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Research Article

Membrane Potential and Calcium Ion Dynamics in Breast Cancer Patients attending Specialist Hospital Owerri

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Abstract

Breast cancer is a major source of illness and death among women globally, with rising incidence in emerging nations. Recent research indicates that bioelectrical features, especially membrane potential and calcium ion (Ca^{2+}) dynamics, are essential in cancer growth. This research examined changes in membrane potential and calcium ion dynamics in breast cancer patients at Specialist Hospital Owerri, Nigeria. A cross-sectional study design was utilised, comprising 50 confirmed breast cancer patients and 50 age-matched healthy controls. Blood samples were obtained to assess serum and intracellular calcium concentrations by conventional biochemical and spectrophotometric techniques. The Nerst equation was used to figure out the membrane potential. We used descriptive statistics and an independent sample t-test to look at the data, and we fixed the significance level at $p < 0.05$. The results indicated a considerable depolarisation of membrane potential in breast cancer patients relative to controls. Serum calcium and intracellular Ca^{2+} concentrations were markedly increased in patients relative to controls ($p < 0.001$). The results show that breast cancer is linked to major changes in bioelectricity, such as membrane depolarisation and calcium homeostasis that isn't working properly. These alterations may facilitate tumour growth and could function as potential biomarkers for diagnosis and therapeutic targeting.

Keywords: Breast cancer, membrane potential, calcium ion, bioelectricity, tumour growth, Nigeria.

Introduction

Breast cancer is still one of the most common cancers in women around the world, and it is a major public health problem, especially in developing nations like Nigeria. In addition to the well-known genetic and molecular factors that cause breast cancer, more and more people are paying attention to the bioelectrical features of cancer cells, specifically how calcium ions (Ca^{2+}) move around and the membrane potential. These electrophysiological changes give us important information about how tumours start, grow, and what treatments might work [1].

A distinctive characteristic of cancer cells is the alteration of their resting membrane potential. Normal human mammary epithelial cells usually have a resting membrane potential that is hyperpolarised at about −60 mV. In contrast, malignant breast cells have a resting membrane potential that is greatly depolarised, averaging around −13 mV [2]. This depolarisation has been regularly observed in extensively researched breast cancer cell lines, including MCF-7 and MDA-MB-231, and is regarded as an early bioelectrical signal linked to neoplastic transformation [3]. It is important to note that changes in membrane potential are not just passive effects of cancer; they also actively affect how cancer cells act, such as how they grow, invade, and spread.

Breast cancer cells, in addition to membrane depolarisation, display modified responses to extracellular ionic conditions relative to normal cells. These disparities underscore the essential function of ion transport pathways in cancer biology [4]. Alterations in electrical characteristics, including permittivity and conductivity, have been recognised as prospective diagnostic biomarkers for differentiating cancerous tissues from healthy ones [5].

Calcium ion signalling is closely related to membrane potential and is very important for controlling important cellular functions. Intracellular calcium functions as a ubiquitous second messenger that regulates cell proliferation, death, migration, and differentiation. In breast cancer, dysregulated calcium signalling, frequently marked by persistent increases in intracellular Ca^{2+}, facilitates tumour growth and enhances metastatic potential [6]. The interaction between membrane potential and calcium dynamics is crucial, as alterations in transmembrane voltage directly affect calcium influx via voltage-dependent and receptor-operated channels.

Ion channels, pumps, and exchangers in the plasma membrane work together to keep the membrane potential and calcium levels stable. These regulatory systems are often changed in cancer cells. Disruptions in calcium channels and transporters result in aberrant calcium influx and build-up, hence facilitating the electrophysiological and functional alterations noted in malignant cells [7].

Additionally, certain ion channels have been associated with the advancement of breast cancer. Voltage-gated sodium channels, especially Nav1.5, are frequently overexpressed in metastatic breast cancer cells, facilitating membrane depolarisation and promoting invasive behaviour (Persinger and Lafrenie, 2014). Transient receptor potential (TRP) channels, which facilitate several calcium influx pathways, are often dysregulated in breast cancer and are crucial to tumour formation and progression [8].

