/length and weight was plotted against the subject's age. The 50th percentile of their age and gender was taken as normal expected height and weight. To determine z-score for weight and height the WHO z-score cards for both the genders were also used.

C. Nutritional Intervention

By providing FDA (India) approved therapeutic Nutritional biscuits, who's chemical analysis was done from Raptakos Brett Test Laboratories, Thane, Maharashtra, India, the children were rehabilitated. The NGO, Shri Satya Sai Institute of Agriculture and Biotechnology, Shri.Satya Sai Seva Kshetra, Aaksa, Malad (west), Mumbai, Maharashtra State, India. 40008**.** Located on 70 acres area land has sponsored therapeutic Nutritional biscuits in charity to the study subjects**.** The therapeutic Nutritional biscuits were having following composition - Sprouted and shed dried green gram (mung), sprouted and shed dried Ragi (Indian name: Nachani) Vanaspati Ghee (Vegetable fat), Sugar, cardamom.

D. Blood Sampling method

From each study and control subjects, morning fasting blood samples, after the 12-14 hours of overnight fast, in the fluoride vaccutainers were collected. As per WHO guide lines and previous studies, immediately after fasting blood withdrawal, the oral glucose load of: 10ml/kg body weight giving 1.75gm/kg [7] was given to all subjects and again after 2hrs PP blood was collected in fluoride vaccutainer. Such kind of blood collection was done at two different periods - first; at the time of admission and second; after three month's nutritional intervention treatment.All the blood samples were centrifuged within 1 hr to obtain plasma, preferably analysis was done immediately. Blood glucose was estimated by glucose auto kit of Span diagnostics (Surat, Gujrath, India) by GOD/POD method with end point kinetic assay, by auto analyzer.

E. Statistical analysis

Data was subjected to analysis by using SPSS S/W version -16 for variance, and differences were identified by Mean, S.D., S.E., 95 % C.I. and Pearson correlation: r values were also determined. P-value was obtained, $P < 0.05$ considered Significant difference, $p < 0.000$ considered Highly Significant difference ,while a) Correlation is considered to be significant at the 0.05 level (2-tailed)., b) Correlation considered very significant at the 0.01 level (2-tailed). C) Correlation considered highly significant at the 0.000 level (2-tailed). And Regression: was done by using SPSS S/W version -16.

III. RESULTS AND ANALYSIS

Table 1. Descriptive statistics of baseline characteristics for Anthropometric measurements at the time of admission.

Equal variances assumed

Baseline chararcteristics	Groups	N	Mean	Std. Deviation	Std. Error Mean
Weight at the time of Admission in kg	Study group	105	8.66	1.58	0.15
	Control group	100	8.83	1.62	0.16
Weight after treatment in kg	Study group	105	14.08	2.61	0.25
	Control group	100	11.28	1.81	0.18
Hight at the time of Admission in cm	Study group	105	84.95	8.63	0.84
	Control group	100	84.91	8.43	0.84
Hight after treatment in cm	Study group	105	91.47	8.29	0.80
	Control group	100	86.12	7.19	0.71
BMI at the time of Admission in %	Study group	105	10.57	0.39	0.28
	Control group	100	10.76	0.28	0.20
BMI After treatment in %	Study group	105	15.53	0.50	0.04
	Control group	100	13.01	0.70	0.07

Table 2. Independent sample test for Anthropometric measurements at the time of admission and after Nutritional Intervention treatment for study and control group

Unpaired t-test for Equality of Means						95% CI of the Difference	
Baseline chararcteristics	**Mean Difference**	**Std. Error Difference**	**t test value**	**df**	**p value**	**Lower**	**Upper**
Weight (kg) (At Admission)	-0.172	0.223	-0.772	203	0.441 (NS)	-0.612	0.268
Weight (Kg) (after treatment)	2.799	0.316	8.857	202	**0.0001**	2.176	3.423
Hight (cm) (At Admission)	0.037	1.192	0.031	203	0.975 (NS)	-2.314	2.387
Hight (cm) (After treatment)	-1.344	1.346	-0.999	203	**0.0001** (S)	-3.997	1.310
BMI (At Admission)	-0.195	0.340	-0.573	203	0.624 (NS)	-1.658	1.268
BMI (After treatment)	2.520	0.085	29.504	203	**0.0001** (S)	2.352	2.688

a) *P < 0.05 considered Significant difference,* ***b)*** *p < 0.000 considered Highly Significant difference*
c) *NS- Not Significant*

