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Impact of Body Mass Index on Survival After Docetaxel Chemotherapy for Metastatic Castration-resistant Prostate Cancer

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Abstract

Background/Aim: Several recent studies, varying in design and the number of baseline parameters analyzed, suggest that a lower body mass index (BMI) is associated with shorter survival in men beginning treatment for metastatic castration-resistant prostate cancer (MCRPC), including treatments such as docetaxel. This study aimed to analyze the impact of BMI and numerous covariates on survival in a homogeneously treated cohort of Caucasian men who received first-line docetaxel for MCRPC.

Patients and Methods: This retrospective analysis included 112 consecutive patients managed between 2009 and 2023. Comorbidity, medications, and blood tests were included. Both, uni- and multivariate tests were performed.

Results: The median age of the patients was 70 years with a median BMI of 26.8 kg/m^2. Most patients were free from serious comorbidities, had bone-only metastases, and experienced metachronous development of metastases. Hemoglobin values were significantly lower in patients with lower BMI (median 11.9 *versus* 13.0 g/dl, p=0.001). Lean patients had numerically shorter survival compared to overweight/obese patients (median 11.8 *versus* 19.4 months, p=0.15). In multivariate analysis of prognostic factors, only three baseline parameters retained statistical significance: serum lactate dehydrogenase (p=0.03), hemoglobin (p=0.007), and the presence of non-bone metastases (p=0.004).

Conclusion: An interaction between BMI and hemoglobin was present in metastatic castration-resistant prostate cancer patients after docetaxel chemotherapy, explaining the observed survival difference between lean and overweight/obese patients. Comorbidities and medications had no significant impact on survival in this population with limited prognosis (median survival 16.1 months).

Keywords: Prostate cancer, anemia, overall survival, nutrition, prognosis, systemic treatment.

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Introduction

Metastatic castration-resistant prostate cancer (MCRPC) represents the final stage of prostate cancer progression, resulting in limited prognosis and often serious clinical symptoms such as bone pain, anemia, cachexia, and dyspnea (1-6). Palliative chemotherapy with docetaxel has long been a mainstay of life-prolonging systemic therapy for MCRPC (7). In recent years, additional options have emerged, including chemotherapy-free alternatives such as abiraterone acetate, enzalutamide, Ra-223, other radioligands, and PARP inhibitors (depending on genetic alterations) (8-12). Due to changes in preceding treatment during the less advanced, hormone-sensitive phase, which may involve upfront administration of docetaxel, abiraterone acetate, enzalutamide, and other drugs (13-17), MCRPC therapy has become less standardized. Docetaxel continues to represent a reasonable option in chemotherapy-eligible and -sensitive patients (18).

Survival after initiation of first-line systemic therapy for MCRPC is highly variable and is influenced by several baseline factors such as performance status (PS), disease extent, and organ function (19). Blood test results may reflect aspects of disease extent and organ function, while also reflecting levels of inflammation and nutritional status (20, 21). An increasing body of evidence suggests that body mass index (BMI) is one of the clinically relevant, readily available prognostic factors that may aid in the prediction of survival (22-28). Some of these studies have focused specifically on docetaxel-treated patients with MCRPC (22, 24). In general, patients with lower BMI had shorter survival than their peers with higher BMI. However, a complex interplay may exist between BMI, the presence of comorbidities and blood test results. It is possible that patients with a low BMI as a result of cachexia and the presence of visceral metastases should be distinguished from those with less advanced disease and different causes of low BMI. The present study was performed to analyze the impact of BMI and numerous covariates on survival in a homogeneously treated cohort of men who received first-line docetaxel for MCRPC.

Patients and Methods

Patients. This study utilized our institutional quality-of-care monitoring database, which has been described before (12, 18, 29). All patients had MCRPC and were managed with first-line docetaxel in various dosing regimens. The latter included the most efficacious dosing every three weeks (75 mg/m^2) together with prednisolone and androgen deprivation therapy (ADT). For patients with anticipated tolerance issues, two lower-dose alternatives were available: weekly or biweekly dosing. The weekly regimen was preferred in frail patients with Eastern Cooperative Oncology Group (ECOG) PS 2, very elderly patients, or those with comorbidity-related concerns. Only patients with ECOG PS 0-2 and appropriate bone marrow function received docetaxel. The number of cycles was adjusted to tolerance, effect, and patient preference. Chemotherapy holidays were provided as needed. After docetaxel, further systemic treatment options included abiraterone acetate, enzalutamide, cabazitaxel or Ra-223. Newer targeted radioligands and PARP inhibitors were not accessible in the time period studied here (2009-2023). We used traditional staging methods, such as radionuclide bone scan, computed tomography or magnetic resonance imaging, rather than routine positron emission tomography. A total of 112 patients, all Caucasian and covered by the public Norwegian healthcare system, had received first-line docetaxel for MCRPC and were included in the study.

