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HEMATOLOGICAL STUDY OF SEVERE ACUTE MALNOURISHED CHILDREN AND ITS CORRELATION WITH FF-SUAGAR AND GLOBULIN

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ABSTRACT

Micronutrient deficiency is the another form of malnutrition. Nearly all deaths linked to micronutrient deficiency are due to a lack of iron, zinc or vitamin. A Present study was designed to assess the efficacy of the study nutritional intervention by monitoring various hematological parameters before and after the Nutritional Intervention treatment (NIT) and to find correlation of MCHC, MCH and MCV with blood sugar level in malnourished children and serum globulin. This was open label prospective parallel group active comparator interventional study, 105 Study and 100 control SAM(Severe Acute malnutrition)children of 1 to 5 years of age and either sex were randomly enrolled. Study group was given NIT, providing 2.5 to 3gm Protein and 90-100 kcal/kg body weight/day for three months. Hb,MCH,MCV,MCHC, Reticulocyte count and Blood sugar level (FF and PP),total protein, albumin and globulin of both groups were estimated before and after the NIT. Before NIT **P** value for Hb,MCH,MCV,MCHC,reticulocyte count and Blood sugar level (FF and PP) were insignificant and after NIT all were noted to be significant**.**The Correlations of sugar –FF with MCHC, and MCV as well as globulin with MCH were noted to be significant. The investigators conclude that study nutritional intervention is the effective food supplement for the recovery of impaired hematological and anemia status in SAM children while Sugar-F has significant correlations with MCV and MCHC similarly serum globulin has significant correlation with MCH.

KEYWORDS: Hemoglobin,MCV,MCH,MCHC,Anemia**,**Malnutrition,Correlations.

INTRODUCTION

Protein–energy malnutrition is defined on the basis of anthropometric criteria as, -The fall below 2 standard deviations (-2S.D.) under the normal weight for age (underweight), height for age (stunting) and weight for height (wasting) is known as malnutrition.".[1] Micronutrient deficiency: This is the another form of malnutrition. A long-term lack of nutritious food or having an infection such as worms, can result in a lack of vitamins and minerals in a child's diet. Micronutrient deficiencies represent a serious risk to a child's health: they account for one-third of all malnutrition-related child deaths, and 10% of all children's deaths.[2,3] Nearly all deaths linked to micronutrient deficiency are due to a lack of vitamin A, zinc or iron. The prevalence of under nutrition and anemia is also greater among children belonging to scheduled castes and scheduled tribes and the lowest wealth quintile in Maharashtra.[4,5]

At study site the high prevalence of iron deficiency anemia was noted, which leads to low immunity and increases the scope of opportunistic infections to grab the children, which results into anorexia followed by small intake of low quality food thus children step into the malnourishment. Consequently anemia was noted to be a key factor in the development of malnourishment. To stop this vicious cycle, development and evaluation of nutritional intervention which could overcome the iron deficiency and other related issues with it, was the highest need at study site. Therefore in present study, development and supplementation of study nutritional intervention and its evaluation has been carried out by estimating various hematological parameters such as –Hemoglobin, MCH,MCV,MCHC, Reticulocyte count as well as blood sugar(FF/PP),total protein, albumin and globulin levels.

MATERIAL

Following kits, chemicals and important instruments were used to estimate various blood parameters:

1) **Serum Total Protein kit : Company:** Span Diagnostics Ltd., Surat, Gujarat.

Method: Modified Biuret, End point assay.[7]

Material supplied with the kit: Biuret Reagent **;** Protein standard: 6.5 g/Dl.[7]

2) **Albumin test kit: Company:** Span diagnostics

Method: Bromocresol Green,End point assay. [8]

Material supplied with the kit : i) Albumin reagent: 1x100 mL. ,

ii) Albumin standard. 4 g/dL[8]

3) Blood Sugar kit: Company : Span diagnostics, **Method:** GOD-POD[209]

Material supplied with the kit:

Reagent no.1-Glucose reagent -100mM/L -10 vials

Reagent no.2-Glucose diluents -10mM/L- 2x500mL

Reagent no.3-Glucose standard -100mg/dL-1x5 ml

Reagent no.4-Glucose standard-400mg/dL-1x2.5ml[209]

4) Chemicals for Reticulocyte count : New Methylene Blue (10g/L)[212]

Company : Beacon,diagnostics,India.

