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Original Research Article

Chemotherapy response rate in seminoma and non-seminoma testicular cancer cases

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ABSTRACT

Background: Testicular cancer is a malignancy in the form of a solid tumor that occurs in men, with a global prevalence that has been steadily increasing. The most commonly found types are seminoma and non-seminoma, particularly among individuals aged 20-34 years. In recent decades, 9,560 new cases of testicular cancer were recorded in the United States in 2019. In Indonesia, based on data from January 1995 to December 2004, the majority of testicular cancer cases were also seminoma and non-seminoma types, with patient ages ranging from 18 to 72 years.
Methods: Data analysis was performed using SPSS software version 29.0, employing univariate and bivariate analyses. Univariate analysis was used to describe the characteristics of the study samples. Ratio-scale data were described using the mean as a measure of central tendency and standard deviation (SD) as a measure of dispersion. Categorical data were described in percentages (%) and numbers (n), and presented in tables. Differences in chemotherapy response outcomes among testicular cancer patients, which are categorical data, were analyzed using bivariate analysis. Comparative tests were conducted to assess differences in the success rates of chemotherapy responses in testicular cancer patients. Both tests were considered statistically significant if they had a $p<0.05$.
Results: Complete chemotherapy response in seminoma and non-seminoma testicular cancer was nearly similar at 66.7% and 68.8%, respectively; partial chemotherapy responses in seminoma and non-seminoma were 13.3% and 18.8%; however, progressive chemotherapy responses tended to be higher in seminoma (20.0%) than in non-seminoma (12.5%).
Conclusions: The success rate of chemotherapy response in testicular cancer cases was mostly complete (67.7%), while partial and progressive responses were 16.1% each, and there was no difference in chemotherapy response between seminoma and non-seminoma tumors.

Keywords: Chemotherapy, Testicular cancer, Tumor markers

INTRODUCTION

Testicular cancer is a solid tumor malignancy that predominantly affects men during their most productive years, with peak incidence occurring between the ages of 15 and 45. Specifically, the highest diagnostic rates are observed in young men aged 20 to 34, a demographic in which the global prevalence has been steadily increasing over the past four decades.[1,2] In 2019, an estimated 9,560 new cases of testicular cancer were diagnosed in the United States alone. Within the context of Southeast Asia, including Indonesia, similar demographic trends are evident. A decade-long retrospective analysis in

Indonesia (1995-2004) demonstrated that testicular malignancies disproportionately affect individuals in their prime, with the mean age of diagnosis at 33 years, predominantly comprising seminoma and non-seminoma subtypes.[3] Although testicular cancer accounts for only about 1% of all male neoplasms and 5% of urological malignancies, it remains a critical focus in urologic oncology due to its significant impact on quality of life, survivorship, and its high potential for cure when diagnosed accurately and treated with a timely multimodal approach.

The etiopathogenesis of testicular cancer is complex and deeply rooted in genetic and embryological developmental anomalies. Most testicular cancers originate from germ cell tumors (GCTs), which are believed to develop from tumorigenic events occurring in utero, leading to germ cell neoplasia in situ (GCNIS).[4] At the genetic level, specific alterations, such as the pathognomonic isochromosome on the short arm of chromosome 12 (i12p), are consistently identified in adult germ cell tumors. Furthermore, dysregulation in the KITLG-KIT signaling pathway and the overexpression of embryonic transcription factors such as NANOG, SOX17, and OCT3/4 facilitate the accumulation of mutations, promoting cellular proliferation and suppressing apoptosis.[5,6] These cellular aberrations manifest clinically as painless scrotal masses, and due to their aggressive biological behavior, they exhibit a strong propensity for early lymphatic and hematogenous metastasis.