Methods. Data were analyzed retrospectively. BMI (kg/m^2) was calculated from the chemotherapy dosing notes in the hospital's electronic health record, which contained body weight and height before administration of the first docetaxel cycle. At the same timepoint, routine blood tests were performed. All patients had complete information available, *i.e.*, no missing values. Actuarial survival from the day of docetaxel initiation was calculated with the Kaplan–Meier method and compared between subgroups with the log-rank test. A single patient was alive at the time of

analysis in October 2024 and censored at 25 months of follow-up. Date of death was recorded in all other patients. Associations between different variables of interest were assessed with the chi-square or Fisher's exact probability test (two-tailed). The impact of continuous variables such as BMI and blood test results on survival was examined in univariate Cox analyses. A multivariate forward conditional Cox analysis of prognostic factors for survival was then performed. A *p*-value <0.05 was considered statistically significant. Statistical analyses were performed with IBM SPSS Statistics 29 (IBM Corp., Armonk, NY, USA).

Results

Baseline characteristics. The majority of patients (54%) belonged to the overweight category (BMI 25.0-29.9), followed by healthy weight (29%) and obesity (17%). The median age was 70 years and typical patients were free from serious comorbidities, had bone-only metastases, and metachronous development of metastases after an initial diagnosis of non-metastatic prostate cancer (Table I). Serum albumin below the institutional lower limit of normal was very rare (2%), while other abnormalities were common [elevated C-reactive protein (CRP), lactate dehydrogenase (LDH), and alkaline phosphatase (ALP), anemia]. The median prostate-specific antigen (PSA) level was 119 µg/l, indicating a relatively high burden of metastases.

Parameters associated with lower BMI. Given that none of the patients were underweight (BMI <18.5), comparisons were made between those with healthy weight (about the first quartile, in exact terms 29%) and all remaining patients (overweight/obese). All baseline characteristics displayed in Table I were compared. A limited number of statistically significant differences emerged. Healthy-weight patients were less often statin users (13 *versus* 32%, *p*=0.04), on anticoagulants and/or cardiovascular prevention drugs (19 *versus* 44%, *p*=0.02), and their hemoglobin values were lower (median 11.9 *versus* 13.0 g/dl, *p*=0.001). However, neither the pattern of metastatic spread nor the comorbidity burden was significantly different. We did not collect information on conditions such as hypercholesterolemia and arterial hypertension. The differences in medication use likely reflect the higher prevalence of these conditions in overweight/obese patients. We also compared median BMI and proportion of men in the two different BMI categories

Table I. *Baseline characteristics (n=112): First-line docetaxel for metastatic castration-resistant prostate cancer; time period 2009-2023; conducted at Nordland Hospital Trust, Bodø, Norway.*

Parameter	n	%
Docetaxel every three weeks	47	42
Docetaxel every two weeks	14	13
Docetaxel weekly	51	46
Docetaxel pre-exposed	6	5
Docetaxel naïve	106	95
Concomitant bisphosphonate users	47	42
Underweight, body mass index <18.5	0	0
Healthy weight, body mass index 18.5-24.9	32	29
Overweight, body mass index 25-0-29.9	61	54
Obesity, body mass index ≥30	19	17
Grade group 5	37	33
Grade group 4	18	16
Grade group 3	11	10
Grade group 1 or 2	36	32
Not biopsied	10	9
De novo distant metastases	44	39
Subsequent distant metastases	68	61
Visceral metastases	21	19
No visceral metastases	91	81
Node metastases alone	11	10
Not node metastases alone	101	90
Bone metastases alone	80	71
Any cardiac comorbidity	19	17
Diabetes mellitus	5	4
Previous other cancer	6	5
Statin users	30	27
Cardiovascular prevention drug users	28	25
Anticoagulant users	14	13
Abnormally low serum albumin	2	2
Abnormally high serum C-reactive protein	55	49
Median hemoglobin, range (g/dl)	12.9, 9.0-16.0	
Median PSA, range (µg/l)	119, 2-3,855	
Median lactate dehydrogenase, range (U/l)	245, 144-1,983	
Median alkaline phosphatase, range (U/l)	172, 47-1,723	
Median age, range (years)	70, 56-86	
Median ECOG PS, range	1, 0-2	
Median Charlson comorbidity index, range	0, 0-3	
Median body mass index, range	26.8, 19.8-38.7	

PSA: Prostate-specific antigen; ECOG PS: Eastern Cooperative Oncology Group performance status.