5) Instruments: **i]** Fully Automated Analyser - Olympus AU-400 : Company : Olympus

ii] Microscope : Binocular, Company : Micron optics

iii] Auto Cell counter - Model-Por-Cam : Company : Rapid diagnostic.

METHOD

This was Open label prospective parallel group active comparator interventional study,105 Study and 100 control SAM children of 1 to 5 years of age and either sex were randomly enrolled. Study group was given NIT, providing 2.5 to 3gm Protein and 90-100 kcal/kg body weight/day for three months. From each test and control subjects morning fasting blood samples were collected in labeled; plain vaccutainers,-such kind of blood collection was done at two different periods-first; at the time of enrollment and second; after three month's nutritional intervention treatment. All blood samples in plain vaccutainer were centrifuged within 1 hr to obtain serum.Instructions and procedures provided by manufacturer in the all kits were followed.

Anthropometric measurements for grading the malnourishment

The age and oedema of each subject was specially noted at the time of enrollment. The weight and height of each subjects were measured as per WHO guidelines. Weight was measured on calibrated regular and infant weighing scales. While Standing height of subjects above two years was measured by stadiometer and length of subjects below two years was measured by infantometer.

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. P-value was obtained, P< 0.05 considered significant difference, p < 0.000 considered highly significant difference.

RESULTS AND OBSERVATIONS

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

Baseline charactcristics	Groups	N	Mean	Std. Deviation	Std. Error Mean
Protein (T) Gm%	Study group	105	4.30	0.32	0.03
	Control group	100	4.30	0.33	0.03
Albumine (A) Gm%	Study group	105	2.54	0.22	0.02
	Control group	100	2.53	0.22	0.02
Globuline (G) Gm%	Study group	105	1.74	0.26	0.03
	Control group	100	1.75	0.27	0.03
Hb g%	Study group	105	6.60	1.71	0.17
	Control group	100	6.41	1.60	0.16
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
MCV f/L	Study group	105	62.40	6.17	0.60
	Control group	100	63.10	6.80	0.68
MCHC g/dL	Study group	105	24.23	1.48	0.14
	Control group	100	24.26	1.36	0.14
MCH pg.	Study group	105	20.70	1.53	0.15
	Control group	100	22.11	7.87	0.79
Reticulocyte count (%)	Study group	105	2.70	0.36	0.04
	Control group	100	2.64	0.46	0.05

Table 2. Independent sample test for Before treatment in study and control group

npaired t-test for Equality of Means						95% C I of the Difference	
Baseline charactcristics	**Mean Difference**	**Std. Error Difference**	**t test value**	**df**	**p value**	**Lower**	**Upper**
Protein (T) Gm%	0.003	0.045	0.063	203	0.950 (NS)	-0.087	0.092
Albumine (A) Gm%	0.007	0.030	0.234	203	0.816 (NS)	-0.052	0.067
Globuline (G) Gm%	-0.004	0.037	-0.113	203	0.910 (NS)	-0.077	0.069
Hb Gm %	0.176	0.232	0.758	203	0.449 (NS)	-0.281	0.632
Amylase Units/dL	0.228	1.303	0.175	203	0.861 (NS)	-2.341	2.798
Lipase units/dL	-0.082	0.186	-0.442	203	0.659 (NS)	-0.448	0.284
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
MCV f/L	-0.748	0.906	-0.825	203	0.410 (NS)	-2.535	1.039

MCHC g/dL	0.069	0.199	0.345	203	0.730 (NS)	-0.323	0.460
MCH pg	-1.443	0.783	-1.843	203	0.067 (NS)	-2.987	0.101
Reticulocyte count (%)	0.011	0.058	0.189	203	0.851 (NS)	-0.102	0.124

