

Outcomes of PSMA radioligand therapy in lymph node-only metastatic prostate cancer: seven-year results from a single centre

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Abstract

Background

Outcomes of Lu-PSMA radioligand therapy in lymph node (LN) only metastatic prostate cancer remain underreported and existing series are small with short follow-up. We report longer-term outcomes from a single tertiary centre.

Methods

We retrospectively reviewed patients treated with prostate specific membrane antigen (PSMA) radioligand theranostics (RLT) for LN-only metastatic prostate cancer between May 2017–May 2025, defined as PSMA-avid nodal disease on ^{68}Ga -PSMA PET/CT without bone or visceral metastases. Outcomes were analysed overall and across three subgroups: metastatic hormone-sensitive disease (mHSPC), mCRPC without prior chemotherapy, and mCRPC with prior chemotherapy. PSA response, PCWG2 progression, prostate cancer-specific survival (PCSS) and toxicity were assessed.

Results

Of 366 patients treated with ^{177}Lu -PSMA RLT, 35 had LN-only disease, 33 were suitable for analysis. The median follow-up time for those still alive at time of analysis was 48 months (IQR 23–74 months). The median number of treatments was three. A PSA decline of $\geq 50\%$ was achieved in 8/11 (73%) patients with mHSPC, all 11 (100%) mCRPC with no prior chemotherapy, and 5/11 (45%) mCRPC with chemotherapy. For those with mHSPC, six patients developed PSA progression with the median time to progression being 47 months (95% CI: 6.9 – not evaluable (NE)). The median time to prostate cancer death was 56 months (95% CI: 26 – NE). Between-group log-rank p values were 0.082 for PSA progression-free survival and 0.003 for prostate cancer-specific survival. No serious haematological or renal toxicity was observed within three months.

Conclusions

In the largest single-centre LN-only ^{177}Lu -PSMA RLT series with extended follow-up, treatment produced high PSA response rates and favourable survival in mHSPC and chemotherapy-naïve mCRPC patients, with minimal toxicity. These findings support prospective evaluation of earlier introduction of Lu-PSMA RLT in LN-only disease.

Introduction

Lymph node metastasis (LNM) from prostate cancer is generally considered by clinicians a non-curative disease, particularly for patients with extra-pelvic LNM outside the field of an extended pelvic lymph

node dissection or whole pelvis radiotherapy. These patients are generally treated with androgen deprivation therapy (ADT), followed by multimodality combinations of androgen receptor pathway inhibitors (ARPI) and chemotherapy. Results from the STAMPEDE trial (1) show that patients with only lymph node metastases have a better overall survival than patients with advanced bone metastatic disease.

In patients who develop LNM after surgery or radiotherapy, standard metastases-directed therapy (MDT) with a salvage pelvic lymph node dissection (PLND) or stereotactic ablative radiotherapy (SBRT) has improved cancer specific survival compared to standard of care with early or delayed ADT (2). In the group of patients where the position of the LNM are not suitable to salvage surgery or SABR, delivering Lutetium- prostate specific membrane antigen (PSMA) (Lu-PSMA) radioligand therapy (RLT) as a metastasis directed therapy (MDT) could also potentially improve prostate cancer specific survival (PCSS) compared to standard of care with ADT.

Treatment with prostate specific membrane antigen Lu-PSMA RLT is based on the delivery of radioactive isotopes to PSMA-positive tumour-associated cells, with subsequent radiation induced cell death. RLT has traditionally been used in patients who progress after standard treatment, including ADT, ARPI's and chemotherapy, with outcomes in the LNM only cohort remaining underreported. These patients traditionally appear to have a better prognosis than patients with bone or skeletal metastasis (3, 4), particularly after progression following chemotherapy (5). Given the improved progression free survival following Lu-PSMA RLT in oligometastatic prostate cancer patients (6), it would be intuitive to suggest that similar improved outcomes might be obtained with a radiopharmaceutical approach to hormone sensitive or hormone naïve lymph node only metastatic disease. However, LNM only RLT series have small patient numbers and short follow-up.

The aim of this study was to evaluate seven years of oncological outcomes following Lutetium-PSMA (Lu-PSMA) RLT in patients with lymph node-only metastatic prostate cancer, and to assess the impact of prior systemic therapies on response and survival, including the outcomes in patients both ADT and chemotherapy naïve. The secondary aim was to confirm the safety of Lu-PSMA RLT with respect to haematological and renal function changes during and shortly after treatment.

