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Clinical Profile and Genomic Mutational Landscape of Prostate Cancer in Brunei Darussalam from 2018 to 2024 A Retrospective Descriptive Study

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Abstract

Background

Prostate cancer has become one of the leading causes of malignancy affecting men in Brunei Darussalam, with steadily rising incidence over the past decade. However, its clinical, pathological, and molecular characteristics remain poorly described. This study aims to address this knowledge gap by providing a detailed profile of prostate cancer's clinical and genomic profile in the country.

Methods

In this retrospective cohort study, medical records of 212 prostate cancer patients receiving 478 treatments received between January 2018 and December 2024 at The Brunei Cancer Centre (TBCC), Jerudong Park Medical Centre (JPMC) were analyzed. Demographic, clinical, laboratory, treatment and genomic data were extracted from BruHIMS,laboratory records, clinical notes, and Next Generation Sequencing report. Data was processed and analyzed using RStudio version 4.3.3 and Python 3.10 (Google Colab) following the STROBE checklist.

Results

Most patients were aged 71–80 years (39.15%), of Malay ethnicity (63.73%), and presented with advanced-stage disease (65%), with frequent metabolic comorbidities; 80% were alive with median survival follow up of 2.25 years. Nearly one-third underwent NGS, revealing diverse genomic alterations, including actionable DDR-related mutations. Median baseline PSA was high (11.1ng/mL, IQR 1.2–85.5), declining to (0.54 ng/mL, IQR 0.07–4.83) post-treatment. Treatment modality showed significant difference between age ($p < 0.001$), marital status ($p < 0.001$), survival status ($p < 0.001$), disease severity ($p < 0.001$), PSA level ($p < 0.001$), and several haematological and biochemical parameters. The younger patients (50–60 years) had the highest body weight with late-stage presentation, indicating an emerging metabolic risk profile in this subgroup.

Conclusion

This study offers the first comprehensive clinical and genomic profile of prostate cancer in Brunei, highlighting the urgent need for earlier detection, broader genomic testing, and tailored management strategies. The dataset lays a robust foundation for developing AI-driven precision oncology tools to improve outcomes for prostate cancer patients.

1.Background

Prostate cancer (PCa) is the fourth most frequent cancer in males, and it's steadily rising across Southeast Asia.[1] Despite being traditionally viewed as a disease of Western countries, recent epidemiological trends show increasing incidence and mortality among Southeast Asia men.[2, 3] The shift was driven by ageing populations, lifestyle modification, environmental conditions and improved diagnostic capacities for early cases.[2, 3] However, this shift in the Southeast Asia region was heterogeneous, as countries have different capacities and policies regarding cancer screening, surveillance, and awareness of early cancer detection.[3, 4] Moreover, the true epidemiology and genomic characteristics of prostate cancer in Southeast Asia remain underexplored, restricting the ability of policymakers and clinicians to implement evidence-based prevention and management strategies.

In Brunei Darussalam, prostate cancer has emerged as one of the leading causes of malignancy affecting men, with incidence rising over the past decade. [5, 6, 7, 8] According to the Brunei Darussalam Cancer Register Report (BDCR), a total of 272 new cases of prostate cancer were diagnosed between 2012 and 2021. A higher incidence rate was observed in the most recent five years (2017–2021) compared to 2012–2016, with the peak incidence occurring among individuals aged 75 to 79 years.[6] Furthermore, according to Leong et al, the age-standardized mortality for prostate cancer in Brunei Darussalam was 9.2% out of 1636 male patients from 2011 to 2020, with second second-highest mortality rate in 2020 and with a regression trend showing a stable trend.[5]

Despite the reported incidence and mortality of prostate cancer, the evidence on its clinical, pathological and molecular characteristics remains limited in Brunei Darussalam. The lack of comprehensive data creates gaps in understanding prostate cancer patterns and planning effective interventions. The Brunei Darussalam Multisectoral Action Plan for the Prevention and Control of Noncommunicable Diseases (BruMAP-NCD) 2021–2025 sets a mission to priorities and improve prevention, early detection, and management of NCDs and their risk factors.[9] The need for precise, robust local evidence, including demographic, clinical, and molecular, including genomic insights, is essential to support national strategies and improve the early detection and management of cancer care in Brunei Darussalam.

The primary objective of the study is to describe the clinical profile of prostate cancer in Brunei Darussalam between 2018 and 2024, while simultaneously examining the genomic mutational landscape of patients who underwent next-generation sequencing (NGS). This population study with comprehensive overview will strengthen the understanding of prostate cancer in Brunei Darussalam and inform future directions in precision oncology including development of artificial intelligence tools, cancer screening and management strategies.

2.Methods

2.1. Study Design and Data Source

The retrospective cohort population study followed a STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) checklist [Supplementary Table 4] for cohort studies to ensure methodological rigour and transparent reporting.[10] The study included all prostate cancer patients treated between 1st January 2018 and December 2024 at the Brunei Cancer Centre (TBCC)at Jerudong Park Medical Centre (JPMC). All cancer patients in the country are referred, registered and treated in the centre. Eligibility criteria included male patients with a histologically confirmed diagnosis of prostate cancer who underwent at least one treatment modality at the Brunei Cancer Centre. Patients with secondary metastatic prostate cancer or those with multiple primary tumours were excluded from the study.

A total of 212 eligible prostate cancer cases with 478 time index observations over 6 years were identified without sampling, as shown in Fig. 1. Demographic, clinical and treatment data were extracted from the Brunei Darussalam Healthcare Information and Management System (BruHIMS), JPMC laboratory database, and digital and manual clinical notes. The next-generation sequencing (NGS) results from HRRT tissue assays(Homologous Recombination Repair mutation analysis in tissue), PCDI (Prostate Cancer Diagnostic Investigation) panels, and Foundation Medicine Liquid (FML) testing were included for patients who underwent somatic genomic profiling.

2.2. Data structure and preprocessing

Two levels of data were presented: case level and cohort structure level. At the case level, demographics (age, race, marital status), medical history (comorbidities and family cancer history), lifestyle factors (smoking status), anthropometric measurements (height, weight, BMI), tumour characteristics (Gleason scores, tumour staging, ECOG Performance Status) and survival outcomes were collected. At the cohort level, treatment detail information and 23 laboratory parameters, including before and one year after treatment PSA, full blood count, liver and renal function markers, ESR, calcium, and phosphate, were extracted. Absolute/relative PSA changes were computed along with tumour staging, Gleason Grade Groups, and NCCN prognostic risk groups were harmonised due to the lack of standardised recoding.[12] Next-generation sequencing information of the reported patient was categorised into DNA damage repair (DDR) and non-DDR alterations. [13] All laboratory parameters except PSA in cooperated in the study were baseline tests taken before each treatment decision change for patients. This enables oncologist to review a patient's blood results before deciding on the next treatment. All datasets were cross-checked across multiple sources and validated by an oncologist before analysis to ensure data quality.Additionally data cleaning, harmonization of tumor staging and Gleason Garde Groups, removal of duplicate records was conducted as a part of data preprocessing.

