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Original Research Paper

Assessment of Hematological Parameters, Serum Calcium, Iron & TIBC In Gastrointestinal Cancer Patients Before And After Treatment

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ABSTRACT

Introduction: To compare the hematological parameters, serum calcium, serum iron, and TIBC levels between gastrointestinal cancer patients and healthy control participants before and after treatment. **Material & Methods:** The study was carried out at SMS Medical College and Hospital Jaipur, India, in the Biochemistry Department in collaboration with the Radiotherapy and Oncology Department. ESR, Hb, TRBC, WBC, Platelet, serum Calcium, Iron and TIBC levels were evaluated in 150 patients with gastrointestinal cancer before and after therapy and in healthy control participants using specified inclusion and exclusion criteria. The serum Calcium, Iron and TIBC levels were estimated in the biochemistry lab using an auto analyzer, and the Hematological parameters were estimated from an automated hematology analyzer in the pathology lab. The t-test was used to assess the significance of the variance in the mean values of the observed parameters between study groups. When assessing the data, p-values less than 0.05 were considered significant. **Results:** Our findings of gastrointestinal cancer patients revealed an increase in ESR and WBC count and a decrease in Hb, TRBC, and Platelets counts compared to healthy control subjects. Significantly lower serum Iron concentration, significantly higher serum Calcium and Total Iron Binding Capacity were seen in cancer patients compared to healthy control participants. **Conclusion:** Significantly high or low values of the aforementioned test parameters can have serious consequences and delay the initiation of the subsequent phase of therapy. Risk of numerous complications can be decreased with regular monitoring.

Keywords: ESR, Hb,Platelets, TIBC, TRBC, WBC.

INTRODUCTION

Cancer chemotherapy and radiation therapy are used to harm or eradicate cancer cells. Some of the body's normal cells, including the blood cells, may also be damaged by these treatments[1]. Certain chemotherapy drugs can damage bone marrow that produces blood cells, which are extremely vulnerable to the side effects of chemotherapy due to their rapid growth. Low blood cell counts may result from chemotherapy treatment and dosage. If a patient receives radiation therapy in large areas of the body, particularly to the large bones that contain the most bone marrow, such as the pelvis, legs, and torso, he/she might experience low levels of red blood cells. The likelihood that radiation therapy will significantly affect platelet count is low. Cancers that spread (metastasize) branch off from a tumor can spread to other parts of the body, including bone marrow. The malignant cells can displace other cells in bone marrow, making it difficult for the bone marrow to produce the blood cells your body needs. Low blood counts are unusually caused by this which can lead to serious complications that may postpone the next round of treatment. Monitoring blood cell counts allows a doctor to prevent or reduce the risk of complications[2]. Cancer patients are often too tired and weak to be as active as usual. Being inactive can increase calcium in the blood because bones release calcium when they are not being used. Certain types of specific biological used for cancer treatment can also cause hypercalcemia. These therapies include growth factors, interleukins, and tumor necrosis factors. Hormone therapy can also increase the amount of calcium in the blood[3]. Hypercalcemia in the patient with a history of cancer presents a wide range of clinical settings and may be severe enough to require hospitalization[4]. Anemia could be caused by cancer or cancer treatment. During cancer treatment, bone marrow might not be able to produce enough red blood

cells. This makes cancer patients likely to become anemic[5]. Anemia of cancer is characterized by inefficient erythropoiesis and abnormal iron metabolism. Iron deficiency can occur rapidly in patients with cancer due to blood loss or insufficient intake or poor absorption of iron by the digestive tract. There is also 'functional failure' due to retention of iron in macrophages and decreased iron availability for erythropoiesis despite adequate iron storage in the reticuloendothelial system[6]. The acute-phase protein hepcidin is currently considered to be the principal cause of aberrant iron metabolism because it leads to a decrease in both intestinal iron absorption and the release of iron by macrophages. Cytokines released in inflammatory processes are able to increase the production of hepcidin and consequently reduction in the amount of circulating iron[7]. Aysegul Aksan, et al[8] reported Iron deficiency, with or without anemia, is the most frequent hematological manifestation in individuals with cancer, and is especially common in colorectal cancer patients. Iron in particular is a vital micronutrient that plays an essential role in many biological functions, in the context of which it has been found to be closely linked to cancer biology. In particular, iron is vital for the optimal functioning of the immune system, playing major roles in a multitude of different immune processes and pathways. Iron deficiency impacts crucial mechanisms such as immune surveillance, gene regulation and cell apoptosis, all of which are key to host defense against malignant transformation and tumor growth.

