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CORRELATIONS OF WEIGHT FOR HEIGHT % WITH SERUM TRIGLYCERIDE AND TOTAL CHOLESTEROL AFTER NUTRITIONAL REHABILITATION IN MALNOURISHED TRIBAL CHILDREN

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ABSTRACT- Globally 165 million children under-five years of age are stunted. Hence development of local therapeutic nutritional intervention is recommended by WHO. Present study was designed to find the efficacy of the nutritional intervention for the recovery of impaired lipid metabolism and correlation of weight for height% with cholesterol, triglyceride in malnourished children. 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 Anthropometric, and Biochemical parameters were measured before and after the nutritional therapy. Before the nutritional intervention treatment P values for Serum Total cholesterol, Triglyceride, Weight for height %, were insignificant suggestive of similar baseline characteristics at enrollment. After nutritional intervention treatment P values for Serum Total cholesterol, Triglyceride, Weight for height % were highly significant. The r value of Pearson correlation coefficient for triglycerides in the study group and its ANOVA model was very significant, showing poor positive correlation with weight for height % while for total cholesterol it was found to be insignificant. Depending on results we conclude that it is the most effective food supplement for the speedy recovery of the impaired lipid metabolism in SAM children and the use of weight for height % as a anthropometric marker for the pre-indication of fatty liver in malnourished children.

Keywords- SAM, Triglyceride, Cholesterol, Weight for height %, Correlation, Nutritional intervention

I. INTRODUCTION

The world's no. one health risk is hunger it kills more people every year than AIDS, malaria, tuberculosis together [1] .It was found that 740 million people in the world do not have enough to eat [2] In developing countries one out of six children roughly 100 million are found to be underweight. [3]. These statistics suggestive of the severity of the malnutrition problem worldwide and also denotes the thrust area for the Research. Lewis et al 1964, has shown that altered lipid metabolism and fatty liver are characteristic features in children suffering from kwashiorkor. Lipids are closely related with liver abnormalities, such as fatty liver. Serum free fatty acids (FFA) are elevated commonly in kwashiorkor, and this has usually been one of the explanations for the fatty liver, which is often found in this condition [4],[6] ; In this context , hypothesis of this study was to correlate weight for height % with serum triglyceride and cholesterol , to trace out whether this simple anthropometric parameter has potentials to reveal, impaired lipid metabolism and the fatty liver status of the malnourished children or not. And also to verify that whether this parameter could recommend as a initial alarming pre-indicator or marker for the primary diagnosis and predictions about the impaired status of serum lipids and liver. Development of edema is noted in the late stages of malnutrition, it could be probably very useful, to predict the impaired lipid status prior to the development of edema, and fatty liver by using simple and commonly available tool. It was necessary that pre-indicator having predictive potentials to detect impaired lipid status, should be as simple as it could be used even at the remote forest and hilly areas where no doctor, no laboratory facility is available, almost all the responsibilities have shouldered on the nonclinical staff in developing countries. Therefore in present study we have attempted to highlight probable basic anthropometric marker such as weight for height %, which was not much studied by this angle till date. The accounting of such preliminary, predictive, alarming pre-indicator could help to start necessary and timely action such as Childs mobilization to city hospital for better treatments. More devastating conditions usually developed after the development of oedema and impaired fatty liver, which could be avoided by using such simple basic predictive indicator and thus the arrest at borderline period of its development could be possible. Which can further help to reduce the mortality rate in PEM, due to such timely arrest of borderline period.