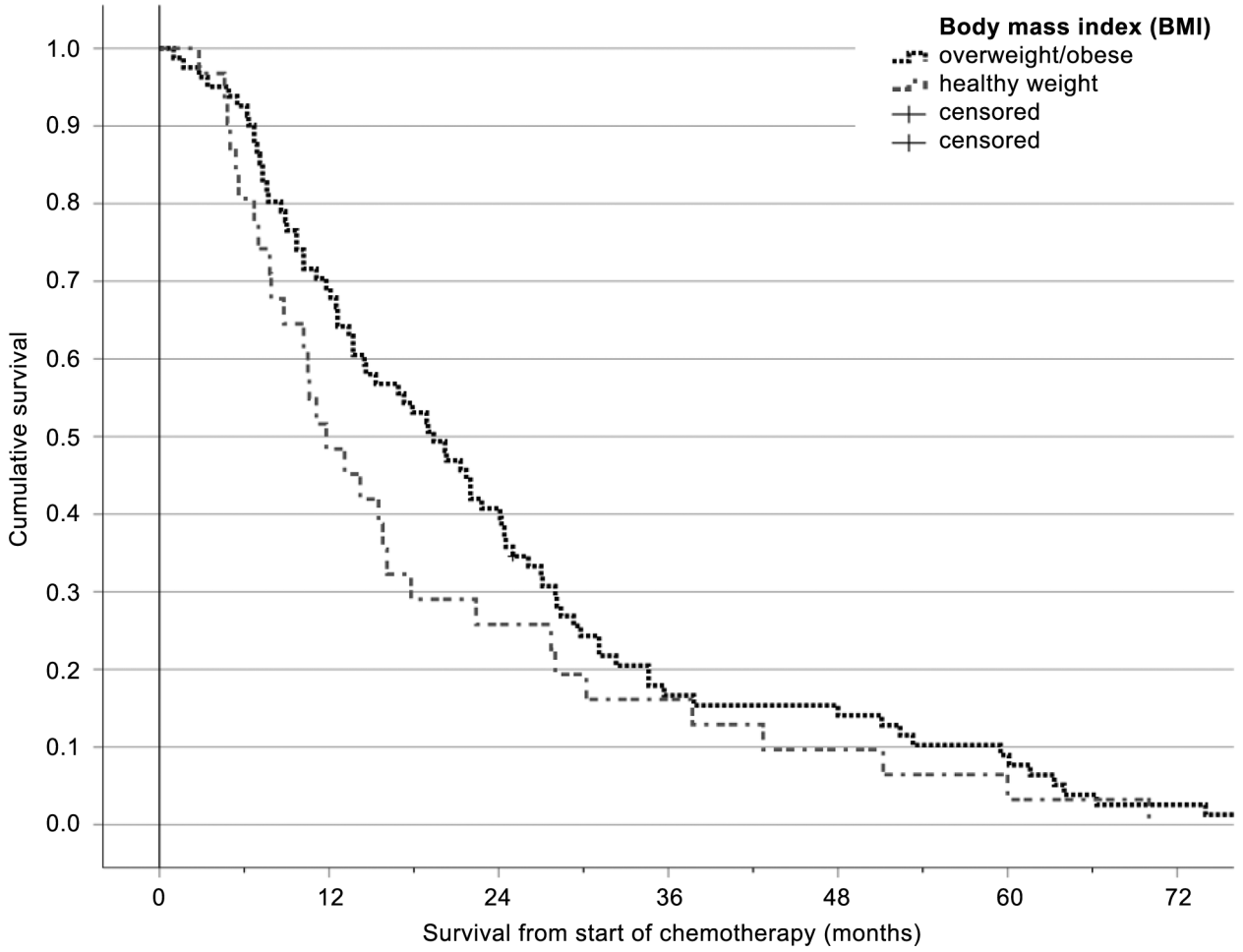

Figure 1. *Actuarial Kaplan–Meier survival curves for patients treated with first-line docetaxel for metastatic castration-resistant prostate cancer stratified by BMI (n=32 and 80, respectively), p=0.15.*

analyzed here in patients with a different pattern of spread such as visceral metastases or bone metastases alone. All these comparisons revealed similar distributions and *p*-values >0.2.

