Vol 7 No 3 August 2026

E-ISSN:2722-0877

RESEARCH ARTICLES

Comparison of the incidence of blood disorders in urological cancer patients undergoing chemotherapy at Dr. Pirngadi Hospital, Medan City

Hasroni Fathurrahman[1], Syahril Ananda Siregar[2]

Medical Education, Faculty of Medicine, University of Muhammadiyah North Sumatra [1,2]

Email correspondence : s.nanda1112002@gmail.com

ABSTRACT

Introduction: Blood disorders such as anemia, leukopenia, and thrombocytopenia are common in urological cancer patients undergoing chemotherapy. This study aims to compare the incidence of blood disorders between patients with bladder cancer, testicular cancer, prostate cancer, and penile cancer who undergo chemotherapy at Dr. Pirngadi Hospital Medan City. **Methods:** This study used a retrospective design with comparative analysis. Data were taken from the medical records of patients who underwent chemotherapy during the 2019-2023 period. The sample consisted of 72 patients, namely 50 bladder cancer patients, 10 testicular cancer patients, 7 prostate cancer patients, and 5 penile cancer patients. Data were analyzed using descriptive statistical tests and analysis. Kruskal-Wallis Test. **Results**: showed that a decrease in hemoglobin was the most common blood disorder, especially in penile (100%) and bladder (84.6%) cancers. Leukopenia is rare, while leukocytosis is more commonly found, especially in penile cancer (60%). Platelet disorders were dominant in bladder cancer, with a decrease and increase of 12.8% each. The Kruskal-Wallis test showed no significant difference in the decrease in hemoglobin, leukocytes, or platelets between types of urological cancer ($p > 0.05$), except for an increase in hemoglobin which showed a significant difference ($p < 0.05$). **Conclusions:** there was no significant difference in blood disorders between types of urological cancer, although haematological monitoring remains important to prevent chemotherapy complications.

Keywords: *Blood disorders, urological cancer, chemotherapy*

INTRODUCTION

Urological cancers are a group of malignant diseases that attack the urinary system and male reproductive organs, including cancers of the prostate, bladder, kidneys, testicles, and penis. Prostate cancer

E-ISSN:2722-0877

is the second most common type of cancer in the world, with about 1.4 million new cases each year. Bladder cancer is also significant, with 573,000 new cases and 213,000 deaths worldwide in 2020.[1] Testicular cancer is the main tumor that appears in young men, with an incidence of 3-11 cases per 100,000 men per year.[2] Prostate cancer is one of the most common cancers in men globally, with 1.41 million new cases and 375,304 deaths in 2020, accounting for 14.1% of all new cancer cases in men.[3] Meanwhile, penile cancer, although less common, has an age-adjusted incidence rate (ASIR) of 0.8 per 100,000 population, with 36,068 new cases, and caused 13,211 deaths worldwide, with an age-adjusted mortality rate (ASMR) of 0.29 per 100,000 population in the same year. [4]

The pathophysiology of each type of urological cancer shows complexity due to the presence of genetic changes and specific biological mechanisms. Prostate cancer begins with an intraepithelial neoplasia of the prostate (PIN) that can progress to adenocarcinoma, triggered by hormonal and genetic factors. [5,6,7] Bladder cancer generally originates in the urothelium with mutations in certain genes, spreading from the non-invasive stage to invasion into deeper tissues. [1] Testicular cancer develops from undifferentiated germ cells, with risk factors such as cryptorchidism playing a role in malignant transformation. [8] Penile cancer, which is mostly squamous cell carcinoma, is linked to HPV infection, inflammatory conditions, and poor genital hygiene.[9,10] Each of these types of cancer requires a different approach to diagnosis and treatment based on its stage and biological characteristics.