Methods

We performed a retrospective case series of patients with lymph node-only metastatic prostate cancer treated with Lu-PSMA RLT at a tertiary referral centre. Before commencing Lu-PSMA RLT, all patients signed a detailed informed consent form. This consent form includes an 'opt-in' option for a theranostics research database developed at I-Med Radiology Network, Wesley Hospital. The hospital Medical Advisory Committee authorized the use of Lu-PSMA RLT at our institution in 2017. The study was conducted in accordance with the Declaration of Helsinki and was approved by the UnitingCare Health Human Research Ethics Committee (reference 2024.12.406). The study period was between May 2017 and May 2025 to allow a minimal follow up period of 12 months. All patients with LNM were diagnosed

with ^{68}Ga -PSMA PET/CT PSMA imaging, with a PSMA avid area corresponding to an anatomical lymph node on the CT scan.

Lu-PSMA was initially synthesised on site with the Modular Lab easy automated synthesizer (Eckert & Ziegler, Berlin, Germany). Subsequently I&T-PSMA was used due to lack of availability of PSMA-617. The Lu-PSMA (7.0–9.0 +/-10% GBq activity) was administered over 10 minutes under the supervision of a dual-trained Radiologist/Nuclear Medicine Specialist. All patients underwent whole-body and two-bed single-photon emission computed tomography imaging to check the distribution of activity, typically 4 hours after Lu-PSMA administration.

Best PSA response was the percentage change from baseline (prior to Lu-PSMA treatment) and to the nadir level, with PSA-50 and PSA-90 defined as a best PSA response of at least a 50% or 90% drop. We analysed PSA progression as per the Prostate Cancer Trials Working Group 2 (PCWG2) criteria (a PSA increase being $\geq 25\%$ and $\geq 2 \mu\text{g/L}$ above the nadir for patients with initial PSA decline and $\geq 25\%$ and $\geq 2 \mu\text{g/L}$ above baseline for patients without PSA decline) [8].

Adverse events within three months post Lu-PSMA RLT were based on assessment of haematological parameters and renal function assessed with eGFR. Haematological abnormalities were graded according to CTCAE v5 and were measured at baseline and within three months of commencing Lu-PSMA treatment.

Death from prostate cancer was also counted as progression in the case that PSA progression had not been recorded. Analysis time for the time to event outcomes commenced on the date of first Lu-PSMA cycle. Patients were censored at the date of last contact for prostate cancer specific survival, and at date of last PSA test for PSA progression. Death from any other cause was also a censoring event at that date. Survival curves were estimated with the Kaplan-Meier method and compared with the log-rank test. No significance level was set, and the p-values reported should be treated as exploratory and nominal. Stata v19MP was used for analysis.

Results

During the study period, 366 patients were treated with Lu-PSMA PRLT for metastatic prostate cancer with 35 men having only LNM. One overseas patient was lost to follow-up, and another was excluded due to prior radiotherapy for bone oligometastases, leaving 33 patients for analysis (Table 1). The median follow-up time for those still alive at time of analysis was 48 months (IQR 23–74 months). 22 patients (67%) had metastatic castration-resistant prostate cancer (mCRPC), including 11 patients who had received prior chemotherapy; the balance ($n = 11$) had metastatic hormone sensitive prostate cancer (mHSPC) with two of these having already commenced ADT.

Short term serum effects

Within three months of Lu-PSMA treatment, 16 patients had Grade 1 anaemia (7 new compared to baseline), and one had Grade 2 anaemia which was also present at baseline (haemoglobin: 99 to 97 g/L). Four patients had Grade 1 thrombocytopaenia, three of these had newly developed. Four patients had eGFR < 60, with one having an eGFR ≥ 60 at baseline (65 to 57). Among patients with available measurements, no white cell count < $3.0 \times 10^9/L$ was recorded at baseline or within three months of treatment.

