RESEARCH ARTICLE

A Comparative Evaluation of Calcitonin and Carcinoembryonic Antigen Levels with Chemotherapy for Gastrointestinal Cancer Patients in Kerbala Governorate

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ABSTRACT

Background: Carcinoma is now identified as one of the extremely important medical troubles defiance the population. For this reason, we were encouraged to investigate the causes of the spread of cancer in the human body. This study included 100 patients with gastric (50%) and colon (50%) Cancer. 50 of those cancer's patients were on chemotherapy, and 50 of them were without chemotherapy.

Results: The findings pretence a highly important result ($p < 0.01$) augmentation in the levels of carcinoembryonic antigen (CEA) and calcitonin as a tumor markers in patients on gastrointestinal (GIT) cancer. In our study, patients on GIT cancer were classified into two groups, group 1-patient were contend with chemotherapy (65 patients), and group 2, patients were not contended by chemotherapy (35 patients). The findings showed a highly significant ($p < 0.01$) rise in the rank of calcitonin and CEA in chemotherapy-treated patients relative to non-chemotherapy-treated patients.

Conclusion: Gastric and colon cancers are associated with elevated CEA and Calcitonin values. Chemotherapy is not enough drugs to treat cancer, and it is causing to metastasis the cancer when used without radiotherapy. Therefore must use a dependent-dose of radiotherapy with it to save the bone. Calcitonin is a cancer inhibitor in a gastric cancer through inhibits a gastric acid.

Keywords: Calcitonin, CEA, Chemotherapy, Radiotherapy.

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INTRODUCTION

Carcinoembryonic antigen (CEA) is a chemical material detected on some cell's surfaces.[1] CEA is a glycoprotein secreted by gastrointestinal tract cells through embryonic stages of development.[2] CEA is secreted in very little amounts postpartum. In the bloodstream, the levels of CEA are decreased relatively unless assured diseases, including clear shapes of cancer are existing.[3]

Carcinoembryonic antigen (CEA) was a most frequent test in blood. It can also be diagnosed in body fluids and in biopsy tissues.[4]

The preferable use of CEA was as a tumor marker, particularly to gastrointestinal tract cancers.[5] When the CEA level was highly disordered before cancerous organ resection or other management, it may be decreased to normal, and this following the surgery was successful in resecting all of the cancer.[2] The highly significant CEA level was documented cancer recurrences or cancer progression. These must be assured, as the CEA exam by itself was not specific enough to prove a recurrence of a cancer.[6] Additionally, levels >20 ng/mL before therapy may be correlated with cancer, which had already spread (metastatic disease).[7]

Together benign (not harmful) and malignant (cancerous) patients were able to rise up the CEA levels. Generality repeated cancer which led to an increased CEA is rectum cancer and colon cancer.[8] Pancreas, breast, lung, gastric and medullary cancer which others included cancers of the thyroid and ovarian carcinoma. Benign tumor patients were can increased CEA levels include infections, smoking, pancreatitis, inflammatory bowel diseases, liver cirrhosis, and other benign tumors in the same organs that increased CEA suggest cancer.[9] Chemotherapy drug and radiotherapy able led to a tentative increasing in CEA levels attributed to death or destroy the tumor cells, then releasing of CEA into the

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blood vessels.[10] The CEA levels, which changed through the management of cancer usually must be associated with other clinical results. But the disturbing, when the CEA level changes were not diagnosed on the cancer progression.[11]

Calcitonin was a hormone which secreted from the thyroid gland by C cells absolutly[5]. The location of the thyroid gland was inside the lower neck frontally of a human. Calcitonin was responsible for calcium metabolism, the destruction (catabolism), and rebuilding (anabolism) of bone.[12]

The secretion of this calcitonin hormone was regulated directly by the calcium level of the blood[6]. As calcitonin levels begin to increase, the body responds with elevated calcitonin levels. When calcium levels have decreased, calcitonin levels[12] also decrease.

MATERIALS AND METHODS

Patients and Control

Between October 2019 and April 2020, 100 patients with GIT cancer, 50 patients with colon cancer and 50 patients with gastric cancer aged between 30 and 50 years were taken from Al-Hussein Teaching Hospital, Kerbala and diagnosed with oncologists in the same hospital.

The control group was included of 100 controlled people who weren't suffered from any signs or any symptoms of cancer, paired with patients of age who had no history of stomach and colon problems.

Specimen Collection

Five milliliters (5 mL) of venous blood were obtained from patients and monitored early in the morning after a fast night. The specimen were centrifuged at 3000 xg for 15 minutes, and the serum was separated and stored at-20 until analysis.

Determination of CEA

Serum CEA and Calcitonin were identified using the enzyme-linked immunosorbent assay (ELISA) (Human CEA, Calcitonin ELISA package, CSB, CUSABIO, China).

Table 1: The levels of parameters under study in patients on GIT cancer compared with control group.

