

Study the Glucose Level in Obese Breast Cancer Patients

Sheerin H. Abbas*, Rula Dhahir Abdulmohsin

Babylon University, College of Science for Women. Iraq
Email: sheerin.hamza@gmail.com

ABSTRACT

This ponders incorporate the estimation of glucose in corpulent breast cancer patients (50) and compared with control (50) with age approximately (20-50) a long time, where the glucose levels in serum patients found critical increment in glucose levels p-value (0.0000), there is not a relationship between glucose levels and body mass index (BMI) (Kg/M2) p-value (0.075). In conclusion: glucose as it were a source of vitality for cell cancer.

Keywords: Glucose level, breast cancer, obese, cell cancer, body mass index (BMI)

Correspondence:

Sheerin H. Abbas
Babylon University, College of Science for Women. Iraq.
Email: sheerin.hamza@gmail.com

INTRODUCTION

Dangerous development maybe cells ailment signs, and that can be considered a fundamental structure for the body. Thus, the body keeps developing and create other new cells, supplant devastated tissue, and recover wounds. Frequently, cells copy and kick the bucket in a precise way. Presently and after that cells do not create, parcel and kick the bucket within the standard line. Consequently, either the blood or lymph, within the whole body, tend to wind up unusual, further, structure a projection known as a tumor, which can be kind or perilous [1]. Dangerous development may be a critical common medical issue in general [2]. In 2007 dangerous development was the third driving reason for passing in Iraq and the seventh driving reason for dismalness [3]. Small is considered the case of threat in Iraq.

Bosom danger happens when the cells coating the bosom lobules or conduits create unusually and insane. A tumor can outline within the breast lobules and conduits. Both men and women can have bosom harmful development, in show disdain toward the reality that it is unprecedented in men [4]. Cell imperativeness digestion is one of the basic strategies that's affected amid the advance from conventional to threatening cell development. In general, there is a clear balance in glucose digestion within the tumor cells. On the other hand, Glycolysis, which considered a catabolic strategy, can change from one glucose molecule to two pyruvates during the period of two NADH (diminished nicotinamide adenine dinucleotide) and two ATP particles. In the oxygen location, the Pyruvate can face oxidation to CO₂ and water within the pathway of the oxidative phosphorylation, which leads to creating approximately 36 ATP particles.

Furthermore, in oxygen absent, pyruvate is altered to lactic destructive through the pathway of the anaerobic glycolysis. However, the change of glucose to lactic destructive within the sight of oxygen can occur and that can be called the Warburg effect or high-impact glycolysis [5]. The majority of threatening cell development delivers a part of lactate paying small intellect to the openness of oxygen [6]. Some have considered this extended oxygen devouring glycolysis to be the seventh sign of an evolutionary threat [7]. For others, beginning with the suggestion of Hanahan and Weinberg [8], being limitless replication potential, freedom in progress signals, a guarantee against apoptosis, rigidity against anti-growth signaling, supported angiogenesis, tissue aggression, and metastasis, Being unlimited repeat potential, independence in improvement signals, security from apoptosis, disregard for anti-growth signals,

angiogenesis, tissue discontinuity, and metastasis. Within the arrangement of early carcinogenesis, an increase in uncontrolled cells causes cancer cells to move out of the veins, and over these lines, from oxygen and complement supply.

The best way that oxygen and glucose can perform the endogenous cells of a nonvascular tumor is to spread over the storm vault layer and within the peripheral tumor cell layers. However, the fractionated oxygen may drop to superb qualities within 100 mm of the veins [11]. This suggested that hypoxia and hypoglycemia are quickly formed, within the inner mass of the resulting tumor. However, it has been known since the 1920s [12] that cancer cells have a higher rate of glucose employment part over the glycolysis pathway that cannot send pyruvate into the Krebs cycle (oxidative phosphorylation pathway) changes somewhat from pyruvate to lactate: the indicated Warburg effect [7] (Fig. 1). Undoubtedly, many tumors use this glucose in the lactate pathway already within the oxygen site, which explains why the term active glycolysis is so often used as a substitute for the Warburg effect. The glycolytic switch that takes place in threat cells is not actually associated with decreased oxidative phosphorylation [13]. Tumor-expanded glycolysis activity by PET scan has been abused to check for diffuse bruising. This will abuse the expanded ability of tumor cells to request and use normal tissues with differentiated glucose [5].