Survival. Median survival was 16.1 months. At 5 years, 8% were still alive. All baseline characteristics displayed in Table I were included in univariate analysis of prognostic factors for survival. With a *p*-value of 0.19, BMI was not significantly associated with survival in the Cox analysis. When displayed as Kaplan–Meier curves (Figure 1), healthy-weight patients had shorter survival than overweight/obese patients (median 11.8 *versus* 19.4 months, p=0.15). Factors with *p*-value <0.1 are shown in Table II. These included all blood test results, pattern of metastases, presentation setting (*de novo versus* metachronous metastases), and chemotherapy dosing regimen. In the multivariate forward conditional Cox analysis of prognostic factors for survival, only three baseline parameters retained statistical significance: LDH (p=0.03), hemoglobin (p=0.007), and non-bone metastases (p=0.004). Replacing the continuous variable hemoglobin with the dichotomized variable anemia (local laboratory cut-off <13.4 g/dl) did not change any of the results.

Table II. *Univariate prognostic factors for survival (displayed if p<0.1).*

Parameter	Median (months)	*p*-Value	Independent factor after multivariate analysis
Docetaxel every three weeks	22.0		
Other dosing regimen	11.8	0.09	No
Metachronous metastases	19.4		
Primarily metastatic disease	12.1	0.02	No
Bone only metastases	21.3		
Other pattern of metastases	10.2	<0.001	Yes
PSA (μg/l)	Continuous variable	0.04	No
Hemoglobin (g/dl)	Continuous variable	<0.001	Yes
Lactate dehydrogenase (U/l)	Continuous variable	<0.001	Yes
Alkaline phosphatase (U/l)	Continuous variable	<0.001	No
C-reactive protein (mg/l)	Continuous variable	<0.001	No

PSA: Prostate specific antigen.

Discussion

This study was performed as an addendum to several recent publications, which have suggested that BMI is a statistically significant prognostic factor for survival in MCRPC settings including but not limited to docetaxel-treated patients (22-28). In the first docetaxel study (published in 2016, n=63) several body composition parameters (skeletal muscle mass, muscle attenuation, visceral, and subcutaneous fat tissue) were measured at spinal level L3 in addition to BMI (22). Sarcopenia was present in 47% of patients. Measurements of adiposity were not predictive of dose-limiting chemotherapy toxicity. Neither sarcopenia nor sarcopenic obesity was associated with survival. In multivariate analysis of prognostic factors, BMI ≥25 kg/m^2 was a significant predictor of longer survival and both visceral fat index ≥ median and anemia [hazard ratio (HR)=2.8, 95% confidence interval (CI)=1.3-6.1, *p*=0.009] were significant predictors of shorter overall survival. The second docetaxel study (published in 2021, n=170) assessed the geriatric nutritional risk index (GNRI), which was calculated using serum albumin and BMI, with a poor nutritional status defined as GNRI <92 (24). In the study, 45 patients were of poor nutritional status. The median survival was 30.4 months in the good nutritional status group and 11.1 months in the poor nutritional status group (*p*<0.001). In multivariate analysis, poor nutritional status was an independent prognostic factor, together with a high metastatic volume and cumulative docetaxel dose. In the third docetaxel study published in 2022, Verma *et al.* studied 466 patients without data on serum hemoglobin and found a median survival of 16.6, 20.1, and 21.4 months (*p*=0.002) for lean, overweight, and obese patients, respectively (27). After adjusting for baseline and tumor characteristics, overweight (*p*=0.006) and obese (*p*=0.003) patients had significantly better survival compared with lean patients. Martini *et al.* studied 1,577 men with MCRPC treated with docetaxel (25). Their analyses were adjusted for age, PSA, ECOG PS, number of metastases, and prior treatment. Regarding survival, BMI emerged as a protective factor both as a continuous variable and as a categorical variable (obesity: HR=0.71, 95%CI=0.53-0.96; *p*=0.027, relatively to normal weight). No interaction was detected between the BMI categories and the docetaxel dose at any level. In other words, the protective effect of BMI was not related to receiving higher chemotherapy doses.

