

Population-based assessment of treatment patterns for high-risk localized prostate cancer

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KEY MESSAGES

- This retrospective, population-based cohort study examined real-world treatment patterns and outcomes among 31 571 Canadian men with IR and H/vHR LPCa.
- Patients with H/vHR demonstrated lower OS, highlighting the need for more effective treatment strategies in H/vHR LPCa.
- Despite differing clinical outcomes, treatment patterns were similar across both patient groups, with EBRT, RP, and brachytherapy being the most common
- ADT use was limited, pointing to an underuse of long-term ADT among patients receiving RT post-RP, suggesting a potential gap between clinical practice and trial-based recommendations

ABSTRACT

Introduction: High- and very high-risk (H/vHR) localized and locally advanced prostate cancer (LPCa) carry a high burden of distant relapse. In light of emerging data on adjunctive systemic therapy, we aimed to assess present-day, real-world treatment patterns and outcomes among Canadian patients with intermediate-risk (IR) and H/vHR LPCa.

Methods: This retrospective, population-based cohort study identified men aged ≥ 18 years diagnosed with LPCa between 2010 and 2022 in Ontario, Canada. Patients were stratified into IR and H/vHR groups. Baseline patient characteristics and treatment patterns following LPCa diagnosis were described. Clinical outcomes, including median time to mortality, were assessed using Kaplan-Meier analyses. To better characterize androgen deprivation therapy (ADT) exposure, subgroup analyses were performed in patients aged ≥ 66 years.

Results: Among 31 571 eligible patients, 18 365 had IR LPCa and 13 206 had H/vHR LPCa. Within one year from diagnosis, patients with H/vHR were more likely to receive definitive therapy (95.4% vs. 81.5%), with shorter durations between diagnosis and treatment (median 72 days vs. 92 days) compared to patients with IR. Patients with H/vHR demonstrated lower median time to mortality (median 4.9 vs. 5.9 years, $p < 0.0001$). Treatment modalities were comparable between cohorts, with external beam radiation therapy (EBRT), radical prostatectomy (RP), and brachytherapy being the most common. Among patients aged ≥ 66 years, use of ADT post-RP was limited.

Conclusions: Current real-world treatment approaches for H/vHR LPCa emphasize local therapy with surgery and radiotherapy. There are considerable gaps, which may allow improved outcomes through optimized use of systemic therapy.

INTRODUCTION

While localized prostate cancer (PCa) is often considered an indolent condition, high- and very high-risk (H/vHR) localized and locally advanced PCa (LPCa) accounts for ~15% of newly diagnosed cases.^{1,2} Importantly, this subset of LPCa has increased risk of biochemical recurrence (BCR), metastatic progression, and PCa-specific mortality.³

While treatment strategies for LPCa are tailored according to risk, the general treatment paradigm for patients with IR and HR PCa is generally similar: radiotherapy $\pm$ androgen deprivation or surgical extirpation. For patients opting for radiotherapy approaches, those with HR disease are often recommended longer durations of ADT and potentially dose-escalation with brachytherapy boost.^{1,4,5}

In spite of these treatment approaches, biochemical recurrence (BCR) occurs in 20-50% of patients with LPCa within 10 years of initial definitive therapy,⁶ representing a significant and persistent challenge in LPCa care. This substantial recurrence, particularly prominent in H/vHR

LPCa, underscores a significant unmet need in LPCa care and reinforces the urgency of advancing next-generation treatments. In response, ongoing clinical trials are assessing the incorporation of systemic treatments into current paradigms including the use of intensified systemic therapy to potentially broaden the treatment landscape for H/vHR LPCa patients.⁷⁻⁹. To address ongoing clinical uncertainties around treatment selection and sequencing, particularly considering emerging evidence supporting intensified systemic therapy, we aimed to better understand current real-world clinical characteristics, treatment patterns and outcomes in Canadian patients with H/vHR LPCa using real-world health administrative data from Ontario, Canada.

METHODS

Data source

This retrospective population-based cohort study used province-wide linked administrative data from Ontario, Canada including the Ontario Public Drug Program, New Drug Funding Program, and Cancer Activity Level Reporting databases (medication use, radiation therapy, and systemic treatments); Canadian Institute for Health Information Discharge Abstract and Same Day Surgery databases and National Ambulatory Care Reporting System (hospital visits); Ontario Health Insurance Plan Claims and ICES Physician databases (physician services); Ontario Cancer Registry (identification of patients with PCa); Registered Persons Database (demographic information); and Ontario Laboratory Information System (laboratory results).

Study population

Men diagnosed with LPCa (ICD-O-3 code C61.9N) between January 1, 2010, and December 31, 2022 and aged ≥ 18 years at diagnosis were included. Patients were excluded if they had invalid or missing Ontario Health Insurance Plan number; were female; were not Ontario residents at index or ineligible for OHIP coverage at index or 2 years prior to diagnosis; had died prior to diagnosis date; had missing disease stage information; had stage IVb; had stage IV-unspecified; had missing Gleason score; had missing PSA within 6 months prior to index or 4 weeks after index; or were aged < 18 years old. Patients were followed from their diagnosis date (index date) until June 30, 2023.

Exposure definition

Patients were stratified into IR and H/vHR groups. The IR group consisted of patients with AJCC Stage II; Stage IIa; Stage IIb with a Gleason score ≤ 7 and PSA < 20 ng/mL; or Stage IIc with a Gleason score ≤ 7 . The H/vHR group consisted of patients with AJCC Stage IIb with Gleason score ≤ 7 and PSA ≥ 20 ng/mL, or Gleason score ≥ 8 regardless of PSA; Stage IIc with a Gleason score 8; Stage III; or Stage IVa.

Outcome definitions

The primary outcome was treatment patterns, defined by claims for PCa-related treatments within 1 year of diagnosis including pharmacological treatments, radiotherapy, and surgical procedures (treatment codes and definitions listed in Supplementary Tables S1 and S2). Medication records for patients ≥ 65 are represented among Ontario Drug Benefit (ODB) records. As such, subgroup analyses were conducted for a cohort of patients ≥ 66 years old at index, which revealed further granularity among treatment patterns in this population, particularly surrounding the use of ADT. Patients aged ≥ 66 years were included to ensure the availability of at least one complete year of prescription data following their eligibility for public drug coverage.

