Indonesian Journal on Health Science and Medicine
Vol. 2 No. 1 (2025): January
ISSN 3063-8186. Published by Universitas Muhamamdiyah Sidoarjo

https://doi.org/10.21070/ijhsm.v2i1.61

The Relationship between female Breast Cancer and Renal Failure (Article Type: Review)

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Abstract. Following up on existing statistics and evaluating published research, we discovered that a quarter of women worldwide suffer from cancer, specifically breast cancer, which has a high fatality rate.Renal insufficiency and breast cancer etiology seem to be related in a reciprocal way. When choosing a course of treatment, this association should be taken into account. This study investigates the correlation between these two traits in patients diagnosed with breast cancer. This research paper will look into the relationship between this malignancy and kidney failure in order to determine the best treatment method. As a result, all medical and laboratory methodologies and standards must be used to determine the relationship and direct link between the two disorders. This does not indicate that there is no kidney failure owing to the lack of cancer, but due to cancer, an increase in blood calcium can occur, or due to tumor lysis syndrome (TLS), which causes renal failure in a patient who has kidney functions performing without any pathological defects. The patient's basal glomerular filtration rate should be considered by medical professionals when deciding on the optimal course of treatment for each breast cancer patient. Neglect and lack of regular monitoring of body functions, especially kidney function, through tests and monitoring of the glomerular filtration rate can increase the risk of kidney failure. When comparing cancer patients, meaning breast cancer, who suffer from kidney failure with those whose glomerular filtration rate is within the normal range, we find that the first group has high mortality rates. Therefore, these mortality rates must be reduced through scientific study and investigation to reach somewhat acceptable results. Both breast cancer and renal failure seem to share a common pathogenesis. This association should be taken into consideration while selecting a treatment approach. Physicians should consider the baseline glomerular filtration rate (GFR) of patients with breast cancer when deciding on the optimal course of therapy.

Highlights:

1. Investigate breast cancer and kidney failure correlation impacting treatment approaches.
2. High blood calcium or TLS may induce renal failure in patients.
3. Monitor glomerular filtration rate to reduce mortality in affected patients.

Keywords: breast cancer, renal failure, tumor lysis syndrome (TLS), glomerular filtration rate (GFR).

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Introduction

Because of the disease's importance and widespread prevalence, the most recent studies and statistics have addressed it, and their findings have revealed that breast cancer is the most common type of cancer among patients who visit health institutions around the world, making it the most common type of cancer and a source of fear for women.One in six women (15.5%) who die from cancer do so from breast cancer (1).

The death rate from breast cancer dropped sharply from over 60% in 1985 to 15.5% when patients were advised to try alternative treatment modalities. Radiation treatment, surgery, hormone therapy, and chemotherapy are some of these techniques (2,3). These days, anatomical staging, patient preferences, and cancer pathology are taken into account while selecting the best treatment plan (4).

This review outlines the spectrum of acute and chronic renal injuries associated with malignancy and explores the correlations between renal disease and cancer. It emphasizes the critical role of assessing kidney function, particularly regarding the adjustment of chemotherapeutic drug dosages based on renal function. Additionally, the review addresses how renal function can influence oncology outcomes and underscores the need for nephrologists to understand malignancy biology and treatment modalities. Active participation of nephrology services in oncology care is crucial for optimizing patient outcomes.

The adverse effects of cancer treatment are causing increasing worry, despite the fact that patients' survival and quality of life are improving. From minor conditions like baldness, fatigue, and nausea to more serious ones like myelosuppression, leukemia, cardiotoxicity, thrombosis, and ovarian failure, the side effects of chemotherapy can range widely (5). Many chemotherapy medications must be metabolized and excreted in a healthy kidney (6). People with solid tumors often experience renal insufficiency as a secondary effect. It is statistically estimated that glomerular filtration rates (GFRs) are lower than normal in patients with solid malignancies. (7).