Our own study did not include GNRI, because only two patients had abnormally low serum albumin. Imaging-based body composition data were not available in our database. Although not statistically significant, lower BMI was associated with shorter survival. There was however an interaction with serum hemoglobin, which was significantly

lower in patients with lower BMI. Given that serum hemoglobin was a strong prognostic factor for survival, the impact of BMI remained non-significant in all our analyses. In essence, all five docetaxel studies had a variable extent of available baseline parameters, and eventually different multivariate prognostic model outcomes.

In addition to the study's size and its retrospective design, limitations of the present work include the lack of information on patient-reported factors such as nausea, appetite, and recent weight loss, which may also predict a shorter survival. It would be interesting to stratify a larger patient population into different categories of low BMI, such as cancer-related, comorbidity-related, and genuine body composition. Strengths of our study include the homogeneous population (Caucasian men with MCRPC receiving first-line therapy), availability of comorbidity and medication data, and complete blood test information. Interestingly, we found no statistically significant impact of comorbidity and medications on survival. BMI did not significantly correlate with the presence of visceral metastases, *i.e*., more aggressive disease. Analyses of other efficacy endpoints or chemotherapy toxicity were not performed.

Finally, the remaining studies on BMI will be discussed. The Govindan *et al.* study (n=5,231) is unique, because veterans treated with abiraterone acetate or enzalutamide within the Veterans Health Administration were assessed (28). The Charlson comorbidity index was included. BMI was associated with survival and a longest median survival of 29.8 months in BMI ≥30, 23.9 months in BMI 25-30, 15.9 months in BMI 18.5-25, and 9.2 months in BMI <18.5 (p<0.001). In the multivariate analysis, compared to normal BMI, increased mortality was observed in BMI <18.5 and a decreased mortality in BMI 25-30 and BMI >30. Modonutti *et al.* studied patients enrolled in the control arm of five randomized trials (first-line MCRPC therapy), randomly split between training (n=1,636) and validation cohorts (n=700) (26). These authors constructed a novel nomogram. At multivariate analysis, several independent predictors of survival emerged: sites of metastasis (visceral *vs.* bone metastasis), PSA, aspartate transaminase, ALP, BMI, and hemoglobin (≥13 g/dl *versus* <11 g/dl). A nomogram based on these variables was developed, which showed favorable discrimination and calibration characteristics on external validation. However, all patients had ECOG PS 0-1 and LDH was not part of the studied parameters. Despite these drawbacks, given the data source, methods, and study size, this work lends credit to the overall impression from the recent literature, suggesting that lower BMI is prognostically unfavorable in men starting treatment for MCRPC. Instead of BMI, sarcopenia assessed on computed tomography images has also been studied, with inconsistent results (30).

It is also relevant to note that recent studies have highlighted several trends in MCRPC treatment, *e.g*., treatment initiation at lower PSA level and often with androgen receptor axis-targeted agents, which allow for treatment of elderly patients unfit for docetaxel (31). These developments impact on second-line systemic therapy, which also has life-prolonging potential. Recent data on second-line treatment suggested that time from first-line androgen receptor axis-targeted agents to progression under 12 months was associated with shorter survival in the nest line consisting of either docetaxel or androgen receptor axis-targeted agents (32). A subgroup of patients may benefit from docetaxel rechallenge during the course of MCRPC, depending on initial response and toxicity profile (33).

Conclusion

An interaction between BMI and hemoglobin was present, explaining the observed survival difference between lean and overweight/obese patients. Comorbidity and medications had no significant impact on survival in this population with limited prognosis (median survival 16.1 months).

Conflicts of Interest

The Authors declare that they have no conflicts of interest in relation to this study.

Authors' Contributions

CN participated in the design of the study and performed the statistical analysis. CN, LS, AD, and ECH conceived the study and drafted the article. All Authors read and approved the final article.

