Comparative study of the some complication in B-thalassemia major patients with c...

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Iron overload due to multiple transfusions is the main cause of complications, such as thyroid dysfunctions are well documented in patients with thalassemia major requiring frequent and recurrent blood transfusions [19].

Our study showed that the mean of the FT4 in the patients was in normal range compared with control group, whereas TSH level in patients were significantly higher than control group.

Jain [20] reported the mean serum total T4 and T3 levels in the β -thalassemia patients were significantly lower (p < 0. 001) and TSH level was higher than control group. Other study was demonstrated significant (p < 0. 05) increased TSH (3. 5 \pm 1. 7 μ IU/mI) in the thalassemia patients, when comparison with healthy control group (2 \pm 1. 2 μ IU/mI), those results showed 20% with subclinical hypothyroid [21]. Similarly, other study showed that in the β -thalassemia patients, level of T4 was lower than the control group and TSH level was higher than the control group[22].

Our study was shown that 23. 7 percentages from β -thalassemia patients had subclinical hypothyroidism, which was in good agreement with the study by Malik et al [23]. Additionally, our finding disagrees with Mula-Abed et al., [24] Pirincciogla et al., [25] that the prevalence was 3. 3%. Recent study by Ayoub et al., [26] demonstrated prevalence of hypothyroidism 21. 6 % of β -thalassemia patients in Baghda (high TSH level with normal T4) in their study subjects. Even so, secondary hypothyroidism was rare in β -thalassemia patients, which was not observed. Ghosh, [27] reported the prevalence of Subclinical hypothyroidism about 23. 5% in West Bengal. Other study was

shown that progressive of thyroid dysfunction observed about 35% of β -thalassemia patients who have the age 18. The lack of increasing of TSH in response to low levels of T4 in these patients was indicated a high incidence of defective pituitary thyrotrophic function [28].

However, the variation of prevalence may be related on the age of study population, duration of blood transfusions, ferritin levels and dose of the iron-chelating agent [29, 30]. In this study hypothyroidism was 23. 6% of β -thalassemia patients which depending on the elevation of thyroid stimulating hormone more than 5 μ IU/ml . High prevalence ranges in our study may attributed to in fact that most of patients of β -thalassemia major were selected in the second decade.

There are many mechanisms responsible for thyroid dysfunction in β-thalassemia patients have been suggested, Nevertheless, the exact mechanism is not known. Hypothyroidism may be related to the accumulation of iron in thyroid glands due to blood transfusion by iron overload leading to gland dysfunction [31]. Consequently, the iron stored as ferritinis deposited in organs as hemosiderin, a toxic substance affecting tissues at least partially by inducing oxidative stress [32]. Additionally, formation of free radical and lipid peroxidation were caused to the damage of the mitochondrial, lysosomal, sarcolemma membrane and DNA [33, 34]. Those damages involve practically all organs in the body like spleen, liver and bone narrow. Also, iron poisoning effects on several endocrine glands as the thyroid gland function and gonads [19].

Most thalassemia patients have a delayed or absent puberty occur with appearance disorder in menstrual cycle and anovulation in women. Also, abnormalities spermatic happen and reduced sexual activity in males. The incidence rate of the puberty failure was between 50% to 100% in the different studies [15, 35].

This study indicated in female more 13 years, the level of estradiol(30. 60 ± 14 . 68pg/ml) declined high significant (p < 0. 01) compared with control group(13. 83 ± 9 . 06pg/ml) whereas the mean of testosterone level was 1. 22 ± 0 . 83ng/ml in β -thalassemia patients and 3. 71 ± 1 . 3ng/ml in control group more than 14 years. Mean level of testosterone was highly significantly lower in β -thalassemia patients than in control (p < 0. 001). In β -thalassemiapatients low level estradiol (in females) and testosterone (in males) was noted 9/14(64.28%) and 7/9 (77. 77%) respectively.

Dundar et al., [36] was recorded the serum level of estradiol 19. 4 \pm 15. 9 pg/ml significantly reduced (p <0. 001) in the β -thalassemia patients compared with control group (72. 1 \pm 51. 1 pg/ml) in puberty females, and in the males, serum levels of testosterone reduced in the β -thalassemia patients but not statistically significant. Carmina et al., [37] reported the serum level of testosterone(66 \pm 123. 66 ng/ml) in β -thalassemia patients significant decrease with healthy group(331. 98 \pm 173. 76 ng/ml) in the male aged 14-18years . Other finding indicated lower serum levels of estradiol than controls in similar age. Additionally, in study [38] were compared the mean of the serum level of estradiol of patients with control group in the female aged between12. 5-18 years. The results indicated significant (p <0.

05) decreased the level of estradiol (22. 91 ± 17 . 41pg/ml) in the patients, when comparison with control group (108. 17 ± 107 . 45pg/ml).

Sexual immaturity is the most complication of severe thalassemia. The association between hypogonadism and pituitary iron overload is well established, duo to pituitary iron overload begins in the first decade of life prior to the liver and cardiac iron deposition [39, 40]. Pituitary iron overload and iron-induced oxidative stress may result in secondary hypogonadism in thalassemia patients [41].

CONCLUSIONS

Endocrine disorder is the important health problem in beta-thalassemia major patients. So, appropriate treatments are suitable for each endocrine disorder. On the other hand, we should focus our attention to control the negative effects of the iron overload in the many tissues function.

Antioxidant drugs, like the vitamin C and zinc are suggested which may reduce oxidative stress and tissue damage.