Histopathologically, testicular malignancies are broadly classified into seminomatous and non-seminomatous germ cell tumors (NSGCT). The initial and universally accepted management for both subtypes involves radical inguinal orchiectomy, a procedure that serves the dual purpose of definitive histological diagnosis and primary local tumor control. However, given the systemic nature of the disease in a significant proportion of patients, surgical intervention alone is often insufficient. For patients presenting with advanced-stage disease, retroperitoneal lymph node involvement, or distant metastasis, a robust multimodality approach becomes absolutely essential.[7]

Systemic chemotherapy has unequivocally revolutionized the treatment landscape for advanced testicular cancer, elevating it to one of the most curable solid neoplasms. Standard therapeutic regimens primarily rely on platinum-based combinations. The selection of these regimens is rigorously guided by the International Germ Cell Cancer Collaborative Group (IGCCCG) risk stratification, which categorizes patients into good, intermediate, or poor prognostic groups based on primary tumor location, sites of metastasis, and serum tumor marker levels (AFP, HCG, and LDH). Consequently, patients receive tailored chemotherapy cycles, most commonly Bleomycin, Etoposide, and Cisplatin (BEP), Etoposide and Cisplatin (EP), or variations such as VIP (Etoposide, Ifosfamide, and Cisplatin) for those with intermediate or poor-risk profiles.[8]

To objectively evaluate the clinical and anatomical efficacy of these chemotherapeutic regimens, the Response Evaluation Criteria in Solid Tumors (RECIST 1.1) is utilized as the international standard measurement tool. RECIST 1.1 provides a highly standardized, reproducible framework for assessing changes in tumor burden via cross-sectional imaging (CT/MRI). It categorizes clinical outcomes into Complete Response (CR), Partial Response (PR), and Progressive Disease (PD) based on the quantitative sum of diameters (SoD) of target lesions alongside the qualitative assessment of non-target lesions and the potential emergence of new lesions.[9,10] While RECIST focuses heavily on morphometric anatomical changes, the integration of serum tumor marker normalization remains a vital adjunct in the overall clinical assessment of testicular GCTs.

Despite the overall high success rate of platinum-based therapies, there is a profound disparity in clinical response dictated by the tumor's histological subtype. Seminomas are inherently highly chemosensitive. Therapeutic success rates for seminomas reach approximately 95%, frequently resulting in a complete response that eliminates the need for any subsequent post-chemotherapy surgical resection of residual masses, reserving radiation or surgery only for specific isolated recurrences.[11,12]

Conversely, the management of non-seminomas poses a significantly greater clinical dilemma. Following induction chemotherapy, the presence of residual retroperitoneal masses is a frequent clinical scenario. Radiological evidence indicates that post-chemotherapy residual lesions larger than 1 cm carry a substantial risk; there is a 10% probability of harboring viable cancer cells, a 40% chance of containing chemoresistant teratomas, and a 50% likelihood of consisting entirely of necrotic or fibrotic tissue.[13] Because imaging modalities cannot definitively distinguish between viable tumor, teratoma, and necrosis, meticulous follow-up and subsequent surgical interventions, such as Retroperitoneal Lymph Node Dissection (RPLND), are often mandated to prevent late relapses and secondary malignancies.[14,15]

While the efficacy of these established chemotherapy regimens and surgical salvage pathways is well documented in global literature, actual clinical outcomes can vary significantly within local populations. In tertiary referral centers, chemotherapy responses are frequently influenced by differing demographic characteristics, delayed presentation stages, adherence to treatment protocols, and specific clinical referral patterns. As a major Type A referral hospital in Central Java, RSUD Dr. Moewardi routinely manages complex urologic oncology cases, including varied presentations of advanced testicular cancer.

To date, there is a notable lacuna in comprehensive institutional data evaluating the specific chemotherapy responses, survival outcomes, and post-chemotherapy residual tumor profiles among testicular cancer patients treated at this facility.

Therefore, this study aims to systematically evaluate the chemotherapy response in testicular cancer patients at RSUD Dr. Moewardi during the period of January 2022 to December 2024.

By analyzing these outcomes through the lens of RECIST 1.1 and tumor marker kinetics, this research Endeavours to provide a robust, evidence-based foundation to optimize future clinical pathways, refine multidisciplinary management strategies, and ultimately improve the survivorship of testicular cancer patients in this specific clinical setting.

METHODS

Research design

This research is a descriptive analytical study utilizing a retrospective cohort design.

Time and place of research

This research was conducted at the Urology Department of Dr. Moewardi General Hospital, Surakarta, utilizing medical record data from the Outpatient Clinic for the period of January 2022 to December 2024.

Research population and sample

The subjects in this study were all testicular cancer patients who had undergone orchiectomy followed by chemotherapy during the period of January 2022 to December 2024.