Chemotherapy is an important method for the management of urological cancer, designed to destroy rapidly dividing cancer cells, but it can also affect healthy cells, including blood cells. Some of the commonly used chemotherapy regimens are Gemcitabine, Cisplatin, Etoposide, Bleomycin, Docetaxel, and Methotrexate. Gemcitabine and Cisplatin work by inhibiting DNA synthesis as well as forming cross-links that interfere with cell replication, while Etoposide and Bleomycin trigger DNA damage that induces cancer cell apoptosis.[11,12] Docetaxel interferes with the function of microtubules, inhibiting mitosis, whereas Methotrexate acts as a folate antagonist, inhibiting the synthesis of purines and pyrimidines that are essential for DNA replication. [13,14,15,16]

Common side effects of these various chemotherapy regimens include myelosuppression, which can lead to anemia, leukopenia, and thrombocytopenia. Anemia can be the result of decreased red blood cell production, while leukopenia increases the risk of infection, and thrombocytopenia contributes to increased bleeding. [17]

E-ISSN:2722-0877

Blood, as a vital component, plays a role in various bodily functions, including the transport of oxygen through red blood cells and protection against infection by white blood cells. Hematopoiesis, the process of blood cell formation in the bone marrow, involves hematopoietic stem cells (HSCs) that differentiate into different types of blood cells. This process is controlled by growth factors and interactions with cellular niches that support the proliferation and differentiation of HSCs into red blood cells, platelets, and leukocytes. Disorders in hematopoiesis can affect overall health, causing serious hematologic problems. [18,19]

The pathophysiology of blood disorders due to chemotherapy involves myelosuppression, in which the use of cytotoxic agents can inhibit the production of blood cells in the bone marrow. This dysfunction leads to a decrease in the number of red blood cells, white blood cells, and platelets, resulting in anemia, leukopenia, and thrombocytopenia.[20] In addition, the toxicity of chemotherapy can induce difficulties in the regulation of the factors involved in hematopoiesis, exacerbating the risk of infection and bleeding. These disorders create additional challenges in the management of cancer patients, requiring extra attention to support the patient's hematological and overall health during the treatment journey.

Comparative studies related to blood disorders between types of cancer are still rare. This study aims to evaluate and compare blood disorders in urological cancer patients to support improved clinical management.

METHODS

This study uses a descriptive analytical design with quantitative, observational analytical, and cross-sectional approaches. Samples were taken non-probabilistic from the medical records of urological cancer patients undergoing chemotherapy at Dr. Pirngadi Hospital Medan during January 2019–December 2023. Of the 84 patients who met the criteria, 59 were analyzed: 39 bladder cancers, 10 testicles, 5 prostate and 5 penises. Based on this time span there were 115 patients undergoing urological cancer chemotherapy, however, only 84 medical records were available for analysis. Of the available medical records, there are 59 medical records that meet the inclusion and exclusion criteria: 39 bladder cancers, 10 testicles, 5 prostate, and 5 penises

The research sample was selected based on inclusion criteria in the form of patients with a diagnosis of urological cancer who have undergone the first cycle of chemotherapy at Dr. Pirngadi Hospital Medan City and have complete medical records including diagnosis, type and dose of chemotherapy, and laboratory results related to blood conditions. The exclusion

E-ISSN:2722-0877

criteria include urological cancer patients who have not undergone chemotherapy or have undergone more than one cycle, patients with incomplete medical records, and patients who died before the necessary data could be collected. Data collection methods were collected from patients' medical records, including the results of routine blood tests before and after chemotherapy. The hematological data taken included the number of hemoglobin, leukocytes, and platelets. Researchers recorded changes in the number of blood cells to analyze the incidence of anemia, leukopenia, and thrombocytopenia.

Data Analysis The results of the study were analyzed univariate and bivariate using the . Kruskal-Wallis Test. Univariate analysis was conducted to see the distribution of data based on research variables, while bivariate analysis was used to see the relationship between the type of urological cancer and the incidence of hematologic disorders. The data is presented in the form of tables and graphs for easy interpretation. The $p<0.05$ value is considered statistically significant

RESULTS

Most patients had low hemoglobin levels, with distributions in testicular cancer (70% low, 10% normal, 20% high), penile cancer (80% low, 20% normal), prostate cancer (all low), and bladder cancer (75.9% low, 24.1% normal). Overall, 77.6% of patients had low Hb, 18.4% normal, and 4.1% high. The highest hemoglobin levels were found in testicular cancer (12.57 g/dL), followed by penile (11.22 g/dL) and prostate cancer (10.90 g/dL), while the lowest levels were in bladder cancer (11.47 g/dL).