Amid chemotherapy, the glucocorticoid dexamethasone is broadly utilized to thwart responses [18]. In any case, glucocorticoid organization is related to the shortcoming of affront affectability, rises in periphery blood glucose, as well as the concealment of the hypothalamic, pituitary, and adrenal turn for at least three weeks [19].

MATERIALS AND METHOD

Fifty females with stout breast cancer was taken in this ponder and their age are between (20-50) a long time, 5 ml of blood tests collected from each case in test tube and after 30 minutes. The serum isolated by centrifuge to degree the glucose levels. The levels of factual noteworthy investigation were ($p < 0.05$).

RESULTS AND DISCUSSION

The number of breast cancer gather 50 ladies, and the number of control gather are 30 women. The cruel \pm SD for persistent bunches were (43 ± 3.9 a long time), and for control bunches were (42 ± 3 years), glucose level statically critical expanded in hefty breast cancer patients compared with controls where p -value ≤ 0.05 , this growing sense of bosom infection event and all patients

take chemotherapy where there is an association between glucose level and bosom threat the setting was more than 16% of bosom dangerous development cases around the world excited with diabetes which diabetic people will in common have more terrible comes about taking after the therapy of bosom cancer [19]. Another important aspect that has not been considered over is the

probability that bosom harmful development therapy impacts the assimilation of glucose. These come about to concur with Tamas Hickish and et.al. (Glucose Bigotry Amid Adjuvant Chemotherapy for Breast Cancer [2009].) These come about the show all patients respond to treatment. As appear in Tables (1, 2) and Figures (3, 4).

