Hematologic dysfunction and myeloid neoplasm risk in patients treated with lutetium-177 prostate-specific antigen membrane therapy

In 2022, the US Food and Drug Administration approved ^{177}Lu-PSMA-617 (^{177}Lu-PSMA), a radiotheranostic, for metastatic castration-resistant prostate cancer (mCRPC), following the landmark phase III VISION trial that demonstrated improved overall survival (OS) and radiographic progression-free survival (PFS) compared to best standard of care.[1] While ^{177}Lu-PSMA has been shown to improve quality of life,[2] hematologic toxicity is prevalent.[1,3] ^{177}Lu-PSMA targets cells with PSMA expression leading to β-particle emission and single-stranded DNA breaks. We previously identified therapy-related clonal cytopenia of undetermined significance (tCCUS) and therapy-related myeloid neoplasms (tMN) in 4% of patients with metastatic NET (neuroendocrine tumor) who received ^{177}Lu-DOTATATE,[4] a radiotheranostic agent targeting the somatostatin receptor (SSTR). Among prostate cancer patients, the risk of tMN is elevated, with hazard ratios ranging from 2.0-6.5, influenced by treatment exposures.[5,6] However, to date, no studies have evaluated changes to the bone marrow in patients treated with ^{177}Lu-PSMA. While the current indication for ^{177}Lu-PSMA is in patients with mCRPC who have exhausted multiple prior lines of treatments (and whose median OS is approximately 15.3 months), ongoing efforts to advance ^{177}Lu-PSMA to earlier lines of therapy[7] underscore the need for a more in-depth understanding of potential hematologic risks. Here, we evaluated hematologic dysfunction and tCCUS/tMN prevalence in mCRPC patients treated with ^{177}Lu-PSMA. After Mayo Clinic Institutional Review Board approval, we identified adult patients treated at Mayo Clinic (enterprise-wide), with >1 dose of ^{177}Lu-PSMA between January 1, 2022, and December 31, 2023. Patients with <3-month follow-up were excluded from analyses. Cytopenias were defined per NCI CTCAE v5.0 and MN diagnoses was defined per the revised 4th-edition World Health Organization criteria (2016).[8] We evaluated patients who had a bone marrow biopsy (BMB) post-^{177}Lu-PSMA, focusing on those with assessable BM morphology, cytogenetics and molecular genetics. Standard descriptive statistical methods were used, specific tests are noted in Figure legends.

Of 405 patients who received >1 dose of ^{177}Lu-PSMA, 42% (N=172) had follow-up with laboratories at least 3 months post-177Lu-PSMA, before subsequent cytotoxic cancer-directed therapy (*Online Supplementary Figure S1*). The median age at first ^{177}Lu-PSMA was 72.8 years (range, 45.1-93.8). Prior to receiving ^{177}Lu-PSMA, 92.4% (N=159) of patients had a history of chemotherapy (chemo), with 88.9% (N=153) and 23.2% (N=40) receiving docetaxel and/or carboplatin, respectively. Prior radiation therapy (RT) was present in 86.6% (N=149), with 69.8% (N=120) receiving salvage RT, and 12.8% (N=22) receiving palliative RT to sites of metastases only. Additionally, 7.0% (N=12) had a history of ^{223}Radium-dichloride (Xofigo) treatment prior to ^{177}Lu-PSMA, and 13.3% (N=23) had prior treatment with PARPi (inhibitors). Patients received a median of six doses (range, 2-9) of ^{177}Lu-PSMA, amounting to a cumulative median exposure of 1,167 mci (range, 193-2867).

At baseline (<1 month prior to 1st dose of ^{177}Lu-PSMA), the median hemoglobin (HGB) was 12.1 g/dL (range, 7.9-17.9) and anemia was present in 42.0% (N=68, grade 1 - 34.6% [N=56], grade2 - 6.8% [N=11], grade 3 - 0.6% [N=1], grade 4 - none (*Online Supplementary Table S1*; Figure 1). Baseline anemia was not associated with age, prior chemo, or prior RT, but was associated with prior PARPi (*P*=0.011) and Radium-^{223} therapy (*P*=0.013) (*Online Supplementary Table S2*). Among those who were not on subsequent cancer-directed therapy at 3 (N=145), 6 (N=111), and 12 (N=44) months post-^{177}Lu-PSMA, any grade of anemia was present in 69.7% (N=101; *P*<0.001), 75.7% (N=84; *P*<0.001), and 72.7% (N=32; *P*=0.003), with grade 3 anemia present in 8.3% (N=12), 10.8% (N=12), and 13.6% (N=6), respectively, and none with grade 4 anemia (Figure 1). Of those with HGB values available at all follow-up time points (N=29), persistent anemia was present in 58.6% (N=17) and those who were anemic at baseline were more likely to be anemic at 12-month follow-up (*P*=0.003; *Online Supplementary Table S1*; Figure 1). While the median mean corpuscular volume (MCV) was 95.0 fL (range, 74.8-116.7) at baseline and 98.8 fL (range, 80.4-124.4) at 12-months (*P*=0.004), there were no changes in red cell distribution width (RDW) with time (*Online Supplementary Table S1*).