2.3. Statistical Analysis

All statistical analyses were performed using RStudio version 4.3.3 and Python 3.10 (Google Colab). Two analytical datasets were constructed: a case-structured dataset (212 observations) and a cohort-structured dataset (478 longitudinal observations). Descriptive statistics were used to summarise patient characteristics with categorical variables reported as frequencies and

percentages, and continuous variables as means ± standard deviations and medians with interquartile ranges(IQR). Distribution of the numerical variables is check for normal distribution before univariate analysis. PSA baseline and one year post treatment were not normally distributed and transformed into log for univariate analysis.

Univariate analysis was performed at the cohort level (478 observations), examining treatment categories against all other study variables using a mixed-effects model with a random intercept and no additional fixed covariates apart from treatment. This method accounts for within-patient correlation due to repeated treatment episodes, and univariate comparisons of continuous outcomes across treatment modalities, using linear and logistic mixed-effects models with treatment category as the only fixed effect and a random intercept for patient ID. This method provides inference on the treatment effect that is essentially identical to ordinary least squares regression with cluster-robust standard errors.[14, 15, 16] Furthermore, univariate and multivariate regression analysis was conducted for 67 NGS tested patient. In alignment with STROBE recommendations, descriptive analyses were conducted using complete (non-imputed)datasets to preserve clinical validity.

3.Results

3.1. Exploratory analysis on the case structure data set

A total of 212 prostate cancer patients treated at Brunei Cancer Centre (TBCC) between January 2018 and December 2024 were included in the study, describe in Table 1. The majority of patients were aged 71–80 (39.15%), followed by those aged 61–70 (29.25%) and aged over > 80 (23.58). Most patients were Malay (63.73%), with Chinese representing (28.43%). Nearly all patients were married 94%, with 31 missing entries. Patient visits peaked in 2022 (27.36%), with a steady increase over the years and approximately 80% of the patients were alive at the time of analysis. The median follows up duration is 2.25 year (IQR: 1.30–3.52). The median weight of patients is 68.5 kg (IQR: 61.35–77.00), and mean of 69 kg (SD: 13.4), while BMI information of 112 patients with 20.5% classified as obese and 41.9% as overweight.

Table 1
Clinical Profile of Prostate Cancer Patients at Brunei Cancer Centre (TBCC) at case level from January 2018 to December 2024 (*n* = 212)

Clinical Profile	***n* (%)**
Age Group	
50–60 year	17 (8.02)
61–70 year	62 (29.25)
71–80 year	83 (39.15)
More than 80 years	50 (23.58)
Race	
Malay	130 (63.73)
Chinese	58 (28.43)
Ethnic (Dusun/Iban)	9 (4.41)
Others (Indian, German, Caucasian)	7 (3.43)
Missing value	8
Weight and Height	
Weight in kilogram (*n* = 209)	68.95 (61.35–77.00)[1]
Height in centimeter (*n* = 117)	163.20 (149.0-168.0)[1]
BMI Category	
Normal	42 (35.89)
Underweight	2 (1.70)
Overweight	49 (41.88)
Obesity	24 (20.51)
Missing	95
Survival of the patients	
Alive	170 (80.19)
Passed Away	42 (19.81)
Follow-up information	
Median Follow up duration (*n* = 212)	2.25 (2.22)[1]
NGS Test	
Tested	67 (31.6)
Not Tested	145 (68.4)
Gleason Grade Grouping	
Group 1	23 (13.22)
Group 2	31 (17.82)
Group 3	13 (7.47)

NGS=Next Generation Sequencing; [1]Median (Interquartile Range)

Clinical Profile	*n* (%)
Age Group	
Group 4	30 (17.24)
Group 5	77 (44.25)
Missing	38
Tumor Type	
Non-Specific	145 (68.39)
mHSPC	39 (18.40)
mCRPC	24 (11.32)
nmCRPC	4 (1.89)
Staging	
Stage I/II	25 (12.63)
Stage III	44 (22.22)
Stage IV	129 (65.15)
Missing	14

NGS=Next Generation Sequencing; [1]Median (Interquartile Range)

Comorbidities were common, including hypertension (67%), hyperlipidemia (55%), diabetes (34.8%) and heart disease (25%), described in supplementary table 1. Along with the metabolic syndrome, around 10% patients reported chronic kidney disease and benign prostatic hypertrophy (BPH), followed by gout (9%) and hepatitis (5.2%). 22% of patients have a family history of cancer, with 21% reporting smoking. In terms of cancer-specific characteristics, 38 patients hadn't reported their Gleason information, and most of the prostate cancer histological outcomes presented with Gleason Grade Group 5 (44.25%), followed by Grade Groups 4 and 2 (~ 17% each), indicating a high proportion of aggressive cancers. More than 65% of patients presented with metastasis Stage IV (IVA, IVB), followed by Stage III (22.22%) and Stage I/II (12.63%). More than 18% reported prostate cancer cases are metastatic hormone-sensitive prostate cancer, and 11% metastatic castration resistant prostate cancer.

3.2. Genomic Mutation Landscape from Next-Generation Sequencing (NGS) data

About one-third (31.60%) had undergone Next Generation Sequencing (NGS) testing, 32 patient had significant gene mutations. One patient also demonstrated PD-L1 expression, suggesting potential eligibility for immunotherapy. The diverse genetic landscape of the study cohort is highlighted in Fig. 2.The most commonly observed genetic mutations include TP53 (*n* = 5), BRCA2(*n* = 3), KRAS (*n* = 3) and DNMT3A (*n* = 2), followed by single genetic mutation occurrences in DK12, CKIT, PIK3CA, PDGFRA, PTEN, ARID1A, ASXL1, KDM6A, TMPRSS2, CHEK2, TET, and NF1. Furthermore, there variant of unknown genetic significance includes MSH3 (*n* = 5), ATM (*n* = 5), POLD1 (*n* = 3), NTHL1 (*n* = 3), POLE (*n* = 2), MET (*n* = 2) followed by single mutation occurrences in SMARCA4, BRCA1, PMS2, APC, BRIP1, EGFR, CTNNA1, MUTYH, STK11, BARD1, AXIN2, BLM, PALB2, RECQL4, SMARCE1, BRAF, MYC, RB1, TERT, and DIS3L2.