Table 3. Descriptive statistics of baseline characteristics Before treatment in study and control group

Baseline chararcteristics	Groups	N	Mean	Std. Deviation	Std. Error Mean
Sugar_FF mg/dL	Study group	105	69.60	3.49	0.34
	Control group	100	69.70	3.40	0.34
Sugar _PP mg/dL	Study group	105	101.44	10.44	1.02
	Control group	100	100.90	10.64	1.06

Equal variances assumed

Table 4. Independent sample test for Before treatment in study and control group

Unpaired t-test for Equality of Means						95% C I of the Difference	
Baseline chararcteristics	**Mean Difference**	**Std. Error Difference**	**t test value**	**df**	**p value**	**Lower**	**Upper**
Sugar_FF mg/dL	-0.109	0.482	-0.226	203	0.821 (NS)	-1.059	0.840
Sugar _PP mg/dL	0.558	1.472	0.379	203	0.705 (NS)	-2.345	3.461

P < 0.05 considered Significant difference, p < 0.000 considered Highly Significant difference NS- Not Significant

Table 5. Descriptive statistics of baseline characteristics After treatment in study and control group

Baseline chararcteristics	Groups	N	Mean	Std. Deviation	Std. Error Mean
Sugar_FF mg/dL	Study group	105	74.401	3.007	0.293
	Control group	100	69.601	3.514	0.351
Sugar _PP mg/dL	Study group	105	84.303	8.437	0.823
	Control group	100	100.301	12.269	1.227

Equal variances assumed

Table 6. Independent sample test for After treatment in study and control group

Unpaired t-test for Equality of Means						95% C I of the Difference	
Baseline chararcteristics	**Mean Difference**	**Std. Error Difference**	**t test value**	**df**	**p value**	**Lower**	**Upper**
Sugar_FF mg/dL	4.772	0.456	10.464	203	**0.0001**	3.873	5.671
Sugar _PP mg/dL	-15.957	1.465	-10.894	203	**0.0001**	-18.845	-13.069

P < 0.05 considered Significant difference, p < 0.000 considered Highly Significant difference NS- Not Significant