Radiation therapy is an alternative treatment for breast cancer; however, it may lead to adverse effects including lymphedema, nephritis, cardiotoxicity, and gastrointestinal complications (8,9). To achieve optimal outcomes from breast cancer therapy, it is crucial to identify and address potential side effects promptly through prevention or treatment strategies. According to several research, there is a correlation

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between a higher risk of cancer and reduced renal function. This could be the outcome of elderly patients having longer life expectancies. Nonetheless, the prevalence of malignancies remains higher in younger individuals with renal failure than in the general population. An elevated inflammatory response that can result in cancer is one of the potential causes (10,11).

Historically, the association between renal disease and malignancy has been recognized, but its significance has only recently gained prominence with the establishment of a specialized field in nephrology known as onconephrology. In the 21st century, patients with malignancies represent an increasingly significant cohort for nephrology consultations and critical care nephrology services. A wide array of renal complications can arise in malignancy patients, impacting both their short-term outcomes and the effective management of their underlying oncological conditions. These kidney-related issues present substantial challenges for both oncology and nephrology (12).

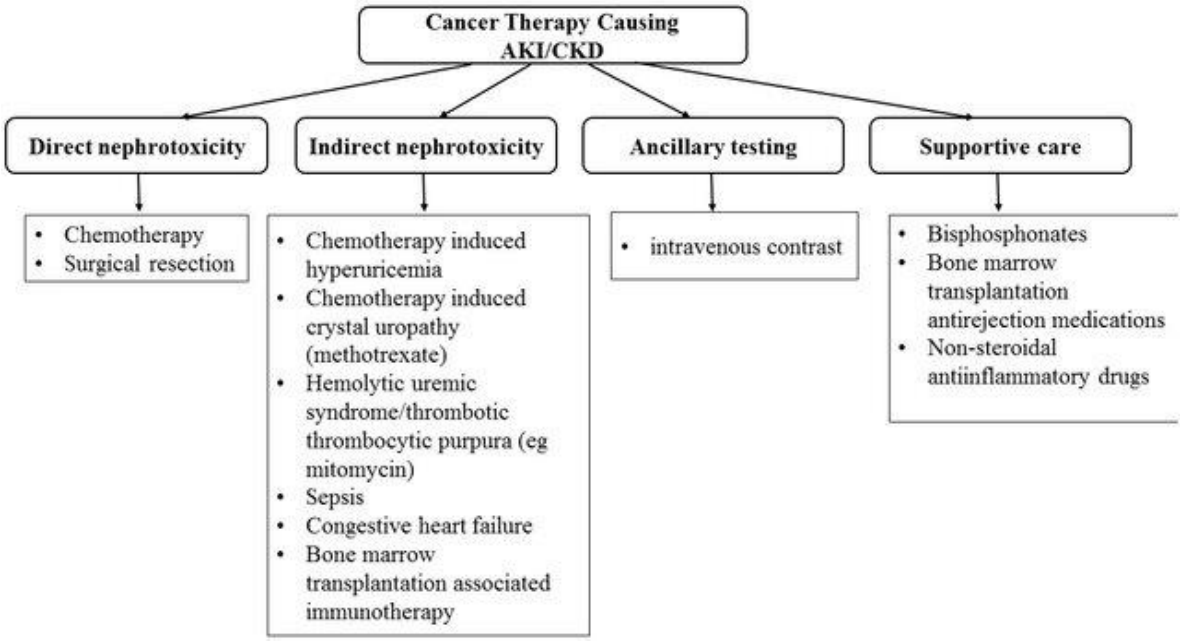

Figure 1: Oncological therapies associated with acute kidney injury (AKI) and/or chronic kidney disease (CKD).

Certain treatments, including chemotherapy, antibiotics, and specific bone-targeted agents, exhibit nephrotoxicity and may necessitate dose adjustments or interruptions to avert renal damage. Nephrologists are crucial in the detection and

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management of renal impairment in oncology patients. They can also provide guidance on renal protection when nephrotoxic agents require dose modifications or interruptions, and in the context of novel therapies or combinations. Effective patient management requires close collaboration between oncologists and nephrologists. This article reviews the interplay between cancer and kidney disease and evaluates the impact of various treatments on renal function, with a focus on considerations for monitoring kidney function (13) .