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

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

Baseline chararcteristics	Groups	N	Mean	Std. Deviation	Std. Error Mean
Protein (T) Gm%	Study group	105	6.904	0.519	0.051
	Control group	100	4.805	0.287	0.029
Albumine (A) Gm%	Study group	105	4.117	0.487	0.048
	Control group	100	2.804	0.191	0.019
Globuline (G) Gm%	Study group	105	2.700	0.207	0.020
	Control group	100	1.930	0.259	0.026
Cholesterol mg%	Study group	105	154.701	10.752	1.049
	Control group	100	116.936	7.534	0.753
Triglyceride mg/dL	Study group	105	52.913	10.185	0.994
	Control group	100	21.030	1.992	0.199
Hb Gm %	Study group	105	12.20	0.384	0.038
	Control group	100	8.510	1.480	0.148
Ferritin ng/mL	Study group	105	40.507	10.412	1.016
	Control group	100	11.740	9.237	0.924
Cortisol (morng) µg/dL	Study group	105	18.508	4.656	0.454
	Control group	100	33.100	4.705	0.471
Growth Hormon ng/mL	Study group	105	5.840	2.491	0.243
	Control group	100	13.130	2.482	0.248
Amylase Units/dL	Study group	105	93.007	12.750	1.244
	Control group	100	23.401	7.278	0.728
Lipase units/dL	Study group	105	50.809	5.479	0.535
	Control group	100	11.207	5.208	0.521
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
MCV f/L	Study group	105	89.602	7.502	0.732
	Control group	100	65.010	4.896	0.490
MCHC g/L	Study group	105	34.310	1.734	0.169
	Control group	100	26.201	2.509	0.251
MCH pg	Study group	105	30.803	2.307	0.225
	Control group	100	22.400	2.420	0.242
Reticulocyte count (%)	Study group	105	1.504	0.677	0.066
	Control group	100	2.703	0.371	0.037

Equal variances assumed

Table 4. 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**
Protein (T) Gm%	2.119	0.059	35.954	203	**0.0001**	2.003	2.235
Albumine (A) Gm%	1.323	0.052	25.386	203	**0.0001**	1.220	1.426
Globuline (G) Gm%	0.807	0.033	24.759	203	**0.0001**	0.743	0.872
Cholesterol mg%	37.765	1.303	28.992	203	**0.0001**	35.197	40.333
Triglyceride mg/dL	31.883	1.037	30.746	203	**0.0001**	29.839	33.928
Hb g%	3.707	0.150	24.703	202	**0.0001**	3.411	4.003
Ferritin ng/mL	28.733	1.377	20.863	203	**0.0001**	26.018	31.449
Cortisol (morng) µg/dL	-14.617	0.654	-22.352	203	**0.0001**	-15.906	-13.328
Growth Hormon ng/mL	-7.288	0.347	-20.978	203	**0.0001**	-7.973	-6.603
Amylase Units/dL	33.888	1.460	23.218	203	**0.0001**	31.011	36.766
Lipase units/dL	39.613	0.747	53.004	203	**0.0001**	38.139	41.086
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
MCV f/ L	24.552	0.890	27.602	203	**0.0001**	22.798	26.306
MCHC g/dL	8.154	0.300	27.177	203	**0.0001**	7.563	8.746
MCH pg	8.419	0.330	25.498	203	**0.0001**	7.768	9.070
Reticulocyte count (%)	-1.188	0.077	-15.463	203	**0.0001**	-1.339	-1.036

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

Table 5. Descriptive statistics for gender (Before treatment) in both groups

Before treatment		**Study group (N=105)**			**Control group (N=100)**		
Characteristics	Gender	N (Sample size of Gender)	Mean	Std. Deviation	N (Sample size of Gender)	Mean	Std. Deviation
Hb gm%	Male	42	6.47	1.88	46	6.26	1.748
	Female	63	6.66	1.61	54	6.54	1.462

Table 6. Comparison in gender (Before treatment) for their characteristics in both groups

Study group (**Before** treatment) (N=105)							
Characteristics	Mean Difference	Std. Error Difference	t test value	df	p value	95% CI Lower	95% CI Upper
Hb gm%	-0.194	0.342	-0.568	103	0.571 (NS)	-0.874	0.485
Control group (**Before** treatment) (N=100)							
Hb gm%	-0.276	0.321	-0.861	98	0.392 (NS)	-0.913	0.361

Table 7. Descriptive statistics for gender (After treatment) in both groups

After treatment		Study group (N=105)			Control group (N=100)		
Characteristics	Gender	N (Sample size of Gender)	Mean	Std. Deviation	N (Sample size of Gender)	Mean	Std. Deviation
Hb gm%	Male	42	12.21	0.37	40	8.37	1.64
	Female	63	12.22	0.39	60	8.60	1.37

Table 8. Comparison in gender (After treatment) for their characteristics in both groups