The distribution of leukocytes showed a majority of normal (63%), with cancers of the testicles (60% normal, 30% high), prostate (60% normal, 40% high), penile (60% leukocytosis), and bladder (69% normal, 31% leukocytosis) cancers. The highest leukocyte levels were found in penile cancer (11.85 x10^3/µL), followed by prostate cancer (10.65 x10^3/µL) and bladder (9.94 x10^3/µL), with the lowest levels in testicular cancer (8.61 x10^3/µL).

Platelet levels are mostly normal (78%), with testicular (80% normal), penile (80% normal, 20% thrombocytosis), prostate (all normal), and bladder (72% normal, 21% thrombocytosis, 7% thrombocytopenia). The highest platelet levels were found in bladder cancer (319.03 x10^3/µL), followed by the penis (298.00 x10^3/µL) and prostate (296.60 x10^3/µL). The lowest levels were in testicular cancer (214.33 x10^3/µL).

Most patients had normal urea levels (84%), with cancers of the testicles (all normal), penile (80% normal, 20% high), prostate (80% normal, 20% low), and bladder (79% normal, 21% high). The highest urea levels were found in penile cancer (33.80 mg/dL), followed by bladder (31.33 mg/dL) and prostate (23.60 mg/dL). The lowest levels were found in testicular cancer (21.80 mg/dL).

Creatinine levels are mostly normal (69%), with cancers of the testicles (60% normal, 20% low, 20% high), penis (80% normal, 20% high), prostate (all normal), and bladder (66% normal, 31% high, 3% low). The highest creatinine levels were

E-ISSN:2722-0877

found in bladder cancer (1.069 mg/dL), followed by prostate (0.956 mg/dL) and penis (0.938 mg/dL), while the lowest was in testicular cancer (0.927 mg/dL).

Table 1. Distribution of Blood Parameters in Urological Cancer Patients

Parameters	Categories	Testicles	Penis	Prostate	Urinary
Hb	Low	7	4	5	22
	Normal	1	1	0	7
	Height	2	0	0	0
Leukocytes	Low	1	0	0	0
	Normal	6	2	3	20
	Height	3	3	2	9
Platelet	Low	2	0	0	2
	Normal	8	4	5	21
	Height	0	1	0	6
Urea	Low	0	0	1	0
	Normal	10	4	4	23
	Height	0	1	0	6
Creatinine	Low	2	0	0	1
	Normal	6	4	5	19
	Height	2	1	0	9

Table 2. Descriptive Statistics of Urological Blood Parameters

Parameters		Testicles	Penis	Prostate	Urinary
Hb	Mean (g/dL)	12.57	11.22	10.90	11.47
	Median	12.50	11.20	11.10	11.60
	Min - Max	8.50 - 16.80	9.90 - 13.53	9.45 - 12.60	6.90 - 14.40
	SD	2.63	1.41	1.29	1.84
leukocytes	Mean ($10^3/\mu L$)	8.61	11.85	10.65	9.94
	Median	7.60	11.32	8.46	8.50
	Min - Max	4.07 - 15.54	5.63 - 21.71	6.93 - 17.05	3.46 - 26.61
	SD	3.49	6.05	4.36	5.24
platelet	Mean ($10^3/\mu L$)	214.33	298.00	296.60	319.03
	Median	223.50	272.00	261.00	298.00
	Min - Max	35 - 357	192 - 439	204 - 385	104 - 563
	SD	104.69	91.30	81.41	113.50
Urea	Mean (mg/dL)	21.80	33.80	23.60	31.33
	Median	25.00	26.00	20.00	28.00
	Min - Max	10 - 34	22 - 64	9 - 42	12 - 73
	SD	9.00	17.27	12.50	17.21
Creatinine	Mean (mg/dL)	0.927	0.938	0.956	1.069
	Median	0.745	0.840	1.000	0.950
	Min - Max	0.54 - 2.19	0.68 - 1.36	0.83 - 1.02	0.51 - 2.27
	SD	0.49	0.28	0.08	0.44

Based on the results of the Kruskal-Wallis test, p-value analysis showed that there was no significant difference in hemoglobin, leukocyte, and platelet levels between types of urological cancer in patients undergoing chemotherapy. The hemoglobin variable has a p-value of 0.553, which is greater than 0.05, so there is no significant difference, and the null hypothesis (Ho1) is accepted. For the leukocyte variable, the p-value of 0.578, which also exceeds 0.05, indicates that the difference

E-ISSN:2722-0877

insignificant, so Ho2 is still accepted. The same is true for platelet variables with a p-value of 0.231, which is still above the significance limit, so Ho3 is accepted. Thus, overall, insufficient statistical evidence was found to conclude that there were significant differences in hemoglobin, leukocyte, and platelet levels based on the type of urological cancer in patients undergoing chemotherapy.