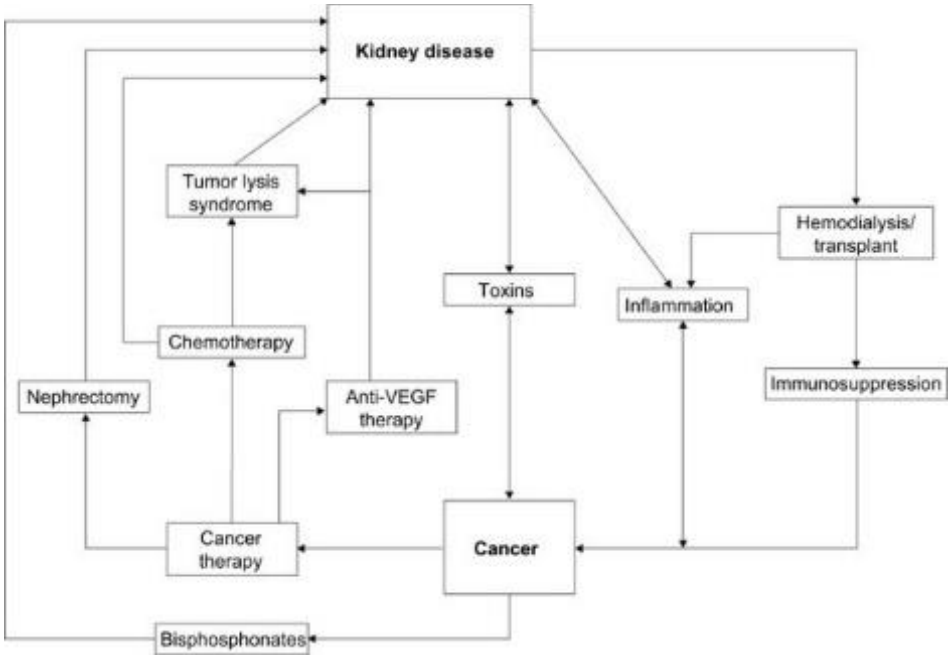

Figure 2: The bidirectional relationship between cancer and kidney disease.

Kidney failure and breast cancer pathogenesis appear to be related on both sides. This association should be taken into account when choosing a treatment plan. The relationship between these two variables in patients with breast cancer is reviewed in this research. The association between renal failure and breast cancer can be divided into three categories:

1. Breast cancer-related renal insufficiency (paraneoplastic syndrome)
2. Renal insufficiency brought on by treatment for breast cancer
3. Patients with impaired renal function who develop breast cancer

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Result and Discussion

1. Breast cancer-related renal insufficiency (paraneoplastic syndrome)

Breast cancer can adversely affect renal function through several mechanisms, often associated with paraneoplastic syndrome. These mechanisms include ectopic hormone secretion, fluid and electrolyte imbalances, and the deposition of antigen-antibody complexes in tubular and glomerular structures, all of which can contribute to renal dysfunction in paraneoplastic conditions(14).

Paraneoplastic glomerular diseases (GNs) are uncommon manifestations in patients with both hematologic and solid organ malignancies, potentially presenting either before or after cancer diagnosis. The lack of established diagnostic algorithms and reliable testing complicates their diagnosis. Due to their rarity and variability, the pathogenesis of most paraneoplastic GNs remains inadequately understood. Recent advancements in understanding paraneoplastic GNs have been driven by the identification of specific target antigens in membranous nephropathy, such as thrombospondin type-1 domain-containing protein 7A and neural epidermal growth factor-like 1 protein. These antigens show promise in distinguishing between primary and paraneoplastic causes of membranous nephropathy (15).

Figure 3: Paraneoplastic glomerular diseases (GNs) .