References

1 van Soest RJ, Efstathiou JA, Sternberg CN, Tombal B: The natural history and outcome predictors of metastatic castration-resistant prostate cancer. Eur Urol Focus 2(5): 480-487, 2016. DOI: 10.1016/j.euf.2016.12.006

2 Shore ND, Drake CG, Lin DW, Ryan CJ, Stratton KL, Dunshee C, Karsh LI, Kaul S, Kernen K, Pieczonka C, Sieber P, Stewart C, Williams M, Concepcion RS: Optimizing the management of castration-resistant prostate cancer patients: A practical guide for clinicians. Prostate 80(14): 1159-1176, 2020. DOI: 10.1002/pros.24053

3 Kulasegaran T, Oliveira N: Metastatic castration-resistant prostate cancer: advances in treatment and symptom management. Curr Treat Options Oncol 25(7): 914-931, 2024. DOI: 10.1007/s11864-024-01215-2

4 Nishino T, Yamamoto S, Numao N, Komai Y, Oguchi T, Yasuda Y, Fujiwara R, Yuasa T, Yonese J: Predictors of progression to castration-resistant prostate cancer after radical prostatectomy in high-risk prostate cancer patients. Cancer Diagn Progn 4(5): 646-651, 2024. DOI: 10.21873/cdp.10376

5 Nieder C, Haukland E, Pawinski A, Dalhaug A: Pathologic fracture and metastatic spinal cord compression in patients with prostate cancer and bone metastases. BMC Urol 10: 23, 2010. DOI: 10.1186/1471-2490-10-23

6 Nieder C, Haukland E, Pawinski A, Dalhaug A: Anaemia and thrombocytopenia in patients with prostate cancer and bone metastases. BMC Cancer 10: 284, 2010. DOI: 10.1186/1471-2407-10-284

7 Tannock IF, de Wit R, Berry WR, Horti J, Pluzanska A, Chi KN, Oudard S, Théodore C, James ND, Turesson I, Rosenthal MA, Eisenberger MA, TAX 327 Investigators: Docetaxel plus prednisone or mitoxantrone plus prednisone for advanced prostate cancer. N Engl J Med 351(15): 1502-1512, 2004. DOI: 10.1056/NEJMoa040720

8 Iannantuono GM, Chandran E, Floudas CS, Choo-Wosoba H, Butera G, Roselli M, Gulley JL, Karzai F: Efficacy and safety of PARP inhibitors in metastatic castration-resistant prostate cancer: A systematic review and meta-analysis of clinical trials. Cancer Treat Rev 120: 102623, 2023. DOI: 10.1016/j.ctrv.2023.102623

9 Patell K, Kurian M, Garcia JA, Mendiratta P, Barata PC, Jia AY, Spratt DE, Brown JR: Lutetium-177 PSMA for the treatment of metastatic castrate resistant prostate cancer: a systematic review. Expert Rev Anticancer Ther 23(7): 731-744, 2023. DOI: 10.1080/14737140.2023.2213892

10 Ito H, Yaegashi H, Okada Y, Shimada T, Yamaoka T, Okubo K, Sakamoto T, Mizokami A: Appropriate patient status for Ra-223 treatment in the treatment sequence for castration-resistant prostate cancer. Cancer Diagn Progn 2(4): 462-470, 2022. DOI: 10.21873/cdp.10129

11 Vigneswaran HT, Warnqvist A, Andersson TML, Leval A, Eklund M, Nordström T, Eloranta S, Schain F, Dearden L, Liwing J, Mehra M, Nair S, Pettersson A, Akre O, Aly M: Real world treatment utilization patterns in patients with castration-resistant prostate cancer. Scand J Urol 55(4): 299-306, 2021. DOI: 10.1080/21681805.2021.1936626

12 Nieder C, Haukland E, Mannsåker B, Pawinski A, Yobuta R, Norum J: Initiation of systemic therapy during the last 30 days of life in patients with metastatic castration-resistant prostate cancer. Anticancer Res 39(1): 335-340, 2019. DOI: 10.21873/anticanres.13116

13 Sweeney CJ, Chen YH, Carducci M, Liu G, Jarrard DF, Eisenberger M, Wong YN, Hahn N, Kohli M, Cooney MM, Dreicer R, Vogelzang NJ, Picus J, Shevrin D, Hussain M, Garcia JA, DiPaola RS: Chemohormonal therapy in metastatic hormone-sensitive prostate cancer. N Engl J Med 373(8): 737-746, 2015. DOI: 10.1056/NEJMoa1503747