Table 3. Kruskal-Wallis Table comparison of blood disorders in chemotherapy urological cancerUrology

Types of Cancer	N	Mean Rank (kat_leukosit)	Rank (kat_trombosit)	Rank (kat_hb)
Testicles	10	27.65	22.60	19.30
Penis	5	24.20	31.40	28.00
Prostate	5	19.50	26.60	23.50
Urinary	29	25.17	24.45	26.71
Total	49	-	-	-

Variable	Kruskal-Wallis H	df	Asymp. Sig.
Hemoglobin	2.094	3	0.553
Leukocytes	1.972	3	0.578
Platelet	4.300	3	0.231

Note:a = Kruskal-Wallis Testb = Grouping variable: type of cancer

DISCUSSION

Hemoglobin

Most urological cancer patients undergoing chemotherapy have anemia, with a prevalence of 78.2%. Prostate cancer has the highest rate of anemia (100%), followed by cancers of the penis (82%), bladder (76.5%), and testicles (72%). This anemia is mainly caused by the toxic effects of chemotherapy on the bone marrow, nephrotoxicity that inhibits the production of erythropoietin, as well as inflammation due to cancer that triggers chronic disease anemia.[21]

In testicular cancer, 22% of patients had high hemoglobin levels, which can be caused by the production of ectopic erythropoietin by tumors or the rebound effects of bone marrow regeneration after the suppression phase due to chemotherapy. This variability can also be attributed to the younger age of the patient, which allows for better recovery of hematopoiesis.[22]

Penile cancer shows a 100% prevalence of anemia, possibly due to tumor bleeding, the effects of chemotherapy on hematopoiesis, as well as secondary infections that increase the production of hepcidin and inhibit the release of iron for erythropoiesis.[23]

In prostate cancer, anemia occurs in 100% of patients and can be exacerbated by androgen deprivation therapy (ADT), which suppresses erythropoietin production and inhibits the proliferation of erythroid progenitor cells. The anemia that occurs is

E-ISSN:2722-0877

usually normocystic, normochromic, and progressive.[24,25]

Bladder cancer has the lowest hemoglobin levels (average 11.35 g/dL) with a wide range (6.85–14.50 g/dL). This variability reflects differences in disease stages and individual responses to therapy. Platinum-based chemotherapy, such as cisplatin, has nephrotoxic effects that inhibit the production of erythropoietin as well as myelotoxic effects that suppress erythropoiesis. [26]

Bladder cancer has the lowest hemoglobin levels (average 11.35 g/dL) with a wide range (6.85–14.50 g/dL). This variability reflects differences in disease stages and individual responses to therapy. Platinum-based chemotherapy, such as cisplatin, has nephrotoxic effects that inhibit the production of erythropoietin as well as myelotoxic effects that suppress erythropoiesis.

Leukocytes

Most urological cancer patients undergoing chemotherapy had normal leukocyte levels (63%) while 35% developed leukocytosis and 2% developed leukopenia. Leukopenia is most commonly found in testicular cancer (10%) and bladder cancer (7.7%), while in prostate and penile cancers there are no cases of low leukocytes.

Leukopenia in testicular and bladder cancers is caused by the myelotoxic effects of cisplatin, which inhibits the proliferation of progenitor cells in the bone marrow.[27] In testicular cancer, chemotherapy regimens based on bleomycin, etoposide, and cisplatin (BEP) also contribute to a decrease in leukocytes.[28] Docetaxel, which is used in prostate cancer, has a milder bone marrow suppression effect than cisplatin, resulting in a lower incidence of leukopenia. The use of Granulocyte-Colony Stimulating Factor (G-CSF) as supportive therapy helps prevent severe neutropenia.[29]

