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Original Research Paper

Physiological study of Aurora kinase A (AURKA) in relation with Breast Cancer Tissue

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Abstract: Breast cancer, a widespread and frequently fatal condition impacting women globally, results in over 40,000 deaths each year. The disease presents in multiple forms, such as benign tumors, normal tissue, in situ carcinoma, and invasive carcinoma. Despite advancements in early identification and treatment efficacy, 20% to 30% of women diagnosed with breast cancer develop metastatic illness, often affecting visceral organs, either at the time of diagnosis or as the disease progresses. **Aims**: The aim of this study is to Investigate the expression levels of the biochemical marker AURKA and its function in tumor proliferation. **Methods:** A total of 60 females diagnosed with breast cancer were selected from Al-WATANI Oncology Hospital and Center Labs in Najaf for further laboratory research. Additionally, 30 females, free from any known diseases and apparently healthy, were recruited as control subjects. **Results:** The findings indicate that the patient group demonstrates a higher average value (3.3 ± 1.0) in comparison to the control group (1.6 ± 0.15), reflecting a raw difference of roughly (1.7). Statistical testing reveals no significant difference between the two groups, as indicated by a p-value of 0.095. **Conclusion**: The data reveals no substantial increase in the biochemical marker AURKA, as shown by the results. This marker may still be significant in breast cancer progression, likely due to its role in cell cycle regulation and tumor development.

Keywords: Breast cancer; Aurora kinase A (AURKA)

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1. Introduction

Breast cancer, a common and frequently lethal disease impacting women globally, results in over 40,000 deaths each year. This condition is categorized into four principal types: benign tumors, normal tissue, in situ carcinoma, and invasive carcinoma [1]. Following non-melanoma skin cancer, it represents the leading cause of cancer-related mortality in women worldwide. Most breast cancers are detected through mammography or physical examination; primary care physicians frequently serve as the initial point of contact. The National Cancer Institute's Breast Cancer Risk Assessment Tool provides an estimate of breast cancer risk over a five-year period; however, it is not intended to assess risk in individuals carrying BRCA1 or BRCA2 gene mutations.[2]. DNA damage or mutations progress to a degree that surpasses the threshold of self-repair, leading to in situ carcinoma, characterized by diseased cells restricted within the ducts. However, they have not yet invaded the surrounding tissues [3]. The disease's genesis is complex, necessitating a thorough investigation of its molecular underpinnings and related risk factors. Tumorigenesis in breast cancer is influenced by multiple factors. Research highlights the complex interplay among genetic, environmental, and lifestyle elements that contribute to its onset. Comprehending these characteristics is crucial for the early prevention and detection of breast cancer [4].
The Aurora kinase family consists of three highly conserved serine-threonine kinases: A, B, and C (with gene names AURKA, AURKB, and AURKC). These intracellular enzymes are essential regulators of cell division, playing pivotal roles in controlling cell proliferation and growth. [5]
AURKA is significantly expressed in around 73% of breast cancer patients, contributing to treatment resistance and reducing median survival time.[6] Aurora kinases, owing to their essential role in oncogenesis, have become a central area of interest in translational cancer research. Aurora kinase inhibitors (AKIs) are engineered to specifically obstruct the ATP-

The binding site of these kinases hinders mitotic progression, thereby inducing cell death in cancerous cells [7]. AURKA has been identified as an initiator of early metastatic processes. Analysis of clinical tumor samples revealed an association between the presence of N-AURKA and diminished patient survival. Our results clarify a molecular link between two crucial pathways in cancer metastasis, suggesting nuclear AURKA as a critical upstream regulator of the HIF1 transcription complex and a potential target for anti-metastatic treatment [8].
Additionally, AURKA is expressed in all somatic cells and exhibits elevated levels in rapidly proliferating organs, including hematopoietic cells, the colon, testis, and mammary gland. It is also detectable in tissues characterized by low or absent rates of cell division, where it contributes to a range of physiological processes during interphase [9]. This study aims to investigate the physiological changes in breast cancer tissues compared to normal tissues. It will also examine the expression levels of the biochemical marker AURKA and its role in tumor growth. The goal is to assess overall health and see how this marker relates to AURKA expression in the tissue.