Furthermore, genomic profiling revealed that 40% of mutations were associated with DNA damage repair (DDR) pathways, while 60% were non-DDR. DDR alteration was most commonly observed in BRCA2, TP53 and ATM, which have known implications for target therapy. The subgroup analysis on the 67 NGS tested patients is presented in the supplementary Table 2. The univariate analysis with the Fisher's exact test revealed only significance with the race category while subsequent multivariable logistic regression found no significant associations across any of the variables while limitations of the current study sample size for NGS might caution the interpretation of the result.

(**A)**

3.3. Exploratory analysis on Cohort Structure dataset

The cohort-structured dataset includes 478 observations incorporating longitudinal laboratory and treatment data, as shown in Table 2. The treatment age group corresponding to the treatment, the minimum age group of treatment received by patient is 46 years and around 10% of patient are in 46–60 years, while around 36% of patients are presented in 61–70 year and 71–80 year. Liver function makers showed mean ALT and AST levels of 26.85 ± 23.17 U/L and 28.01 ± 33.88 U/L, respectively. In renal indicators, a mean creatinine of 94.25 ± 65.58 μmol/L and urea of 5.37 ± 1.90 mmol/L, with only 177 observations reported. While reviewing the electrolytes, the mean sodium at 139.53 ± 3.47 mmol/L, potassium at 4.60 ± 6.47 mmol/L, and chloride at 105.11 ± 3.60 mmol/L.

Haematological parameters show RBC (4.18 ± 0.71 × 10^{12}/L), haemoglobin (121.84 ± 18.72 g/L), neutrophils (4.43 ± 1.93 × 10^{9}/L), and platelets (247.58 ± 83.43 × 10^{9}/L) were reported. The prostate-specific antigens (PSA) biomarker showed substantial variability, with a high median (11.1ng/mL, IQR 1.2–85.5) and mean (132.25 ± 329.44 ng/mL) baseline decreasing to median (0.54 ng/mL, IQR 0.07–4.83) and mean (43.29 ± 164.63 ng/mL) post-treatment respectively, reflecting differential treatment responses. 80.13% of patients were managed as outpatients, and the majority were newly diagnosed cases (94.14%), while around 5.86% experiencing relapse. Treatment patterns indicated that ADT monotherapy (31.59%), local therapy (27.20%), and ARPI-based hormonal/targeted combinations (26.99%) constituted the predominant modalities, followed by chemotherapy ± ADT/ARPI (8.58%) and active surveillance (5.64%).

Table 2
Laboratory and Treatment Profile of Prostate Cancer Patients at Brunei Cancer Centre (TBCC) at cohort level from January 2018 to December 2024 (*n* = 478)

Age groups corresponding to treatment	***n (%)* Mean (SD)**			**Median (IQR)**	
46–60 year	47(9.83)				
61–70 year	172(36.40)				
71–80 year	174(35.98)				
More than 80 years	85(17.78)				
Laboratory Test					
Alanine transaminase (ALT)	440	26.85	(23.17)	21	(14–31)
Aspartate transaminase (AST)	443	28.01	(33.88)	22	(18–27)
Calcium (Ca)	336	2.58	(5.49)	2.3	(2.19–2.39)
Chlorine (Cl)	447	105.1	(3.60)	105	(103–107)
Creatinine (Cr)	449	94.25	(65.58)	85	(74–99)
Potassium (K)	436	4.60	(6.47)	4.3	(4.9–4.6)
Sodium (Na)	448	139.53	(3.47)	140	(138–142)
Urea (Ur)	177	5.37	(1.90)	4.3	(4.0–6.6)
Phosphorus (PO4)	356	1.02	(0.26)	1.05	(0.8–1.21)
Prostate Specific Antigen (PSA) baseline	465	132.25	(329.44)	11.1	(1.2–85.5)
PSA after treatment	416	43.29	(164.63)	0.54	(0.07–4.83)
Red Blood Cell (RBC)	461	4.18	(0.71)	4.18	(3.76–4.71)
Haemoglobin (Hb) in g/dL	461	121.84	(18.72)	124	(110–135)
Mean corpuscular volume (MCV)	461	88.69	(8.03)	89.0	(86.0–93.0)
Mean corpuscular haemoglobin (MCHC)	461	331.29	(35.90)	330	(320–338)
White Blood Cell (WBC)	461	7.86	(16.62)	6.9	6.9(5.6–8.2)
Neutrophils (Neutro)	461	4.43	(1.93)	4.05	(3.2–5.4)
Monocytes (Mono)	453	0.58	(0.29)	0.5	(0.4–0.7)
Eosinophils (Eosino)	461	0.26	(0.28)	0.20	(0.10–0.30
Basophils (Baso)	451	0.04	(0.06)	0.01	(0.01–0.10)
Platelets (Plat)	461	247.58	(83.43)	236	(190–290)
Place of Treatment					
Inpatient care	95			19.87	
Outpatient care	383			80.13	
Relapse					
New case	450			94.14	
Relapse	28			5.86	
Treatment Category Merged					
ADT Monotherapy	151			31.59	

Age groups corresponding to treatment	*n (%)* Mean (SD)	Median (IQR)
Local Therapy	130	27.20
APRI based Hormonal/Targeted Combination Therapy	129	26.99
Chemotherapy ± ADT/ARPI	41	8.58
Active Surveillance	27	5.64

3.4. Univariable analysis of different Treatment Modalities

The univariable analysis on the longitudinal dataset between treatment modalities and multiple variables demonstrates a significant difference between the groups, demonstrated in Table 3 and supplementary Table 3. Age distribution varied substantially by treatment modality ($p < 0.001$), with patients aged > 80 years most frequently receiving ADT monotherapy or APRI-based hormonal/targeted therapy, while younger patients (50–60 years) were more commonly managed with local therapy. Survival status was varied between treatment type ($p < 0.001$), where deceased patients were disproportionately treated with APRI-based therapy or chemotherapy. NGS testing status also differed significantly across treatment groups ($p < 0.001$), with a higher proportion of untreated or mutation-negative reported patients receiving ADT monotherapy or local therapy, whereas mutation-positive patients were more evenly distributed. When reviewing comorbid conditions and clinical factors, the presence of hepatitis disease ($p = 0.036$), relapse status ($p < 0.001$) and inpatient versus outpatient ($p < 0.001$) revealed statistically significant differences between the different treatment groups.