In contrast, leukocytosis is more common than leukopenia, especially in penile cancer (60%), followed by prostate cancer (40%), bladder cancer (31%), and testicular cancer (30%). This increase in the number of leukocytes is generally related to a systemic inflammatory response due to tumor tissue necrosis or secondary infection. [30] In prostate and bladder cancer, urinary tract infections are often a triggering factor for leukocytosis. In testicular cancer, bleomycin is known to cause systemic inflammatory syndrome, which contributes to an increase in the number of leukocytes. [31]

Statistically, the highest average leukocyte levels were found in penile cancer ($11.85 \times 10^3/\mu L$), followed by prostate ($10.65 \times 10^3/\mu L$) and urinary ($9.94 \times 10^3/\mu L$) cancers, while testicular cancer had the lowest average leukocytes ($8.61 \times 10^3/\mu L$). The widest range of leukocyte values was found in bladder cancer ($3.46–26.61 \times 10^3/\mu L$), showing greater variation in immune response and chemotherapy effects than other types of cancer.

E-ISSN:2722-0877

Platelet

Most urological cancer patients undergoing chemotherapy had normal platelet levels (78%). Thrombocytopenia is more common than thrombocytosis, especially in testicular (20%) and bladder (21%) cancers. There were no cases of low platelets in prostate and penile cancers, and high platelets were only found in bladder cancer (21%). Thrombocytopenia is generally caused by cisplatin's toxicity to megakaryocytes, and in bladder cancer it can be aggravated by the infiltration of tumor cells into the bone marrow. [32] Patients undergoing chemotherapy with a high-dose regimen are more at risk of significant platelet decreasing, thereby increasing the potential for bleeding. Therefore, regular monitoring of platelet count is essential during therapy.

Meanwhile, thrombocytosis is less common, found in only 21% of bladder cancer patients. This increase in platelet count is likely due to paraneoplastic mechanisms, in which tumors stimulate the production of thrombopoietin or trigger a systemic inflammatory response, leading to reactive thrombocytosis. This thrombocytosis is often temporary and can occur during the recovery phase after a chemotherapy cycle.[33]

Statistically, the highest average platelet levels were found in bladder cancer patients (319.03 × 10^3/μL), followed by penile (298.00 × 10^3/μL) and prostate (296.60 × 10^3/μL), while testicular cancer patients had the lowest average platelets (214.33 × 10^3/μL). The widest range of platelet values was found in bladder cancer patients (104–563 × 10^3/μL), reflecting greater variation in response to chemotherapy and tumor effects than in other types of cancer.

Urea and creatinine

Urea and creatinine are two important parameters in assessing kidney function in urological cancer patients undergoing chemotherapy. Urea is a product of protein breakdown produced by the liver and excreted through the kidneys, while creatinine is a byproduct of muscle metabolism that is also filtered by the kidneys.[34] Elevated urea and creatinine levels often indicate decreased kidney function, which can be caused by direct kidney damage due to nephrotoxic side effects of chemotherapy drugs, dehydration, or infection. In contrast, low levels are rarely a major concern, although they can indicate metabolic problems such as malnutrition.[35]

During chemotherapy, kidney damage or disturbance of the body's fluid balance can inhibit the kidneys' ability to filter body waste, leading to increased levels of urea and creatinine in the blood.[35] High urea can interfere with the production of erythropoietin, a hormone that stimulates the formation of red blood cells, thus risking anemia in patients.[36] In addition, kidney damage can also affect the immune system, leading to a decrease in the number of

E-ISSN:2722-0877

leukocytes (leukopenia) and increasing its susceptibility to infection.[37] This is caused by the disruption of the kidneys' filtration process, which reduces the body's effectiveness in managing waste and maintaining fluid and electrolyte balance.

Increased levels of urea and creatinine can also contribute to a decrease in platelet counts (thrombocytopenia). Kidney disorders can alter the balance of body fluids and electrolytes, which play a key role in platelet function. In addition, chemotherapy itself can suppress the production of platelets in the bone marrow, worsening the condition of thrombocytopenia in patients.[38] Thus, fluctuations in urea and creatinine levels not only provide an idea of the patient's kidney function, but are also directly related to changes in hematological parameters such as hemoglobin, leukocytes, and platelets, which can affect the treatment and recovery of urological cancer patients.