MATERIAL AND METHODS

Study subjects: The study comprised 150 gastrointestinal cancer patients and 50 healthy control. The patients and healthy volunteers were categorized as follows: Group 1 consists of 50 normal healthy volunteers, Group 2 of 50 esophageal cancer patients, Group 3 of 50 gastric cancer patients and group 4 of 50 colon cancer patients. All patients and healthy volunteers were recruited from the radiotherapy dept. at SMS Medical College and Hospital, Jaipur from July 2011 to December 2012. The processes were compliant with the responsible committee's ethical guidelines. Each participant gave their written consent to participate in the study. A screening proforma with inclusion and exclusion criteria was used to screen patients. Patients with biopsy-proven gastrointestinal malignancies who had not received any treatment met the inclusion criteria. Patients with old diagnosis who have previously received treatment were excluded from consideration. The criteria for inclusion in the control group were subjects who were able to comprehend the study's purpose and were willing to participate. Participants in the control group were excluded if they had a history of gastrointestinal or hepatic issues, severe physical ailments, overt infectious diseases, or autoimmune disorders. Before the start of the treatment, each individual had peripheral venous blood samples drawn using an aseptic technique from the antecubital vein. Blood was collected and allowed to coagulate in a plain sample vial.

Study Methods: Biochemical investigations were carried out with the aid of an auto analyzer, by utilising commercially available reagents and kits. The procedure given in the manuals, accompanying the kits, were strictly adhered to. CBC was analyzed by an automated hematology analyzer, Sysmex Xt – 1800i.

Reference Values:

ESR: Less than 20 mm in 1st hour

Hb: (Female) = 11.2 – 15.7 g/dl (Male) = 13.7 – 17.5 g /dl

TRBC: (Female) = 3.93 – 5.22 × 10^6 / µl or mill / cumm (Male) = 4.63 – 6.08 × 10^6 / µl or mill / cumm

WBC: (Female) = 3.98 – 10.04 × 10^3 /µl or 1000 / cumm (Male) = 4.23 – 9.07 × 10^3 / µl or 1000 / cumm

Platelets: (Female) = 182 – 369 × 103 / µl or lakhs / mL (Male) = 163 – 337× 103 / µl or lakhs / mL

Calcium: Adults = 8.5 to 10.5 mg/dl Children = 10.0 to 12.0 mg/dl

Iron Adults (female)= 35.0 to 145.0 µg/dl (male) = 60.0 to 160.0 µg/dl

TIBC: Adults = 250.0 to 400.0 µg/dl

Statistical Analysis: Data will be analyzed using the SPSS version 10.0 (SPSS Inc., USA) and MedCalc to determine the significance of the observed differences, p-values less than 0.05 were considered significant.

RESULTS

This study comprised 150 gastrointestinal cancer patients and 50 healthy control subjects. The study's findings and observations in Table 1, 2 & 3 are listed below: The average ESR in healthy control participants was 12.70 (SD = 5.82). The mean value was found to be 40.10 (SD=30.58) in individuals with esophageal cancer before therapy and 17.30 (SD=7.36) after therapy. The mean value was found to be 29.90 (SD=22.84) in individuals with gastric cancer before therapy and 17.40 (SD=8.64) after therapy. The mean value was found to be 30.70 (SD=17.55) in individuals with colon cancer before therapy and 15.80 (SD=6.95) after therapy. When compared to healthy control participants, the ESR values of patients with esophageal, gastric and colon cancer were significantly higher before treatment and after treatment. The average Hb in the healthy control participants was 13.02 (SD=1.55). Patients with esophageal cancer had a mean value of 9.92 (SD=2.01) before treatment and