Time-to-metastasis was assessed from index to the first date of metastatic disease progression identified based on bone scans, metastatic disease diagnoses, and American Joint Committee on Cancer (AJCC) staging from physician encounter notes. Time-to-castration resistance was assessed from index to first date of castration resistance identified from laboratory tests, diagnosis code for castrate resistant prostate malignancy, or an indication in physician encounter notes. Time-to-metastatic castration-resistant PCa (mCRPC) was assessed from index to the latest of the metastasis or castration resistance dates. All-cause mortality was evaluated.

Statistical analysis

Data were analyzed descriptively for the overall cohort, then stratified by risk group (IR, H/vHR). Categorical variables were presented as counts and percentages; continuous variables were summarized with medians and interquartile ranges (IQR). All variables were stratified by definitive treatment subgroup (i.e., EBRT or radical prostatectomy). Time-to-clinical outcomes were assessed using Kaplan-Meier (KM) analyses. Small cell values were reported as a range according to ICES reporting standards to reduce the risk of re-identification.

The overall cohort and those aged ≥ 66 years were stratified by LPCa risk and categorized into six mutually exclusive post-diagnosis treatment groups: RP alone, brachytherapy alone, EBRT alone, EBRT + brachytherapy, EBRT + brachytherapy given as salvage, or no RP/RT. Treatment patterns and time from diagnosis to treatment were reported stratified by regional Local Health Integration Network (LHIN) and compared using chi-square tests.

Ethics

Advarra Institutional Review Board granted a Waiver of Consent for this study. The analysis was performed at ICES. ICES is a named prescribed entity, under section 45(1) of the Personal Health Information Protection Act, 2004 and has statutory authority to conduct health services research without consent using anonymized administrative data.

RESULTS

Demographic and baseline characteristics

From 2010-2022, we identified 31,571 patients with non-metastatic PCa who had sufficient baseline data for risk stratification and met inclusion criteria. Among these patients, 18,365 were diagnosed with IR LPCa and 13,206 were diagnosed with H/vHR LPCa. Patient characteristics (Table 1) varied between the IR and H/vHR cohorts. The median (IQR) age of included patients was 67 (61-73) in the IR and 68 (63-75) in H/vHR cohort. The overall median (IQR) PSA value was 8 (6-13), with a median value of 7 (5-10) in the IR cohort and 11 (7-23) in the H/vHR cohort. Among the total cohort, 64.3% had a Gleason score of 7, with 81.7% (n=15,013) in the IR cohort and 40.0% (n=5,287) in the H/vHR cohort.

Treatment patterns

Treatment patterns among patients with LPCa who received treatment within 1 year of diagnosis are described in Table 2. A significantly higher proportion of patients in the IR cohort (18.5%) received no active therapy within 1 year of diagnosis compared to the H/vHR cohort (4.6%; $p<0.0001$; standardized difference=0.44). Among those treated, the median [IQR] time between diagnosis and treatment was longer for the IR cohort (92 [62-138] days) compared to the H/vHR cohort (72 [42-115] days). Across both cohorts, the most common treatment modality was EBRT (IR: 40.1%; H/vHR: 47.2%), followed by RP (IR: 39.7%; H/vHR: 47.0%), brachytherapy (IR: 11.3%; H/vHR: 4.9%), and a combination of EBRT and brachytherapy (IR: 11.1–11.2%; H/vHR: 4.8-4.9%). Other treatments were less frequent (IR: ~2.9%; H/vHR: ~5.5%). A minimal number of patients were treated with ARPIs within one year of diagnosis, which were not approved for LPCa patients in Canada during the study period; fewer patients in the IR cohort were treated with ARPIs (0.005-0.03%) vs. the H/vHR cohort (~0.4%). Considering mutually exclusive treatment groups at any time following diagnosis for the overall cohort (Supplementary Table S3), a lower proportion of patients in the IR cohort received surgery alone (n=7,725, 42.1%) compared to the H/vHR cohort (n=6,282, 47.6%). Similarly, a lower proportion of patients in the IR cohort received EBRT alone (n=5,853, 31.9%) compared to the H/vHR cohort (n=4,511, 34.2%). The number of patients who did not undergo either RP or RT was comparable between cohorts, however the proportion was marginally greater in the IR cohort (n=2,474, 13.5%) compared to the H/vHR cohort (n=1,733, 13.1%).

For patients aged ≥ 66 years, granular details regarding the receipt of ADT are available, forming an important and informative subset analysis. Among patients aged ≥ 66 years (Table 3), 78.1% of patients in the IR cohort and 95.2% of patients in the H/vHR cohort underwent active treatment at any time within the one year following diagnosis ($p<0.0001$; standardized difference=0.519). Patients aged ≥ 66 years within the IR cohort experienced longer time to treatment, with a median [IQR] of 82 [54-124] days from diagnosis to treatment compared to 57 [32-91] days in the H/vHR cohort. Patients aged ≥ 66 years also had different treatment patterns compared to the total cohort, notably reduced treatment with RP. Within the IR cohort aged ≥ 66

years, EBRT represented the most frequent treatment modality ($n=5,394$, 52.6%), followed by RP ($n=2,145$, 20.9%) and LHRH agonist/antagonist monotherapy ($n=1,934$, 18.9%). In the H/vHR cohort aged ≥ 66 years, the most frequent treatment modality was LHRH agonist/antagonist monotherapy ($n=5,502$, 66.9%), followed by a combination of LHRH and a first-generation antiandrogen ($n=4,878$, 59.3%) and EBRT ($n=4,372$, 53.1%).

Considering mutually exclusive treatment groups for the sub cohort aged ≥ 66 (Supplementary Table S3), most patients received EBRT only, with a greater proportion observed in the IR cohort ($n=4,847$, 47.3%) vs. the H/vHR cohort ($n=3,756$ 45.6%). In contrast, fewer patients in the IR cohort received surgery alone ($n=2,264$, 22.1%) than in the H/vHR cohort ($n=2,447$, 29.7%). Furthermore, 18.2% of the IR cohort ($n=1,871$) and 19.2% of the H/vHR cohort ($n=1,576$) received no RP or RT treatment.

