

Metabolic Correction Therapy as Adjuvant Treatment for Breast Cancer Patients: A Case Report

Marilís Navarrete¹, Jomaris Centeno¹, Michael Gonzalez^{2,*}, Miguel J. Berdiel³

¹ San Juan Bautista School of Medicine, Caguas, Puerto Rico

² University of Puerto Rico, Medical Sciences Campus, School of Public Health, San Juan, Puerto Rico

³ Berdiel Clinic, Ponce, Puerto Rico

Abstract

Breast cancer is the most common cancer in women worldwide. In the United States, is the second leading cause of cancer deaths in women. In Puerto Rico between 2008 and 2012, breast cancer was the most diagnosed type of cancer and the leading cause of cancer deaths among women. This is a case of 54 years old female diagnosed with stage IV right breast carcinoma. The patient complaints were weakness and a right breast ulcer. She started a metabolic correction therapy, which consisted of high intravenous vitamin C infusions, a nutritional supplementation plan, and Paleolithic diet. During treatment, both Glycohemoglobin and Carcinoembryonic Antigen levels decreased significantly, the right breast ulcer decreased in size, and the patient's quality of life improved. Over the years, vitamin C studies have demonstrated a cytotoxic action against malignant cells. Based on the results from this case, we advocate continue studying possible adjuvant treatments for cancer patients, involving IV infusions of vitamin C and metabolic correction plans.

Corresponding Author : Michael Gonzalez, University of Puerto Rico, Medical Sciences Campus, School of Public Health, San Juan, Puerto Rico, Email: michael.gonzalez5@upr.edu

Keywords : breast cancer

Received : Oct 28, 2017

Accepted : Jan 03, 2018

Published : Jan 13, 2018

Editor: Hassan Ebrahim, University of Louisiana at Monroe, 1800 Bienville drive-Monroe-Louisiana-71201-USA. Email: hebrahim@pharm.helwan.edu.eg

Introduction

Breast cancer is the most common cancer in women worldwide, according to the World Health Organization (WHO).¹ Excluding skin cancers, breast cancer is the most common cancer and the second leading cause of cancer deaths in women in the United States (U.S.).^{2,3} Based on 2010-2014 cases of breast cancer data from the National Cancer Institute in U.S., 124.9 per 100,000 women per year were new cases and 21.2 per 100,000 women died.⁴ More recently, the National Cancer Institute estimates 252,710 new breast cancer cases and 40,610 deaths in 2017.⁴ Breast cancer is most common in White and African American women between 55-64 years old.^{3,4} In Puerto Rico between 2008 and 2012, breast cancer was the most diagnosed type of cancer and the leading cause of cancer deaths among women.⁵

One important blood test in the diagnosis of cancer is the Carcinoembryonic Antigen (CEA) test. CEA is a glycoprotein used as a nonspecific tumor marker to determine cancer activity.⁶ Increased CEA levels have been detected in several cancers, such as breast cancer.⁶

After the imaging tests, blood tests, and biopsy, the patient will receive a diagnosis that will be categorized by stages. In the clinical setting, the TNM system is used.³ This system is based on the size of the breast tumor (T), whether the cancer has reached any nearby lymph node (N) and whether the cancer has metastasized (M).³ Then the TMN information is combined and the stages are classified by numbers (0, I, II, III, and IV).³ The lower the number, the less the cancer has spread. For example, stage 0 refers to carcinoma in situ and stage IV is the most advanced cancer.³ When the female breast cancer is discovered at stage I or some stage II (also called localized cancers) the patient has a better chance of survival.^{3,4} According to the National Cancer Institute, the 5-year survival for localized female breast cancer is 98.9%.⁴ Meanwhile, metastasized cancers (also called distant stage) have a 5-year survival rate of 6%.⁴

On one hand, breast cancer conventional treatments available are surgery, chemotherapy, radiation, hormonal, and/or targeted therapies.³ On the other hand, we propose a metabolic correction therapy as another available treatment tool for all cancer

patients. Metabolic correction explains how nutrients (vitamins and minerals), at the cellular level can prevent tissue damage and even revert disease thus preserving health by optimizing enzymatic function.⁷ In other words, metabolic correction explains how improving enzyme activity and efficiency will also improve cellular physiology and metabolism.⁷

The Bio-Energetic Theory of Carcinogenesis states that cancer is a disease of metabolism, due to damage to the mitochondria.⁸ This damage shifts the oxidative metabolism of the cell to a non-oxidative metabolism; in consequence, increasing the process of fermentation, which is characteristic of cancerous cells.⁸

In this case, metabolic correction therapy consisted of a low carbohydrate diet (Paleolithic diet), hyperbaric oxygen, and intravenous (IV) vitamin C. First, as the primary energy source of the cancerous cell is glucose, we recommend a low carb Paleolithic diet to starve the cancer cells of glucose.⁸ Second, the hyperbaric oxygen treatment enhances the amount of oxygen available and increases its delivery to the tissues.⁹ Cancer cells obtain its energy from glycolysis in a oxygen deprived environment, so the hyperbaric oxygen can alter tumor hypoxia by producing hyperoxia, which results in reactive oxygen species (ROS) that induce cellular damage.¹⁰ In addition to promoting the formation of hydrogen peroxide in malignant tissue.¹¹