Thrombocytopenia was present in 10% of patients at baseline (grade 1 - 7.5% [N=12], grade 2 - 1.9% [N=3], grade 3 - 0.6% [N=1], grade 4 - none) with a median platelet (PLT) count of 208x10^9/L (range, 28-387). Baseline PLT count was not associated with age, prior chemo, RT, or Radium-^{223} use, but was associated with prior PARPi use (*P*=0.044; *Online Supplementary Table S1*). At 3 (N=141), 6 (N=108), and 12 (N=41) months post-treatment, among those not on subsequent cancer-directed therapy, thrombocytopenia was present in 36.9% (N=52; *P*<0.001), 32.4% (N=35; *P*<0.001) and 24.4% (N=10; *P*=0.182), respectively. At 3 (N=116), 6 (N=90), and 12 (N=44) months, no patients had grade 4 thrombocytopenia, while grade 3 thrombocytopenia was present in 2.8% (N=4), 7.4% (N=8), and 0% (N=0), respectively. In patients assessable at all three follow-up time points (N=25),

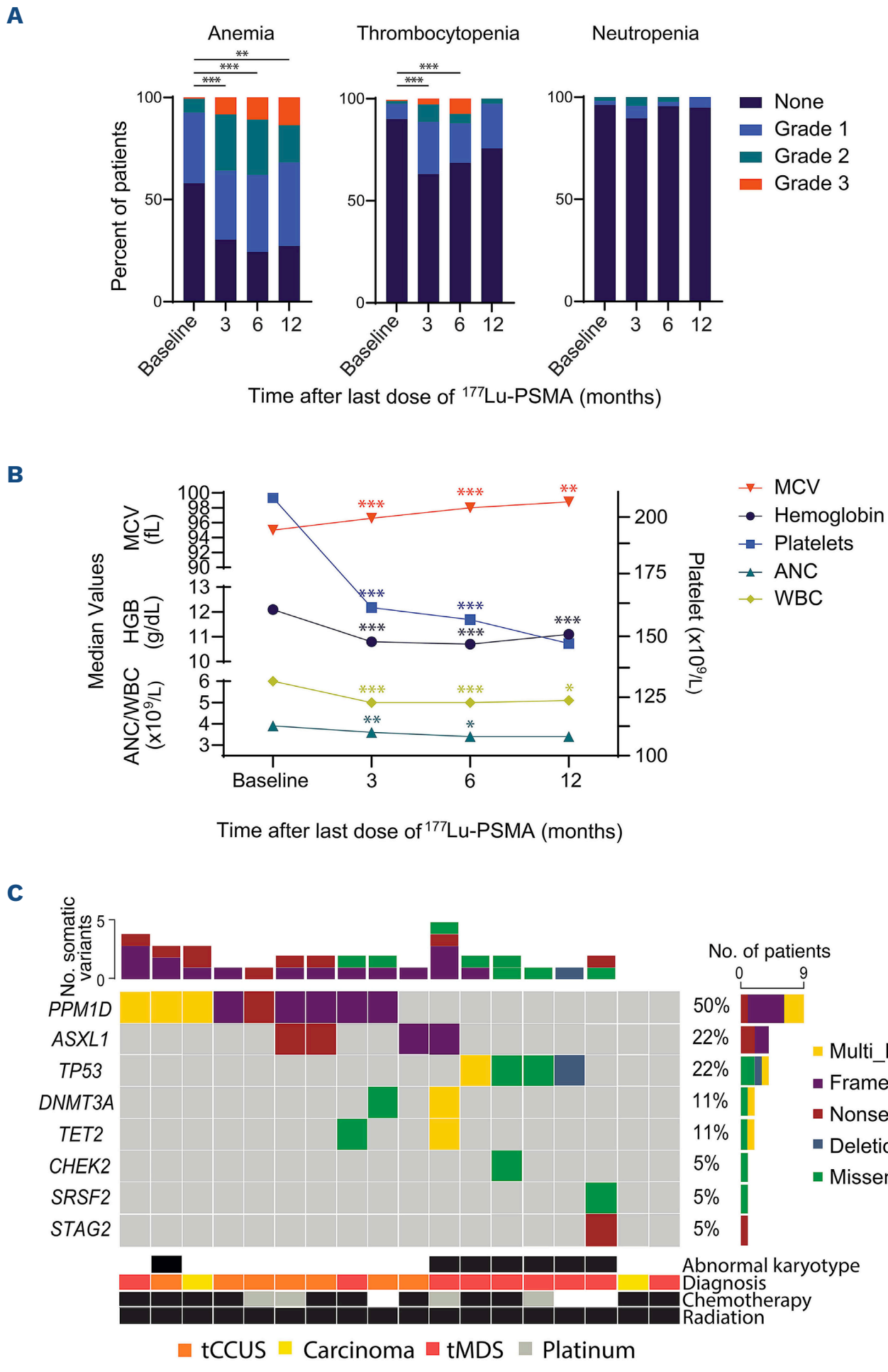

Figure 1. Hematologic dysfunction in metastatic prostate cancer patients treated with *177Lu-PSMA, despite not being on concurrent cytotoxic cancer-directed therapies (other than androgen deprivation therapy). (A) Percent of patients with anemia, thrombocytopenia and neutropenia at baseline, 3, 6, and 12 months after last dose of 177Lu-PSMA. Pairwise comparisons for each time point compared to baseline were assessed using paired *t* test for continuous variables and McNemar tests for binary variables. *P*<0.05 were considered significant. **P*<0.05; ***P*<0.01; ****P*<0.001. (B) Median laboratory values at baseline (0-1 month prior to first dose, N=162), 3 (N=145), 6 (N=113), and 12 (N=45) months after last dose. Pairwise comparisons for each time point compared to baseline were assessed using paired *t* test for continuous variables and McNemar tests for binary variables. *P*<0.05 were considered significant. **P*<0.05; ***P*<0.01; ****P*<0.001. (C) Oncoplot of pathogenic variants among 16 (of 18) assessable patients. Two patients had no variants identified. ANC: absolute neutrophil count; WBC: white blood cell count; HGB: hemoglobin; MCV: mean corpuscular volume; tCCUS: therapy-related clonal cytopenia of undetermined significance; tMDS: therapy-related myeloid neoplasm.

Table 1. Demographic and clinical characteristics of prostate cancer patients undergoing bone marrow biopsy with comprehensive molecular testing after ^{177}Lu-PSMA Therapy.