Understanding that RP alone may not be curative in many patients, we sought to understand the use of subsequent therapies post-RP, particularly as it relates to the use of ADT among patients aged ≥ 66 years (Table 4). ADT after RP was observed in 6.2% of patients ($n=275$) up to 6 months post-RP, and 8.7% ($n=389$) up to 12 months post-RP. For those receiving ADT with concurrent RT ($n=125$, 2.8% up to 6 months after RP; $n=240$, 5.4% up to 12 months after RP), the median ADT duration was 4 and 3 months for patients up to 6- and 12-months post-RP, respectively. Few patients received ADT with non-concurrent RT ($n=17$, 0.4% up to 6 months after RP; $n=43$, 1.0% up to 12 months after RP); among them, the majority received ADT first, followed by RT. For those receiving ADT without RT ($n=133$, 3.0% up to 6 months after RP; $n=106$, 2.4% up to 12 months after RP), the median duration of ADT prescription was 4 and 6 months for patients up to 6- and 12-months post-RP, respectively.

Regional differences

Regional differences in treatment patterns were observed (Table 6; $p<0.0001$ across all groups). RT was the most common treatment across most regions for IR and H/vHR groups, ranging from 58.9% (LHIN E) to 61.4% (LHIN N) for the IR group and 55.0% (LHIN E) to 70.2% (LHIN D) for the H/vHR group. Treatment patterns also differed by region when considering mutually exclusive treatment groups (Supplementary Table S4).

Median time from diagnosis to initiation of surgery or RT differed by region (Table 7; $p<0.0001$ across all groups). Wait times ranged from 85-151 and 79-119 days for surgery and 110-154 and 165-247 days for RT in the IR and H/vHR group, respectively. The longest wait times for surgery across both risk groups were observed in LHIN I, while the longest wait times for RT were observed in LHIN K for the IR cohort and LHIN N for the H/vHR cohort. Wait times also differed when considering mutually exclusive treatment groups (Supplementary Table S5).

Real-world clinical outcomes

We then examined a number of clinically important endpoints for patients with LPCa. For each, we estimated the proportion of patients experiencing these events, and the median time to the

event for those who experienced an event. IR patients demonstrated more favorable clinical outcomes compared to H/vHR (Table 5). Metastatic progression occurred less commonly and, when occurring, significantly later ($p < 0.0001$) in the IR cohort, with a median time to metastasis of 4.3 years vs. 2.6 years in H/vHR. Importantly, progression to CRPC and the initiation of first CRPC therapy were also significantly less common and prolonged in the IR group vs. H/vHR group. Mortality events were less common among patients with IR vs H/vHR disease (18 vs. 29%) and, when occurring, were later following diagnosis (median 5.9 years vs. 4.9 years, $p < 0.0001$).

DISCUSSION

In this real-world study of 31,571 Canadian patients diagnosed with LPCa, treatment patterns were similar across patients with IR and H/vHR, despite differing clinical outcomes. Few patients received intensified systemic therapy within 1 year of diagnosis. These findings offer valuable insights into real-world treatment patterns and outcomes among Canadian men with LPCa and underscore a significant unmet clinical need.

Patients with H/vHR LPCa experienced significantly poorer clinical outcomes than patients with IR, including rates of metastasis, castration-resistance, the initiation of first CRPC systemic therapy, and mortality. With respect to mortality, significantly more patients with H/vHR disease died during follow-up (29 vs. 18%), though due to limitations of the available data, the cause of death was not able to be ascertained. Among those who died, patients with H/vHR disease died substantially earlier, at a median of 4.9 vs 5.9 years following diagnosis. This 12-month difference in time to mortality is clinically significant, and highlights the need for more effective treatment strategies, particularly in H/vHR LPCa. Importantly, despite these disparate clinical outcomes, treatment patterns were quite comparable between these risk groups. While a previous study also found a difference in OS between risk groups, the median OS reported for both the IR and HR groups was > 8 years.¹⁰ It bears noting that this prior analysis provides a Kaplan-Meier estimate of survival (with censoring of individuals not experiencing the event) where the present analysis presents time to death (with exclusion of those not dying during observation). This analytic distinction almost certainly explains the observed differences between the studies. However, it bears further noting that in the present study, 12.7% of patients were untreated and $< 10\%$ received ADT post-RP. The study reporting longer OS included only treated patients and ADT use was substantially higher (34% for IR group and 78% for HR).¹⁰ Real-world treatment patterns among patients with IR and H/vHR LPCa revealed broadly comparable management strategies across risk groups. In both cohorts, the majority of patients initiated treatment within one year of diagnosis. Standard of care local therapies such as RP and EBRT were the most frequently administered modalities. Notably, patients in the H/vHR cohort had significantly higher treatment rates and generally lower durations between diagnosis and treatment relative to the IR cohort, reflecting the appropriate clinical prioritization of patients with more aggressive disease. These findings are consistent with previously reported real-world treatment patterns among patients with LPCa in the United States, which showed that primary

treatment involves RP or RT,^{1, 11} with HR patients in particular demonstrating higher treatment rates, including higher rates of concurrent ADT.¹⁰ Despite clinicians prioritizing aggressive disease, the similarity in treatment approaches between risk groups and poorer outcomes experienced by patients with H/vHR LPCa in the present study suggests that current real-world practices may not be optimized for the treatment of H/vHR disease. These findings highlight the importance of developing treatment strategies that address the distinct therapeutic needs of patients with H/vHR LPCa.

The results further demonstrated that among patients aged ≥ 66 years who received ADT with concurrent RT post-RP as initial treatment, the median duration of ADT was 4 months within the first 6 months post-RP and 3 months within the first 12 months post-RP, indicating limited duration of ADT administration. In contrast, evidence from the RADICALS-HD trial supports long-term ADT (24 months) over short-term ADT (6 months) in improving metastasis-free survival.¹² Our real-world findings therefore suggest a potential gap between clinical practice and trial-based recommendations, pointing to the underutilization of long-term ADT among patients receiving RT post-RP.