When reviewing tumour-related characteristics, including Gleason grade ($p < 0.001$), cancer stage ($p < 0.001$), risk group ($p < 0.001$), and tumour type ($p < 0.001$), demonstrated differences between treatment groups, with advanced or metastatic disease more commonly managed with APRI-based therapy or chemotherapy, while lower-risk or localized tumors were primarily treated with local therapy or active surveillance. Among the biochemical and haematological markers, significant differences were observed between serum sodium levels ($p = 0.025$),potassium level ($p = 0.028$), baseline($p < 0.001$) and post-treatment PSA ($p < 0.001$), haemoglobin concentration ($p < 0.001$) and neutrophil count ($p < 0.001$). Patients undergoing active surveillance had markedly lower PSA levels and higher haemoglobin values compared to those receiving systemic therapies, while those on chemotherapy or APRI-based regimens showed elevated neutrophil counts and more pronounced PSA elevation at baseline and follow-up. No significant differences were found for liver enzymes (ALT/AST), renal function markers (creatinine, urea), electrolytes (Cr, Cl, PO4), haematological (MCV, MCHC, WBC, monocytes, eosinophils, basophils, platelets) or body weight.

Table 3
Univariable analysis of Prostate Cancer Patient with different treatment modalities at Brunei Cancer Centre (TBCC) at cohort level from January 2018 to December 2024 (*n* = 478)

Categorical Variable	***n* (%)**					***p*-value[1]**
	ADT Monotherapy	**APRI based Hormonal/ Targeted Combination Therapy**	**Active Surveillance**	**Chemotherapy ± ADT /ARPI**	**Local Therapy**	
Age Group Corresponding to Treatment						**< 0.001***
46–60 year	10 (21.3%)	9 (19.1%)	1 (2.1%)	10 (21.3%)	17 (36.2%)	
61–70 year	44 (25.6%)	42 (24.4%)	10 (5.8%)	18 (10.5%)	58 (33.7%)	
71–80 year	61 (35.1%)	45 (25.9%)	10 (5.7%)	11 (6.3%)	47 (27.0%)	
>80 year	36 (42.4%)	33 (38.8%)	6 (7.1%)	2 (2.4%)	8 (9.4%)	
Year of First Visit						0.139
2024	40 (31.0%)	20 (15.5%)	13(10.1%)	13(10.1%)	43 (33.3%)	
2023	27 (32.9%)	22 (26.8%)	5 (6.1%)	6 (7.3%)	22 (26.8%)	
2022	21 (35.6%)	14 (23.7%)	3 (5.1%)	4 (6.8%)	17 (28.8%)	
2021	20 (34.5%)	20 (34.5%)	2 (3.4%)	3 (5.2%)	13 (22.4%)	
2020	13 (22.8%)	17 (29.8%)	2 (3.5%)	7 (12.3%)	18 (31.6%)	
2019	15 (30.0%)	22 (44.0%)	1 (2.0%)	4 (8.0%)	8 (16.0%)	
2018	15 (34.9%)	14 (32.6%)	1 (2.3%)	4 (9.3%)	9 (20.9%)	
Race						0.405
Malay	88 (29.6%)	86 (29.0%)	17 (5.7%)	29 (9.8%)	77 (25.9%)	
Chinese	49 (41.2%)	24 (20.2%)	6 (5.0%)	6 (5.0%)	34 (28.6%)	
Ethnic	5 (21.7%)	7 (30.4%)	1 (4.3%)	2 (8.7%)	8 (34.8%)	
Other	3 (21.4%)	4 (28.6%)	2 (14.3%)	1 (7.1%)	4 (28.6%)	
Marital						**< 0.001***
Married	120 (31.6%)	99 (26.1%)	26 (6.8%)	30 (7.9%)	105 (27.6%)	
Single/Others	9 (39.1%)	7 (30.4%)	0 (0.0%)	3 (13.0%)	4 (17.4%)	

*Statistically significant [2] Linear regression model with Fixed effect model for ID

Categorical Variable	*n* (%)					*p*-value[1]
	ADT Monotherapy	APRI based Hormonal/ Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	
Survival						**< 0.001***
Alive	141 (32.5%)	107 (24.7%)	26 (6.0%)	32 (7.4%)	128 (29.5%)	
Passed Away	10 (22.7%)	22 (50.0%)	1 (2.3%)	9 (20.5%)	2 (4.5%)	
NGS Tested with Result						**< 0.001***
Not Tested	115 (36.2%)	77 (24.2%)	24 (7.5%)	15 (4.7%)	87 (27.4%)	
Tested Negative	17 (19.1%)	30 (33.7%)	2 (2.2%)	16 (18.0%)	24 (27.0%)	
Tested Positive	19 (26.8%)	22 (31.0%)	1 (1.4%)	10 (14.1%)	19 (26.8%)	
Family History						0.473
Absent	114 (31.2%)	105 (28.8%)	18 (4.9%)	30 (8.2%)	98 (26.8%)	
Present	37 (32.7%)	24 (21.2%)	9 (8.0%)	11 (9.7%)	32 (28.3%)	
Diabetes						0.269
Absent	101 (33.4%)	74 (24.5%)	21 (7.0%)	23 (7.6%)	83 (27.5%)	
Present	50 (28.4%)	55 (31.2%)	6 (3.4%)	18 (10.2%)	47 (26.7%)	
Hypertension						0.092
Present	115 (34.8%)	81 (24.5%)	19 (5.8%)	26 (7.9%)	89 (27.0%)	
Absent	36 (24.3%)	48 (32.4%)	8 (5.4%)	15 (10.1%)	41 (27.7%)	
Heart disease						0.169
Absent	108 (30.2%)	95 (26.5%)	20 (5.6%)	38 (10.6%)	97 (27.1%)	
Present	43 (35.8%)	34 (28.3%)	7 (5.8%)	3 (2.5%)	33 (27.5%)	
Hyperlipidaemia						0.460
Present	89 (32.5%)	75 (27.4%)	11 (4.0%)	24 (8.8%)	75 (27.4%)	
Absent	62 (30.4%)	54 (26.5%)	16 (7.8%)	17 (8.3%)	55 (27.0%)	
Gout						0.894

*Statistically significant [2] Linear regression model with Fixed effect model for ID