2. Methodology

Study Design and Patients

A total of 60 females diagnosed with breast cancer were selected from hospitals and laboratory centers in Najaf. Subjects were initially diagnosed by doctors utilizing ultrasonography and PET scans and subsequently referred to as additional laboratory studies. Thirty female subjects, devoid of any known diseases and seemingly healthy, were chosen as control participants. Sixty women diagnosed with breast cancer were enrolled, including those aged 40 to 65 years with metastatic breast cancer, who had chemotherapy. Each patient had 5 ml of venous blood taken using a disposable needle. After transferring 2 ml of the material into an EDTA tube and 3 ml into a gel tube, the gel tube was left to coagulate at room temperature for 10 minutes. Subsequently, it was centrifuged at 1200 RPM for seven minutes. Thereafter, the serum was extracted and placed into new, disposable Eppendorf tubes. The tubes were thereafter preserved in a -20 °C deep freezer until analysis commenced. Samples demonstrating hemolysis have been eliminated from research.

Histological processing of breast tissues

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Tru cut biopsies were collected after completion of procedure. Tissues were removed and preserved in 10% formaldehyde. Tissues were then paraffin embedded and processed for hematoxylin and eosin processing and sectioned using a microtome.

Collection of Blood Samples

A total of 5 mL of venous blood was collected from each patient using a disposable needle. Subsequently, 2 mL of the sample was transferred into an EDTA tube, while the remaining 3 mL was placed in a gel tube. The gel tube was allowed to clot at room temperature for 10 minutes before centrifugation at 1200 rpm for 7 minutes. Serum was then separated and transferred into new disposable Eppendorf tubes, which were stored at -20 °C in a deep freezer until analysis. Samples exhibiting hemolysis were excluded from further evaluation.

Measurement of Human Aurora Kinase A (AURKA)

Aurora kinase A (AURKA) facilitates tumor growth linked to 20q gain and correlates with disease recurrence [10].

This kit employs an Enzyme-Linked Immunosorbent Assay (ELISA) methodology. The assay plate has been pre-coated with a Human AURKA antibody. When a sample containing AURKA is added, it binds to the antibodies that are fixed to the wells. Following this, a biotinylated Human AURKA Antibody is added, which then binds to the AURKA present in the sample. Subsequently, Streptavidin-HRP is introduced, and it binds to the Biotinylated AURKA antibody. After incubation, any unbound Streptavidin-HRP is removed through a washing step. The substrate solution is then added, leading to color development that is directly proportional to the amount of Human AURKA in the sample. The reaction is then stopped by adding an acidic stop solution, and the absorbance is measured at 450 nm.

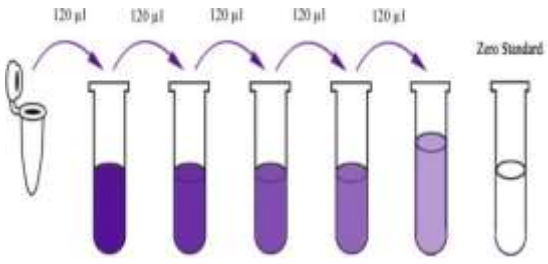

Fig 1: AURKA Standard Reagents preparation

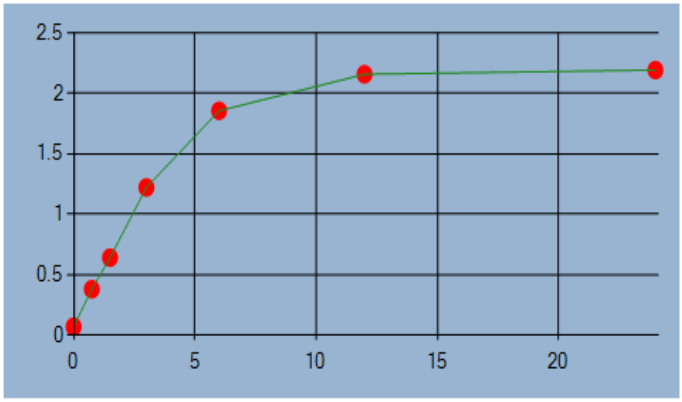

Fig 2: Standard curve for AURKA

Statical analysis

Data analysis was performed using GraphPad Prism v6, and the results were presented as means of standard error (SE) values. Independent samples t-tests were conducted to evaluate group differences, with statistical significance defined as a p-value less than 0.05. Graphical representations were generated utilizing Microsoft Excel 2010.