The treatment landscape for PCa has evolved substantially over the past decade, resulting in longer survival among patients.¹³ Although intensified systemic therapy represents an evolving treatment paradigm, only a small proportion of patients in this study received such therapy within 1 year of PCa diagnosis. The use of ARPIs remained minimal throughout the course of disease progression, largely due to their lack of approval for LPCa during the study period. However, emerging clinical trial evidence has demonstrated promising results regarding the feasibility, toxicity, and potential benefit of ARPIs in HR LPCa, indicating that this subgroup benefit meaningfully from intensified systemic therapy.¹⁴ Notably, findings from the STAMPEDE trial, investigating the addition of the ARPI abiraterone acetate $\pm$ enzalutamide to ADT in LPCa, revealed that combination therapy with abiraterone acetate significantly improved metastasis-free survival compared to ADT alone in HR LPCa,¹⁵ supporting the utility of ARPIs such as abiraterone acetate in improving long-term outcomes in this patient population. Several ongoing phase III trials, including PROTEUS and ATLAS, aim to further clarify the role of ARPIs in improving clinical outcomes in this patient population.^{8, 12, 14, 16} Given the potential benefit of treatment intensification strategies in LPCa, further investigation on the use of ARPIs with ADT may improve clinical outcomes for patients with HR disease.

Variations in patients receiving RT, surgery, and no treatment, and time to treatment across LHINs suggest potential differences in access to care and clinical practice across Ontario. Differences in access to care were also reflected in the wait times for treatments, with patients in some LHINs experiencing significantly longer delays to treatment initiations. These regional differences highlight potential inequities in access to care that could contribute to variability in patient outcomes across Ontario.

A retrospective, population-based cohort design allowed us to leverage data from a large sample of Canadian LPCa patients. However, several limitations should be considered. The

study is limited to Ontario patients and may not be generalizable to the Canadian population. Administrative data lacks detailed patient-specific clinical information such as radiological test results and disease severity. The ODB database does not contain medication records for most individuals <65 years, precluding the ability to assess treatment patterns among younger patients. ODB lacks information on drugs administered in the inpatient setting and drugs not paid for using public funding (e.g., out-of-pocket, private insurance, manufacturer drug access programs) and therefore, may not include complete medication records for all patients. Furthermore, there was insufficient granularity available in the datasets available through ICES to allow for meaningful comparative effectiveness analyses. Lastly, cause-specific mortality was not captured due to privacy-related data limitations, and as such, we cannot comment on OS and mortality due to PCa or other causes.

CONCLUSIONS

Current real-world treatment approaches for H/vHR LPCa emphasize local therapy with surgery and radiotherapy, suggesting considerable gaps in treatment. Alternative therapeutic strategies aimed at improving clinical outcomes in patients with H/vHR LPCa are needed, such as optimized use of systemic therapy.

REFERENCES

1. Freedland SJ, Nair S, Lin X, et al. A US real-world study of treatment patterns and outcomes in localized or locally advanced prostate cancer patients. *World J Urol* 2023;41:3535-42. <https://doi.org/10.1007/s00345-023-04680-w>
2. Cooperberg MR, Broering JM, Carroll PR. Time trends and local variation in primary treatment of localized prostate cancer. *J Clin Oncol* 2010;28:1117-23. <https://doi.org/10.1200/JCO.2009.26.0133>
3. Serrano NA, Anscher MS. Favorable vs unfavorable intermediate-risk prostate cancer: A review of the new classification system and its impact on treatment recommendations. *Oncology (United States)* 2016;30.
4. Parker C, Castro E, Fizazi K, et al. Prostate cancer: ESMO clinical practice guidelines for diagnosis, treatment and follow-up. *Ann Oncol* 2020;31:1119-34. <https://doi.org/10.1016/j.annonc.2020.06.011>
5. Schaeffer EM, Srinivas S, Adra N, et al. Prostate cancer, version 4.2023, NCCN clinical practice guidelines in oncology. *J Natl Compr Canc Netw* 2023;21:1067-96. <https://doi.org/10.6004/jncn.2023.0050>
6. Shore ND, Moul JW, Pienta KJ, et al. Biochemical recurrence in patients with prostate cancer after primary definitive therapy: Treatment based on risk stratification. *Prostate Cancer Prostatic Dis* 2024;27:192-201. <https://doi.org/10.1038/s41391-023-00712-z>
7. Sandler HM, McKenzie MR, Tombal BF, et al. ATLAS: A randomized, double-blind, placebo-controlled, phase 3 trial of apalutamide (ARN-509) in patients with high-risk localized or locally advanced prostate cancer receiving primary radiation therapy. *J Clin Oncol* 2016;34:TPS5087. https://doi.org/10.1200/JCO.2016.34.15_suppl.TPS5087
8. Kibel AS, Gleave M, Brookman-May SD, et al. PROTEUS: A randomized, double-blind, placebo (PBO)-controlled, phase 3 trial of apalutamide (APA) plus androgen deprivation therapy (ADT) versus PBO plus ADT prior to radical prostatectomy (RP) in patients (pts) with localized or locally advanced high-risk prostate cancer (PC). *J Clin Oncol* 2022;40:TPS285. https://doi.org/10.1200/JCO.2022.40.6_suppl.TPS285
9. Yamaguchi N, Yumioka T, Iwamoto H, et al. Biochemical recurrence prediction in high-risk prostate cancer patients, following robot-assisted radical prostatectomy. *Yonago Acta Med* 2016;59:288.
10. Karsh L, Du S, He J, et al. Differences in real-world outcomes by risk classification for localized prostate cancer patients after radiation therapy. *Prostate* 2024;84:1047-55. <https://doi.org/10.1002/pros.24720>
11. Lowrance WT, Elkin EB, Yee DS, et al. Locally advanced prostate cancer: A population-based study of treatment patterns. *BJU Int* 2012;109:1309-14. <https://doi.org/10.1111/j.1464-410X.2011.10760.x>
12. Parker CC, Kynaston H, Cook AD, et al. Duration of androgen deprivation therapy with postoperative radiotherapy for prostate cancer: A comparison of long-course versus short-course androgen deprivation therapy in the RADICALS-HD randomised trial. *Lancet* 2024;403:2416-25. [https://doi.org/10.1016/S0140-6736\(24\)00549-X](https://doi.org/10.1016/S0140-6736(24)00549-X)
13. Azad AA, Kostos L, Agarwal N, et al. Combination therapies in locally advanced and metastatic hormone-sensitive prostate cancer. *Eur Urol* 2025. <https://doi.org/10.1016/j.eururo.2025.01.010>