ID	Prior therapy			Time from 1st administration to bone marrow biopsy, years				^{177}Lu-PSMA			Bone marrow biopsy				Outcome
	Chemo (N of cycles)	RT	Other (duration)	Chemo	RT	Other	^{177}Lu	Age, years	N of doses	Total mci	Diagnosis	Dysplasia	Gene mutation by NGS (%)	Cytogenetics	
19	*RCHOP (6) Docetaxel (6)	Salvage + Palliative	PT112 (4 months)	4.1	5.6	0.4	1.7	76	6	1,200	tMDS	Yes	*PPM1D*: c.1525del, p.Asp509Thrfs*5 (2) *PPM1D*: c.1535dup, p.Asn512Lysfs*16 (9) *PPM1D*: c.1535del, p.Asn512Ilefs*2 (2) *PPM1D*: c.1654C>T, p.Arg552* (5)	46,XY[10]	Deceased; MDS/prostate cancer progression
22	Docetaxel (6) Carboplatin (6)	Palliative	-	9.4	9.4	-	0.4	77	3	590	tMDS	Yes	*TP53*: c.713G>T, p.Cys238Phe (82)	45-48,XY,add(2)(q21),-5,-7,+8,+0-2r,+0-2mar[cp17]/92-94,idemx2[cp3]	Deceased; Treated with HMA/Ven, MDS/Prostate cancer progression
17	None	Salvage + Palliative	-	-	15.0	-	0.7	85	6	1,186	tMDS	Yes	*SRSF2*: c.284C>G, p.Pro95Arg (44) *STAG2*: c.775C>T, p.Arg259* (38)	XY,del(20)(q11.2q13.3)[14]/47,sl,+8[4]/46,XY[2]	Deceased; stroke
2	Docetaxel (6)	Salvage + Palliative	PARPi (6 months)	2.2	12.0	2.9	1.3	74	6	1,168	tCCUS	No	*PPM1D*: c.1422del, p.Glu475Lysfs*8 (3) *PPM1D*: c.1540A>T, p.Lys514* (3) *PPM1D*: c.1654C>T, p.Arg552* (2)	45,XY,-7[6]/46,XY[14]	Deceased; sepsis/acute renal failure
5	Docetaxel (6)	Salvage	Radium-223 (6 cycles, 0.82 mci)	1.9	9.4	3.0	0.9	68	6	1,167	Carcinoma (10%)	No	No pathogenic variants detected	45,X-Y[10]/46,XY[10]	Deceased; prostate cancer progression
23	Docetaxel (12) Cabazitaxel (8)	Palliative	-	7.9	4.7	-	0.9	82	6	1,184	Carcinoma (5%)***	No	*PPM1D*: c.1528C>T, p.Gln510* (4) *PPM1D*: c.1535dup, p.Asn512Lysfs*16 (3) *PPM1D*: c.1561del, p.Met521* (9)	46,XY[20]	Deceased; prostate cancer progression
7	Docetaxel (6)	Palliative	-	6.1	0.2	-	0.8	84	6	1,144	tMDS	Yes	^No pathogenic variants detected	46,XY[20]	Deceased; prostate cancer progression
24	Docetaxel (9) Carboplatin (6)	Palliative	-	8.9	53.1	-	0.8	77	6	1,180	tMDS	Yes	*ASXL1*: c.1934dup, p.Gly646Trpfs*12 (4) *DNMT3A*: c.2238del, p.Asp747Metfs*32 (15) *DNMT3A*: c.2246G>A, p.Arg749His (2) *TET2*: c.651del, p.Val218Trpfs*32 (6) *TET2*: c.3085G>T, p.Glu1029* (5)	46,XY,add(5)(q15),-7,+0-1mar[cp5]/46,XY[15]	Stable; on luspatercept
3	Docetaxel (6)	Palliative	-	1.3	6.3	-	0.4	68	3	589	tMDS	Yes	*PPM1D*: c.1596_1632dup, p.Pro545Lysfs*3 (26) *TET2*: c.1422del, p.Arg1261Cys (6)	46,XY[20]	Deceased; MDS/prostate cancer progression
14	None	Salvage + Palliative	-	-	12.1	-	4.7	84	6	799	tCCUS	No	*PPM1D*: c.1422del, p.Glu475Lysfs*8 (42) *DNMT3A*: c.2711C>T, p.Pro904Leu (41)	46,XY[20]	Stable; transfusions and aranesp
9	Docetaxel (6)	Palliative	-	6.1	3.7	-	0.5	74	3	567	tCCUS	No	*PPM1D*: c.1451dup, p.Leu484Phefs*5 (5)	46,XY[20]	Deceased; unknown
1	Docetaxel (6) Carboplatin (3)	Salvage	-	2.9	2.3	-	0.7	82	4	791	tCCUS	No	PPM1D: c.1666dup, p.Ser556Lysfs*4 (9) ASXL1: c.4276G>T, p.Glu1426* (14)	46,XY[20]	Stable