3.Results and discussion

Women with breast cancer have been separated into 3 groups according to grades. The percentage as well as the number regarding each group represented grade I (13) 22%, grade II represented (38) 63 % grade III represented (9)15%. Also, the age for patients and control ,were between (40 -65 years) and the age be divided into Two groups, patients were (40-52 years) 43% and (53-65 years) 57%, while control were (40-52 years) 66.7 % and (53-65 years) 33% Finally, the BMI , patients with normal weight represented (6) 10 % with expected value (18-24 kg/m2) while (17) 28 % of patients with overweight expected value (25-29 kg/m2) and (37) 62 % of patients Suffered from obese with exapted value (30-36 kg/m2).

AURKA level

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The results in (figure 3) indicates that the patient group exhibits a higher average value (3.3 ± 1.0) compared to the control group (1.6 ± 0.15), representing a raw difference of approximately of (1.7) When comparing the groups using statistical testing of difference there is no statistical significance between the values in the two groups the p value is (0.095).

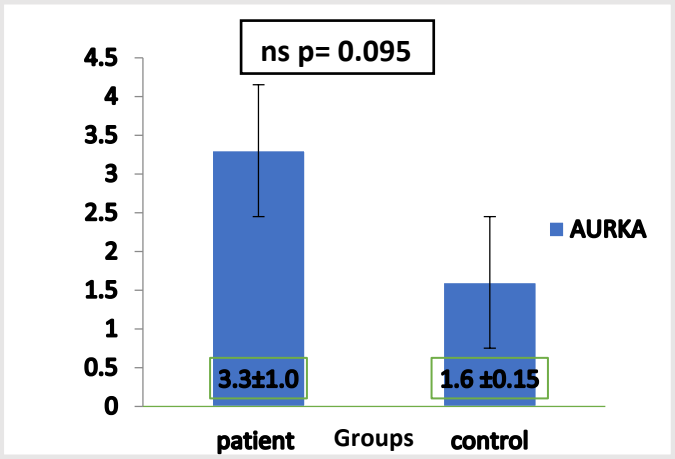

Fig 3: AURKA level in breast cancer women and control group

The present results indicated that there is no significance increase of AURKA levels in patients experiencing breast cancer compared also with control group, these results is acceptable with the result of [11] who explained that there is no statistical significant differences were found between the concentrations The study [12] indicates that AURKA expression is significantly lower in the blood of breast cancer patients compared to the control group. This finding is consistent with [13], which showed that reduced AURKA expression is linked to the progression from in situ to invasive breast carcinoma, and that AURKA expression differs between normal and invasive breast tissue. In contrast to our findings, [14] reports higher AURKA expression in younger breast cancer patients, along with connections between elevated Aurora-A/AURKA levels and aggressive tumor characteristics, such as increased tumor cell proliferation and shorter survival times Furthermore, [15] contradicts our results, as it found that the AURKA gene was highly expressed in breast cancer patients compared to those without the disease, and that this expression was significantly associated with poorer survival outcomes.

The docking analysis indicated that hsa-miR-32-3p could modulate AURKA, a conclusion supported by the observed binding energy and specific interaction patterns.

As noted in [16], the disparate outcomes observed in estrogen receptor-positive and negative patients could have significant clinical ramifications. It is plausible to hypothesize that aurora kinase A inhibitors might demonstrate reduced efficacy in estrogen receptor-negative carcinomas, given the lack of prognostic association with AURKA. Consequently, our observations regarding AURKA's differential performance across various molecular subtypes of breast cancer may elucidate the conflicting findings regarding its prognostic significance.

Conclusions

The data do not reveal any notable increase in the biochemical marker AURKA, according to the results. Nevertheless, AURKA may remain relevant to breast cancer progression, owing to its involvement in cell cycle regulation and tumor development.

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Author's Contributions

Shahad Mohammed Hameed was responsible for the conception and design of the study, carrying out the experimental work, performing data analysis, and writing the manuscript.
Professor Dr. Adhraa Baqer Hassan participated in the experimental work and provided supervision throughout the research process.

Ethics The study was approved by the medical ethics committee at the University of Kufa in 2025.

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