14. Devos G, Devlies W, De Meerleer G, et al. Neoadjuvant hormonal therapy before radical prostatectomy in high-risk prostate cancer. *Nat Rev Urol* 2021;18:739-62. <https://doi.org/10.1038/s41585-021-00514-9>
15. Attard G, Murphy L, Clarke NW, et al. Abiraterone acetate and prednisolone with or without enzalutamide for high-risk non-metastatic prostate cancer: A meta-analysis of primary results from two randomised controlled phase 3 trials of the STAMPEDE platform protocol. *Lancet* 2022;399:447-60. [https://doi.org/10.1016/S0140-6736\(21\)02437-5](https://doi.org/10.1016/S0140-6736(21)02437-5)
16. Williams S, Davis ID, Sweeney C, et al. Randomised phase 3 trial of enzalutamide in androgen deprivation therapy (ADT) with radiation therapy for high risk, clinically localized prostate cancer: ENZARAD (ANZUP 1303). *J Clin Oncol* 2018. https://doi.org/10.1200/JCO.2018.36.6_suppl.TPS156

DATA STATEMENT: The data presented in this study are available on request from the corresponding author as the data are not publicly available due to privacy and legal restrictions. This study made use of de-identified data from the ICES Data Repository, which is managed by the Institute for Clinical Evaluative Sciences with support from its funders and partners: Canada's Strategy for Patient-Oriented Research (SPOR), the Ontario SPOR Support Unit, the Canadian Institutes of Health Research and the Government of Ontario. The opinions, results and conclusions reported are those of the authors. No endorsement by ICES or any of its funders or partners is intended or should be inferred

FIGURES AND TABLE

Table 1. Baseline patient characteristics by LPCa risk						
Variable	Label	Total	Intermediate	High- or very high	p	Standardized difference
		N=31 571	n=18 365	n=13 206		
Age at PCa diagnosis	Mean (SD)	67.5 (8.8)	66.6 (8.6)	68.6 (9.0)	<0.0001	0.222
	Median (Q–Q3)	67 (61–73)	67 (61–73)	68 (63–75)	<0.0001	0.194
Income quintile	1– Lowest, n (%)	4828 (15.3%)	2728 (14.9%)	2100 (15.9%)	<0.0001	0.029
	2, n (%)	5913 (18.7%)	3353 (18.3%)	2560 (19.4%)		0.029
	3, n (%)	6305 (20.0%)	3634 (19.8%)	2671 (20.2%)		0.011
	4, n (%)	6714 (21.3%)	3918 (21.3%)	2796 (21.2%)		0.004
	5 – Highest, n (%)	7811 (24.7%)	4732 (25.8%)	3079 (23.3%)		0.057
Rural residence	Urban, n (%)	27 302 (86.5%)	15 973 (87.0%)	11 329 (85.8%)	0.0023	0.035
	Rural, n (%)	4269 (13.5%)	2392 (13.0%)	1877 (14.2%)		0.035
LHIN	LHIN A	3992 (12.6%)	2383 (13.0%)	1609 (12.2%)	<0.0001	0.024
	LHIN B	3830 (12.1%)	2205 (12.0%)	1625 (12.3%)		0.009
	LHIN C	3751 (11.9%)	2234 (12.2%)	1517 (11.5%)		0.021
	LHIN D	3239 (10.3%)	1761 (9.6%)	1478 (11.2%)		0.053
	LHIN E	2761 (8.7%)	1629 (8.9%)	1132 (8.6%)		0.011
	LHIN F	2493 (7.9%)	1462 (8.0%)	1031 (7.8%)		0.006
	LHIN G	2313 (7.3%)	1395 (7.6%)	918 (7.0%)		0.025

	LHIN H	1746 (5.5%)	1037 (5.6%)	709 (5.4%)		0.012
	LHIN I	1637 (5.2%)	932 (5.1%)	705 (5.3%)		0.012
	LHIN J	1588 (5.0%)	861 (4.7%)	727 (5.5%)		0.037
	LHIN K	1,379 (4.4%)	811 (4.4%)	568 (4.3%)		0.006
	LHIN L	1,216 (3.9%)	648 (3.5%)	568 (4.3%)		0.040
	LHIN M	1168 (3.7%)	717 (3.9%)	451 (3.4%)		0.026
	LHIN N	458 (1.5%)	290 (1.6%)	168 (1.3%)		0.026
PSA value	Mean (SD)	16.6 (74.1)	8.8 (29.9)	27.4 (108.1)	<0.0001	0.234
	Median (Q1-Q3)	8 (6–13)	7 (5–10)	11 (7–23)	<0.0001	0.647
PCa stage	Stage II, n (%)	24 936 (79.0%)	18 365 (100.0%)	6571 (49.8%)	<0.0001	1.421
	Stage III, n (%)	6635 (21.0%)	0 (0.0%)	6635 (50.2%)		1.421
Gleason score	Score ≤6, n (%)	4041 (12.8%)	3249 (17.7%)	792 (6.0%)	<0.0001	0.368
	Score=7, n (%)	20 300 (64.3%)	15 013 (81.7%)	5287 (40.0%)		0.945
	Score ≥8, n (%)	7230 (22.9%)	103 (0.6%)	7127 (54.0%)		1.499
Charlson comorbidity index (CCI)	CCI=0, n (%)	8247 (26.1%)	4815 (26.2%)	3432 (26.0%)	<0.0001	0.005
	CCI=1–2, n (%)	5494 (17.4%)	3074 (16.7%)	2420 (18.3%)		0.042
	CCI ≥3, n (%)	1263 (4.0%)	630 (3.4%)	633 (4.8%)		0.069
	CCI: Missing, n (%)	16 567 (52.5%)	9846 (53.6%)	6721 (50.9%)		0.054
	Mean (SD)	6.8 (6.1)	6.5 (5.9)	7.1 (6.4)	<0.0001	0.085