Study group (After treatment) (N=105)							
Characteristics	Mean Difference	Std. Error Difference	t test value	df	p value	95% CI Lower	95% CI Upper
Hb gm %	-0.011	0.077	-0.138	102	0.890 (NS)	-0.164	0.142
Control group (After treatment) (N=100)							
Hb g%	-0.231	0.303	-0.763	98	0.447	-0.831	0.37

Table 9. Correlations of Sugar-FF with MCV and MCHC after treatment in study group

Sugar-FF	**MCV**	**MCHC**
Sample size (N)	105	105
Pearson Correlation r	0.227*	-0.271*
p value	0.02 (Significant)	0.005 (very Significant)
Interpretation	poor positive correlation	Poor negative correlation

Table 10. Correlations of Globulin with MCH after treatment in study group

Globulin	**MCH**
Sample size (N)	105
Pearson Correlation r	-0.201*
p value	0.004 (very Significant)
Interpretation	Poor negative correlation

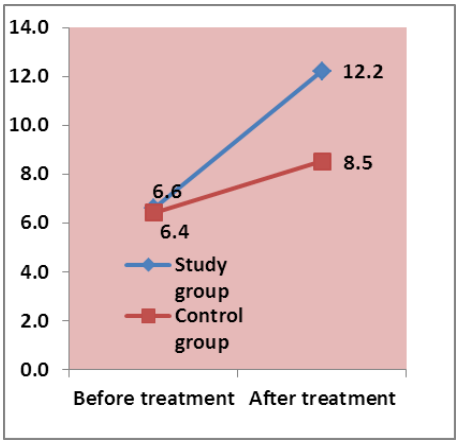

Figure – 1 HB (Gm%):

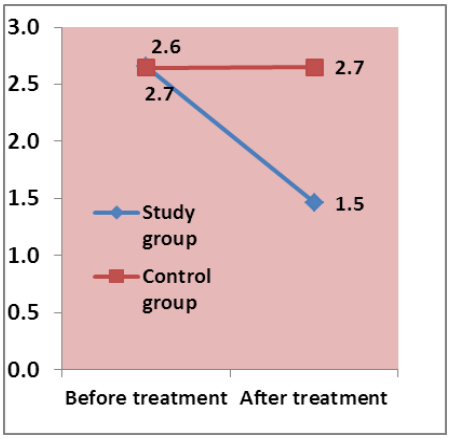

Figure 2 - Reticulocyte count (%)