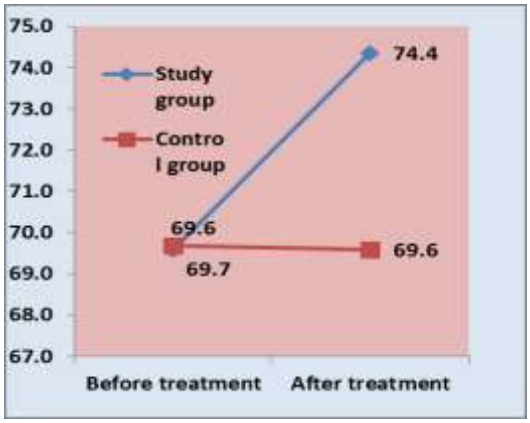

Figure 1. Sugar_FF mg/dL

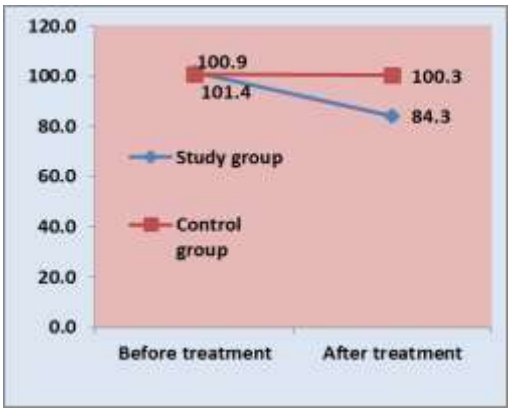

Figure 2. Sugar _PP mg/dL

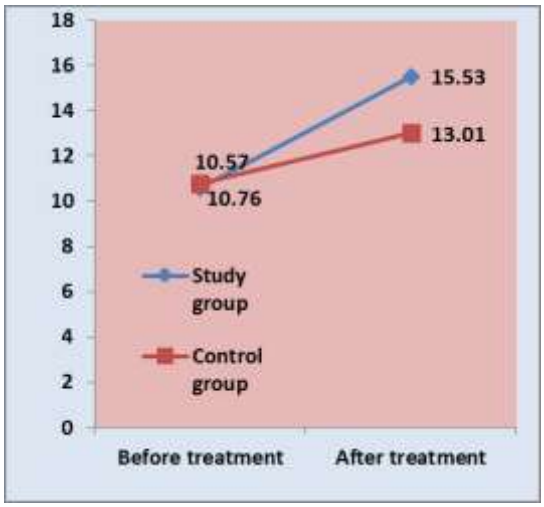

Figure 3. BMI

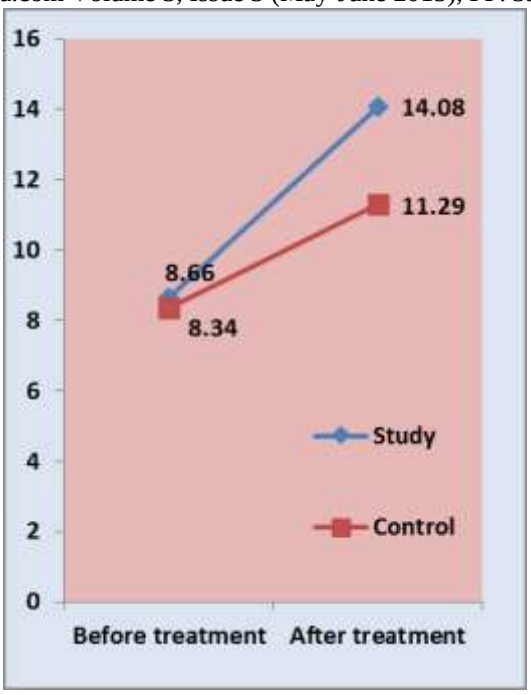

Figure 5. Weight in Kg

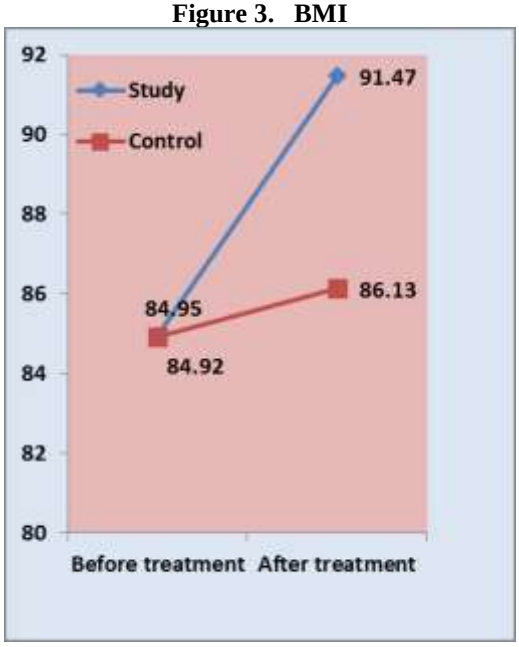

Figure 4. Height in Cm

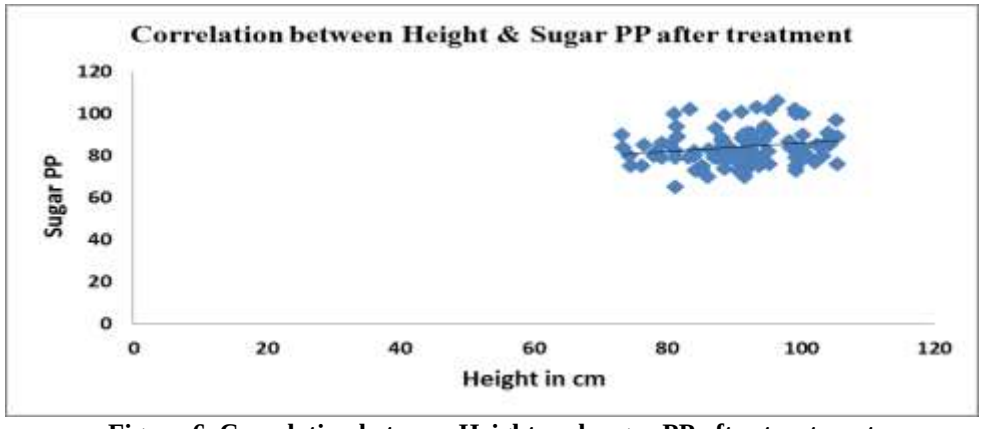

Figure-6. Correlation between Height and sugar PP after treatment

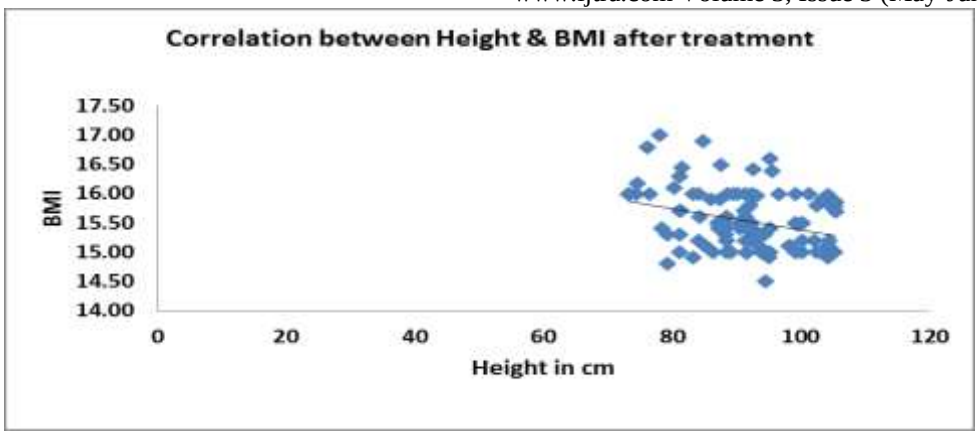