II. MATERIALS AND METHODS

A. Enrollment of Subjects

After getting Institutional ethics committee permission, the enrollment of all subjects has been conducted at the four good conditioned PHC centers of town Dhadgaon, District Nandurbar, Maharashtra State, India, between the period of 2009 to 2012. This was Open label prospective parallel group active comparator interventional study. 105 test and 100 control SAM children without infection, of 1 to 5 years of age and either sex were randomly enrolled step by step. The published random number table was used as a method of generating randomization. Their diagnosis to -3Z score and categorization to SAM was done by PHC medical officers. Only Test group was given treatment of study nutritional intervention therapy, providing 2.5 to 3gm Protein and 90-100 kcal /kg body Weight/day, 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. Their Anthropometric, and Biochemical parameters were measured before and after the therapeutic nutritional biscuits therapy. Patient Information Sheet was provided to the parents of subjects. Consent forms and Case Record forms were filled up at the time of enrollment for all test and control subjects. Biscuit distribution record sheet with signature/thumb of parents as well as follow up cards were also recorded. Deworming of all test and control Subjects was done by Albendazole (Ankur drugs and pharmaceuticals, Solan,HP.,India) before the start of the project. Similarly before enrollment, screening of malaria, and sickle cell anemia by rapid kits (company: Bio Lab Diagnostics India private Ltd. ISO 9001-2008) has been done by PHC center staff as one of their mandatory routine duties; which duly helped to fulfill inclusion criteria of the present study.

B. Anthropometric measurements

All enrolled 105 test and 100 control subjects were classified into kwashiorkor, marasmus, and marasmic kwashiorkor by using welcome classification system [5] The weight, height, weight for height%, of each subjects were measured as per WHO guidelines before and after the nutritional intervention treatment. Standing height of subjects above two years was taken by stadiometer while length of subjects below two years was taken by infantometer. Similarly Weight was measured by infant and regular weighing scales. Height/length and weight was plotted on WHO growth standard charts for height and weight against the subject's age. 50th centile of their age and gender was taken as normal expected height, and weight. WHO z-score cards also used for both genders for weight and height to determine z-score.

C. Intervention

The children were rehabilitated with FDA (India) approved therapeutic Nutritional biscuits, having basic ingredients, sprouted green gram (moong), sprouted Ragi(Indian name: Nachani) Vanaspati Ghee (Vegetable fat), Sugar,cardamom, Its Chemical analysis was done from Raptakos Brett Test Laboratories, Thane, Maharashtra, India. 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 to the study subjects**.**

D. Blood Sampling method

From each test and control subjects morning blood samples in to plain tubes were collected at two different periods, first at the time of enrollment and second after three month's treatment of nutritional intervention. Samples were centrifuged within 1 hr to obtain serum, and transported by maintaining cold chain of 2 to 8^{0}C immediately within 24 hr from study site and stored at -80^{0}C(CRYO scientific)until analyzed for Triglycerides, and total Cholesterol preferably most of the time analysis was done immediately after arrival of samples in the laboratory from field.

E. Laboratory methods

Serum total cholesterol, and triglycerides were estimated before and after giving nutritional intervention therapy on fully automated analyzer- olympus-AU400. Serum total cholesterol was estimated by total cholesterol auto kit of Span Diagnostics, Surat, Gujrath, India by and CHOD-PAP method with end point assay, while serum triglyceride was determined by triglyceride auto kit of Biolab diagnostics, Boisar, Maharashtra, India by GPO-PAP method with end point assay.

F. 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

A. Biochemical

Table 1. **Descriptive statistics of baseline characteristics before treatment in study and control group**

Baseline chararcteristics	**Groups**	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Serum total Cholesterol mg/dL	Study group	105	104.87	8.05	0.79
	Control group	100	105.98	8.24	0.82
Serum Triglyceride mg/dL	Study group	105	21.04	1.96	0.19
	Control group	100	21.09	1.98	0.20

Equal variances assumed

Table 2. Independent sample test before treatment in 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**
Serum total Cholesterol mg /dL	-1.113	1.138	-0.978	203	0.329 (NS)	-3.357	1.130
Serum Triglyceride mg/dL	-0.052	0.275	-0.189	203	0.850 (NS)	-0.593	0.490

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

Table3. **Descriptive statistics of baseline characteristics after treatment in study and control group**

Baseline chararcteristics	**Groups**	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Serum total Cholesterol mg/dL	Study group	105	154.701	10.752	1.049
	Control group	100	116. 936	7.534	0.753
Serum Triglyceride mg/dL	Study group	105	52.913	10.185	0.994
	Control group	100	21.030	1.992	0.199