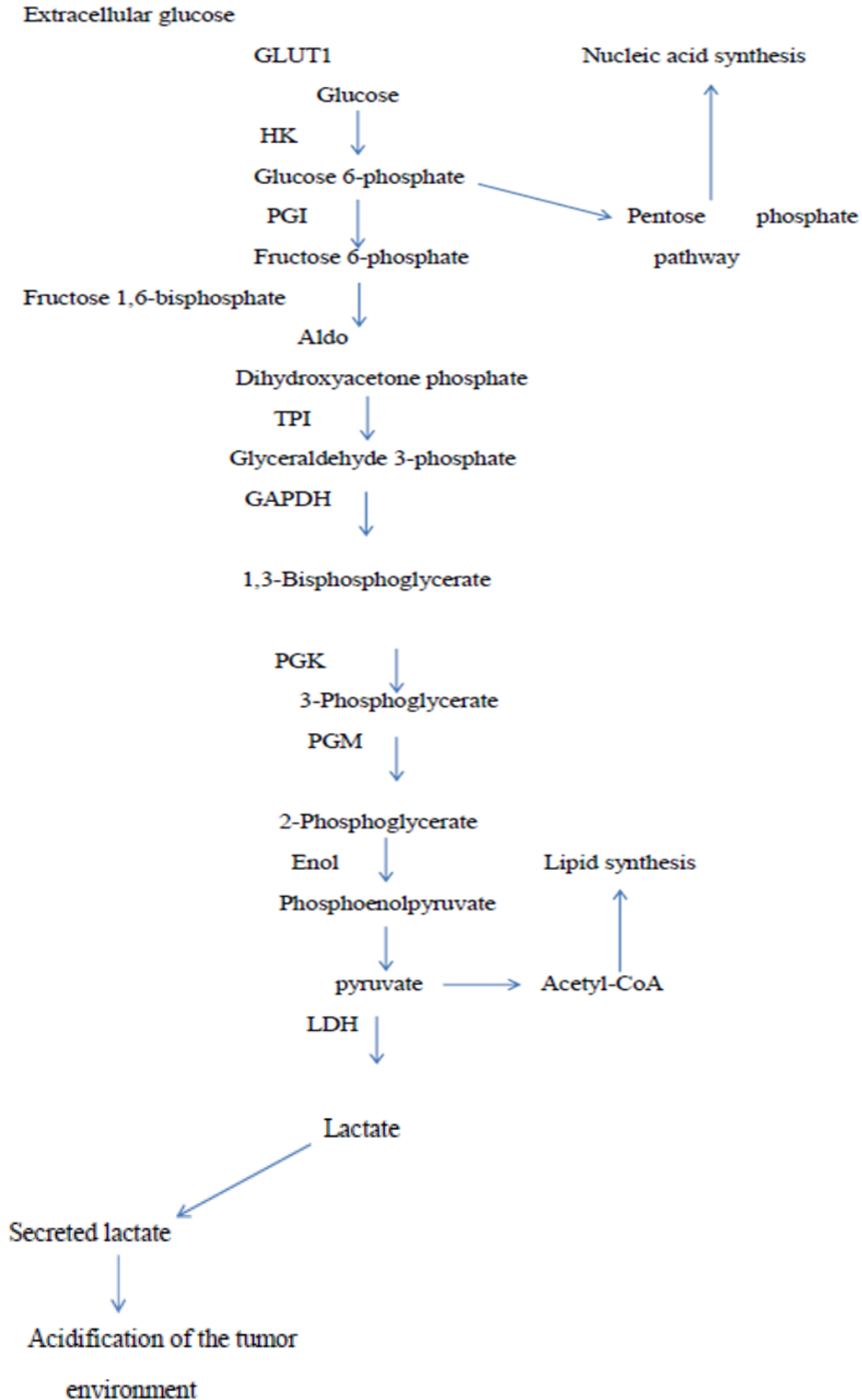


Table 1. Standard deviation and Mean for age and glucose level.

Parameter	N.O. of patients	Sta. Deviation	Mean	P-Value
Age years	50	3.946	43	0.0001
Glucose level mg/dl	50	27.9	149	

Table 2. Standard deviation and Mean for control age and glucose level.

Parameter	NO. of control	Sta. Deviation	Mean	P-Value
Age	30	3.003	42	0.0001
Glucose level mg/dl	30	7.342	80.45	

The level of blood glucose is controlled by biochemical forms and is kept in a moderately contract run amid the day. Fasting glucose level (after 12-16 hours of fasting) in people is 3.5-5.5 mM in blood plasma. Precise estimation of blood glucose is of awesome significance within the conclusion and administration of diabetes. At somewhat lifted fasting glucose levels diabetes or impeded glucose resilience cannot be securely analyzed, in these cases, glucose resistance test gives an alternative. Obesity can be considered a risk factor that can be led to diabetes. New Epidemiologic studies have mentioned the extended

threat of a couple of sorts of malady in relationship with diabetes, counting liver, pancreatic, and bosom. The type 2 of diabetes is portrayed through raised blood sugar brought approximately via insulin hindrance beside extraordinary flaws in affront outflow. Right off the bat within the headway of the turmoil as insulin opposition makes, extended outflow of affront keeps blood glucose levels decently commonplace. At final β -cell work within the pancreas rots, affront levels drop, and blood glucose begins to rise [20, 21]. As appear in Table - 3.

Table 3. Standard deviation and Mean for BMI and breast cancer patients.

Parameter	NO. of patients	Sta. Deviation	Mean	P-Value
Glucose level mg/dl	50	27.9	149	0.075
BMI Kg/M ²	50	4.738	32.27	

When the specific proteins that expect to be work in glucose assimilation have been upregulating, the metabolic turn to oxygen outlaying glycolysis can be noticed in critical development cells, which is known as the Warburg effect. In the glycolytic pathway, glucose using can be led to metabolic intermediates production, which causes the critical development of cells and make it back to the rapid extension. For example, glucose 6-phosphate, which is one of the intermediates, is employed for the incorporation of nucleic destructive via the pentose phosphate path in order to speed the DNA

replication. The lipid mixture is necessary for the path of action of layers in isolating cancer cells. In general, producing the lactate by the tumor cells can be affected by the tumor environment and that will lead to producing certain forte which can support tumor development, and limiting the locomotion of a few anti-cancer solutions. Whereas the glycolysis in ordinary cell deliver pyruvate with oxygen shows and happening in the cytoplasm of the cell whereas in less oxygen the glycolysis delivers lactate this happening practicing muscle [22].