Kruskal-Wallis Statistical Analysis

The results of the Kruskal-Wallis test showed no significant differences in hemoglobin, leukocyte, and platelet levels between types of urological cancer in chemotherapy patients ($p > 0.05$). This indicates that the variation in levels is more influenced by other factors such as the individual response, the patient's condition, or chemotherapy regimen, rather than the type of cancer. The myelosuppressive effects of chemotherapy appear to be similar in all groups.

This study refers to various relevant studies to support the discussion of hematological disorders in urological cancer patients undergoing chemotherapy. Based on the journal *Pharmacon* by Annisa Nabilah, hematological side effects such as anemia and leukopenia are complications that often occur during chemotherapy.[39] In addition, research from *Airlangga University e-journal* by Anna Febriani and Yuly Rahmawati emphasized that chemotherapy can cause immune thrombocytopenia, which risks triggering bleeding in cancer patients[21].

The results of this study reinforce the findings of various previous studies that showed that chemotherapy has toxic effects on the hematopoietic system. Thus, this study emphasizes the importance of regular hematologic monitoring during chemotherapy to minimize the risk of more serious complications.

CONCLUSION

Most urological cancer patients undergoing chemotherapy had low hemoglobin levels (77.6%), with prostate cancer showing the lowest hemoglobin levels (100% anemia), followed by penile cancer (80%), testicular cancer (70%), and bladder cancer (75.9%). The highest hemoglobin levels were found in testicular cancer (12.57 g/dL), and lowest in bladder cancer (11.47 g/dL). The majority of patients had normal leukocyte levels (63%), with the

E-ISSN:2722-0877

highest levels in penile cancer (11.85 × 10^3/μL) and lowest in testicular cancer (8.61 × 10^3/μL). Normal platelet levels were found in 78% of patients, with the highest levels in bladder cancer (319.03 × 10^3/μL) and lowest in testicular cancer (214.33 × 10^3/μL). Most patients had normal levels of urea (84%) and creatinine (69%), with the highest levels of urea in penile cancer (33.80 mg/dL) and creatinine in bladder cancer (1.069 mg/dL). Although there were no significant differences between urological cancer types in hemoglobin, leukocyte, and platelet levels ($p > 0.05$), monitoring of hematological parameters remains important to prevent chemotherapy complications such as anemia, leukopenia, or thrombocytopenia, with interventions tailored based on the patient's condition.

REFERENCES

1. Lopez-Beltran A, Cookson MS, Guercio BJ, Cheng L. Advances in diagnosis and treatment of bladder cancer. BMJ. 2024; 384:e076743.
2. Giona S. The Epidemiology of Testicular Cancer. Urol Cancers. 2022; 107–16.
3. Zhang W, Cao G, Wu F, Wang Y, Liu Z, Hu H, et al. Global Burden of Prostate Cancer and Association with Socioeconomic Status, 1990–2019: A Systematic Analysis from the Global Burden of Disease Study. J Epidemiol Glob Health [Internet]. 2023; 13(3):407–21. Available from: https://doi.org/10.1007/s44197-023-00103-6
4. Study P based, Fu L, Tian T, Yao K, Chen X feng, Luo G, et al. Global Pattern and Trends in Penile Cancer Incidence: Corresponding Author: ResearchGate. 2022; 8:1–15.
5. Murray TBJ. The Pathogenesis of Prostate Cancer. In: Bott SRJ, Ng KL, editors. Mandala of Health. Brisbane (AU); 2021.
6. Dorothy A. Shead M, Tanya Fischer, MEd M, Kidney S. Kidney cancer guidelines for patients English. Int kidney cancer Coalit. 2022;
7. De Silva F, Alcorn J. A Tale of Two Cancers: A Current Concise Overview of Breast and Prostate Cancer. Cancers (Basel). 2022; 14(12).
8. Katabathina VS, Vargas-Zapata D, Monge RA, Nazarullah A, Ganeshan D, Tammisetti V, et al. Testicular germ cell tumors: Classification, pathologic features, imaging findings, and management. Radiographs. 2021; 41(6):1698–716.
9. Bernhard MC, Zwick A, Mohr T, Gasparoni G, Khalmurzaev O, Matveev VB, et al. The HPV and p63 status in penile cancer are linked with the infiltration and therapeutic availability of neutrophils. Mol Cancer Ther. 2021; 20(2):423–37.
10. Sofiani VH, Veisi P, Rukerd MRZ, Ghazi R, Nakhaie M. The complexity of human papilloma virus in cancers: a