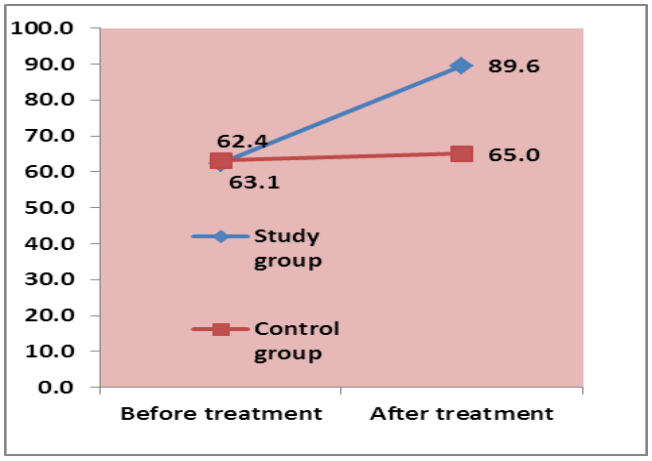

Figure-3 MCV fL

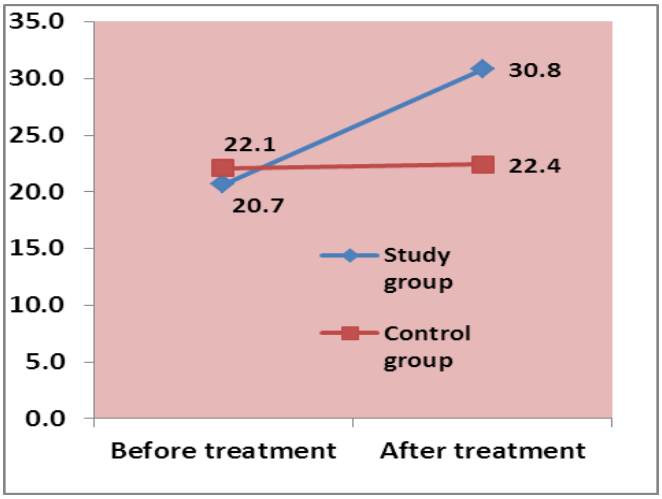

Figure 4. - MCH pg

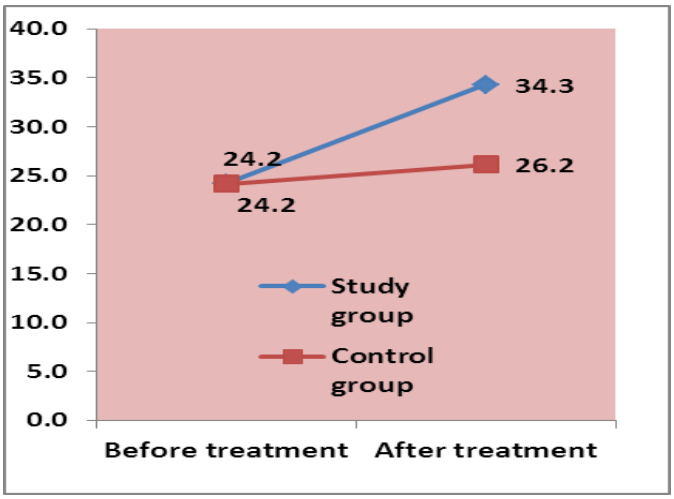

Figure 5. - MCHC g/dL

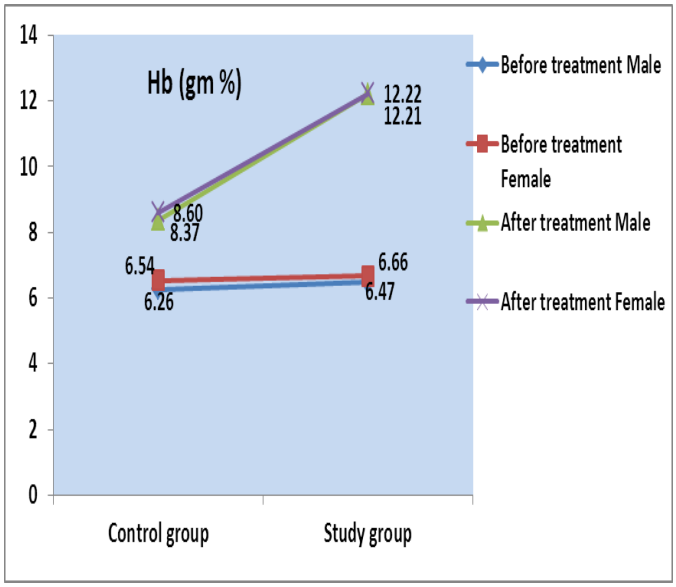

Figure -6 Hb (Gm%) in male and Female

DISCUSSION

MCV: This value represents the average size of the red blood cell and it varies with age.When anemia becomes significant enough to interfere with hemoglobin synthesis microcytosis occurs (low MCV).

MCH: Initially with iron deficiency the MCH is normal, but with advancing anemia the red blood cell becomes more hypochromic and this change is evident by a low MCH.[6]

Hemoglobin: Before : In this study before the nutritional intervention treatment P value was noted as P= 0.449 which is insignificant, suggestive of similar baseline hemoglobin characteristics at the time of enrollment.

After the nutritional intervention treatment serum hemoglobin levels in study group, was found to be increased to normal levels. P value for hemoglobin after the nutritional intervention treatment was noted as P= 0.0001 which is highly significant.

Gender

Study Group: Similarly after comparison of the gender in study group before nutritional intervention treatment both gender had similar Hb status (P=0.571).While after nutritional intervention treatment in study group both gender had similar Hb status (p=0.890).

Control Group: After comparison of the gender in control group before nutritional intervention treatment both gender had similar Hb status (P=0.392). While after the treatment period, in control group both gender had similar Hb status (p=0.447).

MCV, MCH, MCHC and Reticulocyte count (RC):

i) MCV:

Before: Before nutritional intervention, both groups had similar MCV (p=0.410) Nutritional intervention resulted in significant improvement in MCV (p=0.0001) in study group as compared to control group.

MCH: Before nutritional intervention, both groups had similar MCH (p=0.067) Nutritional intervention resulted in significant improvement in MCH (p=0.0001) in study group as compared to control group.

iii) MCHC:

Before nutritional intervention, both groups had similar MCHC (p=0.730) Nutritional intervention resulted in significant improvement in MCHC (p=0.0001) in study group as compared to control group.

iv) Reticulocyte count (RC): Before nutritional intervention, both groups had similar RC (p=0.851) Nutritional intervention resulted in significant improvement in RC (p=0.0001) in study group as compared to control group.