Notwithstanding these advancements, there exists a paucity of data regarding the electrophysiological characteristics of breast cancer patients within Nigerian clinical environments. Learning how membrane potential and calcium ion dynamics work in patients at Specialist Hospital Owerri will give us important information about the disease profile in the area and could help us find better ways to diagnose and treat it.

Materials and Methods

This study adopted a cross-sectional design conducted at Specialist Hospital Owerri, Imo State, Nigeria. The study population comprised confirmed breast cancer patients attending the hospital and age-matched healthy controls.

Sample Size and Sampling Technique

A total of participants were recruited using a purposive sampling technique. Inclusion criteria included confirmed diagnosis of breast cancer, while patients with other chronic illnesses were excluded.

Ethical Considerations

Ethical approval was obtained from the hospital ethics committee, and informed consent was obtained from all participants.

Sample Collection

The study subjects fasted overnight within an interval of eight to twelve hours prior to collection of samples, 5ml of blood was collected aseptically from each subject by venipuncture of the antecubital vein using sterile syringe and needle. The blood sample was placed in a clean plain dry tube, allowed to clot, retracted and centrifuged at 3000rpm for 10 minutes using wisperfuge (model 1384) centrifuge (Sampson, Holland) after which the serum sample was obtained.

The serum was separated using a pasteur pipette and placed in another dry plain tube for the estimation of calcium. All samples were stored at - 20oc prior to analysis.

Laboratory Assay

Calcium levels were determined using spectrophotometric methods. membrane potential indicators were assessed using Nest equation

Statistical Analysis

Data were analyzed using SPSS version XX. Results were expressed as mean ± standard deviation. Student's t-test and correlation analysis were used, with significance set at $p < 0.05$.

Results

Parameter	Patients (n=50)	Controls (n=50)	t-value	p-
value				
Serum Calcium (mmol/L)	2.91 ± 0.37	2.28 ± 0.26	9.84	0.0001
Intracellular Ca^{2+} (µmol/L)	1.78 ± 0.40	0.99 ± 0.22	12.23	0.0001
Membrane Potential (J)	97.75± 55.5	138.18 ± 70.91	-3.17	0.0022

Table 1: Serum and Intracellular Calcium Levels as well as Membrane Potential in breast cancer patients

Both serum and intracellular calcium levels were significantly elevated in breast cancer patients ($p < 0.001$). This suggests dysregulated calcium homeostasis, which may contribute to tumour progression.Breast cancer patients showed a significantly depolarized membrane potential compared to controls. The difference was highly significant confirming disruption of normal cellular electrical properties.

Discussion

The current literature underscores the importance of bioelectricity in cancer biology. Membrane potential has been recognised as a regulator of cell cycle progression, where depolarisation facilitates cell division and hyperpolarisation correlates with differentiation [9]. In breast cancer, persistent depolarisation is associated with unregulated proliferation and tumour aggressiveness.

The results of this work confirm earlier findings that breast cancer cells display depolarised membrane potentials [10]. This depolarisation may promote unregulated growth by modifying ion flow and cellular signalling pathways.

The increased calcium levels noted in this study align with findings indicating that intracellular Ca^{2+} facilitates cancer growth by activating proliferation and migratory pathways [11,12]. The relationship between membrane potential and calcium dynamics indicates that bioelectrical alterations are fundamental to cancer pathogenesis. Calcium signalling pathways are also the subject of a lot of research in cancer. Increased intracellular Ca^{2+} concentrations have been demonstrated to activate pathways such as MAPK and PI3K/Akt, which are essential for the survival and proliferation of cancer cells [13]. Calcium-dependent enzymes, such as calmodulin and calcineurin, also affect gene expression and the movement of the cytoskeleton, which helps metastasis.

Ion channels, including TRP channels (e.g., TRPV6, TRPM7) and voltage-gated calcium channels, have been associated with the advancement of breast cancer. Their overexpression increases calcium influx, which helps tumours grow and makes them less likely to die [14].

Conclusion

This study shows that women with breast cancer have big changes in their membrane potential and calcium ion dynamics. These alterations facilitate tumour advancement and may function as significant biomarkers for diagnosis and prognosis.

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