Rucaparib in Men With Metastatic Castration-Resistant Prostate Cancer Harboring a BRCA1 or BRCA2 Gene Alteration

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PURPOSE BRCA1 or BRCA2 (BRCA) alterations are common in men with metastatic castration-resistant prostate cancer (mCRPC) and may confer sensitivity to poly(ADP-ribose) polymerase inhibitors. We present results from patients with mCRPC associated with a BRCA alteration treated with rucaparib 600 mg twice daily in the phase II TRITON2 study.

METHODS We enrolled patients who progressed after one to two lines of next-generation androgen receptor-directed therapy and one taxane-based chemotherapy for mCRPC. Efficacy and safety populations included patients with a deleterious BRCA alteration who received at least 1 dose of rucaparib. Key efficacy end points were objective response rate (ORR; per RECIST/Prostate Cancer Clinical Trials Working Group 3 in patients with measurable disease as assessed by blinded, independent radiology review and by investigators) and locally assessed prostate-specific antigen (PSA) response (≥ 50% decrease from baseline) rate.

RESULTS Efficacy and safety populations included 115 patients with a BRCA alteration with or without measurable disease. Confirmed ORRs per independent radiology review and investigator assessment were 43.5% (95% CI, 31.0% to 56.7%; 27 of 62 patients) and 50.8% (95% CI, 38.1% to 63.4%; 33 of 65 patients), respectively. The confirmed PSA response rate was 54.8% (95% CI, 45.2% to 64.1%; 63 of 115 patients). ORRs were similar for patients with a germline or somatic BRCA alteration and for patients with a BRCA1 alteration. A higher PSA response rate was observed in patients with a BRCA2 alteration. The most frequent grade 3 treatment-emergent adverse event was anemia (25.2%; 29 of 115 patients).

CONCLUSION Rucaparib has antitumor activity in patients with mCRPC and a deleterious BRCA alteration, but with a manageable safety profile consistent with that reported in other solid tumor types.
We evaluated rucaparib as treatment for men with metastatic castration-resistant prostate cancer (mCRPC) associated with a BRCA gene alteration who had received prior taxane and androgen receptor–directed therapy.

Knowledge Generated
A substantial proportion of patients achieved a confirmed radiographic response with rucaparib treatment in both blinded, central independent radiology review– and investigator-assessed analyses. Furthermore, we provide evidence of radiographic and prostate-specific antigen responses across subgroups based on baseline characteristics (eg, number of prior lines of therapy) and genomic characteristics (eg, gene, zygosity, and alteration types).

Relevance
Men with mCRPC and a BRCA alteration who receive a poly(ADP-ribose) polymerase inhibitor in this setting achieve higher objective and prostate-specific antigen response rates than those observed with previously approved therapies in an unselected population. These data illustrate the potential benefit of rucaparib in patients with mCRPC associated with a BRCA alteration.

Efficacy was assessed in the overall efficacy population (all evaluable patients regardless of measurable disease status), independent radiology review (IRR)–evaluable population (patients who had measurable disease at baseline per blinded, central IRR assessment), and investigator-evaluable population (patients who had measurable disease at baseline per investigator assessment).

Efficacy and safety analyses included all patients meeting the above criteria enrolled by May 8, 2019. The visit cutoff date for safety analyses was September 13, 2019. The visit cutoff date for efficacy analyses was December 23, 2019, to allow for more complete assessment (≥ 32 weeks of follow up) of the efficacy end points.

Analysis Outcomes
The primary end point was objective response rate (ORR) by blinded IRR per modified RECIST v1.1 and Prostate Cancer Clinical Trials Working Group 3 (PCWG3) criteria (IRR-evaluable population), supported by confirmed ORR by investigator assessment (investigator-evaluable population).

Secondary end points included duration of response (DOR) for radiographic response, rate of confirmed locally assessed prostate-specific antigen (PSA) response (≥ 50% decrease from baseline confirmed by a consecutive measurement ≥ 3 weeks later), time to PSA progression, radiographic progression-free survival (rPFS), overall survival (OS), and safety.