Figure-7 Correlation between Height and BMI after treatment

Table 7. Correlations of Height with Sugar PP and BMI after treatment in study group

Height	**Sugar-PP**	**BMI**
Sample size (N)	105	105
Pearson Correlation r	0.209*	-0.305**
p value	0.033 (Significant)	0.002(Very Significant)
Interpretation	Poor positive correlation	Poor negative correlation

Figure- 8. Correlations of Weight with BMI after treatment

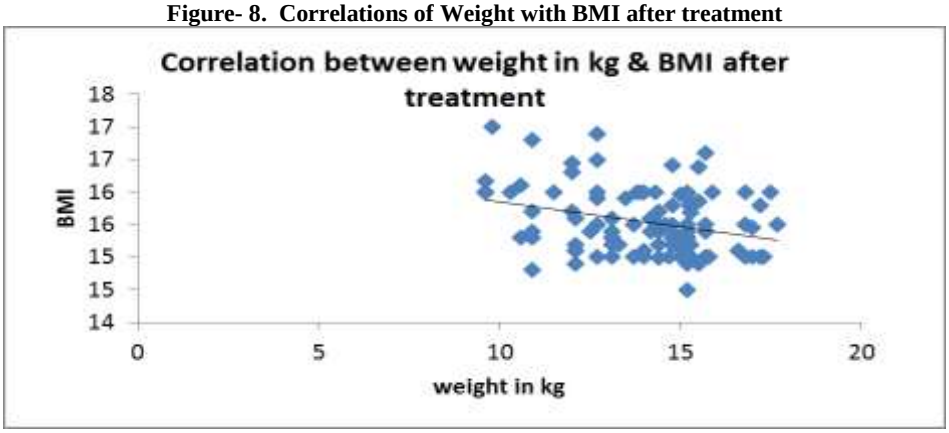

Figure-9. Correlations of Weight with Sugar PP after treatment.