14 James ND, Sydes MR, Clarke NW, Mason MD, Dearnaley DP, Spears MR, Ritchie AW, Parker CC, Russell JM, Attard G, de Bono J, Cross W, Jones RJ, Thalmann G, Amos C, Matheson D, Millman R, Alzouebi M, Beesley S, Birtle AJ, Brock S, Cathomas R, Chakraborti P, Chowdhury S, Cook A, Elliott T, Gale J, Gibbs S, Graham JD, Hetherington J, Hughes R, Laing R, McKinna F, McLaren DB, O'Sullivan JM, Parikh O, Peedell C, Protheroe A, Robinson AJ, Srihari N, Srinivasan R, Staffurth J, Sundar S, Tolan S, Tsang D, Wagstaff J, Parmar MK, STAMPEDE investigators: Addition of docetaxel, zoledronic acid, or both to first-line long-term hormone therapy in prostate cancer (STAMPEDE): survival results from an adaptive, multiarm, multistage, platform randomised controlled trial. Lancet 387(10024): 1163-1177, 2016. DOI: 10.1016/S0140-6736(15)01037-5

15 Armstrong AJ, Szmulewitz RZ, Petrylak DP, Holzbeierlein J, Villers A, Azad A, Alcaraz A, Alekseev B, Iguchi T, Shore ND, Rosbrook B, Sugg J, Baron B, Chen L, Stenzl A: ARCHES: A randomized, phase III study of androgen deprivation therapy with enzalutamide or placebo in men with metastatic hormone-sensitive prostate cancer. J Clin Oncol 37(32): 2974-2986, 2019. DOI: 10.1200/JCO.19.00799

16 Chi KN, Agarwal N, Bjartell A, Chung BH, Pereira de Santana Gomes AJ, Given R, Juárez Soto Á, Merseburger AS, Özgüroğlu M, Uemura H, Ye D, Deprince K, Naini V, Li J, Cheng S, Yu MK, Zhang K, Larsen JS, McCarthy S, Chowdhury S, TITAN Investigators: Apalutamide for metastatic, castration-sensitive prostate cancer. N Engl J Med 381(1): 13-24, 2019. DOI: 10.1056/NEJMoa1903307