Exploratory subgroup analyses of confirmed ORR and confirmed PSA response rate were performed based on baseline disease characteristics (number of prior lines of therapy, measurable disease status, presence of hepatic metastases, and age) and genomic characteristics (BRCA1, BRCA2, and other prespecified DDR gene that may confer sensitivity to PARP inhibition, as well as disease progression after one to two lines of next-generation AR-directed therapy for prostate cancer and one prior taxane-based chemotherapy for castration-resistant disease. Patients who were previously treated with a PARP inhibitor, mitoxantrone, cyclophosphamide, or platinum-based chemotherapy or with an active secondary malignancy were excluded. Patients were enrolled irrespective of measurable disease status (Data Supplement).

Full eligibility criteria are described in the protocol (Data Supplement).

Patients received a starting dose of 600 mg oral rucaparib twice daily. Dose reductions, in decrements of 100 mg, were permitted for grade ≥ 3 or persistent grade 2 treatment-emergent adverse events (TEAEs).

The study was approved by national or local institutional review boards and performed in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines of the International Council for Harmonisation. Patients provided written informed consent before participation.

Patient Populations
Efficacy and safety populations included patients with a deleterious BRCA alteration identified before enrollment who received ≥ 1 dose of rucaparib 600 mg. Patients with a non-BRCA DDR gene alteration (without a deleterious BRCA alteration) were not part of this analysis (data reported previously).
or BRCA2, germline or somatic alteration, zygosity, and alteration types).

Safety was assessed by monitoring TEAEs, vital signs, laboratory testing, and physical examination. Dose intensity was calculated as the actual dose received divided by the first dose.

**Procedures**

Patients were screened for the presence of a deleterious somatic or germline alteration in BRCA1, BRCA2, or other DDR gene through central genomic testing of plasma or tumor tissue (archival or contemporaneous), or through local testing. Central testing was performed by Foundation Medicine.12,13 Germline testing was performed by Color Genomics.14,15 Additional details are provided in the Data Supplement.

Patients received rucaparib until radiographic disease progression (soft tissue or bone lesion) assessed by investigator per modified RECIST/PCWG3 criteria, unequivocal clinical progression, unacceptable toxicity or inability to tolerate additional treatment, loss to follow up, or withdrawal of consent. Tumor assessments by computed tomography or magnetic resonance imaging and bone scans were performed during screening, every 8 weeks for 24 weeks, then every 12 weeks thereafter. PSA assessments were conducted every 4 weeks.

TEAEs were monitored from the first dose of rucaparib until 28 days after the last dose. TEAEs were coded using Medical Dictionary for Drug Regulatory Activities v20.116 and graded according to the National Cancer Institute Common Terminology Criteria for Adverse Events v4.03.17 Additional details are provided in the Data Supplement.

**Statistical Analyses**

A Simon 2-stage design was used as a futility rule. Assuming a total sample size of 83 patients in the IRR-evaluable population, characteristics of the design included a null hypothesis of ORR = 20%, type I error rate of 5% (one sided), and 90% power when the true response rate is 35%. However, the final number of patients was to be defined based on regulatory considerations. Confirmed ORR and PSA responses were summarized descriptively with frequencies and 95% CIs (Clopper-Pearson).

DOR, time to PSA progression, and rPFS were summarized using Kaplan-Meier methodology. DOR was defined as the time from the date of the first confirmed response to the date progression was first documented plus 1 day. Time to PSA progression was defined as the time from the first rucaparib dose to the date of an increase ≥ 25% and absolute increase of ≥ 2 ng/mL above the nadir in PSA plus 1 day. PSA increases must have been confirmed by a consecutive assessment conducted ≥ 3 weeks later; early rises (< 12 weeks) were not considered in determining PSA progression.18 rPFS was defined as the time from first rucaparib dose to the date of first objective evidence of radiographic progression or death due to any cause, whichever occurred first.

Best change from baseline in the sum of the diameter of target lesions and PSA were summarized graphically. Statistical analyses were performed using SAS v9.4 (SAS Institute, Cary, NC).