Hypercalcemia is a common paraneoplastic manifestation observed in patients with breast cancer. Approximately 80% of cases of humoral hypercalcemia of malignancy (HHM) are attributed to humoral factors secreted by tumor cells. The primary mechanism responsible for elevated calcium levels is the secretion of parathyroid hormone-related protein (PTH-rP), which acts through the receptor activator of nuclear factor kappa-B ligand (RANK-L) pathway to stimulate osteoclastogenesis and increase bone resorption. Additionally, elevated levels of parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D can also contribute to hypercalcemia in this context. The remaining 20% of hypercalcemia cases in breast cancer are associated with osteolytic bone metastases, which release calcium from the bone matrix into the bloodstream. It is also important to consider that primary hyperparathyroidism can induce hypercalcemia independently of breast cancer(16).

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Serum calcium levels and renal function, as measured by GFR, are correlated in both directions. Research indicates that when serum calcium levels rise, renal function significantly declines. Nephrolithiasis and chronic kidney disease are the fundamental causes of renal failure brought on by hypercalcemia (CKD). Serum calcium levels are said to be correlated with the severity of renal disease. The kidneys' capacity to eliminate excess calcium from the urine decreases as CKD progresses. Hypercalcemia can subsequently lead to secondary and tertiary hyperparathyroidism. This process establishes a vicious cycle in which chronic kidney disease (CKD), induced by malignancy-associated hypercalcemia, further exacerbates the hypercalcemic state due to diminished renal functio (17).

In managing renal impairment secondary to cancer-induced hypercalcemia, it is essential to avoid the use of calcium and other hypercalcemic agents. Adequate hydration should be maintained through the administration of normal saline solution. Bisphosphonates are utilized to inhibit bone resorption and mitigate mineral loss. When hypercalcemia is driven by excessive 1,25-dihydroxyvitamin D, corticosteroids are typically recommended to counteract this effect (18).

Glomerulonephritis (GN) is an uncommon but possible main manifestation or subsequent consequence of malignancy. The most prevalent kind of GN caused by solid tumors is membrane nephropathy, and paraneoplastic GNs typically exhibit nephrotic syndromes. While GI and lung tumors are more likely to coexist with breast cancer than paraneoplastic GN, certain findings suggest that these problems could happen. In addition to membranous nephropathy (MN), breast cancer has also been associated with minimal change disease (MCD), membranoproliferative glomerulonephritis (MPGN), rapidly progressive glomerulonephritis (RPGN), and focal segmental glomerulosclerosis (FSGS).

There have also been reports of thrombotic microangiopathy (TMA) and IgA nephropathy. Individuals who have GN growth that is inexplicable should be evaluated for undetected cancers, This includes individuals with breast cancer, especially those who are older than 60 years, smokers, or have glomerulonephritis (GN) that is resistant to treatment. In most cases, paraneoplastic GN completely resolves after the underlying cancer is treated, either with or without immunosuppressants. When GN returns after a period of remission in a patient whose cancer has been cured, it may indicate a recurrence of cancer (19).

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Tumor lysis syndrome (TLS) is characterized by a significant increase in serum phosphate, uric acid, and potassium due to malignant cell lysis. If not treated promptly, this imbalance in serum composition might result in serious damage to kidneys and irreversible renal failure. TLS is typically thought of as a lymphoma and leukemia consequence. Solid tumors, such as breast cancer, can also induce TLS, though they are uncommon.

Monitoring serum urea, potassium, phosphate, and uric acid levels as well as maintaining a sufficient urine output volume are important preventative measures for this illness. Large tumors and rapidly responding to therapy, including chemotherapy, produce dynamic alterations in the tumor immune milieu that differ depending on subtype and pathological response. The immune response during therapy is more predictive of treatment outcome than immunological characteristics in paired baseline samples, despite their substantial association. TLS can be caused by a variety of causes, including metastatic illness, increased azotemia, hyperuricemia, and high LDH concentrations.(20)

Proper hydration is essential for both TLS prevention and treatment. Loop diuretics can reduce urine production and potassium secretion when used in moderation. Since urine alkalization can result in kidney damage, it is no longer advised. Uric acid-lowering medications like allopurinol or rasburicase are crucial for the treatment of TLS (21).