Best PSA response

The median best PSA response was – 83% (IQR: -99% to -46%), and 10 (30%) recording a nadir PSA of ≤ 0.1 ng/mL. Only one patient had a rise in PSA, from 2.8 ng/mL to 23 ng/mL. PSA-50 was achieved in 8 patients with mHSPC (73%), 11 mCRPC – no chemo (100%), and 5 (45%) mCRPC – chemo (Fig. 1). The corresponding number of patients per disease state with PSA-90 were 6 (55%), 4 (36%), and 3 (27%) respectively. A PSA-50 decline occurred in one of three patients with pelvic nodal disease, 14 of 17 with retroperitoneal/abdominal nodal disease, and nine of 13 with thoracic or supraclavicular nodal disease.

PSA progression

During follow-up, 22 patients experienced PSA progression with median time to progression of 16 months (95% CI: 9.5–46). For those with mHSPC, 6 events occurred with the median time to progression being 47 months (95% CI: 6.9 – not evaluable (NE)). The corresponding observed events and median time for mCRPC – no chemo was 6 events, 31 months (95% CI: 5.9 – NE), and for mCRPC – chemo: 10 events, 10 months (95% CI: 5.9–15) (Fig. 2). There was weak evidence that the three PSA survival curves were different from each other (Log-rank $p = 0.082$).

Prostate cancer specific survival

Sixteen patients died from prostate cancer. One further patient was recorded as dying from liver cancer and was censored at that date. The median time to prostate cancer death was 56 months (95% CI: 26 – NE). For those with mHSPC, two events occurred and median PCSS was not reached (95% CI: 17–NE). For mCRPC without prior chemotherapy, four events occurred and median PCSS was 69 months (95% CI: 23–NE); for mCRPC with prior chemotherapy, ten events occurred and median PCSS was 26 months (95% CI: 10–48) (Fig. 3).

Discussion

Our study is one of the largest series analysing the outcomes of PSMA-Lu in lymph node only metastatic prostate cancer with more patients and longer follow up compared to our 2021 report (7). The patients with mCRPC who had undergone prior chemotherapy had smaller PSA 50% response and higher PCSM

than the chemotherapy naïve patients. The mHSPC patients had promising responses, with median PCSS not reached during follow-up despite most being ADT-naïve at treatment.

The literature is sparse on patients with only LNM at initiation of PSMA-Lu. In the small series of Zisser et al, nine out of 10 patients responded to their first PSMA-RLT course with a mean PSA decline of $71.8 \pm 25.2\%$, with seven demonstrating a PSA decline of $\geq 50\%$ (8). The median progression-free survival was 21 months, and median overall survival was not reached during the median observation time of 52 months. In the Rasul et al publication survival was improved in the nine patients with only LNM compared to the overall group of 46 patients, with a worse prognosis in the bone metastatic cohort (9). von Eyben et al (10) has previously reported on 35 patients identifying the group with only LNM had a better overall survival than with LNM associated with one or two bone metastases.

Early introduction of PSMA RLT prior to chemotherapy may benefit patients with only LNM. In the Enza-P RCT of chemotherapy and APRI naïve mCRPC, the combination of the ARPI enzalutamide with Lu-PSMA RLT was associated with improved overall survival (HR 0.55; 95% CI 0.36 – 0.84, $p = 0.0053$) compared to enzalutamide alone (11). The rationale for the synergy is that ARPI inhibits repair mechanisms that deal with the DNA breaks that are induced by the irradiation of PRLT.

Most international guidelines recommend that patients with mHSPC are treated with ADT combined with an ARPI if they are fit for the regimen. Randomised phase III data is lacking in the specific setting of only LNM in mHSPC patients regarding the timing of introduction of systemic therapy. In the LNM only cohort of N1 / M1a hormone sensitive disease, we hypothesise based on our results that early instigation of PSMA-Lu RLT combined with ADT and an ARPI could potentially improve progression-free survival compared with standard of care. This hypothesis is now being tested in the PSMAAddition RCT of patients with PSMA PET positive mHSPC (12). An early analysis with a median radiographic progression-free survival follow-up time 19.6 months identifies a 28% reduction in the relative risk of radiographic progression or death (HR 0.72, $p = 0.0021$) in favour of the addition of PSMA-Lu to ADT plus an ARPI. This approach of early PRLT would act as a metastasis directed therapy option. Although this trial was not specific to patients with lymph node-only metastatic disease, these findings provide prospective phase III evidence supporting the earlier integration of PSMA-targeted RLT into the metastatic prostate cancer treatment pathway.