Equal variances assumed

Table 4. Independent sample test after treatment in 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**
Serum total Cholesterol mg/dL	37.765	1.303	28.992	203	**0.0001**	35.197	40.333
Serum Triglyceride mg/dL	31.883	1.037	30.746	203	**0.0001**	29.839	33.928

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

Table 5. Descriptive statistics of baseline characteristics for Anthropometric measurements at the time of admission

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	91.478	8.29	0.80
	Control group	100	86.126	7.19	0.71
Weight for height % at the time of Admission in %	Study group	105	64.866	2.02	0.19
	Control group	100	62.7827	3.86	0.38
Weight for height % After treatment in %	Study group	105	94.152	2.28	0.22
	Control group	100	76.821	4.4811	0.44

Equal variances assumed

Table 6. 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
Weight for height % (At Admission)	2.082	0.42751	4.872	203	0.06(NS)	1.239	2.925
Weight for height % Control (After treatment)	17.3317	0.49315	35.145	203	**0.0001** (S)	16.359	18.304

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

IV. CORRELATIONS

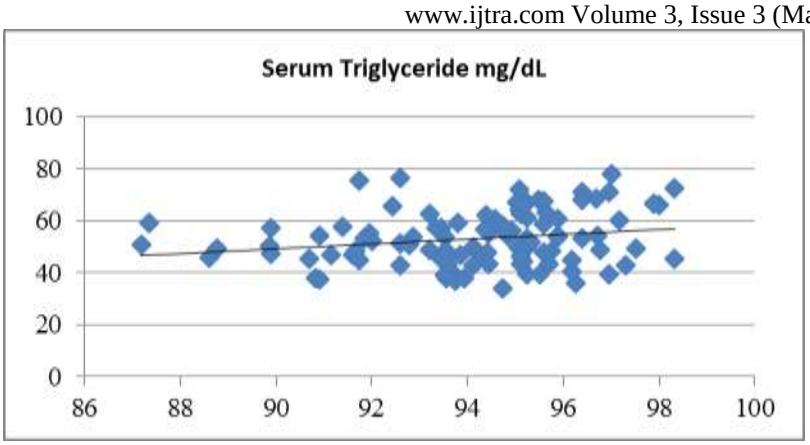

Figure.1.Correlation of Serum Triglyceride (Y-axis) with weight for height % (X-axis) after treatment for study group
r= 0.204, P value(correlation)=0.037 (very significant), Poor positive correlation

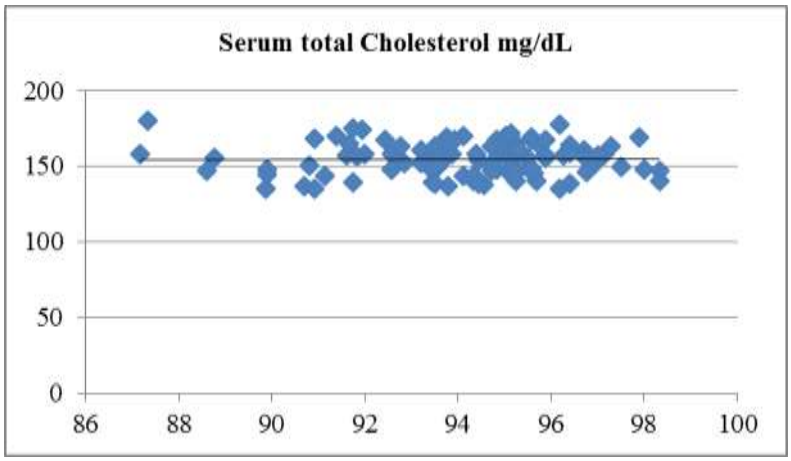

Figure.2. **Correlation of Serum total Cholesterol (Y-axis) with weight for height % (X-axis) after treatment for study group.**
r=0.009, P value (correlation) =0.931(Not Significant), No correlation,