Research sample

The sample consisted of all testicular cancer patients who received chemotherapy treatment at Dr. Moewardi General Hospital between January 2022 and December 2024, and who met the inclusion and exclusion criteria. The sampling technique utilized was consecutive sampling.

Inclusion criteria

Testicular cancer patients who have undergone orchiectomy with histopathological results confirming seminoma or non-seminoma subtypes, patients who underwent chemotherapy with the bleomycin, etoposide, and cisplatin (BEP) regimen for 6 months, patients who have completed post-chemotherapy imaging evaluations (Ultrasound and CT scan) and patients who have undergone tumor marker examinations both before and after chemotherapy were included in the study.

Exclusion criteria

Patients who did not complete the chemotherapy program, patients who did not undergo tumor marker and imaging evaluations before and after chemotherapy and patients who were lost to follow-up were excluded.

Research variables

The independent variable in this study was testicular cancer patients who had undergone orchiectomy followed by chemotherapy at Dr. Moewardi General Hospital. The dependent variable in this study was the success rate of the chemotherapy response.

Operational definitions

Testicular cancer

Operational boundary

Patients with seminoma and non-seminoma testicular cancer who have undergone orchiectomy and received BEP chemotherapy at Dr. Moewardi General Hospital.

Method of examination

Anamnesis, physical examination, and supporting investigations.

Scale

It was nominal.

Chemotherapy response

Operational boundary

Success rate of chemotherapy response evaluated when patient completes chemotherapy cycles, which includes:

Complete response: All cancer or tumor disappears, the diameter of pathological lymph nodes decreases to <10 mm, and tumor markers return to normal.

Partial response: Tumor markers decrease, but residual clinical signs of the disease are still present.

Progressive response: Tumor markers increase, or new lesions appear.

Method of examination

Anamnesis, physical examination, and supporting investigations.

Scale

It was ordinal.

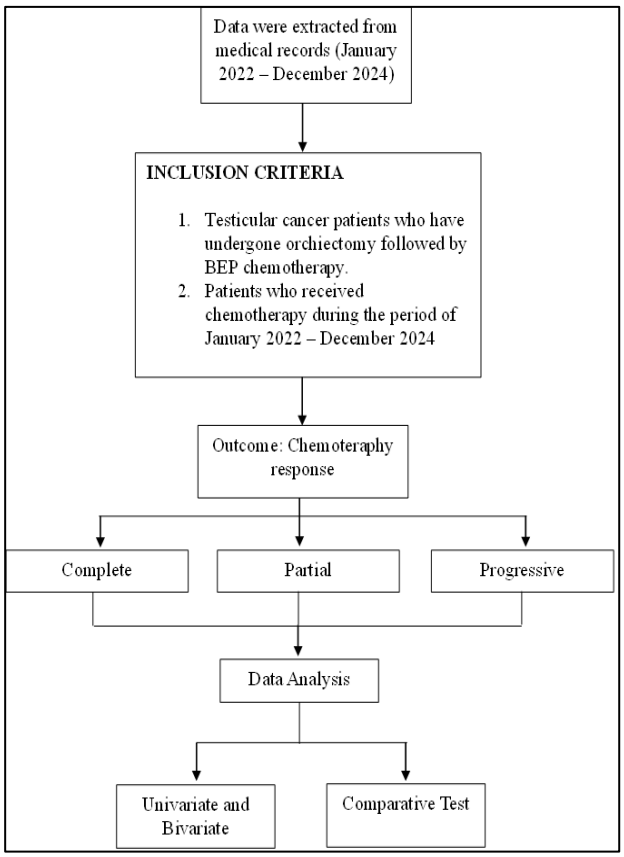
Histopathological results

Seminoma

The most common type of tumor originating from germ cells.

Non-seminoma

A type of cancer that includes yolk sac tumor, embryonal carcinoma, and teratoma.



Figures 1: Research operational flowchart.

Research procedure

The research sample was obtained from patient data based on medical records of those who had undergone operative procedures and received BEP chemotherapy. Furthermore, all data were collected and entered into the research data table. The data obtained in this study are secondary data. The collected data will undergo statistical analysis using SPSS.

Ethical clearance

Before the research was conducted, the researcher requested an ethical clearance certificate from the ethics committee for basic/clinical science research at Dr. Moewardi General Hospital, Surakarta / Faculty of Medicine, Universitas Sebelas Maret, Surakarta.