10.15 (SD=1.98) after treatment. Patients with gastric cancer had a mean value of 9.22 (SD=1.71) before treatment and 10.37 (SD=1.43) after treatment. Patients with colon cancer had a mean value of 9.62 (SD=1.73) before treatment and 10.31 (SD=1.46) after treatment. When compared to healthy control participants, the Hb values of patients with esophageal, gastric and colon cancer both before and after treatment significantly decreased. The average TRBC count in healthy control participants was 5.12 (SD=0.78). Patients with esophageal cancer had mean values of 4.18 (SD=0.95) before treatment and 3.98 (SD=0.74) after treatment. Patients with gastric cancer had mean values of 3.83 (SD=0.76) before treatment and 3.78 (SD=0.82) after treatment. Patients with colon cancer had mean values of 3.79 (SD=0.74) before treatment and 3.69 (SD=0.74) after treatment. When compared to healthy control participants, the TRBC counts of patients with esophageal, gastric and colon cancer are significantly reduced both before and after treatment. The average WBC count in healthy control participants was 6.67 (SD=1.40). Patients with esophageal cancer had mean values of 8.71 (SD=3.43) before treatment and 5.86 (SD=1.32) after treatment. Patients with gastric cancer had mean values of 8.48 (SD=3.42) before treatment and 5.95 (SD=1.40) after treatment. Patients with colon cancer had mean values of 7.81(SD=2.95) before treatment and 5.91 (SD=1.28) after treatment. Compared to healthy control participants, the WBC counts of patients with esophageal, gastric and colon cancer are significantly higher before and significantly lower after treatment. The average Platelet count in healthy control participants was 2.91 (SD=0.69). Patients with esophageal cancer had mean values of 2.56 (SD=1.04) before treatment and 2.42 (SD=1.01) after treatment. Patients with gastric cancer had mean values of 2.61 (SD=1.04) before treatment and 2.52 (SD=0.81) after treatment. Patients with colon cancer had mean values of 2.59 (SD=0.96) before treatment and 2.55 (SD=0.89) after treatment. Patients with esophageal, gastric and colon cancer have reduced platelet counts before and after therapy compared to healthy control participants. The average Calcium level in healthy control participants was 8.31 (SD=0.72). Patients with esophageal cancer had mean values of 8.87 (SD=1.00) before treatment and 8.83 (SD=0.90) after treatment. Patients with gastric cancer had mean values of 8.84 (SD=0.86) before treatment and 8.89 (SD=0.98) after treatment. Patients with colon cancer had mean values of 8.82 (SD=0.83) before treatment and 8.89 (SD=0.97) after treatment. When compared to the healthy control group, patients with esophageal, gastric, and colon cancer have higher calcium levels both before and after treatment. The average Iron level in healthy control participants was 105.38 (SD=28.89). Patients with esophageal cancer had mean values of 64.66 (SD=37.34) before treatment and 93.88 (SD=25.93) after treatment. Patients with gastric cancer had mean values of 77.12 (SD=38.13) before treatment and 92.68 (SD=25.30) after treatment. Patients with colon cancer had mean values of 71.94 (SD=37.53) before treatment and 93.62 (SD=25.69) after treatment. Patients with esophageal, gastric, and colon cancer had low iron levels before and after therapy compared to the healthy control group. The average TIBC level in healthy control participants was 312.76 (SD=38.52). Patients with esophageal cancer had mean values of 333.24 (SD=57.42) before treatment and 337.62 (SD=68.48) after treatment. Patients with gastric cancer had mean values of 333.8 (SD=63.93) before treatment and 331.06 (SD=52.02) after treatment. Patients with colon cancer had mean values of 333.94 (SD=64.87) before treatment and 331.7 (SD=53.88) In comparison to the healthy control group, patients with esophageal, gastric and colon cancer exhibited higher TIBC levels both before and after treatment.

DISCUSSION

Our results were supported by studies by Gabay C et al.,[9]who observed that ESR is an indirect estimate of acute phase protein concentrations and is a sensitive but nonspecific index of plasma protein changes that result from inflammation or tissue damage. ESR is concerned with two things: firstly it indicates some type of infection in the human body and secondly it indicates some type of inflammatory condition. ESR levels are elevated in medical conditions such as severe infection and inflammation going on in the body[10].White blood zcell (WBC) count, or the measurement of white blood cells in the blood, is a reliable and widely used marker that reflects inflammation throughout the body. Various evidence suggested that inflammation is correlated with the development and progression of cancer, and individuals in the highest quartile of WBC count had an increased risk of death from cancer. The association provided an essential relationship between inflammation and cancer mortality, local inflammatory processes that have long been known to be associated with tumor progression may be reflected in the systemic inflammatory marker of higher WBC count[11].Shruti Singh etal.,[12]also found a significant difference in TLC count of oral cancer and oral pre-cancer patients when compared to normal healthy control. Xiaoying Zhou etal.[13]stated that hematological parameters of monocyte-to-lymphocyte ratio (MLR), and platelet distribution width (PDW) can be