Number of GP visits (in the year prior to PCa diagnosis)	Median (Q1-Q3)	5 (3–8)	5 (3–8)	5 (3–9)	<0.0001	0.076
Hospitalization (in the year prior to PCa diagnosis)	n (%)	3120 (9.9%)	1614 (8.8%)	1506 (11.4%)	<0.0001	0.087
Ever resident of long-term care (in the year prior to PCa diagnosis)	n (%)	94 (0.3%)	33 (0.2%)	61 (0.5%)	<0.0001	0.050
Diabetes (any time before PCa diagnosis)	n (%)	7,169 (22.7%)	4,020 (21.9%)	3149 (23.8%)	<0.0001	0.047
Myocardial infarction (MI) (within 5 years prior to PCa diagnosis)	n (%)	491 (1.6%)	269 (1.5%)	222 (1.7%)	0.1255	0.017
Cerebrovascular accident (within 5 years prior to PCa diagnosis)	n (%)	171 (0.5%)	100 (0.5%)	71 (0.5%)	0.9345	0.001
Congestive heart failure (CHF) (any time before PCa diagnosis)	n (%)	1482 (4.7%)	723 (3.9%)	759 (5.7%)	<0.0001	0.084
COPD (any time before PCa diagnosis)	n (%)	5153 (16.3%)	2874 (15.6%)	2279 (17.3%)	0.0001	0.043
Hypertension (any time before PCa diagnosis)	n (%)	18 915 (59.9%)	10 898 (59.3%)	8017 (60.7%)	0.0146	0.028
ED/hospitalization for arrhythmia (within 5 years prior to PCa diagnosis)	n (%)	731 (2.3%)	399 (2.2%)	332 (2.5%)	0.0466	0.023

Dementia (within 5 years prior to PCa diagnosis)	n (%)	911 (2.9%)	459 (2.5%)	452 (3.4%)	<0.0001	0.054
Liver disease (within 5 years prior to PCa diagnosis)	n (%)	237 (0.8%)	139 (0.8%)	98 (0.7%)	0.8806	0.002
Renal disease (within 5 years prior to PCa diagnosis)	n (%)	1233 (3.9%)	673 (3.7%)	560 (4.2%)	0.0092	0.030

ED: emergency department; LHIN: local health integration network; PCa: prostate cancer.

Table 2. Treatment patterns within 1 year of diagnosis by LPCa risk						
Treatment	Label	Total	Intermediate-risk	High- or very high-risk	p	Standardized difference
		N=31 571	n=18 365	n=13 206		
RP	n (%)	13 493 (42.7%)	7286 (39.7%)	6207 (47.0%)	<0.0001	0.148
	Median time from diagnosis to treatment (Q1–Q3), days	103 (72–146)	106 (74–153)	99 (70–139)	<0.0001	0.127
EBRT	n (%)	13 607 (43.1%)	7371 (40.1%)	6236 (47.2%)	<0.0001	0.143
	Median time from diagnosis to treatment (Q1–Q3), days	125 (79–188)	103 (70–154)	154 (102–217)	<0.0001	0.604
Brachytherapy	n (%)	2722 (8.6%)	2076 (11.3%)	646 (4.9%)	<0.0001	0.237
	Median time from diagnosis to treatment (Q1–Q3), days	126 (96–170)	122 (91–162)	143 (111–194)	<0.0001	0.365
EBRT + brachytherapy	n (%)	2691 (8.5%)	2048–2052*	639–643*	<0.0001	0.234
	Median time from diagnosis to treatment (Q1–Q3), days	124 (91–167)	118 (89–157)	140 (105–187)	<0.0001	0.363
Bilateral orchiectomy	n (%)	48 (0.2%)	19 (0.1%)	29 (0.2%)	0.0090	0.029
	Median time from diagnosis to treatment (Q1–Q3), days	65 (26–151)	39 (25–146)	77 (43–174)	0.1085	0.489
Chemotherapy	n (%)	77 (0.2%)	7 (0.0%)	70 (0.5%)	<0.0001	0.093
	Median time from diagnosis to treatment (Q1–Q3), days	121 (82–215)	125 (90–181)	120 (79–220)	0.9083	0.047
ARPIs	n (%)	49 (0.2%)	1–5*	44–48*	NA	NA
	Median time from diagnosis to treatment (Q1–Q3), days	244 (141–300)	NA*	244 (141–300)	NA	NA

Table 2. Treatment patterns within 1 year of diagnosis by LPCa risk						
Treatment	Label	Total	Intermediate-risk	High- or very high-risk	p	Standardized difference
		N=31 571	n=18 365	n=13 206		
PARP inhibitors	n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	NA	NA
Estrogens	n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	NA	NA
Radium-223	n (%)	1–5*	0 (0.0%)	1–5*	NA	NA
Bone agents	n (%)	1082 (3.4%)	500 (2.7%)	582 (4.4%)	<0.0001	0.091
	Median time from diagnosis to treatment (Q1–Q3), days	64 (28–151)	53 (21–104)	81 (36–191)	<0.0001	0.343
Any treatment [‡]	1 - n (%)	27 565 (87.3%)	14 968 (81.5%)	12 597 (95.4%)	<0.0001	0.445
	Median time from diagnosis to treatment (Q1–Q3), days	84 (53–128)	92 (62–138)	72 (42–115)	<0.0001	0.401

Note: Mean and median are estimated among patients who had the treatment (i.e., excluding those without the treatment).

*Sample size suppressed due to the small # for the difference between this group and the group for brachy. [‡]Any treatment, including RP, EBRT, brachytherapy, ADT (including orchiectomy), chemotherapy, ARPIs, PARPi, and radium-223. ADT: androgen deprivation therapy; ARPI: androgen receptor pathway inhibitor; EBRT: external beam radiotherapy; LPCa: locally advanced prostate cancer; PARP: poly(ADP-ribose) polymerase; RP: radical prostatectomy.