**RESULTS**

**Efficacy**

The overall efficacy population included 115 patients who received ≥ 1 dose of rucaparib and had a deleterious BRCA alteration (BRCA1 [n = 13], BRCA2 [n = 102], germline [n = 44], and somatic [n = 71]; Data Supplement). Baseline patient demographics and disease characteristics are shown in Table 1 and the Data Supplement. At baseline, 62 patients had measurable disease per blinded IRR assessment (IRR-evaluable population) and 65 patients had measurable disease per investigator assessment (investigator-evaluable population); 57 patients had measurable disease by both IRR and investigator. Genomic characteristics (eg, germline/somatic status, zygosity, and alteration type) and co-occurring alterations are shown in the Data Supplement.

Median treatment duration for the overall efficacy population was 8.1 months (range, 0.5-30.3 months), and the median follow up was 17.1 months (range, 7.6-31.5 months). As of December 23, 2019, 29 patients (25.2%) remained on treatment.

The confirmed ORR for the IRR-evaluable population was 43.5% (95% CI, 31.0% to 56.7%; 27 of 62), and the confirmed ORR for the investigator-evaluable population was 50.8% (95% CI, 38.1% to 63.4%; 33 of 65; Table 2). Most patients had a best response of stable disease or better (88.7% in the IRR-evaluable population and 89.2% in the investigator-evaluable population). Eight patients had a confirmed complete response in soft-tissue disease per blinded IRR and/or investigator assessment. Among these patients, one had hepatic metastases, one had a non-nodal pelvic mass, and 6 had nodal-only disease at baseline per IRR; one had a BRCA1 alteration and seven had a BRCA2 alteration. Among responders in the IRR-evaluable population, 19 of 27 patients (70.4%) demonstrated a response by the first tumor assessment (week 8; Data Supplement). Median DOR in the IRR-evaluable population was not reached (NR; 95% CI, 6.4 months to NR; Data Supplement) and was 6.4 months in the investigator-evaluable population (95% CI, 5.5 to 11.7 months; Data Supplement). Within the IRR-evaluable population, 15 of 27 patients with a confirmed objective response had a DOR ≥ 6 months; three patients with ongoing responses were followed for < 6 months from the onset of response. In the IRR- and investigator-evaluable populations, 64.5% and 63.1% of patients demonstrated...
TABLE 1. Baseline Patient and Disease Characteristics in TRITON2 Patients With a BRCA Alteration

| Characteristic | Value (N = 115) |
|---------------|----------------|
| Median age, years (range) | 72.0 (50.88) |
| Race, No. (%) | |
| White | 85 (73.9) |
| Black or African American | 7 (6.1) |
| Other | 2 (1.7) |
| Unknown | 21 (18.3) |
| ECOG PS, No. (%) | |
| 0 | 37 (32.2) |
| 1 | 76 (66.1) |
| ≥ 2 | 2 (1.7) |
| Median baseline PSA, ng/mL (range) | 61.1 (0.0-4,782.0) |
| Median baseline ALP, U/L (range) | 98.0 (34.0-5,490.0) |
| Median baseline albumin, g/L (range) | 39.0 (24.0-49.0) |
| Median baseline LDH, U/L (range) | 240 (84.0-2,058.0) |
| Gleason score ≥ 8 at diagnosis, No. (%) | 77 (67.0) |
| Median No. of prior CRPC therapies (range) | 2 (1-8) |
| Prior therapies, No. (%) | |
| Next-generation AR-directed therapy | |
| Abiraterone acetate | 74 (64.3) |
| Enzalutamide | 82 (71.3) |
| Apalutamide | 3 (2.6) |
| Darolutamide | 0 |
| ≥ 2 AR-directed therapies | 42 (36.5) |
| Docetaxel (castration sensitive) | 17 (14.8) |
| Docetaxel (castration resistant) | 108 (93.9) |
| Cabazitaxel | 8 (7.0) |
| Sipuleucel-T | 12 (10.4) |
| Radium-223 | 14 (12.2) |
| Measurable disease (per blinded IRR), No. (%) | |
| Measurable disease | 62 (53.9) |
| Only measurable nodal disease | 41 (66.1) |
| Measurable visceral ± nodal disease | 21 (33.9) |
| Nonmeasurable disease | 53 (46.1) |
| Bone-only disease | 36 (67.9) |
| Other nonmeasurable disease | 17 (32.1) |
| Site of metastases (per blinded IRR), No. (%) | |
| Hepatic | 14 (12.2) |
| Lung | 14 (12.2) |
| ≥ 10 bone lesions (per blinded IRR), No. (%) | 54 (47.0) |

