RESULTS

The serum carcinoembryonic antigen (CEA) levels in group A (NMSC) were averging from 1.8 to 3.96ng/ml and its mean and S.D were 3.07 and 0.74 (table 2).

The serum CEA levels in group B (the control group) were averging from 0.31 to 4.1 ng/ml and its mean and S.D were 1.35 and 0.92 (table 3).

There was a significant difference in serum CEA level between the NMSC group and the control group. As, the correlation between the serum CEA level in the control and the NMSC groups showed these values T = 5.43 and P = <0.05 (Figure 3).

In nonmelanoma skin cancer group, the mean level of CEA in cytosol was 32.93 ($\pm$ 8.86), while the mean level of CEA in the membrane was 25.86 ($\pm$ 11.81) (table 4).

Correlation between the mean serum CEA levels in NMSC group and the control group was demonstrated (table 5). Correlation between the mean CEA levels in the cytosol and membrane of NMSC patients was demonstrated (table 6).

Table (2): Serum CEA in nonmelanoma skin cancer group (Group A).

No.	Sex	Age	Type of cancer	Serum ng/ml
1	M	82	BCCs	1.8
2	M	62	BCCs	3.96
3	M	59	SCC	3.3
4	M	78	SCC	3.8
5	F	65	SCC	2.03
6	M	60	BCCs	3.0
7	M	45	Basal squamous cell carcinoma	3.6
8	F	43	BCCs	3.1

$\bar{X}$ = 3.07 S.D. = 0.74 S.E. = 0.26

Table (3):Serum CEA level in the control group (Group B).

No.	Sex	Age	Serum ng/ml
1	M	28	3.5
2	M	28	1.6
3	F	22	0.4
4	F	23	1.5
5	F	24	1.8
6	F	30	1.3
7	F	25	0.6
8	F	25	4.1
9	F	26	2.5
10	M	27	0.5
11	M	33	0.6
12	M	32	0.53
13	M	38	1.32
14	M	23	0.31
15	M	26	1.2
16	M	27	0.4
17	M	29	0.8
18	M	30	1.35
19	F	32	1.65
20	F	34	2.01
21	M	28	1.32
22	F	23	1.75
23	M	22	0.89
24	F	25	0.95
25	F	30	1.12