Table 7. Correlations of Weight for height % with Serum total Cholesterol and Serum Triglycerides after treatment in study group

Weight for Height (%)	**Serum total Cholesterol**	**Serum Triglyceride**
Sample size (N)	105	105
Pearson Correlation r	0.009	0.204*
p value	0.931 (NS)	0.037 (Very Significant)
Interpretation	Very weak Positive correlation (almost no corrln)	Poor positive correlation

a. Correlation is significant at the 0.05 level (2-tailed).,b.Correlation is very significant at the 0.01 level (2-tailed). C. Correlation is Highly significant at the 0.000 level (2-tailed).

V. REGRESSION

Table-8 Model Summary[b]

Mode l	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	0.204[a]	0.042	0.032	10.0185

a. Predictors: (Constant),weight for height% (After)

b. Dependent Variable: Triglyceride

Table 9. The ANOVA table tests the acceptability of the model from a statistical perspective: ANOVA[b] :

Model 1	Sum of Squares	df	Mean Square	F	Sig.
Regression	449.562	1	449.562	4.479	.037 [a]
Residual	10338.199	103	100.371		
Total	10787.761	104			

a. Predictors: (Constant), weight for height % (After) b.Dependent variable Triglyceride.

Table-10. Coefficients [a]

Model 1	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-32.942	40.579		-0.812	0.419	-113.421	47.537
weight for height% (After)	0.912	0.431	0.204	2.116	0.037	0.057	1.766