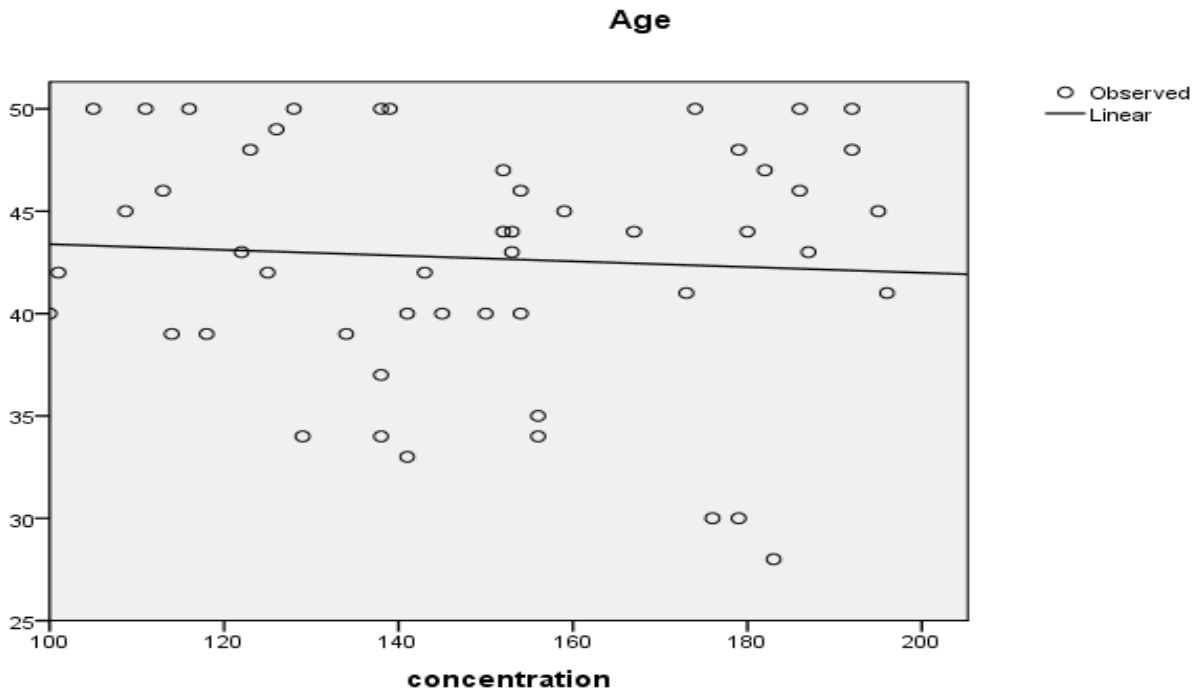


Figure 2. Correlation between glucose level and age of patients.