$\bar{X}$ = 1.35 S.D = 0.92 S.E = 0.18

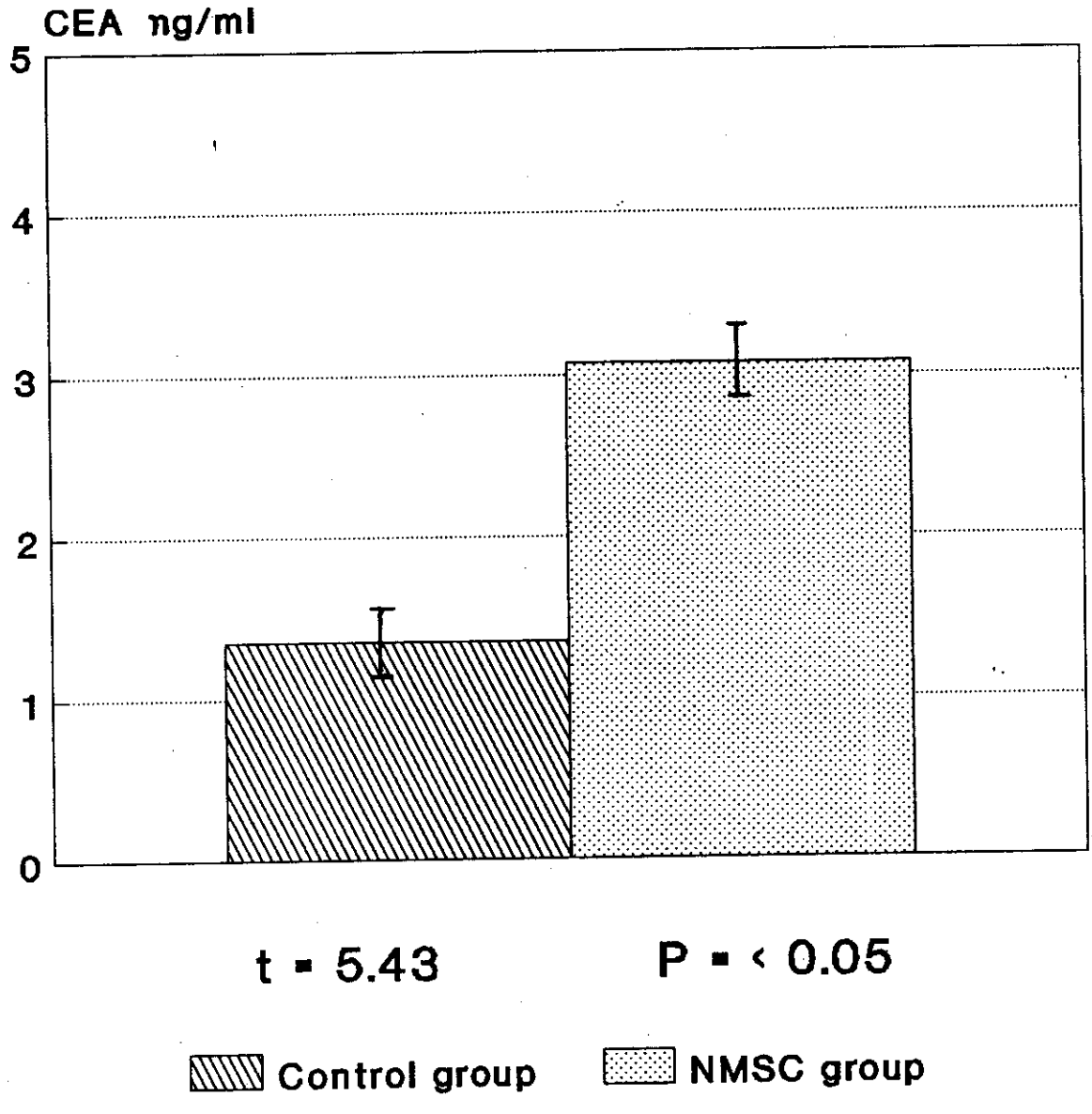

Fig.(3):Comparison between serum CEA ($\bar{X}\pm$S.E) in the control group and in NMSC group.

Table (4): CEA levels in cytosol and membrane fractions in nonmelanoma skin cancer group.

No.	Sex	Age	Type of cancer	Cytosol ng/ml protein	Membrane ng/mg protein
1	M	82	BCCs	29.8	6.7
2	M	62	BCCs	18.5	31.4
3	M	59	SCC	95.9	110.7
4	M	78	SCC	41.5	26.3
5	F	65	SCC	21.6	11.5
6	M	60	BCCs	27.8	11.4
7	M	45	Basal squamous cell carcinoma	12.9	6.4
8	F	93	BCCs	16.2	2.5
				$\bar{X}$ = 32.93	25.86
				S.D.= 25.06	33.42
				S.E.= 8.86	11.81

Table (5): Correlation of the mean serum CEA levels in group of nonmelanoma skin cancer and control group.

Group	($\bar{X}$ ± S.E) ng/ml
Serum NMSC	3.07 ± 0.26
Serum control	1.35 ± 0.18

* Significant difference from control serum.

Table (6): Correlation of the mean levels of CEA in cytosol and membrane of nonmelanoma skin cancer patients.

Group	($\bar{X}$ ±S.E) ng/ml
CEA level in cytosol of NMSC	32.93 ± 8.86
CEA level in membrane of NMSC	25.86 ± 11.81

DISCUSSION

Squamous and Basal cell carcinoma are commonly grouped together and referred to as nonmelanoma skin cancer, because both carcinomas share an origin from the epidermal cell and many common features of the epidemiology and carcinogensis (Harely and Haynes, 1984).