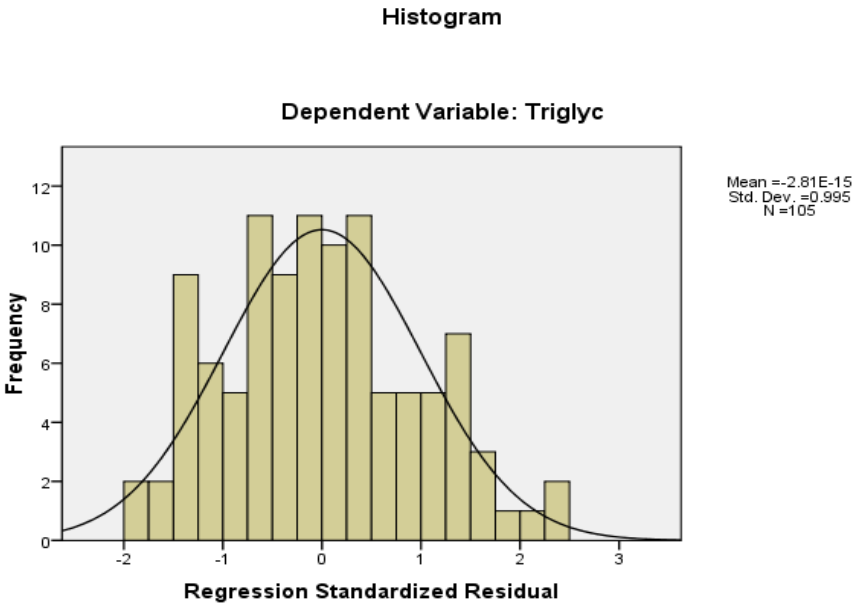

Figure 3: Histogram

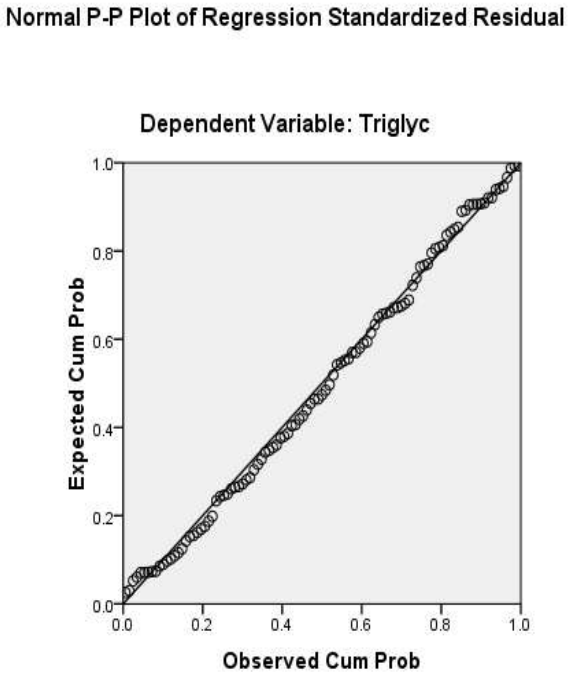

Figure -4: Normal P-P Plot

VI. DISCUSSION

A. Lipids:

Triglycerides: All the enrolled SAM children of both test and control groups have shown reduced serum triglycerides concentrations at the time of admission. (Table-1) While After treatment serum triglyceride concentrations in study group were found to be improved to normal. But control group has not shown such improvements to normal. (Table -3)

Before the nutritional intervention treatment (Table-2) P value was noted as P= 0.850 which is insignificant suggestive of similar baseline triglyceride characteristics at the time of enrollment. **After** the nutritional intervention treatment (Table-4) P value for triglyceride was noted as P = 0.0001 which is highly significant.

Cholesterol : All the enrolled SAM children of both test and control groups have shown reduced serum total cholesterol concentrations at the time of admission.(Table-1) While After treatment serum total cholesterol concentrations in study group were found to be improved to normal. But control group has not shown such improvements to normal.(Table -3) **Before** the nutritional intervention treatment(Table-2) P value was noted as P= 0.329 which is insignificant suggestive of similar baseline characteristics at the time of enrollment. **After** the nutritional intervention treatment (Table-4) P value was noted as P= 0.0001 which is highly significant.

B. Anthropometry

Weight: All the enrolled SAM children of both test and control groups have shown reduced weight at the time of admission. (Table-5) While After treatment weight in study group were found to be improved to normal. But control group has not shown such improvements to normal.(Table -5)

Before the nutritional intervention treatment(Table-6) P value was noted as P= 0.441 which is insignificant suggestive of similar baseline weight characteristics at the time of enrollment. **After** the nutritional intervention treatment (Table-6) P value for weight was noted as P= 0.0001 which is highly significant.

Height: All the enrolled SAM children of both test and control groups have shown reduced height at the time of admission. (Table-5)While After treatment height in study group were found to be improved to normal or close to normal. But control group has not shown such improvements. (Table -5)

Before the nutritional intervention treatment (Table-6) 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 (Table-6) P value for height was noted as **P= 0.0001** which is highly significant.

Weight for Height %: All the enrolled SAM children of both test and control groups have shown reduced weight for height % at the time of admission. (Table-5) While After treatment weight for height% in study group were found to be improved to normal .But control group has not shown such improvements. (Table -5)

Before the nutritional intervention treatment (Table-6) P value for weight for height % was noted as P= 0.06 which is insignificant suggestive of similar baseline height characteristics at the time of enrollment. **After** the nutritional intervention treatment (Table-6) P value for weight for height % was noted as P= 0.0001 which is highly significant.

C. Correlations

In this study we have also focused on the correlation of triglycerides and cholesterol with the weight for height % in study group after the nutritional intervention treatment.

The Pearson correlation coefficient **(Table 7 Fig-2)** for cholesterol in study group after the treatment was noted as r = 0.009 with P value 0.931,which is not significant, showing very weak positive correlation. However the Pearson correlation coefficient **(Table -7,Fig-1)** for triglycerides in the study group after the treatment was noted as r = 0.204 with P value 0.037, which is very significant, showing poor positive correlation. These correlations results were found to be in harmony of the all stated results and scientific interpretations.

D. Regression

Model Summary[b] : (Table-8) :

R: The multiple correlation coefficients, is the linear correlation between the observed and model-predicted values of the dependent variable. R = 0.21 Its value indicates a poor positive relationship however, very significant at $p < 0.037$.