Third, vitamin C or ascorbate is an essential vitamin for humans that even at high levels, is a nontoxic agent.¹¹ When vitamin C is administered intravenously at pharmacological doses, it exerts cytotoxic actions in cancer cells.¹² These actions may produce inhibition of malignant cell proliferation, decrease tumor growth, and increased cellular damage to malignant cells.¹¹ Therefore, metabolic correction therapy can improve the cellular metabolism and produce a better quality of life.

Case Report

Case of a 54 y/o female diagnosed with stage IV right breast carcinoma that was treated at the Berdiel Clinic in Ponce, PR. The patient arrived with no current treatment. Her complaints were weakness and a right breast ulcer of approximately five inches.

The patient initiated a metabolic correction plan, which involved pharmacological doses of Intravenous vitamin C. The vitamin C infusions in the first week were

15g two times a week. The second week, were 25g three times per week. The third week, were 50g three times per week. From the fourth week to three years later, the patient received infusions of 75g three times per week. The main anticancer mechanism for IV Vitamin C is the generation of hydrogen peroxide. Ascorbic acid converts to dehydroascorbic acid that in the presence of oxygen creates hydrogen peroxide. Hydrogen peroxide affects mainly the malignant cell because of their lack of antioxidant enzymes glutathione peroxidase, catalase and superoxide dismutase, while it does not affect the normal cells that produce enough quantities of these enzymes.

In addition, the metabolic correction therapy included a nutritional supplementation plan that consisted of Vitamin D3 10,000 IU qd, Mixed Phospholipids 100mg qd, B complex sublingual 1cc bid, Magnesium Citrate 500mg bid, R-alpha lipoic acid 300mg bid, Acetyl L Carnitine 500mg bid, Omega-3 1g tid, and CoQ10 100mg tid. Also the patient had implemented a Paleolithic diet that primarily consisted of vegetables, free-range poultry, wild-caught fish, and grass-fed meat. Beside that, the patient also could eat nuts and organic berries.

Before the metabolic correction therapy, in 2013, the glycohemoglobin (GHB) (N=4.80-5.90%) and CEA (N=0-3 ng/ml) results were 11% and 4.90 ng/ml, respectively. During the treatment, in 2014, the GHB and CEA were 5.00% and 2.60 ng/ml, respectively. Furthermore, the ulcer decreased in size to approximately 1.5 inches.

An important fact is that the patient's quality of life improved significantly. She was able to perform daily activities, since her weakness decreased and did not experience any secondary side effects. After three years, the patient decided to stop the treatment because she moved to another country.

Discussion

As stated by The Bio-Energetic Theory of Carcinogenesis, cancer is due to damage to the mitochondria.⁸ This damage shifts the metabolism of the cell from oxidative to non-oxidative, which will promote fermentation and produce large amounts of lactic acid.^{8,13} This build-up of lactic acid creates a lower pH in cancer cells, driving out oxygen.¹³ To maintain an alkaline pH (7.4), there has to be an adequate mineral

consumption, so the blood can supply the crucial minerals.¹³ For this reason, the Paleolithic diet is recommended for cancer patients, since it is a low carbohydrate but rich in nutrients diet.

In addition, oxygen is necessary to regulate the pH of the body.¹³ This is one of the reasons why hyperbaric chamber is recommended for cancer patients. When there is an increased amount of oxygen, the metabolism of the cell can shift from anaerobic to aerobic. Consequently, the metabolism of ascorbic acid (AA) can proceed and hydrogen peroxide can be generated.¹²

Throughout the years, vitamin C (or AA) has been studied as a therapy for infectious diseases and cancer.¹⁴ Specifically, since 1952, AA has been proposed as an agent with chemotherapeutic potential.¹¹ This concept was proposed by Dr. William McCormick, and explained that the chemotherapeutic effect of AA results from its actions as reducing/oxidizing agent when administered intravenously in massive repeated doses.¹⁵ Then, Cameron and Pauling, presented the results of a clinical trial in which 100 terminal cancer patients received 10g of AA daily as supplemental therapy and their survival time increased in comparison to the control group that did not receive AA supplementation.¹⁶

The primary source of cancer cells energy is glucose, meaning that cancerous cells have an increased requirement for glucose.¹¹ To compensate this need, the cancer cell increases its number of glucose transporters (GLUTs).^{11,13} As AA and glucose have similar molecular structure, both can enter the cells through GLUTs.¹¹ In most cells, AA enters the cells in the oxidized form, which is dehydroascorbic acid.¹¹ In this way, AA shows its nontoxic chemotherapeutic potential, which compromises the growth of tumor cells.^{11,17}

Furthermore, when AA is administered at high levels, it can exert its cytotoxic action.¹² This action is mediated through hydrogen peroxide generated by the conversion of ascorbate to dehydroascorbate.¹¹ High amounts of hydrogen peroxide induce cell growth arrest, apoptosis, and/or necrosis.^{11,18} As explained before, dehydroascorbate can enter the cancerous cell through GLUTs, convert to ascorbate and generate hydrogen peroxide, via redox reactions. In consequence, hydrogen peroxide inside the cancer cell can slow the tumor cell grow and even induce apoptosis.^{11,13}

In relation to supplementation, the idea is to provide nutrients for metabolic correction⁷, more specifically for mitochondrial repair. This concept based on our Bioenergetic Theory of Carcinogenesis¹³.