The measure of malignancy of SCC may be judged by its local aggressiveness and ability to metastasize (Moller et al., 1979).

Basal cell carcinoma although it is rarely metastasize, it is capable of significant local destruction and disfigurement (Miller, 1991).

Squamous cell carcinoma is prevalent in geographic areas of maximal sun exposure and in the population group which is susceptible to ultraviolet light damage such as the fair-skinned races. The incidence of the tumor increases after the age of 55 years, males are more commonly affected (Vasarinsh, 1982).

Basal cell carcinoma is extremely uncommon in dark skinned races, and less common in chinese, japanese and other mongoloids than in caucasoids. Although it may occur at any age from chilhood, more than three quarters of patients are over 40 year old (Mackie, 1986). The Bccs are the most common

malignant disease that human beings acquire in the course of a life time (Giles et al., 1988).

The clinical presentation of SCC makes clinical diagnosis much more difficult than it is in the BCC (Harley and Haynes, 1984).

The tumors of the skin are the most commonly encountered neoplasms in clinical medicine.Despite this high frequency, the microscopic diversity of these lesions sometimes makes their histopathologic diagnosis difficult and necessitates the use of other specialized studied as electron microscopy or histochemistry. Untill recently, cumulative data on the immunophenotypes of nonmelanoma skin cancer were relatively sparce. However, sufficient informations are now available to make immunohistochemistry a viable approach to make differential diagnosis for this types of tumors (Mark et al., 1988), thus the classical methods for diagnosis which include, clinical presentation and histological picture are in need of other methods of diagnosis which focused on the ultrastructural, biochemical, molecular, genetic, and immunologic factors that underlie the clinical aspects of this tumor (Miller, 1991).

Yet, there is a need for new methods other than clinical examinations, ultrasonography, computerized tomography, and angiographic techniques for diagnosis, follow up,proper stag-

ing, detection of micrometastasis and predicting prognosis. The developing of monoclonal antibodies have allowed the production of numerous new reagents specific for a variety of antigens including those expressed by epithelium (Parker, 1981).

Tumor markers are the biochemical or immunological counterpart of the morphology of the tumors (Sell, 1990).

Tumor markers, which are substances that can be related to the presence or progress of neoplasms, were studied comprehensively as regards the application of their serum level in mass screening, differential diagnosis, detection of micrometastasis and monitoring response to therapy.Unfortunately, measurement of serum level of different biomarkers has proved to be neither specific nor sensitive for detection of cancer (Gomaa et al., 1990).

Recently, the advent of specific immunocytochemical techniques has enabled the demonstration of some of the relevant antigens within histological sections of conventionally processed material. Several studies had shown possible demonstration of CEA in the sections of some lung tumors (Harach et al., 1983).

There are only a very few tumor markers that exhibit sufficient specificity and sensitivity to enable them to be used for screening. A rare example is plasma calcitonin. The best immunoassays demonstrate elevated plasma calcitonin

concentrations in all subjects with medullary carcinoma of the thyroid and basal and/or stimulated concentrations of calcitonin may be used to predict early tumor growth in close family relatives and, where appropriate, allow prophylactic thyroidectomy (Beastall et al., 1991).

Tumor markers have been shown to be of value in the differential diagnosis of benign and malignant diseases as in tuberculosis and bronchial carcinoma (Khalifa et al., 1988) and in non malignant and malignant pleural effusion (El-Ahmady et al., 1988).

Tumor markers to be of value in prognosis, the tumor marker concentration should correlate closely with tumor size and/or activity, so that a modest elevation means a small localized tumor, whilst a greater elevation suggests bulky disseminated malignancy or an aggressive tumor (Beastall et al., 1991).

Serial determinations of tumor marker are important in the follow up of patients who have radiotherapy to detect incomplete resection, recurrence or micrometastasis (Sell, 1990).

Squamous cell carcinoma demonstrates cytokeratin expression over a wide range of a molecular weights, from 40 to 68 kd (Varnaar et al., 1984).