Table 3. Treatment patterns within 1 year of diagnosis by LPCa risk, among patients aged ≥66 years						
Treatment	Label	Total	Intermediate risk	High- or very high-risk	p	Standardized difference
		N=18 482	n=10 254	n=8228		
RP	n (%)	4569 (24.7%)	2145 (20.9%)	2424 (29.5%)	<0.0001	0.198
	Median time from diagnosis to treatment (Q1–Q3), days	103 (71–146)	105 (73–153)	98 (70–141)	0.0003	0.108
EBRT	n (%)	9766 (52.8%)	5394 (52.6%)	4372 (53.1%)	0.4717	0.011
	Median time from diagnosis to treatment (Q1–Q3), days	117 (75–175)	98 (65–148)	145 (94–200)	<0.0001	0.555
Brachytherapy	n (%)	1566 (8.5%)	1135 (11.1%)	431 (5.2%)	<0.0001	0.214
	Median time from diagnosis to treatment (Q1–Q3), days	127 (96–170)	121 (91–161)	146 (112–194)	<0.0001	0.395
EBRT + brachytherapy	n (%)	1548 (8.4%)	1119–1123*	425–429*	<0.0001	0.212
	Median time from diagnosis to treatment (Q1–Q3), days	124 (91–167)	118 (89–155)	141 (105–189)	<0.0001	0.381
Bilateral orchiectomy	n (%)	41 (0.2%)	15–19*	22–26*	0.0706	0.026
	Median time from diagnosis to treatment (Q1–Q3), days	48 (25–112)	39 (25–53)	71 (31–130)	0.1717	0.443
ADT and EBRT, or brachytherapy and EBRT, or RP alone	n (%)	9315 (50.4%)	3420 (33.4%)	5895 (71.6%)	<0.0001	0.830
	Median time from diagnosis to treatment (Q1–Q3), days	97 (58–150)	98 (63–153)	95 (56–149)	<0.0001	0.085
LHRH agonist/antagonist monotherapy	n (%)	7436 (40.2%)	1934 (18.9%)	5502 (66.9%)	<0.0001	1.109
	Median time from diagnosis to treatment (Q1–Q3), days	51 (30–82)	57 (35–91)	49 (29–78)	<0.0001	0.211
	n (%)	6560 (35.5%)	1682 (16.4%)	4878 (59.3%)	<0.0001	0.986

Table 3. Treatment patterns within 1 year of diagnosis by LPCa risk, among patients aged ≥66 years						
Treatment	Label	Total	Intermediate risk	High- or very high-risk	p	Standardized difference
		N=18 482	n=10 254	n=8228		
LHRH + first generation antiandrogen	Median time from diagnosis to treatment (Q1–Q3), days	52 (32–82)	57 (36–89)	50 (30–78)	<0.0001	0.198
Chemotherapy	n (%)	46 (0.2%)	3-7*	39-43*	<0.0001	0.082
	Median time from diagnosis to treatment (Q1–Q3), days	134 (92–245)	121 (90–176)	146 (93–247)	0.6131	0.212
ARPIs	n (%)	40 (0.2%)	1–5*	35–39*	NA	NA
	Median time from diagnosis to treatment (Q1–Q3), days	252 (130-297)	NA*	260 (141–300)	NA	NA
PARP inhibitors	n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	NA	NA
Estrogens	n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	NA	NA
Radium-223	n (%)	1–5*	0 (0.0%)	1–5*	NA	NA
Bone agents	n (%)	996 (5.4%)	455 (4.4%)	541 (6.6%)	<0.0001	0.094
	Median time from diagnosis to treatment (Q1–Q3), days	64 (28–147)	54 (22–106)	79 (35–179)	<0.0001	0.311
Any treatment [‡]	n (%)	15 842 (85.7%)	8007–8011* (78.1%)	7831–7835* (95.2%)	<0.0001	0.519
	Median time from diagnosis to treatment (Q1–Q3), days	69 (42–110)	82 (54–124)	57 (32–91)	<0.0001	0.534

Note: Mean and median are estimated among patients who had the treatment (i.e., excluding those without the treatment).

*Sample size is suppressed due to the small # for the difference between this group and the group for brachytherapy, or between this cohort of patients aged ≥66 years and the overall cohort patients, or between the current table and previous corresponding table.

‡Any treatment including RP, EBRT, brachytherapy, ADT (including orchiectomy), chemotherapy, ARPIs, PARPi, and radium-223.

ADT: androgen deprivation therapy; ARPI: androgen receptor pathway inhibitor; EBRT: external beam radiotherapy; LPCa: locally advanced prostate cancer; LHRH: luteinizing hormone-releasing hormone; PARP: poly(ADP-ribose) polymerase; RP: radical prostatectomy.

Table 4. ADT received after RP surgery among patients receiving RP surgery as their first treatment, and aged ≥ 66 years			
Treatment	Label	Up to 6 months after RP surgery	Up to 12 months after RP surgery
	(Sample size)	n=4468	n=4468
Any ADT	n (%)	275 (6.2%)	389 (8.7%)
ADT with concurrent RT	n (%)	125 (2.8%)	240 (5.4%)
	Mean (SD) duration of ADT prescription, months [†]	3.7 (2.1)	3.6 (2.3)
	Median (IQR) duration of ADT prescription, months [†]	4 (3–6)	3 (2–6)
ADT with non-concurrent RT	n (%)	17 (0.4%)	43 (1.0%)
Having ADT first, then RT	n, (%) among ADT with non-concurrent RT	12–16*	36 (83.7%)
ADT without RT [‡]	n, (%)	133 (3.0%)	106 (2.4%)
	Mean (SD) duration of ADT prescription, months [†]	3.5 (2.6)	6.6 (5.3)
	Median (IQR) duration of ADT prescription, months [†]	4 (1–4)	6 (2–10)

[†]Mean and median values of ADT use are estimated among patients who received ADT. [‡]Without RT means that patients did not receive any RT treatment in the 6 or 12 months after RP surgery. ADT: androgen deprivation therapy; IQR: interquartile range; RT: radiotherapy; SD: standard deviation.