employed as an adjuvant tool for the diagnosis of early esophageal cancer (EEC). Moreover, the value of MLR can represent the invasion depth index. If patients have a high pre-treatment or post treatment platelet count, they might require more aggressive treatment in the future. Poor prognosis with shorter overall survival (OS), time to progressive disease (TPD), and time to metastasis (TM) were observed in nonmetastatic esophageal cancer patients with pre- and post-concurrent chemoradiotherapy (CCRT), high platelet counts (>300,000/μL), particularly at after CCRT, platelet-to-lymphocyte ratio, and neutrophil-to-lymphocyte ratio as well as low lymphocyte percentage[14]. Hematologic test results regarding the baseline host status and hepatic function were related to the response to neoadjuvant chemotherapy (NACT). Hematologic test results can reflect the body status and provide information regarding the potential response to NACT which is a very significant finding[15]. Noriyuki Hirahara etal., [16]stated it is now widely recognized that outcomes in cancer patients are not determined by their tumor features alone. On analyses of the clinical data of esophageal cancer patients to determine the effect of red blood cell distribution width (RDW), platelet distribution width (PDW), and mean platelet volume (MPV) on cancer-specific survival (CSS) it was confirmed that a high RDW was significantly associated with the CSS of esophageal cancer patients after curative esophagectomy. Furthermore, in non-elderly patients, a high RDW was also a significant and independent predictor of poor survival. Fevzi Coşkun Sokmen etal.,[17]reported there was a significant decrease in WBC, Hgb, MCV and PLT levels after adjuvant chemotherapy and this decline was unaffected by metastasis, duration of chemotherapy and blood transfusions. It was also observed that MCV levels decreased significantly after adjuvant chemotherapy, and this decrease was found to be the same in patients with and without metastasis, and in those with and without blood transfusion. In conclusion, chemotherapy-induced changes in complete blood count values are obvious and should be taken into consideration by clinicians. A significant decrease in serum iron concentration and a significant increase in total iron binding capacity was observed in cancer patients as compared to healthy control subjects. Our findings were in accordance with study of Shakhawat Hossain et al., [18]in which they found low serum iron levels ($p<0.05$) significantly increase the risk of oral malignancy. Iron deficiency and anemia, both increase oxidative stress and DNA damage, which might increase carcinogenesis risk, especially in the gastrointestinal (GI) tract. Based on the clinical, epidemiological, and experimental evidence, iron deficiency may increase the risk of developing cancer through the impairment of several iron-dependent metabolic functions that are related to genome protection and maintenance (e.g., immune responses against cancer-initiating cells, metabolism of toxic compounds, and redox regulation of DNA biosynthesis and repair)[19]. Anemia can put a strain or already weakened cancer patient. Cardiac output must rise to maintain adequate oxygen delivery as anemia worsens and symptoms develop. Anemia is also associated with a poorer overall prognosis [20].Hiroshi Sawayama, et al.,[21] stated patients with gastric cancer (GC) are affected by changes in iron status. Before surgery, gastric cancer patients are likely to have iron-defciency anemia and after gastrectomy, patients suffer from low nutritional status, low iron and low vitamin B12. Preoperative Hgb and TIBC were significantly associated with disease free survival (DFS) and overall survival (OS). TIBC, but not Hgb, was independently associated with DFS and OS. Low TIBC was associated with poorer prognosis following curative resection for GC. Overall, the findings indicated preoperative serum TIBC levels of patients undergoing curative gastrectomies for GC is a novel prognostic marker in univariate and multivariate analysis.

A significant increase in calcium levels are seen in our results in cancer patients as compared to healthy control subjects. Our results were consistent with those of Bower M et al.,[22]who observed hypercalcemia in majority of cancer patients. Hypercalcemia is the most common life-threatening complication of cancer occurring in 10% to 20% of adults and rarely in children, cancer related hypercalcemia is the leading cause of hypercalcemia in hospitalised patients. The main pathogenesis of hypercalcemia in malignancy is increased osteoclastic bone resorption, which can happen with or without bone metastasis. The enhanced bone resorption is secondary to different humoral factors that alter calcium regulation and are released by tumor cells locally (at the site of metastatic bone lesions) or systemically. The primary humoral component connected with cancer-related hypercalcemia is parathyroid hormone-related protein, which is produced by many solid tumors. Parathyroid hormone-related protein increases calcium by activating parathyroid hormone receptors in tissue, which causes osteoclastic bone resorption, and increases renal tubular resorption of calcium, cancer cells cause the kidneys to return calcium to the blood after filtering it, rather than excreting the extra calcium out of the body in the urine[23]. Marina Nogueira Silveira et al.,[24]also showed the use of corrected calcium levels as a biomarker of colorectal cancer prognosis holds promise for better