Spindle cell and pleomorphic carcinoma may co-express cytokeratin and viementin in the same neoplastic cells (Zarbo et al., 1986). Another determinant can be found and reflect the level of differentiation in SCC e.g. EMA. All SCC lack CEA (Mark et al., 1988).

Basal cell carcinoma demonstrates intermediate filaments keratin,fibronectin and other antigens such as,hyaluronectin, tenasin, transferrin receptors,EMA,lectin,Pemphigus antigen, involucrin skin calcium-binding protein and lactoperoxidase. CEA is present only in area of keratinization in BCC (Miller, 1991).

The CEA was originally described by Gold and Freedman in (1965) as an antigen present in gasterointestinal adenocarcinoma and in embryonic entodermal tissue of man, later on, it was found in a variety of non intestinal tumors and even in normal adult tissues in low concentration (Milgrom et al., 1981).

Elevated CEA levels are found in non tumor conditions e.g.cigarette smokers (Steven and Mackay, 1973), in pulmonary infections (included tuberculosis), alcholic liver disease, pancreatitis, recent blood transfusion, ulcerative colitis, gasteritis following gasterectomy and colonic polyp (Steven and Macky, 1973),it is also elevated in diseases accompanied by abnormal mucous production such as chronic bronchitis (Hansen et al.,1974) and cystic fibrosis (Wu et al., 1976).

CEA is elevated in cases with cancer condition such as colorectal cancer and it is highly suggestive to diagnose it (Sell, 1990), in patients with breast cancer, elevation of CEA are strongely correlated with metastasis. Determination of CEA level is also very useful in lung cancer as an aid in diagnosis, prognosis and follow up patients.Elevations of CEA in pleural fluids indicate malignant effusion (Sell, 1990).

In colonic cancer,combination of CEA with tumor-associated trypsin inhibitor antigen gives a better specificity for cancer than any other combination of CEA with other Markers (yedema et al., 1987).

A significant number of patients with SCC of cervix and valva were found to have elevated plasma level of CEA (Van nagell et al., 1975).

Untill now there is a lack in the researches to use the tumor markers as a diagnostic, prognostic or even detecting micrometastasis in skin cancer. Our research used CEA as a tumor marker in serum and cancer tissues of patient with NMSC The present results revealed that the mean serum CEA level in NMSC patients was (3.07 ± 0.26). It was significantly higher than that of the mean CEA level in serum controls (1.35 ± 0.18).

However, this difference is located within the normal range of serum CEA (0 - 5 mg/ml) (Sell, 1990).

This high level in NMSC patients may be attributed to the presence of CEA in cytosol and membrane of the cancer tissues.

However, this may be also due to area of keratinization in BCCs as reported by Miller et al. (1991) who said that CEA can be demonstrated in BCC only in area of keratinization.

On the other hand, the higher level of CEA in serum and it's level in cancer tissues of SCC is not in agreement with the research of Mark and his Colleagues (1988) who reported that all SCC lack CEA. This may be attributed to the long duration between the appearance of the tumors and the coming of the patients to seek the medical advice.

SUMMARY AND CONCLUSION

The present study was performed on 33 individuals categorized in two groups, group (A) which include 8 patients of NMSC (BCC, SCC, basal squamous type) and group (B) which include 25 individuals of apparently healthy persons.

This study included measurement of CEA level in serum of NMSC patients, in the tissues of NMSC (cytosol and membrane), and also in the serum of normal control group using enzyme immunoassay.

The findings in this study could be summarized as :

1- The range of CEA level in serum of normal patients were within normal range.
2- There is a significant increase in the level of CEA in serum of patients with NMSC in contrast to it's level in serum of control group.

In conclusion we recommend for future research in this topic to compare the CEA level in the NMSC tissues with its level in normal tissues. Also, more researchs must be done for follow up the patients after operation to see the importance of tumor marker in predicting the recurrence of the disease or micrometastasis before the clinical symptoms of the disease.