In conclusion, AA possesses many therapeutic benefits for cancer patients that can improve the patient's quality life, such as increased appetite, increased energy, and decreased pain, among many others.¹² For this reason, we recommend to continue studying possible adjuvant treatments for cancer patients, involving IV infusions of vitamin C and metabolic correction plans. We suggest a clinical trial as future research to further explore this approach.

References

1. World Health Organization. WHO Position Paper on Mammography Screening. WHO Position Paper Mammogr Screen. 2014;78. <http://www.ncbi.nlm.nih.gov/pubmed/25642524>.
2. Center For Disease Control. Breast Cancer Statistics. U.S. Cancer Statistics Working Group. <http://www.cdc.gov/ncidod/dvbid/westnile/index.htm>. Published 2017.
3. de Rinaldis E, Tutt A, Dontu G. Breast Cancer. Breast Pathol. 2011;352-359. doi:10.1016/B978-1-4377-1757-0.00028-7.
4. Howlader N, Noone A, Krapcho M, et al. Cancer Statistics Review, 1975-2014. National Cancer Institute. <https://seer.cancer.gov/statfacts/html/breast.html>. Published 2016.
5. Zavala-Zegarra D, Tortolero-Luna G, Torres-Cintrón C, et al. Cáncer en Puerto Rico, 2008-2012. Puerto Rico Cent Cancer Regist. 2015;1-131.
6. Tian Y, Li J, Li X, Guo G, Wen X, Nan J. Preoperative Serum Carcinoembryonic Antigen as a Marker for Predicting the Outcome of Three Cancers. Biomark Cancer. 2017;9(0). doi:10.1177/1179299X17690142.
7. Gonzalez MJ, Miranda-massari JR. Metabolic Correction: A Functional Biochemical Therapeutic Approach. Townsend Lett. 2016;(July).
8. Gonzalez MJ, Miranda-Massari JR, Duconge J. The Bio-Energetic Theory of Carcinogenesis: The Origin of Cancer Revisited. 2016;31(2):84-89.
9. Moen I, Stuhr LEB. Hyperbaric oxygen therapy and cancer—a review. Target Oncol. 2012;7(4):233-242. doi:10.1007/s11523-012-0233-x.
10. Daruwalla J, Christophi C. Hyperbaric oxygen therapy for malignancy: A review. World J Surg. 2006; 30(12): 2112-2131. doi:10.1007/s00268-006-0190-6.
11. González MJ, Miranda-Massari JR, Mora EM, et al. Orthomolecular Oncology Review: Ascorbic Acid and Cancer 25 Years Later. Integr Cancer Ther. 2005;4 (1):32-44. doi:10.1177/1534735404273861.
12. Gonzalez MJ, Miranda-Massari JR, Duconge J, Berdiel MJ. Increasing the effectiveness of intravenous Vitamin C as an anticancer agent. J Orthomol Med. 2015;30(1):45-50.
13. Gonzalez MJ, Miranda Massari JR, Duconge J, et al. The bio-energetic theory of carcinogenesis. Med Hypotheses. 2012; 79(4): 433-439. doi:10.1016/j.mehy.2012.06.015.
14. Riordan HD, Hunninghake RB, Riordan NH, et al. Intravenous ascorbic acid: protocol for its application and use. P R Health Sci J. 2003;22(3):287-290. <http://www.ncbi.nlm.nih.gov/pubmed/14619456>.
15. McCormick WJ. Ascorbic acid as a chemotherapeutic agent. Arch Pediatr. 1952;69(4):151-155. <http://www.ncbi.nlm.nih.gov/pubmed/14924799>.
16. Cameron E, Pauling L. Supplemental ascorbate in the supportive treatment of cancer: Prolongation of survival times in terminal human cancer. Proc Natl Acad Sci U S A. 1976;73(10):3685-3689. <http://www.ncbi.nlm.nih.gov/pubmed/1068480>.
17. Gonzalez MJ, Miranda-Massari JR. New Insights on Vitamin C and Cancer. New York, NY: Springer New York; 2014. doi:10.1007/978-1-4939-1890-4.
18. Davies KJ. The broad spectrum of responses to oxidants in proliferating cells: a new paradigm for oxidative stress. IUBMB Life. 1999;48(1):41-47. doi:10.1080/713803463.