understanding the mechanisms behind the aggressiveness of colorectal cancer. Elevation of intracellular Ca^{2+} can improve the proliferation, migration, invasion, metastasis, and drug resistance of colorectal cancer cells, which are often associated with the high expression and aberrant activation of calcium channel proteins in cells. However, when intracellular Ca^{2+} rises to a certain level (Ca^{2+} overload), it will cause depolarization, apoptosis, or some other biological processes of cells, which ultimately results in the death of cancer cell[25]. Ling Wu, et al.,[26] supported that researches conducted during the past few decades have updated our knowledge of significance and mechanisms of calcium signaling. The core role of Ca^{2+} homeostasis in human diseases especially cancer has been sustained by mounting evidence from experimental and clinical data, including multiple examples like those mentioned above. Therefore, calcium signaling is a possible target for the development of novel anticancer therapies. Last but not least, it should be considered how changes in calcium signalling impact downstream responses. For instance, while some channels that are overexpressed in cancer cells may be viable targets, they might also be required for other chemotherapeutics. Therefore, it is important to prioritise taking drug-drug interactions into account in clinical practice.

CONCLUSION

There was an increase in ESR and WBC count and a decrease in Hb, TRBC, and Platelets count found in our results in gastrointestinal cancer patients as compared to healthy control subjects. A significant increase in calcium levels is seen in our results in cancer patients as compared to healthy control subjects. Compared to healthy control subjects, cancer patients had a significantly lower serum iron concentration and a significantly higher total iron binding capacity. It is recommended that as with any putative prognostic discriminant, it is necessary to distinguish between the ability to provide practical therapeutic guidelines, and the ability to enhance the biological understanding of the disease. The clinical implications of the findings must be placed into perspective, particularly when examining end results in adjuvant therapy.

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TABLES:

Table 1: Comparison of Hematological parameters, serum Calcium, Iron & TIBC of healthy control subjects and Esophagus cancer patients

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S. No.	Variable	Control (n=50) Mean±SD (Range)	Esophagus cancer (n=50) Mean±SD (Range)					
			BT	*		**AT**	#	
				't'	**p**		**'t'**	**p**
1.	ESR(mm in 1st hour)	12.70±5.82 (5-25)	40.10±30.58 (5-110)	6.223	0.000	17.30±7.36 (10-45)	3.463	0.000
2.	Hb (gm/dl)	13.02±1.55 (10.5-14.9)	9.92±2.01 (6.1-12.9)	8.622	0.000	10.15±1.98 (7.6-13.9)	8.048	0.000
3.	TRBC(mill/ cumm)	5.12±0.78 (4-6.3)	4.18±0.95 (2.4-5.9)	5.378	0.000	3.98±0.74 (2.2-6.0)	7.502	0.000
4.	WBC(1000 /cumm)	6.67±1.40 (4.6-9.2)	8.71±3.43 (1.7-16.8)	3.886	0.000	5.86±1.32 (3.8-7.9)	2.961	0.003
5.	Platelets (lakhs/ml)	2.91±0.69 (1.8-4.4)	2.56±1.04 (1.1-5.91)	1.984	0.050	2.42±1.01 (1.0-4.47)	2.781	0.006
6.	Calcium (mg/dl)	8.31±0.72 (7.1-9.9)	8.87±1.00 (7.4-12)	3.189	0.001	8.83±0.90 (7.1-12.3)	3.160	0.002
7.	Iron (µg/dl)	105.38±28.89 (52-160)	64.66±37.34 (20-157)	6.097	0.000	93.88±25.93 (54-169)	2.094	0.038
8.	TIBC (µg/dl)	312.76±38.52 (212-435)	333.24±57.42 (245-440)	2.094	0.038	337.62±68.48 (241-489)	2.237	0.027

compared before therapy for patients and healthy control group
compared after therapy for patients and healthy control group
BT-Before Therapy, AT-After Therapy, ESR-Erythrocyte Sedimentation Rate, Hb- Hemoglobin, TRBC-Total Red Blood Cells, WBC- White Blood Cells, TIBC-Total Iron Binding Capacity

Table 2: Comparison of Hematological parameters, serum Calcium, Iron & TIBC of healthy control subjects and Gastric cancer patients