R Square: The coefficient of determination is the squared value of the multiple correlation coefficient. It shows that 4% variation in triglyceride is explained by the model. The model summary table reports the strength of the relationship between the model and the dependent variable.

The ANOVA table tests the acceptability of the model from a statistical perspective: ANOVA [b] Table interpretation: (Table 9):

The significance value of the F statistic is less than 0.05 ($p<0.05$), which means that the variation explained by the model is not due to chance in the present study. While the ANOVA table is a useful test of the model's ability to explain any variation in the dependent variable, it does not directly address the strength of that relationship.

- **Coefficients [a] : (Table 10) :**
  In this table the coefficients of the regression line is shown. In this study, Weight for height (%) showed significant difference at $p < 0.037$
- **Interpretation:** For one unit change in the weight for height% there is 0.91 times in triglyceride level which is statistically significant at $p < 0.037$ and 95% CI (0.06, 1.77) So above table states that the expected triglyceride level is equal to 0.912 x Weight for height% - 32.942. [If an investigator wants to know Triglyceride level of an individual for Weight for height(%) say 95, the predicted triglyceride level would be 0.912x95 – 32.942 = 53.698 ie. 53.7]
- **Histogram: (Fig-3) :** The shape of the histogram should approximately follow the shape of the normal curve. This histogram is acceptably close to the normal curve.
- **Normal P-P Plot of Regression Standardized Residuals : (Fig-4) :**
  The shape of the histogram should approximately follow the shape of the normal curve. This histogram is acceptably close to the normal curve. The P-P plotted residuals should follow the 45-degree line. Neither the histogram nor the P-P assumption is violated.

- **Scientific Interpretations: Triglyceride:** The triglyceride results of the present study are in agreement with the previous studies [4, 6, 7, 5, 8] and are attributed to the facts that, the concentration of serum triglyceride may be influenced by the extent of fatty infiltration of the liver, the concentration being lowest in patients with the greatest extent of fatty liver. This view is supported by[7,8].The importance of fatty liver in kwashiorkor is that prognosis in this disorder correlates hepatic lipase and triglyceridaemia in kwashiorkor **,**with the extent of fatty infiltration of the liver [5] and the variation in the levels of serum triglyceride, may therefore reflect the severity of the disease.In explaining the fatty liver in kwashiorkor many workers have proposed that there is a defective release of very-low-density lipoprotein probably as a consequence of decreased apoprotein synthesis by the liver [7,5,8]. Thus there are two major opposing factors affecting the levels of circulating triglycerides in kwashiorkor; a decreased release of VLDL causing hypotriglyceridaemia while reduced serum hepatic triglyceride lipase causing hypertriglyceridaemia. The mechanisms of deficiency of dietary fat and impaired fat absorption, could be the underlying factors for hypotriglyceridaemia in these malnourished children.[7,5,8] Malnourished children with varying extents of triglyceridaemia may be considered as representing different stages of the equilibrium between the rates of release and catabolism of triglycerides. This probably explains why dietary therapy which has been shown to cause release of liver lipids into circulation [7] leads to a pronounced hypertriglyceridaemia within 2-10 days of dietary rehabilitation in treatment of kwashiorkor followed by a return to normal level at full recovery [5].There is convincing evidence to support the involvement of other mechanisms in the pathogenesis of the fatty liver of childhood malnutrition [9,6,4,10]. In theory, the amount of hepatic triacylglycerol available for export can exceed the rate of removal because of 1 or more of 4 possible mechanisms- either increased hepatic fatty acid (FA) synthesis; impaired FA oxidation by hepatocytes; increased hepatic FA influx secondary to a stimulated rate of lipolysis, impaired whole-body FA oxidation, or both; or impaired removal of triacylglycerol from the liver by VLDL. For example, [6] proposed that hepatic FA availability was increased because of increased FA synthesis from glucose. Fletcher found significantly lower glucose-6-phosphatase activity in the liver tissue of malnourished children with fatty livers than in the liver tissue of children who had recovered from malnutrition [6]. In addition, 2 studies by [4] (Lewis B, 1964) reported faster plasma palmitate flux and higher FA concentrations, which were indicative of a stimulated rate of lipolysis, in malnourished children with fatty livers than in well-nourished children. An increased lipolysis together with the finding that whole-body lipid oxidation is markedly slower in children with kwashiorkor than in well-nourished children [10] will result in an increased influx of free FA into the liver. Furthermore, decreased peroxisomal β-oxidation of FA by the liver has been proposed as one of the mechanisms of the pathogenesis of fatty liver, on the basis of the markedly lower concentration of peroxisomes in the liver of severely malnourished children at autopsy than in the liver of children who had recovered from malnutrition [11].It is therefore possible that the fatty liver of the malnourished child can result from an increased availability of FA for hepatic re-esterification to triacylglycerol and not from impaired VLDL-apo B-100 synthesis.[9] Marvin Reid and Asha Badaloo 2005 have further proposed that, the synthesis of the apolipoprotein moiety of VLDL in severely malnourished children with more liver fat is not impaired relative to the synthesis in those with less liver fat, there seems to be a compensatory response as liver fat increases, suggested by the faster rate of synthesis of apo B-100 in the children with more liver fat. This occurs even in the face of the slower rate of whole-body protein turnover that is characteristic of the severely malnourished state [9].