2. Renal insufficiency brought on by treatment for breast cancer

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Side effects of breast cancer treatment may include renal failure. Endocrine therapy has played a crucial role in the prevention and treatment of breast cancer. Recent guidelines recommend the use of aromatase inhibitors and antiestrogens before menopause, as hormone therapy is a primary and effective treatment for estrogen receptor-positive breast cancer, one of the most common types of cancer in women worldwide. (22). Ovarian hyperstimulation syndrome (OHSS) is a consequence of infertility treatments. Common effects include hypercoagulability and decreased kidney function. When HCG or GnRH stimulants are used for viable egg retrieval, ovarian hyperstimulation syndrome (OHSS) may develop. Special ovarian stimulation methods should be used to minimize the risk(23).

Large volumes of intravascular volume depletion and third-space fluid retention, intraabdominal hypertension, compartment syndrome, and obstructive uropathy can all be brought on by OHSS. Acute renal damage can result from any of these disorders (24).Letrozole and anastrozole are examples of aromatase inhibitors, which are another class of drugs used in hormonal therapy. Case studies indicate that some medications may have adverse effects on the kidneys. For instance, anastrozole has been connected to GN, despite its rarity. These patients had both crescentic and sclerosing GN. Renal function may be restored by oral corticosteroids (25, 26).

An additional uncommon anastrozole adverse effect is Henoch-Schoenlein purpura. HSP regresses in two weeks when anastrozole is stopped without receiving any additional treatment (27). Patients using aromatase inhibitors run the risk of suffering renal damage since these drugs might cause and/or worsen hypercalcemia.(28,29) Anastrozole, in particular, is linked to enhanced renal tubular damage characteristics, according to a recent investigation conducted on female rats (30).

The SERMs include raloxifene and tamoxifene, which are vital drugs for treating breast cancer and preventing it in high-risk individuals (31).

Renal insufficiency patients often tolerate SERSMs without dosage modification (32). Furthermore, SERMs have the ability to decrease calcium and phosphate levels as well as rectify hypercalcemia brought on by cancer or treatment regimens, protecting the kidneys from elevated serum calcium levels. SERMs protect the kidneys against high serum calcium concentrations, while also lowering phosphate and calcium levels and perhaps correcting hypercalcemia caused by cancer or treatment regimens (33,34). Further research is required to elucidate the specific impact; however, preliminary data suggest that selective estrogen receptor modulators (SERMs) may have renoprotective and potentially anti-fibrotic effects that could be advantageous for patients undergoing treatment for breast cancer(35,36).

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Agents used in chemotherapy Alkylating substances Treatment for triple-negative breast cancer is beneficial when using platinum drugs, such as carboplatin and cisplatin (37). Due to renal tubular and vascular damage, these medications can significantly lower serum potassium and magnesium levels and cause a drop in GFR. This might result in acute or chronic kidney insufficiency. Kidney damage risk is decreased when renoprotective medications are taken in conjunction with adequate hydration (38). Cyclophosphamide: This medication is frequently used to treat breast cancer that has spread or returned (39).

Interstitial edema, cortical tubular vacuolization, and glomerular nephritis can all be brought on by cyclophosphamide metabolites. Hemorrhagic cystitis and bladder inflammation are additional adverse symptoms that make diagnosing and treating renal damage more difficult. When administering cyclophosphamide, it is advisable to incorporate an antioxidant or renoprotective agent into the therapeutic regimen (40,41).

