

Table 3: The level of Hb, PCV, MCV, MCH and MCHC in thalassaemia major, minor and control group

Parameters	Study group (mean±SD)		
	Thalassaemia major n=72	Thalassaemia minor n=17	Control n=30
Hb concentration (gm/dl)	10.8±3.0*	11.1±1.18	11.5±1.79
PCV(%)	34.6±8.6 ⁺	36.4±7.10 [♦]	34.3±6.02
MCV(fl)	83.0±6.35 ^{**++}	64.9±1.76 ^{♦♦}	76.3±16.17
MCH(pg)	26.9±3.67 ^{++*}	18.8±1.57 ^{♦♦♦}	28.5±1.57
MCHC(gm/dl)	31.2±3.78 ^{*+}	29.0±2.53 ^{♦♦}	35.5±2.10

* p<0.05 correspondent to control. ** p<0.01 correspondent to control. ♦ p<0.05 correspondent to control. ♦♦ p<0.01 correspondent to control. ♦♦♦ p<0.001 correspondent to control. + p<0.05 correspondent to minor. ++ p<0.01 correspondent to minor. +++ p<0.001 correspondent to minor.

Discussion

Trace elements are increasingly playing a diagnostic, curative and preventive role in many diseases^[10].

Iron overload is one of the major complication in B- thalassaemia which due partly to the ineffective erythropoiesis and the constant hemolysis from early infancy and partly to the frequent blood transfusion required for the treatment of the severe anaemia^[11].

Our study in agreement with other studies found that serum Cu and iron were significantly increased in thalassaemia patients^[12-14]. The close relation between serum Cu and iron can be explained by the importance of copper containing enzymes and co-factors for iron absorption and the effect of copper on the release of iron from the body stores as well as the utilization of iron in haemoglobin synthesis^[15,16]. This will explain the hypercupraemia usually encountered in haemochromatosis which is a principle complication in thalassaemia^[17,18].

In thalassaemia minor serum ferritin and serum Cu level was significantly lower than in thalassaemia major, table 2, figure 1 and

2, which is expected since those patients are non transfusion dependant and have less extend of ineffective erythropoiesis^[11]. Therefore, we expect the increase in serum Cu will be much less than thalassaemia major.

Bashir NA et al^[13] study was similar to our study revealed a significant increase in serum Zn and Cu in thalassaemia major patients, and he had contributed it to impairment in kidney function and to disturbances in the metabolism of Zn and Cu which usually encountered in those patients^[13].

Moreover since Zn concentration in the RBC is approximately 12 time that found in the plasma^[12] and during transfusion of blood a considerable proportion of transfused cells will be destroyed¹¹, therefore we may propose that these patients who are transfusion dependant will have higher serum zinc comparing to control subjects and to thalassaemia minor patients who are not transfusion dependant.

It is well known that liver function as major storage organ for zinc^[19] and since iron overload in the liver may generate oxygen free radical and may induce peroxidative