Categorical Variable	*n* (%)					*p*-value[1]
	ADT Monotherapy	APRI based Hormonal/ Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	
Absent	138 (31.4%)	120 (27.3%)	24 (5.5%)	39 (8.9%)	119 (27.0%)	
Present	13 (34.2%)	9 (23.7%)	3 (7.9%)	2 (5.3%)	11 (28.9%)	
Hepatitis						0.036*
Absent	148 (32.6%)	121 (26.7%)	26 (5.7%)	35 (7.7%)	124 (27.3%)	
Present	3 (12.5%)	8 (33.3%)	1 (4.2%)	6 (25.0%)	6 (25.0%)	
Benign Prostate Hyperplasia						0.989
Absent	137 (31.5%)	117 (26.9%)	25 (5.7%)	38 (8.7%)	118 (27.1%)	
Present	14 (32.6%)	12 (27.9%)	2 (4.7%)	3 (7.0%)	12 (27.9%)	
Chronic Kidney Disease						0.608
Absent	137 (31.7%)	114 (26.4%)	25 (5.8%)	39 (9.0%)	117 (27.1%)	
Present	14 (30.4%)	15 (32.6%)	2 (4.3%)	2 (4.3%)	13 (28.3%)	
Smoking history						0.973
Absent	110 (30.8%)	96 (26.9%)	21 (5.9%)	32 (9.0%)	98 (27.5%)	
Present	29 (32.2%)	23 (25.6%)	4 (4.4%)	9 (10.0%)	25 (27.8%)	
Patient visit						**< 0.001***
Outpatient	131 (34.2%)	96 (25.1%)	26 (6.8%)	24 (6.3%)	106 (27.7%)	
Inpatient	20 (21.1%)	33 (34.7%)	1 (1.1%)	17 (17.9%)	24 (25.3%)	
Relapse						**< 0.001***
No	146 (32.4%)	128 (28.4%)	19 (4.2%)	40 (8.9%)	117 (26.0%)	
Yes	5 (17.9%)	1 (3.6%)	8 (28.6%)	1 (3.6%)	13 (46.4%)	
Gleason Group						**< 0.001***
Grade 5	54 (29.3%)	63 (34.2%)	2 (1.1%)	23 (12.5%)	42 (22.8%)	

*Statistically significant [2] Linear regression model with Fixed effect model for ID

Categorical Variable	*n* (%)					*p*-value[1]
	ADT Monotherapy	APRI based Hormonal/ Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	
Grade 4	26 (35.6%)	15 (20.5%)	4 (5.5%)	4 (5.5%)	24 (32.9%)	
Grade 3	25 (36.2%)	16 (23.2%)	2 (2.9%)	5 (7.2%)	21 (30.4%)	
Grade 2	15 (31.9%)	3 (6.4%)	11 (23.4%)	0 (0.0%)	18 (38.3%)	
Grade 1	9 (31.0%)	7 (24.1%)	3 (10.3%)	2 (6.9%)	8 (27.6%)	
Cancer Staging						**< 0.001***
Stage IV	80 (26.8%)	112 (37.6%)	2 (0.7%)	40 (13.4%)	64 (21.5%)	
Stage III	44 (46.3%)	8 (8.4%)	6 (6.3%)	1 (1.1%)	36 (37.9%)	
Stage I/II	18 (30.5%)	4 (6.8%)	14 (23.7%)	0 (0.0%)	23 (39.0%)	
Risk Group						**< 0.001***
Metastasis	80 (26.8%)	112 (37.6%)	2 (0.7%)	40 (13.4%)	64 (21.5%)	
High Risk	40 (45.5%)	6 (6.8%)	6 (6.8%)	1 (1.1%)	35 (39.8%)	
Low Risk	9 (28.1%)	0 (0.0%)	11 (34.4%)	0 (0.0%)	12 (37.5%)	
Intermediate	10 (33.3%)	5 (16.7%)	3 (10.0%)	0 (0.0%)	12 (40.0%)	
Tumour Type						**< 0.001***
Non specific	123 (36.3%)	65 (19.2%)	27 (8.0%)	21 (6.2%)	103 (30.4%)	
mHSPC	15 (21.1%)	31 (43.7%)	0 (0.0%)	11 (15.5%)	14 (19.7%)	
mCRPC	11 (18.0%)	31 (50.8%)	0 (0.0%)	8 (13.1%)	11 (18.0%)	
nmCRPC	2 (28.6%)	2 (28.6%)	0 (0.0%)	1 (14.3%)	2 (28.6%)	

*Statistically significant [1] Logistic regression model with Fixed effect model for ID

Numerical Variables	**Mean (SD)/Median(IQR)**					**p-value[2]**
	ADT Monotherapy	APRI based Hormonal/Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	

*Statistically significant [2] Linear regression model with Fixed effect model for ID

Categorical Variable		*n* (%)					*p*-value[1]
		ADT Monotherapy	APRI based Hormonal/ Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	
ALT	25.79 (23.70)	25.55 (20.03)	24.85 (17.32)	22.73 (15.00)	31.06 (27.97)	0.132	
AST	30.49 (46.44)	28.84 (37.50)	23.37 (6.01)	26.40 (15.09)	26.39 (18.56)	0.809	
Ca	2.29 (0.16)	3.40 (10.62)	2.36 (0.11)	2.21 (0.21)	2.29 (0.16)	0.267	
Cl	104.96 (3.58)	105.08 (4.33)	105.26 (3.03)	104.40 (3.71)	105.50 (2.88)	0.717	
Cr	102.58 (104.14)	89.82 (34.20)	92.22 (15.86)	97.08 (67.68)	88.74 (27.27)	0.202	
K	4.28 (0.49)	5.45 (12.52)	4.37 (0.34)	4.42 (0.58)	4.26 (0.49)	**0.028***	
Na	139.33 (3.87)	139.15 (3.25)	140.11 (2.06)	138.62 (4.14)	140.28 (3.10)	**0.048***	
Ur	5.47 (1.93)	5.41 (2.08)	5.10 (1.23)	4.75 (1.72)	5.52 (1.91)	0.967	
PO4	1.03 (0.23)	1.00 (0.32)	0.97 (0.23)	1.00 (0.27)	1.07 (0.23)	0.676	
PSA Baseline (mean)	77.80 (209.12)	207.57 (437.45)	2.82 (3.52)	317.04 (475.14)	84.28 (243.60)	**< 0.001***	
Median	13.23 (0.93–54.50)	38.60 (4.40-156.48)	0.50 (0.02-5.05)	137.75 (36.99-435.42)	5.30 (0.30-25.71)		
PSA after Treatment (mean)	22.55 (86.84)	65.98 (240.63)	1.32 (1.88)	113.88 (217.63)	24.66 (101.93)	**< 0.001***	
Median	0.30 (0.03–1.94)	1.80 (0.15–9.33)	0.21 (0.02–2.21)	5.01 (0.59-125.22)	0.20 (0.02–1.98)		
RBC	4.22 (0.70)	4.04 (0.82)	4.65 (0.49)	3.93 (0.62)	4.28 (0.63)	0.393	
Hb(g/dl)	123.08 (18.34)	116.26 (20.34)	138.41 (11.95)	112.12 (18.21)	125.43 (15.23)	**0.021***	

*Statistically significant [2] Linear regression model with Fixed effect model for ID