In our study, of the nine mHSPC patients who are without prior systemic treatments or androgen deprivation therapy, and a median follow up of 50 months, four remain free of secondary systemic directed therapies including ADT at between 24–83 months follow up. This approach has potentially improved patient quality of life outcomes but needs to be balanced against the concern as to whether a delay in the introduction of systemic treatments such as ADT with an ARPI will have a negative impact on long term prostate cancer specific survival. Results from the BULLSEYE randomised phase II trial of 58 patients with oligometastatic PSMA expressing mHSPC have now been reported (13). This is the first prospective study to randomise four cycles of ^{177}Lu -PSMA RLT as MDT compared to the standard of care of deferred ADT. With a median follow up of 27 months, median progression-free survival (per-

protocol) was 25 months (IQR 15-not reached) for intervention group versus 5 months (IQR 3–7) for control group. These results provide prospective randomised evidence supporting PSMA-targeted RLT as a potential ADT-delaying treatment strategy in appropriately selected patients with oligometastatic hormone-sensitive disease.

The LUNAR randomised phase II trial (3) in patients with oligo-recurrent hormone-sensitive prostate cancer supported using PRLT as a metastases directed therapy. The trial used two cycles of neoadjuvant ¹⁷⁷Lu-PNT2002 RLT followed by SBRT. The combination of RLT and SBRT significantly improved progression-free survival compared with SBRT alone (17.6 versus 7.4 months; HR 0.37; $p < 0.0001$), without an increase in severe adverse effects. Although not specific to a LNM only cohort, these results again support the hypothesis that MDT using RLT could be an effective option for delaying systemic treatment escalation in highly selected patients, especially in mHSPC patients with LNM sites not suitable for SABR

Our LNM only patients had no severe adverse effects from Lu-PSMA RLT even in the mCRPC sub-group. Specifically, there were no clinically significant effects on haematological or renal function outcomes. This contrasts to other studies, with Flippot et al reporting that 26 (21%) of 126 patients with mCRPC and prior chemotherapy developed severe anemia, asthenia, and thrombocytopenia after Lu-PSMA RLT, with LNM only comprising 7% of this study (14). Our LNM only patients even with prior chemotherapy have less underlying bone marrow dysfunction. Therefore, they better tolerate the PRLT. Previous data identified high osseous tumour burden, prior taxane chemotherapy and baseline cytopenia as risk factors for clinically severe myelosuppression following PSMA-Lu RLT (15). This may explain the haematological tolerance observed in our LNM-only cohort. Also, we only used a median of three PSMA RLT cycles.

In conclusion, patients with lymph node only metastatic prostate cancer obtain good oncological response to Lu-PSMA RLT. The favourable outcomes may partly reflect the favourable biology of LN-only disease. Our study has strengths. The study provides many new insights regarding the use of RLT for LNM patients. This includes patients who underwent neoadjuvant RLT and a longer follow-up than had been reported in previous studies. The results help to better understand the potential of Lu-PSMA in LNM patients. Our study also has limitations. The study was a retrospective analysis. We did not formally collect QOL data. In Australia TGA (Therapeutic Goods Administration) has approved Lu-PSMA use in the treatment of patients with prostate cancer. However, funding by federal government Medicare is only for RLT treatment of mCRPC after they have undergone one prior taxane chemotherapy and at least one androgen signaling inhibitor. Therefore, patient numbers are limited as patients needed to self-fund the RLT until the recent Medicare reimbursement for mCRPC post chemotherapy and an ARPI. Not all patients have access to clinical trials. Randomised phase III data is lacking in the specific setting of patients with only LNM in mHSPC regarding the timing of introduction of systemic therapy. Therefore, until prospective randomised outcomes are available specifically for patients with lymph node-only metastatic disease, prospective data collection and publication of case series results are required to help better understand the outcomes of Lu-PSMA in the patient cohort with only LNM.

Declarations

Competing interests: The authors declare no competing interests.

Data availability: The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to approval by the relevant institutional ethics committee and applicable patient privacy requirements.

Author contributions: J.W.Y. designed the study, collected data and contributed to drafting the manuscript. A.A., J.K., D.W. and P.V. collected data and contributed to preparation and revision of the manuscript. F.v.E. and W.J.Y. contributed to interpretation of the findings and critical revision of the manuscript. All authors reviewed and approved the final manuscript and agree to be accountable for the work.