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ID	Prior therapy			Time from 1st administration to bone marrow biopsy, years				^{177}Lu-PSMA			Bone marrow biopsy				Outcome
	Chemo (N of cycles)	RT	Other (duration)	Chemo	RT	Other	^{177}Lu	Age, years	N of doses	Total mci	Diagnosis	Dysplasia	Gene mutation by NGS (%)	Cytogenetics	
25	**FOLFOX (4) Docetaxel (4)	Salvage + Palliative	-	4.5	2.7	-	0.7	63	5	814	tCCUS	No	PPM1D: c.1461_1462insT, p.Asp488* (7)	46,XY[20]	Deceased; unknown
34	None	Salvage + Palliative	-	-	6.5	-	1.9	71	2	402	tMDS	Yes	*TP53*: c.827_841del, p.Ala276_Arg280del (74)	46,XY,r(1)(p?36.3q44),der(5)t(5;11)(q13;q21)[12]/46,idem,?inv(3)(p25q11.2)[7]/46,XY[1]	Deceased; Treated with HMA/Ven, Prostate cancer progression
58	Docetaxel (6)	Palliative	-	5.6	3.5	-	1.1	69	6	1,190	tCCUS	No	*PPM1D*: c.1388delG; p.Gly463Valfs* (3) *ASXL1*: c.2077C>T; p.Arg693* (15)	46,XY[20]	Stable
4	Docetaxel (6)	Salvage	-	2.9	10.0	-	2.1	82	6	1186	tMDS	Yes	*TP53*: c.742C>T; p.Arg248Trp (29) *CHEK2*: c.479T>C; p.Ile160Thr (4)	44,XY,dic(5;17)(q11.2;p11.2),dic(7;20)(p11.2;q11.2),del(13)(q12q14)[11], 44,idem,-dic(7;20),+mar[2], 46,XY[5]	Deceased; subarachnoid hemorrhage
6	Docetaxel (3)	Salvage + Palliative	-	2.3	15.5	-	1.5	83	4	783	tCCUS	No	*ASXL1*: c.1934dup; p.Gly646Trpfs*12 (3)	46,XY[20]	Stable
8	Docetaxel (6) Cabazitaxel (6)	Salvage + Palliative	-	5.4	16.2	-	2.1	74	6	1199	tMDS	Yes	*TP53*: c.879_880del p.Glu294Alafs*Ter11 (46) *TP53*: c.659A>G; p.Tyr220Cys (46)	46,XY,add(5)(q13),-11,der(18;22)(p10;q10),+2r[20]	Deceased; MDS progression

*History of lymphoma, **history of colorectal cancer, ***due to carcinoma infiltration of bone marrow, cannot rule out contribution of carcinoma causing cytopenia. ^Panel did not include *PPM1D*. Chemo: chemotherapy; RT: radiation; MCI: millicurie; tMDS: therapy-related myelodysplastic syndrome; tCCUS: therapy-related clonal cytopenia of undetermined significance; NGS: next-generation sequencing; PT112: small-molecular platinum pyrophosphate conjugate; HMA: hypomethylating agent; Ven: venetoclax.

persistent thrombocytopenia was seen in 8% (N=2) and was not associated with thrombocytopenia at baseline (*P*=0.083). At baseline, the median absolute neutrophil count (ANC) was 3.9x10^9/L (range, 0.0-12.9) and neutropenia was present in 3.8% (N=6), with 1.9% (N=3) grade 1, 2 and 3 each. Baseline neutropenia was not associated with age or prior treatments (*Online Supplementary Table S2*). Patients who were neutropenic at baseline were not more likely to be neutropenic at follow-up. There were no patients with persistent neutropenia at 12 months.

BMB, next-generation sequencing (NGS) and cytogenetics were performed in 10.5% (N=18 of 172) of patients, of which 83.3% (N=15) had prior history of chemo (22.2% - platinum), 100% (N=15) had prior RT (salvage - 53.3%, N=8, palliative-46.7%, N=7), and 5.5% (N=1) each had prior Radium-^{223} or PARPi (Table 1). BMB was performed 4.5 years (yrs) (range, 1.3-9.4), 7.9 yrs (range, 0.1-53.1) and 0.9 yrs (range, 0.4-4.7) from first chemo, RT, and ^{177}Lu-PSMA, respectively. Diagnoses included: tMDS in 50.0% (N=9), tCCUS in 38.9% (N=7), and metastatic carcinoma in 11.1% (N=2). Diagnosis of tMDS or tCCUS was not associated with prior treatments (*P*=0.83), older age (*P*=0.52), or total mci exposure (*P*=0.69).

NGS revealed 34 somatic pathogenic variants (PV) in 18 patients, with a median of two PV (range, 0-5) per patient, and median variant allele fraction (VAF) of 8.5% (range, 2-82). The most common PV was *PPM1D*, with 16 PV identified among nine patients (60.0%), followed by *TP53*, with five PV identified among four patients (22.2%, 4 presumed biallaleic; Figure 1). PV in *PPM1D* and *TP53* were mutually exclusive (*P*=0.023), and PV in *TP53* correlated with complex cytogenetics (*P*=0.004). Specific PV or VAF were not associated with age (*P*=0.15), total mci exposure (*P*=0.23), or prior treatment (*P*=0.57). Abnormal karyotype was present in 38.9% (N=7), and five patients (27.8%) had a complex karyotype. The most common karyotypic abnormalities included deletion 5q (N=3, 16.7%) and monosomy 7 (N=3, 16.7%). Abnormal karyotype and was not associated with age (*P*=0.63), total mci exposure (*P*=0.80) or prior treatments (*P*=0.66). PV in *PPM1D* were predominantly found in patients with a normal karyotype (*P*=0.01), whereas PV in *TP53* were found among those with abnormal karyotype (*P*=0.004). With a median follow-up of 1.6 yrs (range, 0.5-5.9) from first ^{177}Lu-PSMA, 72.2% (N=13) of those who had a BMB with molecular testing (N=18) were deceased, with 53.8% (N=7) due to progression of disease (Table 1).

