

## SQUAMOUS CELL CARCINOMA PRESENTING AS TUMOR LYSIS SYNDROME

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### KEYWORDS :

#### CASE REPORT :

68 year old female, known case of CVA and hypertension on medication, presented with loose stools, abdominal distension and bilateral lower limb swelling for the past one week. Lump in right breast, insidious in onset gradually progressed to attain present size. No other complaints and comorbidities. On examination patient is oriented with hypotension [blood pressure of 60/40 mm of Hg], bilateral pedal edema and pallor is present with no obvious axillary lymphadenopathy. Local examination of breast reveals hyperkeratotic patches with ulceroproliferative lesion of 8\*8cm with necrotic and granulation tissue over upper and lower outer quadrant of right breast associated with foul smelling purulent discharge [figure 1]. Patient is treated with i.v fluids, inotropes, intravenous antibiotics, albumin, 4 units of packed cell transfusion and other supportive measures. With persistent hypocalcemia, raised uric acid, phosphate and potassium levels. With suspicious mass over right breast provisional diagnosis of tumor lysis syndrome is made, hence patient is started on tab. febuxostat 40mg od. Edge biopsy taken from lesion and sent for histopathological examination. HPE section shows extensive areas of necrosis and small fragments of hyperkeratotic, hyperplastic and papillomatous epidermis overlying dense inflammatory cell collection with pigment laden macrophages in dermis, epidermis shows nests and islands of vacuolated cells with enlarged nucleus showing prominent nucleoli. One fine fragment shows islands of squamous cells with pleomorphic nuclei and mitotic figures with tumor giant cells seen, features suggestive of squamous cell carcinoma. Based on clinical, laboratory parameters and microscopic features diagnosis of Squamous cell carcinoma presenting as Tumor lysis syndrome is made. Patient underwent bilateral mastectomy, On follow up, patient recovered in clinical and laboratory parameters with aforementioned treatment.



**Figure 1:** shows hyperkeratotic patches with ulceroproliferative lesion of 8\*8cm with necrotic and granulation tissue over upper and lower outer quadrant of right breast.

#### DISCUSSION :

Tumor lysis syndrome (TLS) is a medical emergency that occurs in patients with certain cancers and is caused by the rapid and massive breakdown of tumor cells, either spontaneously, or after the initiation of cytoreductive therapy. The rapid release of intracellular contents causes hyperuricemia, hyperkalemia, hyperphosphatemia, and secondary hypocalcemia. These metabolic abnormalities can lead to significant morbidity, putting patients at risk of severe clinical consequences that include acute kidney injury (AKI), cardiac arrhythmias, pulmonary edema, fluid overload, seizures, and even death [1]. Although it can occur spontaneously, TLS is associated most commonly with rapid cell breakdown after initiation of chemotherapy and the consequent massive release of intracellular contents that overwhelms the body's homeostatic and excretory mechanisms. Potassium is stored primarily in the intracellular compartment, and its rapid and excessive release into the extracellular compartment can lead to hyperkalemia, cardiac arrhythmia, and possibly sudden death. Similarly, hyperphosphatemia results from massive release of intracellularly stored phosphate. It can lead to secondary hypocalcaemia and deposition of calcium phosphate crystals in the kidney [1]. Tumors most frequently associated with TLS are clinically aggressive non-Hodgkin lymphomas (NHLs) and acute lymphoblastic leukemia (ALL), particularly Burkitt lymphoma/leukemia [2,3,4-8]. Other hematologic malignancies that are commonly associated with TLS include anaplastic large cell lymphoma, T-cell or B-cell precursor ALL, acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), and plasma cell disorders. TLS has been rarely described after treatment of some nonhematologic solid tumors [9,10]. These include breast cancer [11-13]. Spontaneous TLS is associated with marked hyperuricemia prior to the initiation of therapy has been described in lymphomas and acute leukemias [14,15]. It usually occurs in patients with bulky, rapidly proliferating, treatment-responsive tumors and has been described in inflammatory breast cancer [16]. The actual incidence of this syndrome is difficult to ascertain. Interestingly, spontaneous TLS is associated with hyperuricemia but frequently without hyperphosphatemia. It has been postulated that rapidly growing neoplasms with high cell turnover rates produce high serum uric acid levels through rapid nucleoprotein turnover but that the tumor is able to reutilize released phosphorus for resynthesis of new tumor cells. In contrast, TLS after chemotherapy is due to cell destruction in the absence of reuptake of phosphorus, and thus, hyperphosphatemia. Effective management involves the combination of treating specific electrolyte abnormalities, and/or acute renal failure, and the use of allopurinol, a xanthine oxidase inhibitor administered to reduce the

conversion of nucleic acid byproducts to uric acid in order to prevent urate nephropathy and subsequent oliguric renal failure [17].

### CONCLUSION:

Tumor lysis syndrome (TLS) is an oncologic emergency that is caused by massive tumor cell lysis. It can occur after initiation of cytotoxic therapy, but also can occur spontaneously. TLS should be included in the differential diagnosis of patients with breast cancer who present with acute kidney injury even if they did not receive chemotherapy. Here, we report a rare case of squamous cell carcinoma of breast presenting as tumor lysis syndrome

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