Benzodiazepines Doxorubicin, epirubicin, and other anthracyclines are commonly used as adjuvant chemotherapy for breast cancer, and it is suggested to use more than one drug association checker database to find drug-drug interactions in, and it is suggested to use more than one drug association checker database to find drug-drug interactions in cancer patient treatment. (42). Due to oxidative stress, anthracyclines can result in glomerular and tubular damage as well as an abrupt increase in serum urea and creatinine (43, 44). The use of some compounds as a treatment has shown antioxidant and anti-inflammatory activity and thus positive results have been achieved in improving kidney function. In clinical studies, some substances used in antioxidant therapy have shown protective effects, while other substances have not given any results in treating kidney diseases. (45,46).

Antemetabolites Gemcitabine and capecitabine, which are pyrimidine analogs, are used to treat metastatic breast cancer. 95% of the time, capecitabine and its metabolites are eliminated in the urine. It is usually well tolerated by people with normal or slightly impaired renal function and has no notable negative effects on the kidney. Patients with advanced renal impairment require a change in capecitabine dosage.

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There have been isolated reports of capecitabine-induced nephrotoxicity, which is entirely curable by stopping the medication (47,48). Renal insufficiency brought on by gemcitabine has been documented more frequently. Gemcitabine nephrotoxicity is linked to microangiopathic hemolytic anemia, thrombocytopenia, and hypertension. When thrombocytopenia, anemia, or hypertension appear or worsen in a gemcitabine-treated patient, thrombotic microangiopathies should be suspected. There is always a good prognosis. Gemcitabine must be stopped as soon as possible in order to restore at least some renal function (49,51).

An inhibitor of dihydrofolate reductase, methotrexate (MTX), is sometimes used to treat breast cancer in addition to other chemotherapeutic medications, or following radiation therapy or surgery (52). Damage to renal tubules may result from MTX and its metabolites. The degree of injury from methotrexate (MTX) is directly proportional to both the plasma concentration and the infusion rate of the drug. Risk factors for MTX-induced nephrotoxicity include low serum albumin levels, male gender, and concomitant use of medications such as furosemide that inhibit MTX. Given that 90% of MTX and its metabolites are excreted via urine, renal impairment from MTX increases the risk of adverse effects by diminishing MTX clearance. Reducing the infusion rate, alkalinizing the urine, and ensuring adequate hydration are effective strategies for mitigating MTX-induced renal damage(53,54).

A semisynthetic vinca alkaloid called vinorelbine prevents cell division and tubulin polymerization. Whether used as first-line therapy or for metastatic or recurring breast cancer, many individuals with the disease found it to be a beneficial treatment (55,56). Vincristine (60) is an additional medication that proves effective in the advanced stages of breast cancer.Vinorelbine and vincristine generally alter neurohypophysis, which leads to improper antidiuretic hormone (SIADH) secretion. One to two weeks after taking vinca alkaloids, SIADH may manifest (57). There have been occasional reports linking the use of vincristine to hemolytic uremic syndrome (58). In a few trials, about 22% of patients receiving cisplatin and vinorelbine together experienced grade 1/2 nephrotoxicity. Whether vinorelbine, cisplatin, or both are at blame for these side effects is unclear (59,60).

Agents antimicrobial In late metastatic cancer, taxanes like paclitaxel and docetaxel are frequently used either by themselves or in conjunction with other medications. Adjuvant and neoadjuvant therapies can also benefit from these (61). Although there is limited data on paclitaxel nephrotoxicity, several studies recommend care due to the drug's potential kidney danger (62). Docetaxel is a taxane that cytochrome P450 metabolizes in the liver.

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Usually, metabolites of it are excreted in feces and detected in bile. Even for patients with renal impairment, dacatetaxel is a safe option due to its kidney-independent pharmacokinetics. Those with a lower GFR do not require a change in docetaxel dosage. A few occurrences of unexpected acute tubular injury have been documented after using docetaxel. Unknown is the process by which these injuries arise (63,64). A plausible rationale could be the activation of apoptosis and oxidative stress in kidney cells subsequent to docetaxel administration. Selenium, via lowering oxidative stress, can reduce docetaxel's nephrotoxic effects.

One common negative effect of using PARPis is an increase in serum creatinine. Rather than a decrease in glomerular filtration rate, renal transporter inhibition is the reason for this increase in serum creatinine. It is dose-dependent and reversible.(65).