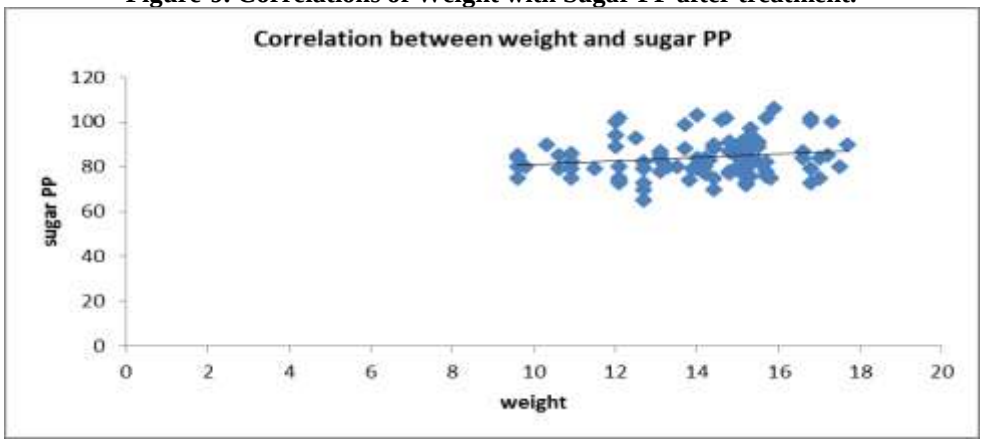

Table 8. Correlations of Weight with BMI and Sugar PP after treatment in study group

Weight	**BMI**	**Sugar PP**
Sample size (N)	105	105
Pearson Correlation r	-0.306*	0.193*
p value	0.001 (Very Significant)	0.048 (Significant)
Interpretation	Poor negative correlation	Weak positive correlation

IV. DISCUSSION

BMI: (Body mass index)

Before the nutritional intervention treatment P value was noted as p = 0.624 which is insignificant suggestive of similar baseline BMI characteristics at the time of enrollment. After the nutritional intervention treatment p value was noted as p = 0.0001 which is highly significant. (Fig: 3, Table: 2)

-The study showed a significant fall, in the body mass index (BMI) of the malnourished children before nutritional intervention treatment. This observation is similar to that of earlier workers [8] who have attributed the fall in the BMI to low food intake, protein deficiency and accompanying of BMI with low levels of serum albumin. These results are also expected to be due to muscle wasting, loss of subcutaneous fat and growth failure in malnourished children.

These results were parallel to those obtained by previous workers [9,10,11,12] who have found significant reduction in body weight. Body weights and heights of children reflect their nutritional and growth status.

BMI, however, does not clearly bring out the entire extent of chronic under- nutrition; for instance those who are stunted and have low body weight may have normal BMI. Both in adults and in children increase in energy intake will result in improvement in BMI. It has also been reported that body fat content for a given BMI is different not only between male and female but also between different countries[5] The currently used norms, were evolved on the basis of data from the developed countries.

Weight and Height: Before the nutritional intervention treatment p value was noted as p = 0.441 which is insignificant suggestive of similar baseline weight characteristics at the time of enrollment. However, after the nutritional intervention treatment p value was noted as p = 0.0001 which is highly significant (Fig: 5 Table: 2)

Before the nutritional intervention treatment p value was noted as p = 0.975 which is insignificant suggestive of similar baseline height characteristics at the time of enrollment. After the nutritional intervention treatment P value was noted as p = 0.0001 which is highly significant. (Fig: 4 Table: 2)

Carbohydrate:

F-BSL: Before: the nutritional intervention treatment p value was noted as p = 0.821 which is insignificant, suggestive of similar F-blood sugar baseline characteristics at the time of enrollment. (Fig.1, Table.4 and 6)

After: the nutritional intervention treatment p value was noted as p= 0.0001 which is highly significant. F-BSL level in study group, after the treatment was found to be increased to the normal level. However control group after the treatment period have not shown any significant rise, the F-BSL values were remained below normal.