Hb, MCV, MCH, MCHC: The results revealed for Hb, MCV, MCH, MCHC, Ferritin, have shown that all the enrolled subjects were having iron deficiency anemia at the time of admission. The study site is a hilly area, tribal people living there on "Sahiyadri" mountain ranges". The anemia associated with severe PEM has a more significant impact on oxygen transport at high altitude than at sea level and requires an adaptive response in the oxygen dissociation curve to satisfy tissue oxygen demands. The anemia of PEM is of great functional importance to those living at higher altitude and who experience hypobaric-hypoxic conditions daily.[7]These geographical conditions along with nutritional deprivation could be the probable reason behind development and worsening of anemia which ultimately reflected as failure to the adoptive mechanism of malnutrition at study site. It has been reported that preschool children (<8years) and adolescents (>15 years) during growth spurts have the greatest physiological demands for iron and are at highest risk of iron deficiency

anaemia.[8] After the finding of a microcytic, hypochromic blood picture, parameters such as low serum ferritin levels, lower values of hemoglobin, MCV ,MCH, MCHC, finally,the response to the therapy are the parameters taken for the diagnosis of iron deficiency,in the present study.

This study comprised of the subjects belonging from the age group 1-5 year. the same age group has been studied by Dhar et. al.(1969). [8] The various factors responsible for clinical manifestations observed in this study were similar to the previous studies are discussed below.

-The present study have shown that all malnourished subjects from both test and control groups had below normal Hb, MCV, MCH and MCHC values before the nutritional intervention therapy.

-Smith et al (1959)[9] Dhar et al (1969)[8] has shown similar observations.While after the nutritional therapy treatment test group have shown significant improvement in Hb, MCV, MCH, MCHC values. These values were found to be raised up to the normal levels, which can be well compared with those reported by, Raman et al (1992).[8] - In this study reticulocyte count was found to be increased in both test and control groups before the nutritional intervention treatment, suggesting good erythropoisis due to iron deficiency and low Hb ,While few other workers have noted impaired erythropoises in malnourished children due to folate deficiency[10]. After the nutritional intervention treatment there was fall down of reticulocyte count to the normal range was noted in study group suggesting improved iron status and Hb status, where -as there was no or very less decrease in RC was noted in control group, suggesting that still iron deficiency with low Hb levels remained in the control group.

-Fondu p. et.al. in their work have shown that in 2 stages the hematological picture could forward in PEM., the erythropoiesis was found normal in a first stage or increased because of the existence of a decreased red cell life span; there was no apparent hypoxia but a low hemoglobin level was noticed. At a more advanced stage of the disease the tissue metabolism falled dramatically, the erythropoiesis was no longer stimulated, and the erythrocyte volume decreased notably.[10] Also anemia in PEM was shown to be associated with, decreased RBC osmotic fragility. which was in agreement with other investigators who attributed it to abnormalities of the red cell membrane lipids that could be due to hepato cellular

derangement within kwashiorkor. [8.11] Thomas et al., Dhar et al (1969) [8,12] and Smith et al [13] has also reported same results - in a study of this kind ,the most reliable evidence of iron deficiency anemia is taken as the response to therapeutic and dietary iron. In both boys and girls from test group, after 90 days of nutritional intervention therapy. Significant Hb levels increased which was noticed as a effect of the supplementation. The response was better in the severe anemic cases. In the lower age groups the effect on the Hb levels was high. The similar results were shown by Dhar et. al.[8]

-Ferrous iron is more easily absorbed than ferric iron, and thus the usual treatment for infants and children is ferrous sulfate. Premature infants are frequently vitamin E deficient due to decreased intake, decreased stores, and poor absorption of vitamin E. Since iron therapy inhibits absorption of vitamin E [14] it could worsen the Vit-E status of these children, which leads the child to other complications, and it could enter into sever malnourishment.