Data analysis

Data analysis was performed using the SPSS 29.0 software program by conducting univariate and bivariate analyses. Univariate analysis was used to describe the characteristics of the research sample. Ratio data were described using the mean as a measure of central tendency and standard deviation (SD) as a measure of dispersion. Categorical data were described in percentages (%) and frequencies (n) and presented in tabular form.

The analysis of differences in the chemotherapy response outcomes of testicular cancer patients, which are categorical data, was performed using bivariate analysis. A comparative test was conducted to assess the differences in the success rate outcomes of chemotherapy responses in testicular cancer patients. Both tests were considered statistically significant if they had a $p<0.05$.[16,17]

RESULTS

This research included 31 testicular cancer patients with the following characteristics:

More than half of the patients (51.6%) in this study were aged >35 years, while the lowest incidence was 12.9 years, aged <15 years. Pathology results revealed seminoma as the most common tumor (48.4%), followed by yolk sac tumor (41.9%).

The distribution of patient characteristics according to chemotherapy response is shown below:

Table 2 demonstrates that the seminoma subtype of testicular cancer is more predominantly found in patients aged >35 years, whereas the non-seminoma subtype is distributed relatively evenly across all age groups, comprising 43.8% in patients aged >35 years, 31.3% in those aged 15-35 years, and 25.0% in the <15 years age group.

The Fisher's exact test yielded a p-value of 0.166 ($p>0.05$), indicating no significant difference in the type of testicular cancer based on age groups. Meanwhile, the age distribution of patients according to the chemotherapy response success rate is presented in Table 3 below.

A complete chemotherapy response was more predominant across all age groups (75.0% in patients aged <15 years, 63.6% in those aged 15-35 years, and 68.8% in those aged >35 years) compared to partial and progressive responses. The Fisher's exact test yielded a $p=1.000$, indicating no significant difference in chemotherapy response among the different age groups. Meanwhile, the analysis of differences in chemotherapy response based on the testicular cancer subtype is presented in Table 4.

Table 1: Patients characteristics.

Characteristics	N (%)
Age (in years)	
<15	4 (12.9)
15-35	11 (31.5)
>35	16 (51.6)
Pathology results	
Seminoma	15 (48.4)
Yolk sac tumor	13 (41.9)
Embryonic	1 (3.2)
Teratoma	2 (6.5)

Table 2: Distribution of patient age according to testicular cancer type.

Age (in years)	Types of testicular cancer N (%)		P value
	Seminoma	Non-seminoma	
<15	0 (0)	4 (25)	0.166
15-35	6 (40)	5 (31.3)	
>35	9 (60)	7 (43.8)	

Table 3: Distribution of patient age according to chemotherapy response.

Types of testicular cancer (in years)	Chemotherapy response N (%)			Total	P value
	Complete	Partial	Progressive		
<15	3 (75)	1 (25)	0 (0)	4 (100)	1.000
15-35	7 (63.6)	2 (18.2)	2 (18.2)	11 (100)	
>35	11 (68.8)	2 (12.5)	3 (18.8)	16 (100)	
Total	21 (67.7)	5 (16.1)	5 (16.1)	31 (100)	

Tabel 4: Differences in chemotherapy response according to testicular cancer type.

Types of testicular cancer	Chemotherapy response N (%)			Total	P value
	Complete	Partial	Progressive		
Seminoma	10 (66.7)	2 (13.3)	3 (20)	15 (100)	1.000
Non-seminoma	11 (68.8)	3 (18.8)	2 (12.5)	16 (100)	
Total	21 (67.7)	5 (16.1)	5 (16.1)	31 (100)	

Table 4 demonstrates that a complete chemotherapy response was observed in the majority (67.7%) of patients. The rates of complete response were comparable between seminoma and non-seminoma testicular cancers, at 66.7% and 68.8%, respectively. Partial responses were recorded in 13.3% of seminoma and 18.8% of non-seminoma cases; however, a progressive chemotherapy response tended to be slightly more frequent in seminomas (20.0%) than in non-seminomas (12.5%). The Fisher's exact test yielded a p=1.000, indicating no statistically significant difference in chemotherapy response between the seminoma and non-seminoma subtypes.