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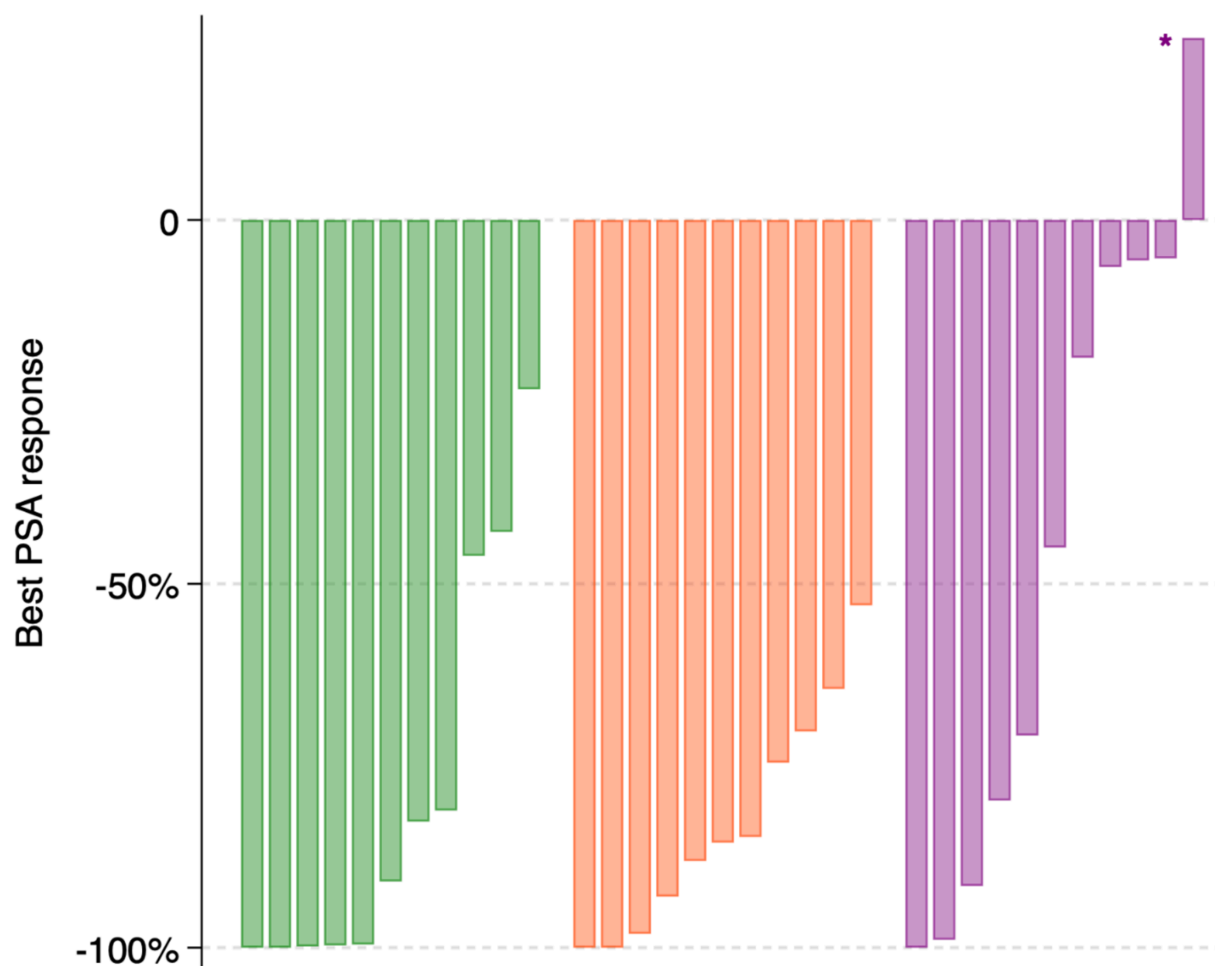
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Table 1

Table 1 is available in the Supplementary Files section.