In addition to the above treatments, bisphosphonate therapy helps reduce bone deterioration in metastatic disease by inhibiting and preventing active osteoclasts. (66).

A sizable fraction of all acute kidney injuries and chronic kidney disorders are caused by a large category of diseases collectively known as drug-induced kidney disease. Tubular damage is caused by bisphosphonates, particularly the more strong one zoledronate. Pamidurat treatment may cause kidney damage mainly by focal segmental glomerulosclerosis following severe stages of nephrotic syndrome. Patients getting IV forms and greater dosages are more likely to experience these renal adverse effects. Because the drug's intravenous form is recommended for malignancy rather than osteoporosis, bisphosphonate nephrotoxicity has not been documented in individuals treated for osteoporosis with oral medicines. To stop renal impairment from getting worse, blood creatinine and urine albumin levels should be checked often(67).

In terms of acute kidney injury, ibandronate is thought to be less harmful than other bisphosphonates. This is reasonable in smaller dosages. Nonetheless, there is less of a difference in the nephrotoxicity of isoniabandrate and other bisphosphonates in patients with breast cancer, who frequently require greater doses of these drugs (68).

Conventional chemotherapeutic agents can cause damage to nearly all nephron compartments—tubules, interstitium, vasculature, and glomerulus—resulting in acute kidney injury (AKI) through various mechanisms. Similarly, targeted therapies such as bevacizumab (Avastin) and emerging immunotherapies can induce a syndrome characterized by AKI, proteinuria, and hypertension(69).

Figure 4: Acute kidney injury (AKI) in oncology patients that is not attributable to oncological treatments.

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Another component of the treatment plan for breast cancer that enhances results and raises patient survival is radiation therapy. Radiotherapy for breast cancer uses a variety of approaches. The best prognosis is being achieved with continuously evolving techniques. The gold standard for treating breast cancer with radiation therapy combines targeted nodal irradiation with whole breast radiotherapy. For certain individuals, partial breast irradiation is the alternative (70,71).

Patients with breast cancer have a decreased risk of developing radiation nephritis than those with other malignancies because radiation is typically restricted to the breast and axillary lymph nodes. Rules are changing, nonetheless, because of metastatic breast cancer. Patients with the disease that has spread to the abdominopelvic region may require symptomatic radiation therapy (72.73).

Under these circumstances, radiation therapy to the kidneys appears to be a viable option for treating breast cancer and ought to be taken into account.

Before the patient experiences the symptoms of radiation nephritis, there is a 6-month latent phase. Clinicians should therefore be aware of this potential or else they may fail to identify the underlying cause of nephritis in some patients. (74).

The acute phase, several months after radiotherapy, is when radiation nephritis first appears clinically. After a year and a half, chronic radiation nephritis can develop. After radiotherapy, including the effect on kidney function, DNA damage, inflammation, fibrosis and RAAS activation

minimizing radiation exposure for kidney function, and lowering the dose of angiotensin-converting enzyme inhibitors (ACEIs) and ARBs to prevent radiation nephritis (75, 76).

3. Patients with impaired renal function who develop breast cancer

When receiving therapy for breast cancer, patients with chronic renal disease may find it difficult to manage their health.Determining the potential link between CKD and breast cancer is also necessary. Here, we made an effort to draw attention to the main issues surrounding the occurrence, progression, and management of patients with lower GFR who had breast cancer. Numerous pieces of data point to a higher cancer risk among CKD patients. Such patients may have a greater incidence of cancer because of higher levels of oxidative stress, toxin buildup, and chronic inflammation (77.78).