Patients	*Carcinoembryonic antigen (CEA) (ng/mL)*	*Calcitonin (ng/mL)*
Gastric cancer Patients n=50 Mean ± Sd.E	83.67 ± 15.67	269.03 ± 69.08
Control n=50 Mean ± Sd.E	2.10 ± 0.141	5.06 ± 0.27
P-value	0.000	0.001
Colon cancer Patients n=50 Mean ± Sd.E	9.41 ± 0.09	4.95 ± 0.176
Control n=50 Mean ± Sd.E	8.46 ± 0.05	3.71 ± 0.09
P-value	0.001	0.000

Statistical Analysis

Versions 26 of the Statistical Package for Social Sciences (SPSS) were applied to analysis of statistical results. The student's t-test was applied to assess the findings. All the facts were manifested as a mean ± standard error (Sd. E). p-value ≤ 0.05 was considered to be important.

RESULTS AND DISCUSSION

The findings appeared a very significant ($p < 0.01$) rise in CEA levels and then a very significant ($p < 0.01$) rise in calcitonin rank in patients with gastric and colon cancer relative to healthy subjects (Table 1).

In an antecedent investigation, Cheryl *et al*. (2006) identified the CEA as a fabulous tumor marker for gastric and colon cancer and was beneficial for precocious demonstration to results, staging, and patient monitoring after ultimate treatment.[13] The CEA as a tumor diagnostic will have a high sensitivity, specificity and positive predictive values to the comparing between patients with benign gastric, colon tumor, and patients with gastric, colon cancer.[14]

In our study, patients with gastric and colon cancer (GIT cancer) were classified into two groups, and group 1 included 65 patients treated with chemotherapy drug, and group 2 included 35 patients who had not been treated with chemotherapy medications. The findings showed a very substantial rise ($p < 0.01$) in CEA and calcitonin levels in patients with gastric and colon cancer that were managed with chemotherapy relative to those who were not treated with chemotherapy (Table 2).

The findings showed a positive correlation between CEA and calcitonin ($r = 0.577$) in patients with colon cancer, but there was a negative correlation between CEA and calcitonin ($r = -0.600$) in patients with gastric cancer when we using the person's correlation coefficient (r) (Table 3).

Although most of the patients were taking chemotherapy, but it did not contribute to the return of the normal level, CEA and calcitonin, that's mean chemotherapy did not show any interest in cancer treatment.[15] The primary aim of chemotherapy was to destroy the cancerous cells and stop carcinoma cells from dividing and from rising, but to see if the drug works.[16]

Increasing CEA during hormone therapy does not indicate that patients are out of the range of choices. This suggests that patients ought to explore the use of other systemic treatments,

Table 2: The degree of parameters under study in GIT cancer patients with and without chemotherapy.

Parameters	*Without chemotherapy drug n = 35*	*With chemotherapy drug n = 65*	*p-value*
	Mean ± Sd.E	*Mean ± Sd.E*	
Carcinoembryonic antigen (CEA) (ng/mL)	18.37 ± 3.35	95.40 ± 17.86	0.000
Calcitonin (ng/mL)	8.80 ± 0.11	9.52 ± 0.097	0.004

Table 3: The degree of correlation between parameters in gastric and colon carcinoma patients.

Parameter	*Parameter*	*Type of Cancer*	*n*	*(r)*	*P-value*
CEA	Calcitonin	Gastric CA.	50	- 0.600	0.781
CEA	Calcitonin	Colon CA.	50	0.577**	0.003

* * The correlation is important at the stage of 0.01.

like chemotherapy, alternative hormone medicines, or factors that target the spread of gastric or colon carcinoma (metastasis).[17]

In beforehand studies, Banfi *et al*. (2001) assessed bone mineral denseness in many patients and reported a lot of chemotherapy dose, leading to a 10% destruction in cortical bone and a 20% loss in trabecular bone and a rise in bone resorption rates.[18] These have been due to non-dose-dependent radiation chemotherapy, which demonstrates toxicity to bone marrow stromal osteoprogenitors and may trigger osteopenia through manage harm to the osteoblast cubicle as a mechanism perspicuous from and condensable to hypogonadism.[19]

Sena *et al*. (2018) showed a strong association with an increased stage of cancer (>377 ng/mL) when preoperative CEA rank were >500 ng/mL. A highly significant variation of preoperative CEA levels was significantly correlated with tumor size and point, distant metastases, decline biochemical remedy, and mortality.[20] Also, in our results, there was a negative correlation between CEA and calcitonin in gastric cancer patients compared to colon cancer patients; G. Wells *et al*. (2016) revealed calcitonin was a cancer inhibitor in gastric carcinomas, during inhibits gastric acid.[21]

CONCLUSION

Carcinoembryonic antigen (CEA) and calcitonin can be clinically useful markers for metastatic bone cancer and for treatment and monitoring. Gastric and colon cancers are associated with elevated CEA and Calcitonin values. Chemotherapy has not enough drug to treat cancers. Therefore, one must use a dependent-dose of radiotherapy to save the bone and bone marrow. Calcitonin is a cancer inhibitor in gastric cancer as it inhibits gastric acid.

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