-The absorption of iron on an empty stomach is about twice to that of a full stomach; therefore it is recommended that the dose be given about an hour prior to a meal. [14] It was noted that at the study site that practically these important things were not followed while supplementing the child with iron therapy. Also early discontinuation of the iron/folate treatment, and non co-operation as well irregularity by the parents and child for the iron treatment was also noted at the study site. Many times multi vitamin syrup supplied by Government is irregular, which creates gap in the treatment and leads to under nutrition.These facts could be attributed to the anemic results of enrolled study subjects before the start of the nutritional therapy. Hence the duration of intervention treatment of present study was 3 months in order to replenish the iron stores.

-Brown M.S., Dallman PR,[14,15] in their study have found that If these iron stores are not replenished because the iron therapy was discontinued too soon, a rapid recurrence of iron deficiency anemia may result. They have found that response to treatment was initially evident by a reticulocytosis peaking in 5 to 10 days from the onset of treatment. During the first week of therapy, they have found that the hemoglobin increased to about 0.25 to 4 g/dL/d and then found to be slowed to about 0.1 g/dL/d.[14] The reticulocytes results of the present study was found to be in accordance with these workers. -Dallman PR, Yip Ret.al.(1993)[14] Muslimatun S. Schmidt MK,(2001)[16] in their studies have called children for follow up to see if there was no improvement,they have attributed the failure of oral iron

therapy as the result of impaired absorption, incorrect diagnosis, ongoing blood loss greater than hemoglobin generation, inadequate dose, ineffective iron preparation, superimposed malignancy or inflammatory disease, or, most commonly, simple noncompliance. [14]According to them compliance can be an issue because of the taste of iron, gastrointestinal distress, or concern of parents that the drops will stain the infant's teeth. These problems could be dealt with by giving the iron with a small amount of food or liquid, preferably something that will enhance the absorption, and by giving the drops in the back of the mouth. The above discussed probable causes of treatment failure could also be attributed to the study site malnourished children also, due to which in spite of consumption of iron syrup, and folate tablets provided by PHC to the children who were enrolled in the present study all were suffering from iron deficiency before the nutritional rehabilitation. Various other factors and conditions responsible for the development of iron deficiency anemia were also studied by different workers, their important findings could also be correlated to the present study results, so those were discussed below – A reticulocytosis and disappearance of anemia was noticed due to the normal diet. To a hypometabolic state with decrease in oxygen requirements gives response as a anemia of starvation. A reduction in oxygen consumption and erythropoietin production, results due to protein deficiency. Similarly a subsequent drop in reticulocyte count and erythropoiesis also resulted due to protein deficiency.[17] At the erythroblast level red cell maturation is blocked and Slightly decrease in the erythropoietin-sensitive stem cell pool occures. [18] The marrow along with reduced erythroid-myeloid ratio, often slightly hypo cellular or cellular. The reticulocyte count may rise with treatment of the infection, thus erythroid precursors may appear in the marrow. By feeding high-protein diets when nutrition is improved (essential amino acids), due to hemodilution there is reticulo-cytosis, a slight fall in hematocrit and then a rise in hematocrit, hemoglobin and red blood cell count.[19] In children with kwashiorkor, although the plasma volume is reduced to a variable degree in proportion to the decrease in lean body mass the total circulating red cell mass decreases as metabolic demands reduces protein deprivation.[20]

Before an increase in red cell mass, an increase in plasma volume may occur during repletion, and despite reticulocytosis the anemia may become more severe. The erythropoietin level increases as the hemoglobin concentration falls[16] and more important, as oxygen demand increases. The increased oxygen demand may in part account for the reticulocytosis. Also, during the repletion period, occult deficiencies of iron, folic acid, and occasionally of riboflavin, vitamin E, and vitamin B12 may become manifest unless these

essential nutrients are supplied in adequate amounts. [16] In kwashiorkor there is a decrease in serum proteins. Most of the trace elements such as iron, copper are carried in serum are protein bound, and therefore changes in the trace element concentration of serum may be influenced by the abnormal serum protein pattern. [21]

Correlation of Sugar-FF with MCV and MCHC were significant at P = 0.02 and P = 0.005 respectively where as correlations were found poor positive at r = 0.227 and poor negative at r = -0.271 respectively for both in the study group after treatment .Correlations of Globulin with MCH was significant at P = 0.004 while correlations was noted to be poor positive at r = -0.201 in study group after treatment.

CONCLUSION: The investigators conclude that study nutritional intervention is the effective food supplement for the recovery of impaired hematological and anemia status in SAM children while Sugar-F has significant correlations with MCV and MCHC similarly serum globulin has significant correlation with MCH.

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