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However, when we narrow down the primary cancer types in the CKD group, we discover that there is no detectable increased risk for breast cancer, and, curiously, it is less common among female CKD patients than in the overall female population. This paradoxical outcome can be explained by a shorter lifespan and an underestimation of the risk. Future research and larger studies may provide light on the precise involvement of CKD in the development of breast cancer. The current breast cancer screening guidelines for those with chronic renal disease are similar to those for the general population (79.80). Breast cancer outcomes are also influenced by CKD. It is a risk factor for death that exists independently of all other cancers, including the cancer of the breast (81.82). In patients with stage IV breast cancer, Ishii T et al. established a link between CKD and increased mortality (83). Patients with breast cancer are more likely to experience potentially deadly consequences such venous thromboembolism (VTE) when they have renal insufficiency. Physicians must to take this elevated risk into account, especially prior to initiating endocrine medication (84).

Cancer-associated thrombosis arises from multiple interconnected mechanisms (85):

1. Hypercoagulability: Cancer cells release pro-coagulant factors like tissue factor (TF), activating the coagulation cascade and leading to a pro-thrombotic state. Elevated levels of these factors, including soluble tissue factor, are linked to malignancies.

2. Endothelial Dysfunction: Cancer-related inflammation and cytokine release damage endothelial cells, increasing thrombus formation. Elevated inflammatory cytokines such as IL-6 and TNF-α are associated with endothelial activation and enhanced thrombotic risk.

3. Platelet Activation: Tumor cells interact with platelets, leading to their activation and aggregation. This process involves platelet surface receptors and tumor-derived factors that promote thrombus formation.

4. Altered Blood Flow: Tumor growth or metastasis can obstruct or compress blood vessels and alter blood flow dynamics, increasing thrombotic risk in both small and large vessels.

5. Immune System Activation: Cancer can stimulate immune responses that affect coagulation pathways, contributing to a pro-thrombotic environment through immune mediators and activated immune cells.

Figure 5: Pathogenesis of cancer - associated Thrombosis

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A patient's GFR must be taken into consideration while adjusting the dosage of chemotherapy drugs to minimize their adverse effects on the kidneys and extrarenal areas. This paper does not address the appropriate pharmacological modification for any of these drugs. It is highly advised that patients have their GFR assessed both before and during their course of medication. The secret to preventing undesired medication removal is timing adjustments between dialysis sessions and drug intake (86).

Conclusion

Several factors must be addressed when investigating the relationship between breast cancer and renal insufficiency. The interplay between breast cancer and renal insufficiency encompasses multifaceted pathophysiological mechanisms that significantly impact clinical management and patient prognosis. Breast cancer patients are at heightened risk for renal complications such as hypercalcemia, tumor lysis syndrome (TLS), and paraneoplastic renal syndromes, each of which can contribute to or exacerbate renal dysfunction. Hypercalcemia, driven by paraneoplastic mechanisms and osteolytic metastases, can impair renal function through calcium overload and subsequent renal tubular damage. TLS, though more commonly associated with hematologic malignancies, poses a risk in breast cancer patients with rapid tumor lysis, leading to acute renal failure due to elevated serum phosphate, uric acid, and potassium. Furthermore, paraneoplastic syndromes, including glomerulonephritis and nephrotic syndrome, can arise as direct or indirect consequences of malignancy, further complicating renal function. Given these complexities, it is crucial to integrate baseline glomerular filtration rate (GFR) assessments into the treatment planning for breast cancer patients. This approach enables tailored therapeutic interventions that mitigate the risk of renal adverse effects while optimizing cancer management. Regular monitoring of renal function throughout the course of treatment and prompt adjustment of therapeutic regimens in response to any decline in GFR are essential strategies to prevent or manage renal failure. Additionally, breast cancer patients with newly diagnosed chronic kidney disease (CKD) exhibit increased morbidity and mortality, highlighting the urgent need for ongoing research to elucidate the mechanisms linking CKD with breast cancer and to develop targeted strategies to enhance patient outcomes. This comprehensive approach aims to balance effective cancer treatment with the

ISSN 3063-8186. Published by Universitas Muhamamdiyah Sidoarjo

https://doi.org/10.21070/ijhsm.v2i1.61

preservation of renal function, ultimately improving survival and quality of life for patients facing this challenging dual diagnosis.

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