NOTE. Visit cutoff date: December 23, 2019.

Abbreviations: AR, androgen receptor; CRPC, castration-resistant prostate cancer; ECOG PS, Eastern Cooperative Oncology Group Performance Status; LDH, lactate dehydrogenase; IRR, independent radiology review; PSA, prostate-specific antigen.

1Does not include luteinizing hormone–releasing hormone analogs, first-generation antiandrogens, hormones, corticosteroids, bone-targeted agents, hematopoietic growth factors, or docetaxel administered for castration-sensitive disease (per protocol, docetaxel in this setting was not counted toward the number of prior metastatic CRPC therapies for study eligibility).

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In the overall efficacy population, 63 of 115 patients had a confirmed PSA response (54.8%; 95% CI, 45.2% to 64.1%; Table 2), and median time to PSA response was 1.9 months (95% CI, 1.3 to 1.9 months). Median time to PSA progression was 6.5 months (95% CI, 5.9 to 7.8 months; Data Supplement). The majority of patients (60.0%) demonstrated a single best PSA reduction ≥ 50% from baseline (Fig 1B).

Although ORRs were similar across subgroups based on baseline clinical or genomic characteristics, some differences were observed with PSA response rates (Fig 2 and Data Supplement). PSA responses were observed among patients with BRCA1 (15.4%; 2 of 13 patients) or monoallelic alterations (11.1%; 1 of 9 patients), albeit at a lower rate than the overall population; however, the number of patients in these subgroups was low. Conversely, a higher proportion of patients with biallelic alterations (75.0%; 27 of 36 patients) and/or homozygous loss (81.0%; 17 of 21 patients) experienced a PSA response compared with the overall population.

Median rPFS was 9.0 months (95% CI, 8.3 to 13.5 months) per blinded IRR assessment (Fig 3) and 8.5 months (95% CI, 8.1 to 11.2 months) per investigator assessment (Data Supplement). Although OS data were not yet mature at the time of the analysis (41% of events reported), the Kaplan–Meier estimate of 12-month OS was 73.0% (95% CI, 62.9% to 80.7%).

Safety

The safety population included 115 patients who received one or more dose of rucaparib. As of the visit cutoff, median treatment duration was 6.5 months (range, 0.5-26.7 months), and median follow up was 13.7 months (range, 4.2-28.2 months). Mean dose intensity was 0.88 (standard deviation, 0.15).

A TEAE of any grade occurred in 114 patients (99.1%), and a grade ≥ 3 TEAE was reported in 70 patients (60.9%; Table 3 and Data Supplement). The most frequent TEAEs (any grade) were asthenia/fatigue (61.7%), nausea (52.2%), and anemia/decreased hemoglobin (43.5%). The most frequent grade ≥ 3 TEAE was anemia/decreased hemoglobin (25.2%). Overall, 32 patients (27.8%) received ≥ 1 transfusion of packed RBCs. Other TEAEs of interest are presented in the Data Supplement.

ALT/AST elevations (any grade) were observed in 38 patients (33.0%)—the majority were grade 1 or 2. These typically occurred within the first 4 weeks of rucaparib treatment, normalized over time with continued treatment, and were not associated with abnormal increases in bilirubin or other criteria for drug-induced hepatotoxicity (Data Supplement). Increases in creatinine—predominantly grade 1 or 2—were also observed with the initiation of
rucaparib and typically stabilized by the third week (Data Supplement) without being accompanied by a change in blood urea nitrogen. Decreased phosphate was observed in 75 (67.6%) of 111 patients (Data Supplement); however, most patients had low baseline phosphate and/or were receiving concomitant medications associated with decreased phosphate (eg, antacids or bone-targeting agents).