Categorical Variable		*n* (%)					*p*-value[1]
		ADT Monotherapy	APRI based Hormonal/ Targeted Combination Therapy	Active Surveillance	Chemotherapy ± ADT /ARPI	Local Therapy	
MCHC	330.09 (12.37)	329.96 (50.40)	336.22 (19.72)	325.98 (12.43)	334.62 (44.08)	0.554	
MCV	88.84 (6.41)	88.59 (11.05)	89.04 (5.77)	88.00 (7.02)	88.79 (7.01)	0.593	
WBC	6.96 (2.12)	10.10 (32.10)	6.41 (1.66)	8.16 (2.87)	6.95 (1.94)	0.263	
Neutrophils	4.24 (1.87)	4.54 (1.86)	3.67 (1.32)	5.75 (2.61)	4.28 (1.75)	**< 0.001***	
Monocytes	0.57 (0.21)	0.62 (0.47)	0.57 (0.20)	0.66 (0.28)	0.56 (0.16)	0.893	
Eosinophils	0.31 (0.36)	0.25 (0.26)	0.23 (0.15)	0.19 (0.20)	0.27 (0.26)	0.305	
Basophils	0.04 (0.05)	0.04 (0.07)	0.05 (0.05)	0.06 (0.07)	0.04 (0.05)	0.380	
Platelets	235.17 (79.35)	251.13 (87.94)	228.89 (57.02)	283.95 (107.31)	243.21 (76.26)	0.882	
Weight(kg)	68.34 (17.98)	69.13 (13.66)	72.81 (13.96)	64.87 (18.56)	70.13 (15.09)	0.193	

*Statistically significant [2] Linear regression model with Fixed effect model for ID

3.5. Univariable analysis between age group, BMI and body weight

The univariable analysis between different age groups with body weight distribution and body mass index (BMI) categories was conducted and illustrated in Fig. 3. The boxplot illustrates the distribution of patient weight across four age groups, showing a statistically significant difference (one-way ANOVA $p = 0.0092$). Patients aged 50–60 years exhibit the highest median weight and the widest variability, including several high-value outliers, suggesting greater heterogeneity in this age group. Weight progressively declines with increasing age, with the > 80 group showing the lowest median weight and a narrower interquartile range. Although the heatmap comparing BMI category distributions across age groups did not show a statistically significant difference ($p = 0.24$), a notably higher proportion of overweight (50%) and obese (33.3%) individuals was observed in the younger age group (50–60 years). This pattern highlights the potential influence of excess body weight and higher BMI proportion in younger prostate cancer patients, which raises concern as relevant clinical and preventive considerations in this population.

4.Discussion

The retrospective study of 212 prostate cancer patients provides a comprehensive assessment of demographic, clinical, pathological, biochemical and genomic landscape in Brunei Darussalam. The findings indicate that the majority of patients presented at an older age, with most cases occurring in individuals aged 71–80 years, consistent with the global disease demographic. [5, 7, 17] The majority of study participants were diagnosed at advanced stages (Stage III–IV) with metastatic conditions. Metabolic comorbidities were common, particularly hypertension, hyperlipidemia, diabetes and obesity. Longitudinal laboratory trends indicate substantial PSA variability across treatment groups. Univariable analyses revealed differences between

treatment modality and age, disease severity, PSA levels, serum sodium levels, red blood cell count, haemoglobin concentration and platelet levels. Additionally, the study findings on younger late-stage patients with higher body weight alert to the important trend in the changing demographic of prostate cancer reported cases.

The study patterns align with regional and international trends, where prostate cancer incidence increases with age and is diagnosed at a late stage in the Asian population.[18, 19, 20] The high proportion of advanced-stage disease presentation reflects the delayed diagnosis of prostate cancer in the Southeast Asia region.[19, 20] Additionally, a higher tumor Gleason grade was reported in the study, consistent with previous studies' findings of Asia males presenting more aggressive pathological features than Western counterparts.[20] Furthermore, the previous study has demonstrated an association between obesity, metabolic syndrome, linked to higher-grade prostate cancer, increased treatment resistance, and poorer survival.[21, 22] The younger age group presented with high rates of overweight and obesity in the study is consistent with global trends in rising metabolic risk among middle-aged men and prostate cancer biology.[22, 23]

The study findings of one-third of patients who underwent NGS testing demonstrate a diverse spectrum of genetic mutations, with 32 patients demonstrating actionable or relevant gene alterations. DNA damage-repaired mutation, particularly BRCA2, TP53 and ATM, consistent with international evidence showing 20–30% involvement of advanced prostate cancers.[24–26] The identification of these genes provides a substantial therapeutic implication for precision oncology with the use of PARP inhibitors such as olaparib in BRCA-mutated disease.[27, 28] Additionally, the study reported variants of uncertain significance, such as MSH3, POLD1, NTHL1, MET, etc., reflecting the evolving genomic landscape of prostate cancer and highlighting the need for broader genomic testing to understand the diverse spectrum of genetic mutations.[29, 30]

As our study findings indicate late-stage presentation, the role of cancer screening might play an important role. In Brunei Darussalam, prostate cancer screening is not explicitly included in the National Health Screening Programme.[31] Globally, the utility of prostate cancer antigen(PSA) screening remains controversial; while PSA tests can reduce prostate cancer-specific mortality, they can also carry significant risks of overdiagnosis and overtreatment.[32, 33] Population-based screening programs in Europe have demonstrated high rates of low-risk cancer detection, leading to unnecessary biopsies and declining quality of life.[31] Due to these potential harms, the American Cancer Society recommended a shared decision-making approach, ensuring patients are fully informed about the benefits, risks, and uncertainties of prostate cancer screening.[35] Countries such as Kenya follow an individualized, shared decision-making approach to prostate cancer screening, and a similar strategy, combined with strengthened community awareness, should be adopted within Brunei’s cancer screening program.[36]

Our study has some limitations. First, although it is a single-center retrospective analysis, the Brunei Cancer Centre manages all national cancer cases, which allows the findings to reasonably reflect prostate cancer outcomes at the national level Second, the observational design led to some missing clinical information, which may reduce the robustness and interpretability of certain results, such as incomplete body mass index (BMI) data to concluding regarding obesity. Only a subset of patients underwent NGS testing, with a lack of a complete genomic sequence database, limiting statistical power and the detection of a complete genomic mutation pattern from the dataset introduce. Additionally, the non-random selection of patients for NGS testing may introduce selection bias. Finally, an inherent limitation of this study is its primarily descriptive nature, rather than being inferential or prognostic. Despite these constraints, the study offers important insights into epidemiology and genomic characteristics of prostate cancer in Brunei. Furthermore, continued analysis is needed to deepen our understanding of variable associations, and the current findings offer a strong foundation for developing artificial intelligence-based precision oncology tools in the prostate cancer continuum in Brunei Darussalam.