To our knowledge, this is the first series to detail tCCUS/tMN in mCRPC patients following ^{177}Lu-PSMA treatment. Limitations of this retrospective analysis include the confounding effects of prior exposure to cytotoxic chemo and RT, which was present in 83.3% and 100.0%, respectively, making it challenging to determine whether ^{177}Lu-PSMA was directly or solely responsible. Additionally, the lack of comprehensive hematologic assessments in these patients, which may relfect providers' perceptions of limited life expectancy or provider difficulty in distinguishing BM disease from treatment toxicity, may underestimate the prevalence of tCCUS/tMN in this population.

Within the VISION trial, 23.4% of patients experienced grade >3 BM suppression and drug-related adverse events leading to death occurred in 0.9%, with etiologies including pancytopenia and BM failure; without tMN diagnosis.[1] Among patients in our cohort who underwent BMB for cytopenias, tCCUS/tMN was identified in 88.9%, with DNA damage response/repair (DDR) pathway mutations present in 72.2% (50.0% with *PPM1D*, 22.2% with *TP53*). It is likely that prior exposures to chemo and RT, followed by ^{177}Lu-PSMA, positively selects hematopoietic progenitor cells with intrinsic resistance mechanisms to apoptosis, in this case, somatic DDR mutations. Over time, these mutant clones result in hematologic dysfunction and tCCUS/tMN. While genomic instability in *TP53* mutant clonal hematopoiesis is a well documented pathway towards MN progression, mechanisms of MN progression with *PPM1D* mutations remain to be ascertained.[9-13] We also highlight the short latency for tCCUS/tMN, akin to reports in ovarian cancer patients receiving platinum agents and PARPi.[14] While the median survival of mCRPC patients receiving ^{177}Lu-PSMA is 15.3 months, current attempts to advance ^{177}Lu-PSMA in earlier stages of disease raise important long-term hematologic safety questions.[1,15-18] These findings underscore the need for further research into mechanisms of clonal evolution and hematologic surveillance strategies in this population.

Authors

Yael Kusne,[1] Osama M. Mosalem,[2] Jaxon Quillen,[3] Skye Buckner-Petty,[3] Miguel Muniz,[4] Oliver Sartor,[4] Geoffrey B. Johnson,[5] Daniel S. Childs[4#] and Mrinal M. Patnaik[6#]

[1]Division of Hematology and Oncology, Mayo Clinic, Phoenix, AZ; [2]Division of Hematology and Oncology, Mayo Clinic, Jacksonville, FL; [3]Department of Biostatistics, Mayo Clinic, Scottsdale, AZ; [4]Department of Medical Oncology, Mayo Clinic, Rochester, MN; [5]Department of Radiology, Mayo Clinic, Rochester, MN and [6]Division of Hematology, Mayo Clinic, Rochester, MN, USA

[#]*DSC and MMP contributed equally as senior authors.*

Correspondence:
Y. KUSNE - Kusne.Yael@mayo.edu

https://doi.org/10.3324/haematol.2024.286808

Received: October 24, 2024.
Accepted: March 26, 2025.
Early view: April 3, 2025.

Disclosures

MMP has received research funding from Stemline Pharmaceuticals, Kura Oncology, Epigenetix, Solu Therapeutics and Polaris Pharmaceuticals. OS is a consultant for ArtBio, Amgen, Astellas, AstraZeneca, Bayer, Clarity, EMD Serono, Fusion, Isotopen, Janssen, MacroGenics, Novartis, Pfizer, Point Biopharma, Ratio, Sanofi, Telix, and TeneoBio; and receives research support from Advanced Acceleratory Applications, Amgen, AstraZeneca, Bayer, In Vitae, Janssen, Lantheus, Merck, Sanofi, and Point Biopharma.

Contributions

YK and OM collected the data and wrote the manuscript. YK, MP and DC designed the study. JQ and SBP performed statistical analyses. MM, OS, GBJ, DC and MP reviewed and edited the manuscript.

Data-sharing statement

The data supporting this study are available upon request to the corresponding author.

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