(Fig.1, Table.4 and 6)

PP-BSL - Before the nutritional intervention treatment p value was noted as p = 0.705 which is insignificant, suggestive of similar baseline characteristics of study and control groups at the time of enrollment. (Fig.2, Table 4 and 6) Similarly, After: the nutritional intervention treatment p value was noted as p = 0.0001, which is highly significant. (Fig. 2, Table.4 and 6)

V. SIGNIFICANT CORRELATIONS OF WEIGHT AND HEIGHT WITH SUGAR- PP AND BMI:

In present study following significant correlation were found for weight and Height with sugar-PP and BMI in study group after nutritional intervention treatment:

1. Significant (p=0.033) correlation of Height with sugar-PP was noted. Its correlation coefficient r = 0.209 indicating poor positive correlation.(Fig.6, Table-7)
2. There was a weak positive and significant correlation was found between weight and sugar-PP after treatment in study group (r = 0.193, p =0.048) (Fig. 9, Table- 8)
3. Where as Correlations of Height with BMI after treatment in study group has shown very significant (p=0.002) and poor negative (r= -0.305) correlations. (Fig.7,Table.7)
4. There was a poor negative and very significant correlation was found between weight and BMI

(Pearson Correlation r = -0.306, p= 0.001) (Fig.8, Table 8.)

All the above stated correlations are significant, and hence expected to help future studies to design their study protocols and also will also help to develop community policies for malnourished children.

The study results have demonstrated some degree of glucose intolerance most probably caused by inadequate response of pancreatic β cells to the increased blood glucose. This was in harmony with the reports of investigators who found sluggish response of serum insulin after glucose or arginine stimulation.[13] Some investigators had been attributed such glucose intolerance to poor insulin release as a result of pancreatic endocrinal insufficiency secondary to deficiency of β cells cytotrophic. [14] The measurements were repeated after normalization of the body weight following 90 days of nutritional rehabilitation. The fasting blood glucose value in the test group have shown improvement significantly in the post-treatment period and returned to the normal value and reached the basal level. Thus the abnormal blood glucose response to oral glucose load was normalized, while the control group has found unable to reach at the normal levels, after treatment period control group has shown still some hypoglycemia, and it was also found that they were unable to reach to the basal levels after two hours of oral glucose load. These findings are in accordance with other workers.[14,4,15] The impaired glucose tolerance observed in kwashiorkor cases before the nutritional treatment, was explained by poor functional response of pancreatic β cells due to diminished rate of protein synthesis secondary to amino acids deficiency.[16]

In addition to the poor initial response of β cells, kwashiorkor cases showed a sustained low C- peptide secretion after the oral glucose load, suggesting presence of insulin antagonism on top of sluggish inadequate response of β-cells. In fact, this finding is compatible with the presence of insulin antagonists such as growth hormone and cortisol, being generally elevated among cases of PEM.[14]

We assume that there is a considerable association between glucose intolerance, and poor β cell response to glucose load. In support of this view, other workers have shown among experimental animals, which were maintained on low protein high carbohydrate contents, plasma insulin levels were low. [4]

Also, it is suggested that the islet cell changes in PEM may be related to free radical damages secondary to depletion of glutathione and other antioxidants, as well as relative deficiency

of zinc. However, this suggestion needs reassessment of β cell function after dietary rehabilitation and supplementation of the possibly claimed deficient vitamins and trace elements. The next few years will undoubtedly present a clarification of free radical nutrient and tissue interactions, and may help to put the concept on a sounder footing.[14]

In kwashiorkor hypoglycemia is a common phenomenon [17-18,19] Theoretically, due to increased glucose clearance or impaired hepatic endogenous glucose production (EGP) hypoglycemia could be caused. Hepatic steatosis and oxidative stress linked to Kwashiorkor. Infections, decreased antioxidant status, or toxins trigger the oxidative stress. [20,21,22] It has been shown that, oxidative stress is stimulated by surplus tissue fatty acids, resulting into cell death and mitochondrial damage irrespective of its cause.[22,23] Impairment of ATP production and [24] mitochondrial function is due to steatosis.[25] Mitochondrial dysfunction and impaired gluconeogenesis are closely linked ,which is indicated by decreased hepatic glucose production rates [26] Finally, in patients with severe malnutrition signs of hepatic failure have been described [27] which is also associated with decreased EGP.