17 Smith MR, Hussain M, Saad F, Fizazi K, Sternberg CN, Crawford ED, Kopyltsov E, Park CH, Alekseev B, Montesa-Pino Á, Ye D, Parnis F, Cruz F, Tammela TLJ, Suzuki H, Utriainen T, Fu C, Uemura M, Méndez-Vidal MJ, Maughan BL, Joensuu H, Thiele S, Li R, Kuss I, Tombal B, ARASENS Trial Investigators: Darolutamide and survival in metastatic, hormone-sensitive prostate cancer. N Engl J Med 386(12): 1132-1142, 2022. DOI: 10.1056/NEJMoa2119115
18 Nieder C, Stanisavljevic L, Dalhaug A, Haukland E: Survival after docetaxel for metastatic castration-resistant prostate cancer in a rural health care setting. Contemp Oncol (Pozn) 28(1): 31-36, 2024. DOI: 10.5114/wo.2024.138842
19 Pinart M, Kunath F, Lieb V, Tsaur I, Wullich B, Schmidt S, German Prostate Cancer Consortium (DPKK): Prognostic models for predicting overall survival in metastatic castration-resistant prostate cancer: a systematic review. World J Urol 38(3): 613-635, 2020. DOI: 10.1007/s00345-018-2574-2
20 Cotogno PM, Ranasinghe LK, Ledet EM, Lewis BE, Sartor O: Laboratory-based biomarkers and liver metastases in metastatic castration-resistant prostate cancer. Oncologist 23(7): 791-797, 2018. DOI: 10.1634/theoncologist.2017-0564
21 Nieder C, Dalhaug A, Pawinski A: Contribution of serum biomarkers to prognostic assessment in patients with oligometastatic prostate cancer. In Vivo 33(2): 465-468, 2019. DOI: 10.21873/invivo.11495
22 Cushen SJ, Power DG, Murphy KP, McDermott R, Griffin BT, Lim M, Daly L, MacEneaney P, O' Sullivan K, Prado CM, Ryan AM: Impact of body composition parameters on clinical outcomes in patients with metastatic castrate-resistant prostate cancer treated with docetaxel. Clin Nutr ESPEN 13: e39-e45, 2016. DOI: 10.1016/j.clnesp.2016.04.001
23 Vidal AC, Howard LE, de Hoedt A, Kane CJ, Terris MK, Aronson WJ, Cooperberg MR, Amling CL, Freedland SJ: Obese patients with castration-resistant prostate cancer may be at a lower risk of all-cause mortality: results from the Shared Equal Access Regional Cancer Hospital (SEARCH) database. BJU Int 122(1): 76-82, 2018. DOI: 10.1111/bju.14193
24 Chang LW, Hung SC, Li JR, Chiu KY, Yang CK, Chen CS, Lu K, Chen CC, Wang SC, Lin CY, Cheng CL, Ou YC, Yang SF, Hsu CY, Ho SH, Wang SS: Geriatric Nutritional Risk Index as a prognostic marker for patients with metastatic castration-resistant prostate cancer receiving docetaxel. Front Pharmacol 11: 601513, 2021. DOI: 10.3389/fphar.2020.601513
25 Martini A, Shah QN, Waingankar N, Sfakianos JP, Tsao C, Necchi A, Montorsi F, Gallagher EJ, Galsky MD: The obesity paradox in metastatic castration-resistant prostate cancer. Prostate Cancer Prostatic Dis 25(3): 472-478, 2022. DOI: 10.1038/s41391-021-00418-0
26 Modonutti D, Majdalany SE, Corsi N, Li P, Sood A, Dalela D, Jamil ML, Hwang C, Menon M, Rogers CG, Trinh Q, Novara G, Abdollah F: A novel prognostic model predicting overall survival in patients with metastatic castration-resistant prostate cancer receiving standard chemotherapy: A multi-trial cohort analysis. Prostate 82(13): 1293-1303, 2022. DOI: 10.1002/pros.24403
27 Verma S, Arora S, Sahoo RK, Singh P, Nayak B, Haresh KP, Das CJ, Shamim SA, Kaushal S, Batra A: Differential effect of body mass index (BMI) on outcomes of patients treated with docetaxel in prostate cancer - An exploratory analysis. Cancer Treat Res Commun 31: 100520, 2022. DOI: 10.1016/j.ctarc.2022.100520
28 Govindan S, Cheranda N, Riekhof F, Luo S, Schoen MW: Effect of BMI and hemoglobin A1c on survival of veterans with metastatic castration-resistant prostate cancer treated with abiraterone or enzalutamide. Prostate 84(3): 245-253, 2024. DOI: 10.1002/pros.24644
29 Nieder C, Dalhaug A, Haukland E: Feasibility and efficacy of sequential systemic therapy for metastatic castration-resistant prostate cancer in a rural health care setting. Scand J Urol 54(2): 110-114, 2020. DOI: 10.1080/21681805.2020.1730435
30 Hiroshige T, Ogasawara N, Kumagae H, Ueda K, Chikui K, Uemura KI, Nishihara K, Nakiri M, Matsuo M, Suekane S, Igawa T: Sarcopenia and the therapeutic effects of androgen receptor-axis-targeted therapies in patients with castration-resistant prostate cancer. In Vivo 37(3): 1266-1274, 2023. DOI: 10.21873/invivo.13204
31 Yamamoto Y, Nishimoto M, Akashi Y, Kiba K, Minami T, Nozawa M, Yoshimura K, Hirayama A, Uemura H, Fujita K: Impact of novel agents on patient characteristics, treatment patterns, and outcomes in patients with metastatic castration-resistant prostate cancer. Anticancer Res 44(7): 3155-3161, 2024. DOI: 10.21873/anticanres.17130
32 Fujiwara M, Fujiwara R, Oguchi T, Komai Y, Numao N, Yamamoto S, Yonese J, Yuasa T: Clinical outcome and prognostic variables of second-line therapy for patients with castration-resistant prostate cancer after failure of first-line androgen receptor axis-targeted therapy. Anticancer Res 42(4): 2123-2130, 2022. DOI: 10.21873/anticanres.15694
33 Hung SC, Chang LW, Li JR, Wang SS, Yang CK, Chen CS, Lu K, Chen CC, Wang SC, Lin CY, Cheng CL, Ou YC, Chiu KY: Docetaxel rechallenge improves survival in patients with metastatic castration-resistant prostate cancer: a retrospective study. In Vivo 35(6): 3509-3519, 2021. DOI: 10.21873/invivo.12653