Conclusion

This study provides the first comprehensive overview of the demographic, clinical, biochemical, and genomic characteristics of prostate cancer patients managed at the Brunei Cancer Centre. The findings reveal that most patients present at an advanced stage with metastatic disease and frequently exhibit metabolic comorbidities, reflecting broader regional patterns of delayed diagnosis and increasing metabolic risk among Asian populations. The identification of key DRR genomic alterations highlights significant precision therapeutic opportunities, while several variants of uncertain significance highlight the need for broader genomic testing. Although limited by its retrospective design, incomplete clinical information and partial genomic testing coverage, the study offers valuable insights into emerging disease patterns, including the concerning trend of younger, metabolically at-risk patients presenting

with late-stage disease. These findings highlight the urgent need to strengthen early detection strategies, consider the role of risk-based prostate cancer screening, and expand genomic testing capacity to support more personalised and effective cancer care in Brunei Darussalam. Future studies employing multivariable and causal inference methods will provide deeper insights into the factors driving differences across treatment modalities. Additionally, this comprehensive dataset lays a solid foundation for developing AI-driven risk stratification models and clinical decision support tools.

Abbreviations

AI : Artificial intelligence

APA. : AI-enabled Prescription Advisory

DNA : Deoxyribonucleic acid

EHR. : Electronic Healthcare Records

IQR : Interquartile Range

NCCN: National Comprehensive Cancer Network

NGS : Next Generational Sequencing

PCa : Prostate cancer

PSA. : Prostate-specific antigen

PARP. : Poly (ADP-ribose) polymerase

STROBE : STrengthening the Reporting of Observational Studies in Epidemiology

WHO : World Health Organization

Declarations

Ethics approval and consent to participate:

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Medical and Health Research and Ethics Committee (MHREC), Ministry of Health, Brunei Darussalam (Approval No. MHREC/MOH/2024/22(1); approved on 25 January 2025). Following ethical approval, access to electronic health records of participants under the supervision of the Brunei Cancer Centre (TBCC) was permitted. All data were fully anonymized prior to analysis, and the requirement for informed consent to participate was waived by the MHREC in accordance with national regulations.

Consent for publication:

Permission to publish the findings of this study was obtained from the relevant institutional authorities. All data were anonymized prior to publication.

Competing interest:

All authors do not have a conflict of interest to declare.

Clinical Trial Number

Not applicable.

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Author Contribution

All authors involved in conceptualizations of this report. HMT was responsible for data curation, investigation, methodology, resources, visualization, and writing the original draft. HAR was involved in methodology development, supervision, methodology, resources, and writing, reviewing, and editing the manuscript. OAM provided supervision, methodology, and reviews. LN and MSA contributed through supervision, writing, reviewing, and editing the manuscript. USS and TT contributed through conceptualization, methodology and resources. All authors approve the content of the manuscript.

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Data Availability

The datasets generated and/or analysed during the current study are not publicly available. All data were collected by the research team from The Brunei Cancer Centre(TBCC) as part of this retrospective study. The data supporting the findings of this study are available from the corresponding author upon reasonable request and with permission from the Brunei Cancer Centre and the relevant institutional ethics committee.

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Figures

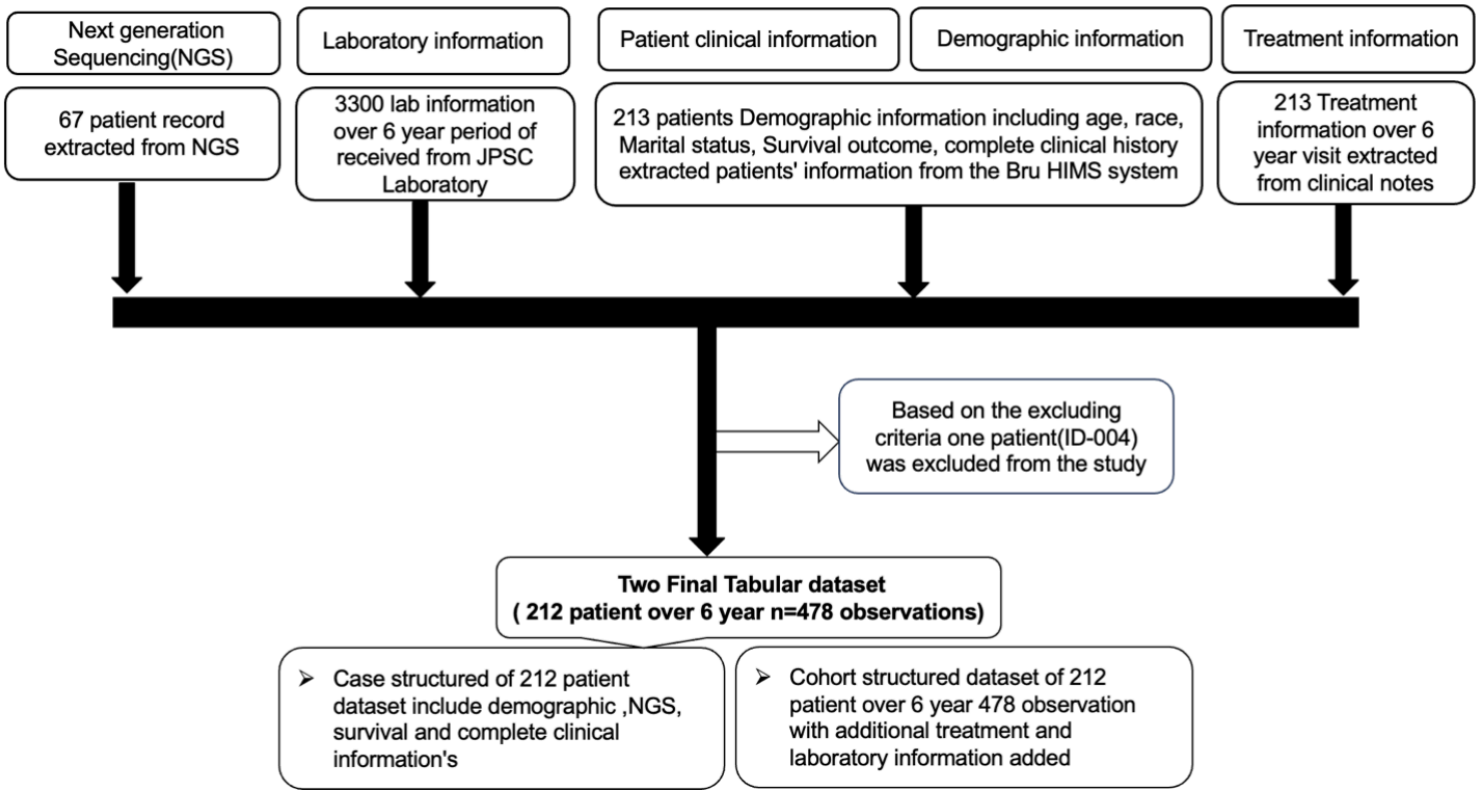
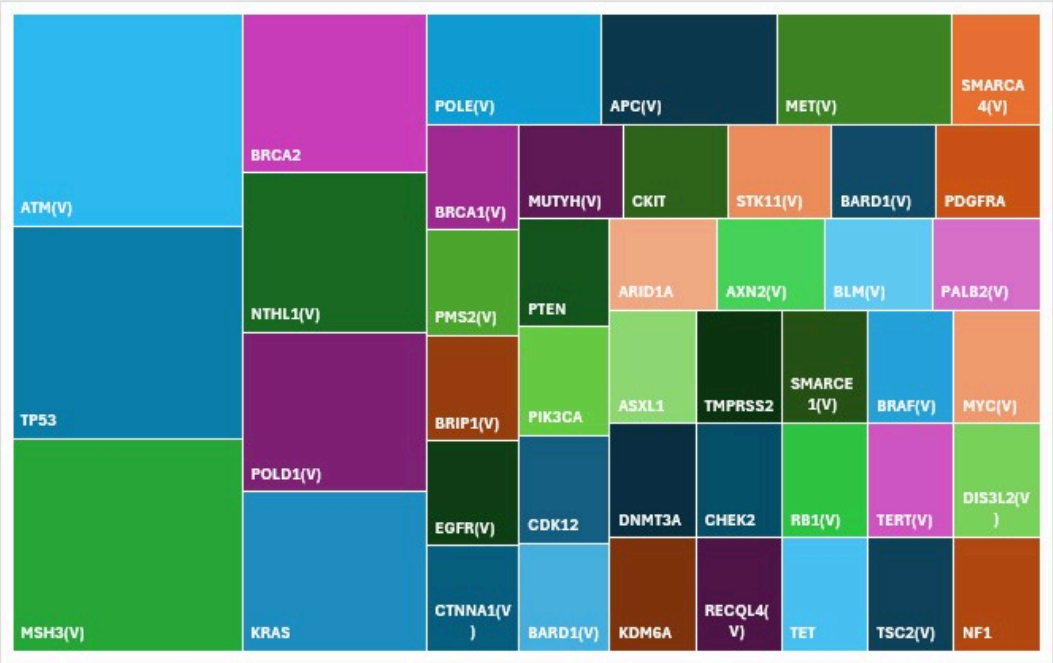