Hypoglycemia could relate to increased glucose clearance, decreased hepatic glucose production, glucose absorption. Because of insufficient glucose production, young children generally develop hypoglycemia after 24 hr as they have a limited capacity for fasting [28-29] From children with kwashiorkor, in biopsies , Glycogen content has been found to be high [30] low [31] or normal [17]. In this study, due to overnight fast, the depleted glycogen stores and differences in EGP are related to alterations in gluconeogenesis than glycogenolysis. Deficiencies in amino acids used for gluconeogenesis are suggested in studies on prolonged fasting in young children. [28] A severe derangement in the child's metabolic system is indicated by hypoalbuminemia, reflecting a disturbance in protein metabolism [32,33] potentially cytokine-mediated capillary leak or cytokine-mediated inhibition of albumin synthesis [34] and redistribution of albumin to the extra vascular space.[20,21] In children with kwashiorkor, as compared to marasmus and healthy children there could be decreased EGP but it could be related to the oxidative stress and degree of hypoalbuminemia. In children with different forms of severe malnutrition, to distinguish the severity of metabolic derangements, clinical criteria alone is insufficient. Hypoglycemia is a common complication in malnourished children and can lead to brain damage and ultimately death can occur due to hypoglycemia, since the main fuel for the brain is glucose. During the initial stabilization phase of the treatment of severe malnutrition preventing hypoglycemia is vital [32-33]

In children with severe malnutrition there are many underlying causes for the development of hypoglycemia. First, because of muscle wasting, in a malnourished child, the quantity of stored glucose in the body is reduced. Second, mechanisms for re-establishing glucose equilibrium are impaired and thus protein and fat cannot be converted into glucose. Third, glucose is used for the immune response to infections, which is common in malnourished children. -Fourth absorption of glucose is impaired and fifth- the child not being fed for several hours due to long journey to a hospital and the process of admission to the hospital.[32,18,19] In children with severe malnutrition the signs of hypoglycaemia include, lethargy, loss of consciousness, limpness, and a body temperature of less than 36.5^0C. reatment should be started immediately if hypoglycaemia is suspected, Since hypoglycaemia can lead to death during the first two days of treatment.[35] If the hypoglycaemic child should be given 50 ml of 10% glucose or 10% sucrose. Then after every 30 mins, the F-75 diet for two hours should be provided to the child [35] This effect in edematous kwashiorkor might be due to defective glycolytic pathway rather than insulin distribution in larger fluid space as suggested by Alleyne et.al., might be due to increased half life of insulin or to a defect in the homeostatic glycogenolytic pathway in these children should have been able to effect a prompt correction of hypoglycaemia. Fatty degeneration in the liver of the child with kwashiorkor have been documented but glycogen storage has shown to be normal [33] The blood glucose response to glycogen [33,34] and epinephrine stimulation has been shown to be blunted in kwashiorkor.[34] The lack of prompt and spontaneous recovery from hypoglycemic state observed in kwashiorkor, is therefore in agreement with the findings in previous studies that a defect in glycolytic pathway usually exists in kwashiorkor.

This defect may be due to hypothalamoendocrine insensitivity to the stress of hypoglycemia with consequent inadequacy of hormonal backup for glycogenolysis or to in adequate enzyme activity at the hepatocellular level. An insensitive hypothalamo-endocrine pathway failing to sense and respond appropriately to hypoglycemia would be an added factor to the sluggish self correction of hypoglycemia in children with kwashiorkor. [33,34]

Post-glucose insulin was significantly less, showing a delayed response, in kwashiorkor only, resembling the diabetic type of insulin response. These changes were reversed after treatment. During starvation, insulin levels decrease and glucagon levels increase, resulting in the conversion of glycogen to glucose and the stimulation of gluconeogenesis, which involves the synthesis of glucose from lipid and protein breakdown products. Subsequent refeeding after starvation causes an increase in insulin release and an increased shift of phosphate, glucose, potassium, magnesium, and water to intracellular compartments often resulting in edema (ie, pulmonary) after fluid administration, as seen in this patient. [16, 36-39] Hyperglycemia stimulates secretion of insulin which brings down the blood glucose to normal level. Hyperglycemia stimulates insulin secretion by directly acting on the islet cells, and insulin increases the rate of oxidation of glucose. Similarly, hypoglycemia depresses insulin secretion and the blood glucose level is elevated to normal levels. The defect in NIDDM is attributed to decreased number of insulin receptors. These are cells responding to insulin and to which insulin binds specifically, located on the plasma membrane. [40] When there is a lack of insulin, the hexokinase reaction by which glucose is converted to glucose-6-phosphate and which is the initial reaction for oxidation of glucose is retarded leading to utilization of glucose by the body. As a result, the concentration of glucose increases in blood, contributing to hyperglycemia. As glycolysis and tricarboxylic acid cycle are subsequently retarded, the body derives its energy requirements by increasing the oxidation of fats. The diabetics have to monitor their blood glucose levels to determine the complications.[40] Eleven of the above mentioned 54 marasmic children were studied by previous worker, the stress of insulin-induced hypoglycemia.[40] Eight out of 11 cases showed no response by raised hGH levels