E-ISSN:2722-0877

narrative review. Infect Agent Cancer [Internet]. 2023; 18(1):1–14. Available from: https://doi.org/10.1186/s13027-023-00488-w

11. Hu J, Chen J, Ou Z, Chen H, Liu Z, Chen M, et al. Neoadjuvant immunotherapy, chemotherapy, and combination therapy in muscle-invasive bladder cancer: A multi-center real-world retrospective study. Cell Reports Med [Internet]. 2022; 3(11):100785. Available from: https://doi.org/10.1016/j.xcrm.2022.100785
12. Mirowski M. Drug Development. 2024;
13. Lawrence H. Development of Prostate Cancer Therapy. Contin med educ [Internet]. 2019; 46(8):521–7. Available from: https://journal.lppmunindra.ac.id/index.php/SAP/article/view/2731
14. Vale CL, Fisher DJ, Godolphin PJ, Rydzewska LH, Boher JM, Burdett S, et al. Which patients with metastatic hormone-sensitive prostate cancer benefit from docetaxel: a systematic review and meta-analysis of individual participant data from randomised trials. Lancet Oncol. 2023 Jul; 24(7):783–97.
15. Stecca CE, Alt M, Jiang DM, Chung P, Crook JM, Kulkarni GS, et al. Recent Advances in the Management of Penile Cancer: A Contemporary Review of the Literature. Oncol Ther. June 2021; 9(1):21–39.
16. Joshi VB, Chadha J, Chahoud J. Penile cancer: Updates in systemic therapy. Asian J Urol [Internet]. 2022; 9(4):374–88. Available from: https://www.sciencedirect.com/science/article/pii/S2214388222000315
17. Mitchell E, Pham MH, Clay A, Sanghvi R, Pietsch S, Hsu JI, et al. The long-term effects of chemotherapy on normal blood cells. bioRxiv [Internet]. 2024 Jan 1;2024.05.20.594942. Available from: http://biorxiv.org/content/early/2024/05/21/2024.05.20.594942.abstract
18. Seth J, Sharma S, Leong CJ, Vaibhav V, Nelson P, Shokravi A, et al. The Use of Hematopoietic Stem Cells for Heart Failure: A Systematic Review. Vol. 25, International Journal of Molecular Sciences. 2024.
19. Poller WC, Nahrendorf M, Swirski FK. Hematopoiesis and Cardiovascular Disease. Circ Res. 2020; 126(8):1061–85.
20. Lyman GH, Kuderer NM, Aapro M. Improving outcomes of chemotherapy: established and novel options for myelotherapy in the COVID-19 era. Front Oncol. 2021; 11(July):1–11.
21. Febriani A, Rahmawati Y. Hematological Side Effects Due to Chemotherapy and Its Management. J Respiration. 2019; 5(1):22.
22. Shevchenko JA, Nazarov K V, Alshevskaya AA, Sennikov S V. Erythroid Cells as Full Participants in the Tumor Microenvironment. Vol. 24, International Journal of Molecular Sciences. 2023.