Figures



	PSA-50, n (%)	PSA-90, n (%)
mHSPC	8 (72)	6 (55)
mCRPC – no prior chemo	11 (100)	4 (36)
mCRPC – prior chemo	5 (45)	3 (27)

Figure 1

Best PSA response by disease state. Green: mHSPC; orange: mCRPC without prior chemotherapy; purple: mCRPC with prior chemotherapy. *PSA increased from 2.8 to 23 ng/mL.

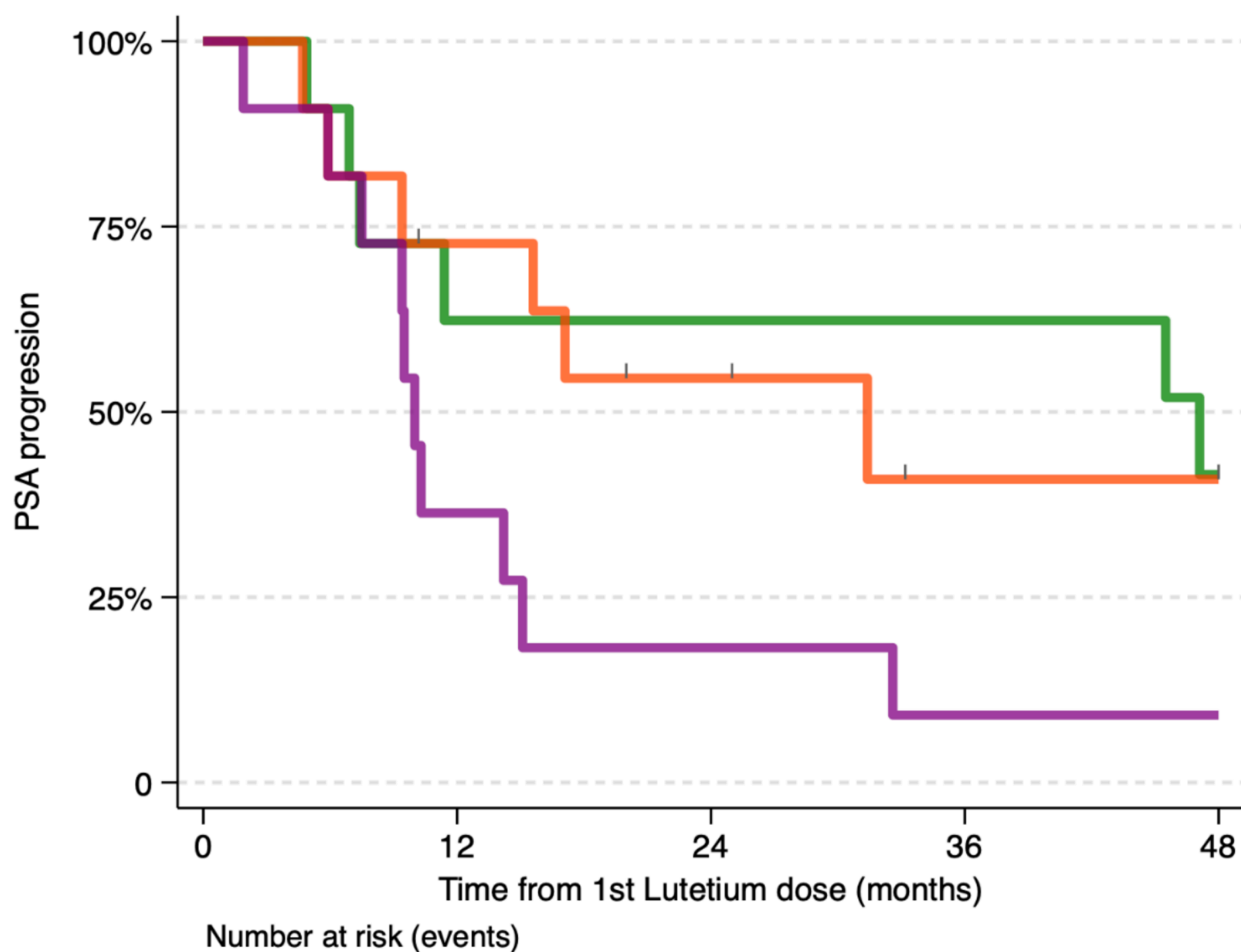


Figure 2

Kaplan–Meier curves for PSA progression-free survival according to disease state. Green: mHSPC; orange: mCRPC without prior chemotherapy; purple: mCRPC with prior chemotherapy. Twenty-two progression events occurred; median time to PSA progression was 16 months (95% CI 9.5–46).