Table 5. Clinical outcomes for patients with intermediate and high risk localized prostate cancer						
	Label	Total	Intermediate-risk	High- or very high-risk		Standardized difference
Outcome	(Sample size)	N=31 571	n=18 365	n=13 206	p	
Time to diagnosis of metastatic disease, days	n (%)	11 474 (36%)	5435 (30%)	6039 (46%)	<0.0001	0.338
	Mean (SD)	1461.3 (1236.9)	1666.1 (1268.4)	1276.9 (1177.9)	<0.0001	0.318
	Median (IQR)	1281 (253–2345)	1576 (439–2605)	958 (188–2091)	<0.0001	0.307
Time to castration resistance (CRPC), days	n (%)	1245 (4%)	285 (2%)	960 (7%)	<0.0001	0.281
	Mean (SD)	1496.2 (847.0)	1955.9 (839.9)	1359.7 (800.1)	<0.0001	0.727
	Median (IQR)	1311 (798–2048)	1930 (1280–2555)	1162 (725–1828)	<0.0001	0.767
Time to first mCRPC treatment, days	n (%)	775 (2%)	149 (1%)	626 (5%)	<0.0001	0.241
	Mean (SD)	1721.5 (892.9)	2211.1 (898.0)	1605.0 (851.9)	<0.0001	0.692
	Median (IQR)	1583 (975–2324)	2207 (1520–2806)	1449 (927–2164)	<0.0001	0.717
Time to prostate cancer event, days [†]	n (%)	19 808 (62.7%)	10 522 (57.3%)	9286 (70.3%)	<0.0001	0.274
	Mean (SD)	460.6 (723.0)	464.9 (761.6)	455.8 (676.5)	0.3783	0.013
	Median (IQR)	161 (91–406)	134 (78–399)	187 (115–412)	<0.0001	0.275
Overall mortality (time to death)	n (%)	7219 (23%)	3343 (18%)	3876 (29%)	<0.0001	0.264
	Mean (SD)	2016.9 (1122.0)	2171.3 (1136.3)	1883.8 (1092.3)	<0.0001	0.258
	Median (IQR)	1946 (1109–2839)	2155 (1287–3022)	1798 (1001–2661)	<0.0001	0.258

Note: Mean and median are estimated among patients who had the outcome (i.e., excluding those without the outcome).

[†]PCa event including BCR, RT, or bone agents. Time to outcome is estimated from index to the event in days. ADT: androgen deprivation therapy; CRPC: castrate-resistant prostate cancer; IQR: interquartile range; SD: standard deviation.

Table 6. Treatment with any surgery or radiotherapy in intermediate- and high-risk localized prostate cancer by LHIN						
LHIN	Intermediate-risk			High/very high-risk		
	No active treatment	Surgery	Radiotherapy	No active treatment	Surgery	Radiotherapy
LHIN A	310 (13.0%)	998 (41.9%)	1290 (54.1%)	202 (12.6%)	753 (46.8%)	1076 (66.9%)
LHIN B	251 (11.4%)	1031 (46.8%)	1131 (51.3%)	226 (13.9%)	815 (50.2%)	978 (60.2%)
LHIN C	317 (14.2%)	919 (41.1%)	1170 (52.4%)	220 (14.5%)	636 (41.9%)	959 (63.2%)
LHIN D	208 (11.8%)	730 (41.5%)	1010 (57.4%)	147 (9.9%)	677 (45.8%)	1038 (70.2%)
LHIN E	226 (13.9%)	722 (44.3%)	796 (48.9%)	152 (13.4%)	648 (57.2%)	623 (55.0%)
LHIN F	201 (13.7%)	555 (38.0%)	801 (54.8%)	125 (12.1%)	506 (49.1%)	695 (67.4%)
LHIN G	226 (16.2%)	551 (39.5%)	738 (52.9%)	117 (12.7%)	472 (51.4%)	563 (61.3%)
LHIN H	116 (11.2%)	449 (43.3%)	546 (52.7%)	72 (10.2%)	340 (48.0%)	472 (66.6%)
LHIN I	123 (13.2%)	403 (43.2%)	483 (51.8%)	82 (11.6%)	337 (47.8%)	449 (63.7%)
LHIN J	169 (19.6%)	336 (39.0%)	432 (50.2%)	119 (16.4%)	323 (44.4%)	460 (63.3%)
LHIN K	122 (15.0%)	365 (45.0%)	412 (50.8%)	91 (16.0%)	296 (52.1%)	336 (59.2%)
LHIN L	94 (14.5%)	291 (44.9%)	333 (51.4%)	95 (16.7%)	220 (38.7%)	375 (66.0%)
LHIN M	84 (11.7%)	315 (43.9%)	368 (51.3%)	57 (12.6%)	217 (48.1%)	273–277*
LHIN N	27 (9.3%)	114 (39.3%)	178 (61.4%)	28 (16.7%)	65 (38.7%)	109–113*
p-value between LHINs	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

*Sample size suppressed due to the small patient counts. LHIN: local health integration network.

Table 7. Median (IQR) days from prostate cancer diagnosis to initiation of surgery or radiotherapy in intermediate– and high–risk localized prostate cancer by LHIN				
LHIN	Intermediate–risk		High/very high–risk	
	Any surgery	Any radiotherapy	Any surgery	Any radiotherapy
LHIN A	101 (70–148)	133 (77–364)	90 (66–126)	222 (135–393)
LHIN B	89 (63–129)	127 (82–360)	83 (57–118)	237 (146–523)
LHIN C	132 (91–195)	134 (85–391)	118 (83–163)	184 (138–333)
LHIN D	119 (89–168)	118 (74–298)	112 (82–146)	165 (84–338)
LHIN E	123 (85–173)	120 (69–247)	114 (82–148)	181 (120–393)
LHIN F	112 (78–172)	133 (85–280)	97 (69–140)	191 (119–348)
LHIN G	105 (71–162)	142 (83–387)	100 (67–147)	222 (133–466)
LHIN H	104 (74–154)	134 (81–337)	97 (70–129)	191 (116–344)
LHIN I	151 (90–211)	125 (70–308)	119 (81–166)	175 (125–336)
LHIN J	139 (72–202)	139 (63–401)	107 (64–153)	172 (95–329)
LHIN K	94 (66–148)	154 (91–492)	84 (65–124)	202 (141–526)
LHIN L	112 (82–154)	153 (73–365)	96 (75–134)	165 (77–344)
LHIN M	112 (76–164)	110 (67–179)	115 (74–152)	154 (110–328)
LHIN N	85 (62–119)	153 (104–502)	79 (64–99)	247 (167–496)
p-value between LHINs	<0.0001	<0.0001	<0.0001	<0.0001

LHIN: local health integration network.