Figure 1

Data Flow Diagram of the Study Cohort along with Extracted Data Sources

(A)



(B)

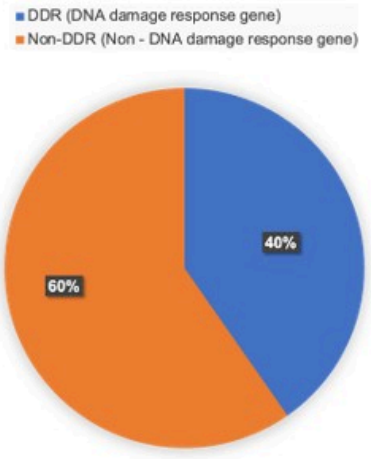


Figure 2

(A) Mutation Gene (B) DDR or Non-DDR gene diagnosed in the prostate cancer patient in Brunei Cancer Canter (TBCC) from January 2018 to December 2024 (n=32)

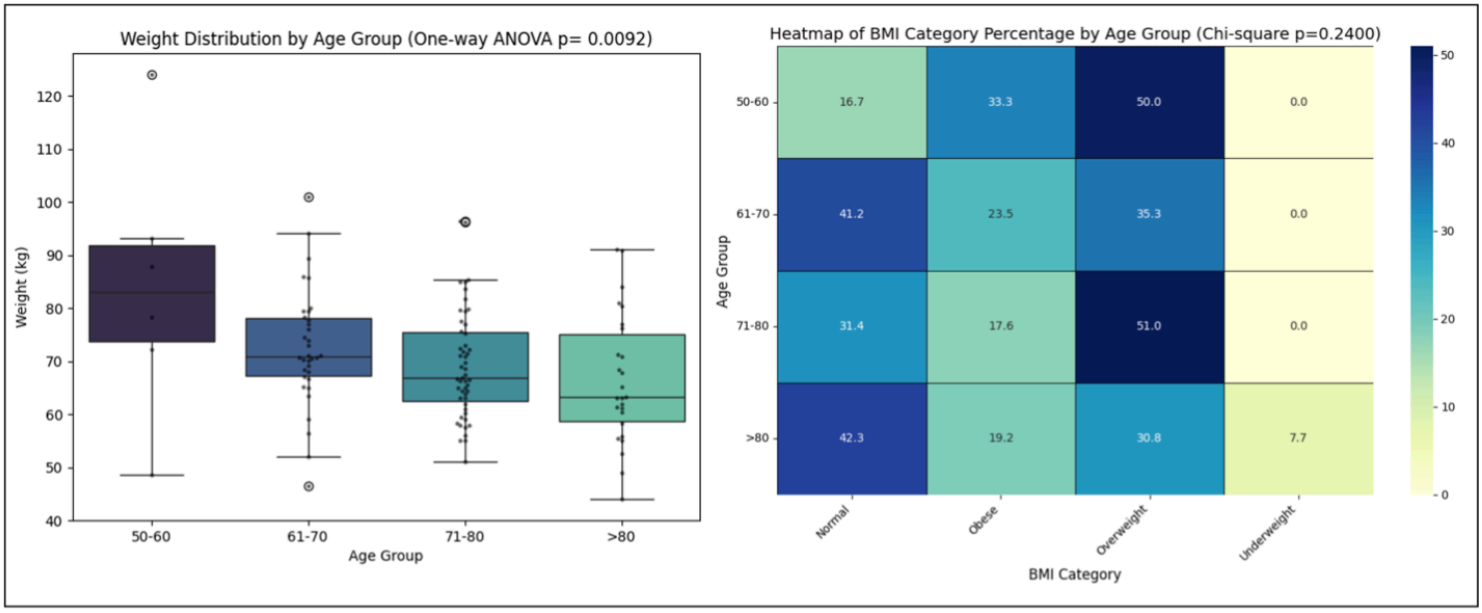


Figure 3

Univariable analysis between different age groups with body weight distribution (boxplot) and body mass index (BMI) categories (heatmap) ofProstate Cancer at Brunei Cancer Centre (TBCC) at case level from January 2018 to December 2024 (n=212)

Supplementary Files

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- Supplementaryfiles.docx