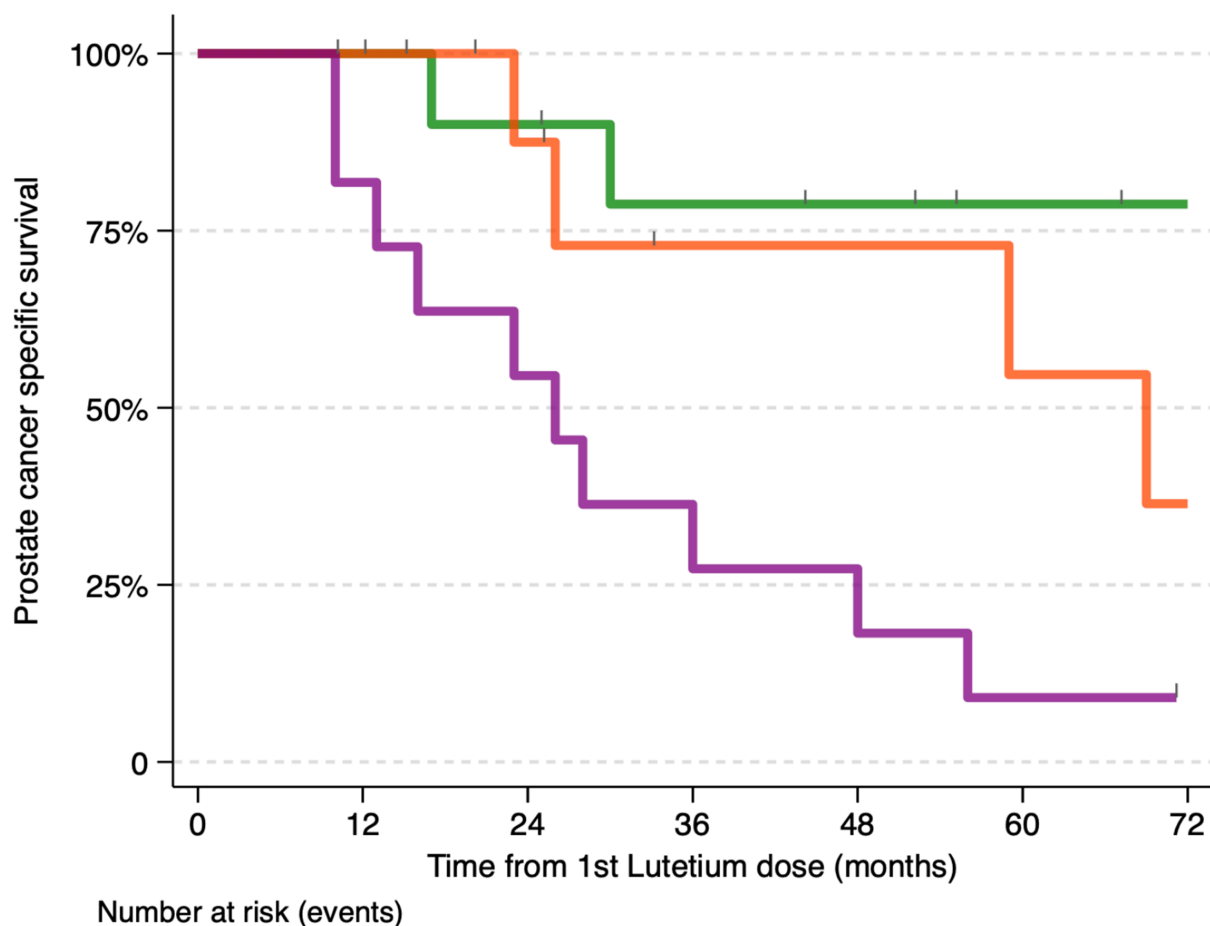


Figure 3

Kaplan–Meier curves for prostate cancer-specific survival according to disease state. Green: mHSPC; orange: mCRPC without prior chemotherapy; purple: mCRPC with prior chemotherapy. Sixteen prostate cancer-specific deaths occurred; median PCSS was 56 months (95% CI 26–NE).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Table1.xlsx](#)