to insulin hypoglycemia. This non responsiveness was partially corrected by replacement of high protein diet for 4–6 weeks. The role of the impaired hypothalamic-pituitary response to induced hypoglycemia in the metabolic adjustments in nutritional deprivation requires further elucidation. Golden and Ramadath[41] who found lower plasma zinc concentration in association with nutritional edema. As well as having catalytic, structural and regulatory roles in enzymes that participate in the metabolism of carbohydrate, protein, lipids and nucleic acids, zinc has also been found to have regulatory functions in the binding of insulin to the cell membrane. In humans, Zinc is also associated with pre secretory insulin hormones in the pancreatic β-cells, and its repletion in patients with total parenteral nutrition is associated with increased insulin secretion.[40,42,43] Hypoglycaemia is uncommon in malnourished children with normal basal cortisol.[16,24,44] An impairment in insulin availability related to the disturbed glucose clearance in malnutrition.[46] Plasma albumin concentrations correlated with glucose clearance rates.[45] In both kwashiorkor and marasmus insulin responses were strongly impaired. In the malnourished groups there was no peripheral or hepatic insulin resistance indication. [46] Spoelstra MN have shown that in both children with marasmus as well as kwashiorkor , glucose clearance rates are affected, and it is correlated with plasma albumin concentrations. An Impairment in insulin availability is related to disturbed glucose clearance in malnutrition. [24] Normal levels of blood sugar have been reported in the adult subjects suffering from nutritional edema and also to both insulin and adrenaline they show normal responses.[32] Gopalan et.al. have reported contrary findings of it.[26] They have noticed low levels of fasting blood sugar and impaired oral glucose tolerance in the subjects with nutritional edema. Low levels of fasting blood sugar reported by waterlow et.al. in children suffering from kwashiorkor.[30]

GH levels were poorly suppressed by the meal but some infants had further elevations, possibly in response to protein. After partial rehabilitation, fasting Blood sugar(BS) and FFA and BS elevations after the meal were normal. A slight improvement in insulin release was apparent. Fasting GH levels and responses to the meal were normal. Fasting, minimally treated children with marasmic kwashiorkor (MK) had normal or low BS, normal or low IRI (Immunoreactive insulin), normal FFA, and probably normal GH levels.[46,47] D.R.Hadden have found, there was considerable delay in BS elevation, moderately delayed glucose disappearance, and very poor or un-measurable insulin release after the test meal; FFA and GH were poorly suppressed. After partial rehabilitation they have noted that, fasting BS was normal, IRI was still low, BS elevations and disappearance improved. IRI responses modestly improved, and GH responses were normal.[46,47] These results suggest that defective energy-linked mitochondrial function impairs gluconeogenesis and that hypoglycemia occurs when oxidative phosphorylation becomes uncoupled and hepatic glycogen stores are subsequently depleted. [46,47]

VI. CONCLUSION

The abnormal blood glucose response to oral glucose load was normalized after nutritional intervention treatment, thus considering the above discussed improvements and supportive results, investigators recommend that the study nutritional intervention has potentials for normal body functioning related to carbohydrates such as ; glucose clearance, digestion, absorption, hepatic glucose production as well as it has potentials to meet to the normal metabolic end points of the glucose and thus shows carbohydrate compliance of the nutritional intervention with the body. Investigators conclude that it is the most effective food supplement for the speedy recovery of glucose intolerance.

Similarly investigators also conclude that body mass index can be used as an anthropometric index for assessment of nutritional status as it reflects the effect of both acute and chronic energy deficiency as well as excess. Investigators further recommend that the currently used cut off points of BMI can be used as an indicator of various metabolic functions- especially carbohydrates, work capacity and health indices and which should need to be more studied.

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