E-ISSN:2722-0877

23. Rorimpandey NG, Rambert GI, Wowor MF. Overview of Interleukin 6 and Hepidin in Chronic Diseases That Can Cause Anemia. Med, Scope, J., 2023; 5(1):64–74.
24. Zhou W, Su Y, Zhang Y, Han B, Liu H, Wang X. Endothelial Cells Promote Docetaxel Resistance of Prostate Cancer Cells by Inducing ERG Expression and Activating Akt/mTOR Signaling Pathway. Front Oncol. 2020; 10(December):1–14.
25. Karim MU, Tisseverasinghe S, Cartes R, Martinez C, Bahoric B, Niazi T. Early Versus Delayed Androgen Deprivation Therapy for Biochemical Recurrence After Local Curative Treatment in Non-Metastatic Hormone-Sensitive Prostate Cancer: A Systematic Review of the Literature. Vol. 17, Cancers. 2025.
26. Zhang D, Luo G, Jin K, Bao X, Huang L, Ke J. The underlying mechanisms of cisplatin-induced nephrotoxicity and its therapeutic intervention using natural compounds. Naunyn Schmiedebergs Arch Pharmacol [Internet]. 2023; 396(11):2925–41. Available from: https://doi.org/10.1007/s00210-023-02559-6
27. Yuliandra Y, Nasif H, Ermayanti S, Sulistyowati L, Juwita DA. Hematologic Toxicities of Chemotherapy in Lung Cancer Patients: A Retrospective Study in Dr. M. Djamil Hospital Padang, Indonesia. Indones J Clin Pharm. 2019; 8(2):129.
28. Bagus I, Pramana P, Hakim L, Djatisoesanto W, Hardjowijoto S. Predictive factors of bleomycin, etoposide and cisplatin chemotherapy failure in patients with testicular seminoma a retrospective cohort study. Med Udayana [Internet]. 2019; 8(6):2597–8012. Available from: https://ojs.unud.ac.id/index.php/eum
29. Poon DMC, Chan K, Chan TW, Ng B, Siu S, Ng J, et al. Prevention of docetaxel-associated febrile neutropenia with primary granulocyte colony-stimulating factor in Chinese metastatic hormone-sensitive and castration-resistant prostate cancer patients. Asia Pac J Clin Oncol. 2021 Apr; 17 Suppl 3:39–47.
30. 30. Zhang Q, Li Y, Zhang Y, Deng Z, Ding Y. Case report of penile squamous cell carcinoma continuous treatment with BRCA2 mutation. World J Surg Oncol [Internet]. 2024; 22(1):50. Available from: https://doi.org/10.1186/s12957-024-03305-9
31. Gülle S, Çelik A, Birlik M, Yılmaz O. Skin and lung fibrosis induced by bleomycin in mice: a systematic review. Rheumatism [Internet]. 2024 Mar 22; 76(1 SE-Reviews). Available from: https://www.reumatismo.org/reuma/article/view/1642
32. Moik F, Ay C. Treatment of VTE in the thrombocytopenic cancer patient. Hematology [Internet]. 2024 Dec 6;

E-ISSN:2722-0877

2024(1):259–69. Available from: https://doi.org/10.1182/hematology.2024000551

33. Tsantes AG, Petrou E, Tsante KA, Sokou R, Frantzeskaki F, Domouchtsidou A, et al. Cancer-Associated Thrombosis: Pathophysiology, Laboratory Assessment, and Current Guidelines. Vol. 16, Cancers. 2024.
34. Sarofah T, Ritonga F. Comparison of Blood Urea and Creatinine Levels in Patients with Chronic Kidney Failure Pre and Post Hemodialysis. A Student ... [Internet]. 2022; 4:40–4. Available from: https://journal.binawan.ac.id/index.php/bsj/article/view/214
35. Paradise NI. DESCRIPTION OF CREATININE AND UREA LEVELS IN PATIENTS WITH CHRONIC KIDNEY FAILURE BEFORE AND AFTER HEMODIALYSIS AT R.S.U RANGE ASAHAN SRI [Internet]. Vol. 8, T-Shirt GL Dergisi. 2020. 147–154 p. Available from: https://doi.org/10.1016/j.jnc.2020.125798%0Ahttps://doi.org/10.1016/j.smr.2020.02.002%0Ahttp://www.ncbi.nlm.nih.gov/pubmed/810049%0Ahttp://doi.wiley.com/10.1002/anie.197505391%0Ahttp://www.sciencedirect.com/science/article/pii/B9780857090409500205%0Ahttp:
36. Colombo G, Altomare A, Astori E, Landoni L, Garavaglia ML, Rossi R, et al. Effects of Physiological and Pathological Urea Concentrations on Human Microvascular Endothelial Cells. Vol. 24, International Journal of Molecular Sciences. 2023.
37. Espi M, Koppe L, Fouque D, Thaunat O. Chronic Kidney Disease-Associated Immune Dysfunctions: Impact of Protein-Bound Uremic Retention Solutes on Immune Cells. Vol. 12, Toxins. 2020.
38. Hutapea RD, Widaningsih Y, Mangarengi F, Muhadi D. Analysis of Urea, Creatinine, and Platelet Indices in Hypertensive Patients. Indonesia, J Clin Pathol Med Lab. 2021; 27(2):117–21.
39. Setiawan SD. The effect of chemotherapy on cancer patients on. pharmacon J. 2015; 10(1):94–9.