DISCUSSION

Based on patient characteristics, it was found that testicular cancer was more frequently observed in patients aged >35 years (51.6%), followed by 31.5% in the 15-35 years age group. This finding is consistent with the perspective of Gaddam et al., who stated that testicular cancer is the most common malignancy in males aged 15 to 45 years, driven by multifactorial causes including genetic and environmental factors.[18]

Based on cancer types, the non-seminoma subtype was slightly more predominant (51.6%) compared to seminoma (48.4%). The slightly higher prevalence of non-seminomas is associated with differences in their growth rates. Seminoma is a type of testicular cancer, primarily a germ cell tumor developing from sperm-producing cells, characterized by a slower growth rate and a higher cure rate, especially if detected and treated early.[19] Non-seminoma testicular cancer also originates from germ cells but, unlike seminoma, tends to grow more rapidly and consists of various cell types such as yolk sac, embryonal, and teratoma.[20]

Patient age in this study was not associated with the type of testicular cancer. Although age is a risk factor for

testicular cancer incidence, which typically occurs between 15 and 45 years of age, both subtypes can develop at any age within this range. The peak incidence for seminoma is at 25-29 years, whereas for non-seminoma, it is at 35-39 years.[21] Furthermore, age did not correlate with the chemotherapy response in testicular cancer patients, as chemotherapy remains highly effective regardless of whether the cancer is in an advanced or metastatic stage. The cure rate for testicular cancer is generally high, even in disseminated cases. Similar findings from a study at Saiful Anwar General Hospital also demonstrated no significant relationship between age and chemotherapy response in testicular cancer cases.[22]

The testicular cancer subtype was also not associated with the chemotherapy response, as both seminoma and non-seminoma subtypes predominantly exhibited a complete response. Both seminomas and non-seminomas are generally highly responsive to chemotherapy; however, seminomas tend to demonstrate a slightly better overall response rate, particularly in the early stages. Both types of testicular germ cell tumors (TGCT) are highly curable, and even the majority of patients with metastatic disease can still achieve remission. This evidence is supported by a study at Dr. Soetomo Hospital, which stated that although seminoma is more curable than non-seminoma, non-seminoma is also chemosensitive. This was indicated by the lack of significant correlation between tumor stage or elevated serum tumor markers and chemotherapy response ($p=0.769$ and $p=0.363$, respectively). Non-seminoma patients, whether in stage I or already presenting with metastasis, exhibited relatively similar chemotherapy responses.[23]

Both testicular cancer subtypes, seminoma and non-seminoma, are highly curable, and the vast majority of patients, even those with metastasis, still have a favorable prognosis for cure. This high cure rate is not attributed to the indolent nature of testicular cancer, but rather to its extreme sensitivity to chemotherapy (and radiotherapy for seminomas). The chemosensitivity of testicular cancer is associated with the p53 marker, a tumor suppressor gene that is frequently mutated in cancers generally but remains unmutated in testicular cancer. Oct4, an embryonic transcription factor, is also homogeneously expressed in seminoma and embryonal carcinoma components of non-seminomas, and its interaction with p53 plays a crucial role in the response to chemotherapy.[24]

The findings of this study regarding the lack of difference in chemotherapy response between seminoma and non-seminoma testicular cancer patients contrast with those reported by Fabrianta et al who demonstrated a higher success rate in the seminoma subtype compared to non-seminoma.[22] This discrepancy may stem from varying patient characteristics, such as genetic factors, cancer stage, comorbidities, environmental factors, and other clinical risk factors that can influence the response to chemotherapy.

This study acknowledges several limitations, specifically the incomplete availability of preoperative clinical data and a constrained sample size due to its single-center design.

CONCLUSION

In this study, the majority of testicular cancer patients achieved a complete chemotherapy response following BEP treatment, with an overall complete response rate of 67.7%. No statistically significant difference in chemotherapy response was observed between seminoma and non-seminoma subtypes. These findings suggest that platinum-based chemotherapy remains highly effective in both histopathological subtypes of testicular cancer. Furthermore, this study provides local institutional evidence regarding chemotherapy outcomes in testicular cancer patients at a tertiary referral center, which may support future clinical decision-making and optimization of treatment strategies.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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