Cholesterol: A decrease in total blood lipids was reported by previous workers [12,13,14,15] a decrease in total serum cholesterol, and reduction in the level of both o-lipoproteins [14] and 8-lipoproteins [15] was also shown by previous studies. The serum total cholesterol results of this study are also found to be in the harmony with the result of above workers, who have reported a significant reduction in serum total cholesterol level in PEM cases. The same two mechanisms; deficiency of dietary fat and impaired fat absorption which are involved in the reduction of triglycerides and phospholipids could be the underlying factors for hypo cholesterol level in these malnourished children. In the present study the scenario of lipid was also noted to be improved to the normal after the administration of nutritional intervention to the study group, Serum levels of triglycerides and total cholesterol returned to the normal, probably indicative of the increased release of VLDL. It was proposed that changes in the lipid composition of the small intestinal mucosa and in phospho lipid distribution as well as in the fatty acid profile may alter membrane fluidity and organization. These alterations appear to affect the activity of membrane-bound hydrolytic enzymes [16]. In other study it is shown that, in well-fed pre-school children, serum lipids decrease with advancing age and that this pattern was abolished in malnourished children [17] while supply of dietary fat has been reflected to recovered fat absorption and loss of edema and improvement in fatty liver is also an indication of recovery from hypotriglyceridaemia and hypocholesterolemia. On the basis of above results showing lipid status of study group after receiving study nutritional intervention indicated that study nutritional intervention has potentials for normal functioning of digestion and absorption of fat as well as it has potentials to meet to the normal metabolic end points of the lipids and shows lipidic compliance of the nutritional intervention with the body.

However the assessment of clinical correlation between triglyceride and weight for height % was found to be very significant with the support of above results.

Anthropometry: After accounting the anthropometric data recorded during this study, which indicate that, chronic malnutrition appeared to be a more pressing problem than acute malnutrition in this area, as indicated by the levels of stunting and underweight compared to the levels of wasting. It is necessary therefore, to educate the mothers in this study area on the importance of

feeding their children appropriately in their early life. The very significant correlation of triglycerides with weight for height % after the nutritional intervention treatment to the SAM enrolled child also supports strength of nutritional intervention in the recovery of SAM children.

VII. CONCLUSION

Considering the above discussed improvements and supportive results, we recommend that the study nutritional intervention has potentials for normal functioning of digestion and absorption of fat as well as it has potentials to meet to the normal metabolic end points of the lipids and shows lipidic compliance of the nutritional intervention with the body. We conclude that it is the most effective food supplement for the speedy recovery of the impaired lipid metabolism in SAM children. We also conclude that weight for height % can be suggested as an anthropometric marker for the pre-indication of impaired lipid metabolism and fatty liver.

VIII. ACKNOWLEDGMENT

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