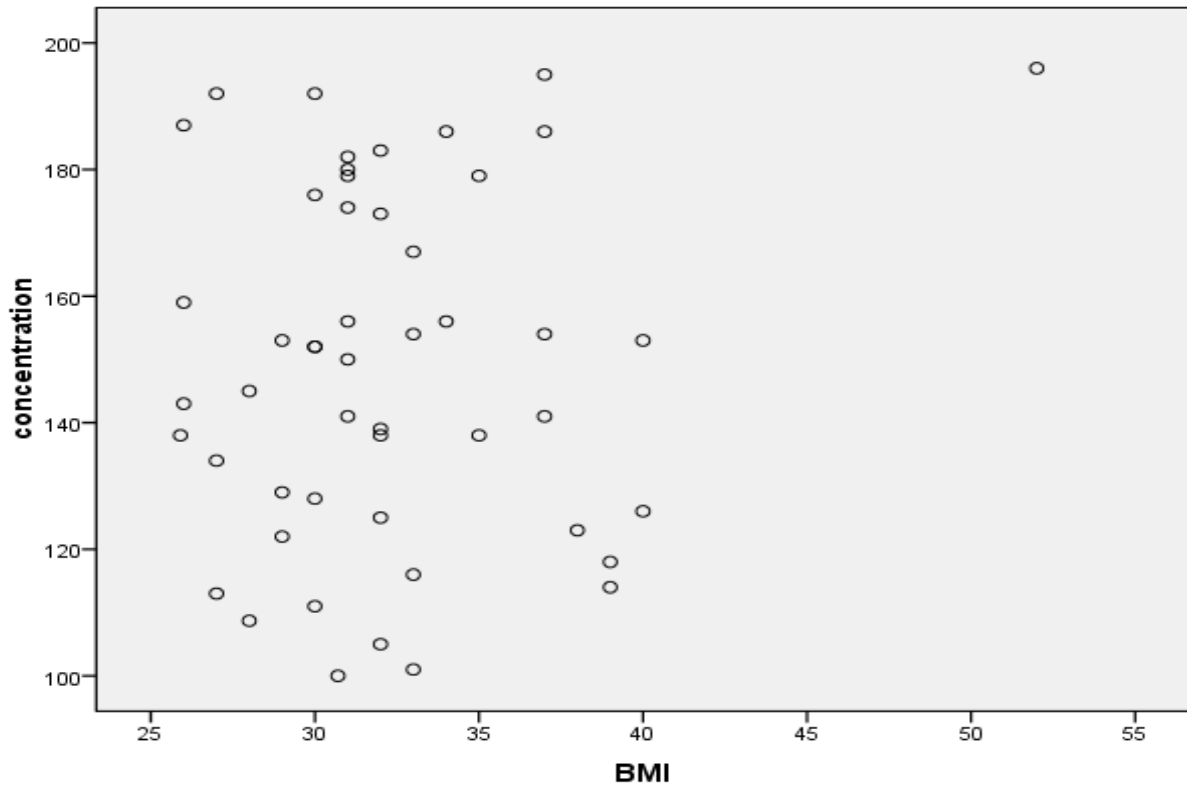


Figure 3. correlation between Body Mass Index and glucose level.

REFERENCES

1. Bech, A.G., 2012. Breast Cancer in Australia: An Overview (No. 71). AIHW.
2. Jemal, A., Siegel, R., Ward, E., Murray, T., Xu, J. and Thun, M.J., 2007. Cancer statistics, 2007. CA: a cancer journal for clinicians, 57(1), pp.43-66.
3. Iraqi Ministry of Health. Department of vital statistics 2008 report.

4. World Health Organization, 2006. Guidelines for the early detection and screening of breast cancer.
5. House, S.W., Warburg, O., Burk, D. and Schade, A.L., 1956. On respiratory impairment in cancer cells. *Science*, 124(3215), pp.267-272.
6. Vander Heiden, M.G., Cantley, L.C. and Thompson, C.B., 2009. Understanding the Warburg effect: the metabolic requirements of cell proliferation. *Science*, 324(5930), pp.1029-1033.
7. Yeung, S.J., Pan, J. and Lee, M.H., 2008. Roles of p53, MYC and HIF-1 in regulating glycolysis—the seventh hallmark of cancer. *Cellular and Molecular Life Sciences*, 65(24), p.3981.
8. Hanahan, D. and Weinberg, R.A., 2000. The hallmarks of cancer. *Cell*, 100(1), pp.57-70.
9. Shaw, R.J. and Cantley, L.C., 2006. Ras, PI (3) K and mTOR signalling controls tumour cell growth. *Nature*, 441(7092), pp.424-430.
10. Helmlinger, G., Yuan, F., Dellian, M. and Jain, R.K., 1997. Interstitial pH and pO₂ gradients in solid tumors in vivo: high-resolution measurements reveal a lack of correlation. *Nature medicine*, 3(2), pp.177-182.
11. Warburg, O., Wind, F. and Negelein, E., 1927. The metabolism of tumors in the body. *The Journal of general physiology*, 8(6), p.519.
12. Munck, A., 1971. Glucocorticoid inhibition of glucose uptake by peripheral tissues: old and new evidence, molecular mechanisms, and physiological significance. *Perspectives in Biology and Medicine*, 14(2), pp.265-289.
13. Wolf, I., Sadetzki, S., Catane, R., Karasik, A. and Kaufman, B., 2005. Diabetes mellitus and breast cancer. *The lancet oncology*, 6(2), pp.103-111.
14. Cavaghan, M.K., Ehrmann, D.A. and Polonsky, K.S., 2000. Interactions between insulin resistance and insulin secretion in the development of glucose intolerance. *The Journal of clinical investigation*, 106(3), pp.329-333.
15. Mahler, R.J. and Adler, M.L., 1999. Type 2 diabetes mellitus: update on diagnosis, pathophysiology, and treatment. *The Journal of Clinical Endocrinology & Metabolism*, 84(4), pp.1165-1171.
16. Denniston Topping Caret 5th Edition Carbohydrate Metabolism Chapter 21.