S. No.	Variable	Control (n=50) Mean±SD (Range)	Gastric cancer (n=50) Mean±SD (Range)					
			BT	*		AT	#	
				't'	p		't'	p
1.	ESR(mm in 1st hour)	12.70±5.82 (5-25)	29.90±22.84 (10-105)	5.518	0.000	17.40±8.64 (10-55)	3.188	0.001
2.	Hb (gm/dl)	13.02±1.55 (10.5-14.9)	9.22±1.71 (6.5-13.0)	11.609	0.000	10.37±1.43 (8.3-13.3)	8.878	0.000
3.	TRBC(mill/ cumm)	5.12±0.78 (4-6.3)	3.83±0.76 (1.9-5.2)	8.317	0.000	3.78±0.82 (2.1-5.1)	8.280	0.000
4.	WBC(1000 /cumm)	6.67±1.40 (4.6-9.2)	8.48±3.42 (4.1-17.8)	3.455	0.000	5.95±1.40 (3.7-8.2)	2.544	0.012
5.	Platelets (lakhs/ml)	2.91±0.69 (1.8-4.4)	2.61±1.04 (1.12-4.99)	1.700	0.092	2.52±0.81 (1.11-4.40)	2.552	0.012
6.	Calcium (mg/dl)	8.31±0.72 (7.1-9.9)	8.84±0.86 (7.8-12)	3.352	0.001	8.89±0.98 (7.8-12.0)	3.377	0.001
7.	Iron (µg/dl)	105.38±28.89 (52-160)	77.12±38.13 (28-161)	4.176	0.000	92.68±25.30 (50-160)	2.337	0.021
8.	TIBC (µg/dl)	312.76±38.52 (212-435)	333.8±63.93 (213-489)	1.993	0.049	331.06±52.02 (250-421)	1.998	0.048

*Compared before therapy for patients and healthy control group
compared after therapy for patients and healthy control group
BT-Before Therapy, AT-After Therapy, ESR-Erythrocyte Sedimentation Rate, Hb- Hemoglobin, TRBC-Total Red Blood Cells, WBC- White Blood Cells, TIBC-Total Iron Binding Capacity

Table 3: Comparison of Hematological parameters, serum Calcium, Iron & TIBC of healthy control subjects and Colon cancer patients

S. No.	Variable	Control (n=50) Mean±SD (Range)	Colon cancer (n=50) Mean±SD (Range)					
			BT	*		AT	#	
				't'	p		't'	p
1.	ESR(mm in 1st hour)	12.70±5.82 (5-25)	30.70±17.55 (5-80)	6.881	0.000	15.80±6.95 (10-50)	2.417	0.017
2.	Hb (gm/dl)	13.02±1.55 (10.5-14.9)	9.62±1.73 (6.3-12.7)	10.318	0.000	10.31±1.46 (8.3-13.1)	8.967	0.000
3.	TRBC(mill/ cumm)	5.12±0.78 (4-6.3)	3.79±0.74 (2.2-5.3)	8.679	0.000	3.69±0.74 (2.6-5.2)	9.353	0.000
4.	WBC(1000 /cumm)	6.67±1.40 (4.6-9.2)	7.81±2.95 (4.5-18.6)	2.460	0.015	5.91±1.28 (3.5-7.9)	2.836	0.005
5.	Platelets (lakhs/ml)	2.91±0.69 (1.8-4.4)	2.59±0.96 (1.18-4.56)	1.898	0.060	2.55±0.89 (1.0-4.5)	2.237	0.027
6.	Calcium (mg/dl)	8.31±0.72 (7.1-9.9)	8.82±0.83 (7.2-12.1)	3.308	0.001	8.89±0.97 (7.9-12.3)	3.411	0.000
7.	Iron (µg/dl)	105.38±28.89 (52-160)	71.94±37.53 (25-169)	4.991	0.000	93.62±25.69 (54-169)	2.150	0.034
8.	TIBC (µg/dl)	312.76±38.52 (212-435)	334.94±64.87 (241-481)	2.078	0.040	331.7±53.88 (248-433)	2.021	0.045

*Compared before therapy for patients and healthy control group

compared after therapy for patients and healthy control group

BT-Before Therapy, AT-After Therapy, ESR-Erythrocyte Sedimentation Rate, Hb- Hemoglobin, TRBC-Total Red Blood Cells, WBC- White Blood Cells, TIBC-Total Iron Binding Capacity