Treatment interruption due to a TEAE occurred in 65 patients (56.5%), most commonly because of anemia/decreased hemoglobin (21.7%), thrombocytopenia/decreased platelets (13.9%), and asthenia/fatigue (9.6%). Dose reduction due to a TEAE occurred in 47 patients (40.9%), most commonly due to anemia/decreased hemoglobin (13.0%), asthenia/fatigue (9.6%), and thrombocytopenia/decreased platelets (7.0%). Overall, 73 patients (63.5%) had either a treatment interruption or dose reduction due to a TEAE.

Nine patients (7.8%) discontinued because of a TEAE, one patient each due to acute respiratory distress syndrome; ALT/AST increased; anemia; balance disorder; cardiac failure; decreased appetite, fatigue, and weight decreased; leukopenia and neutropenia; pneumonia; and prolonged QT. There were three deaths as a result of TEAEs, including one each from pneumonia and prolonged QT, considered unrelated to rucaparib, and one from acute respiratory distress syndrome, considered related to rucaparib by the investigator (additional details are in the Data Supplement).

**DISCUSSION**

Rucaparib treatment demonstrated significant clinical activity in men with mCRPC associated with a **BRCA** alteration, resulting in meaningful radiographic and PSA responses, including complete responses in soft-tissue disease, with a manageable safety and tolerability profile. Based on these results, rucaparib was granted accelerated approval by the US Food and Drug Administration for the treatment of men with a deleterious **BRCA** mutation (germline and/or somatic) associated mCRPC who have been treated with AR-directed therapy and a taxane-based chemotherapy.19

Baseline characteristics in the efficacy population were as expected for a population of patients with mCRPC receiving third-line or later treatment. More than one third of patients were treated with ≥ 2 next-generation AR-directed therapies.

For men with mCRPC with disease progression after next-generation AR-directed therapy and taxane-based chemotherapy, response to previously approved therapies has been historically poor, with confirmed ORRs ranging from 8%-15% and PSA response (≥ 50% decrease) rates ranging from 8%-39%.20-22 Of note, these data come from studies of men with mCRPC who were not selected based on a potential predictive biomarker (eg, **BRCA** alteration).

Data from TRITON2 illustrate the importance of genomic screening to identify men who may benefit from treatment with a PARP inhibitor.2,4,23 TRITON2 patients with mCRPC associated with a **BRCA** alteration who received rucaparib had substantially higher RECIST and PSA response rates than those typically observed with other treatments in an unselected population, including responses in clinically relevant subgroups. For example, although the numbers are small, the confirmed ORR of 46.2% (6 of 13) among patients with hepatic metastases (Fig 2) is potentially clinically important as hepatic metastases are known to be an indicator of poor prognosis in mCRPC.24

RECIST and PSA responses were observed in patients with **BRCA2** and **BRCA1** alterations, germline and somatic alterations, and all categories of alteration zygosity. Although the rates of confirmed PSA response were higher in **BRCA2** versus **BRCA1** and biallelic versus monoallelic subgroups, the numbers of patients in the **BRCA1** and monoallelic subgroups were small. Due to the limited number of tissue

### TABLE 2. Rate of Response to Rucaparib Treatment

| Response                        | Investigator-Evaluable Population (n = 65) | IRR-Evaluable Population (n = 62) |
|--------------------------------|-------------------------------------------|----------------------------------|
| Confirmed ORR, No. (%; 95% CI)  | 33 (50.8; 38.1 to 63.4)                   | 27 (43.5; 31.0 to 56.7)          |
| Complete response               | 4 (6.2)                                   | 7 (11.3)                         |
| Partial response                | 29 (44.6)                                 | 20 (32.3)                        |
| Stable disease                  | 25 (38.5)                                 | 28 (45.2)                        |
| Progressive disease             | 6 (9.2)                                   | 6 (9.7)                          |
| Not evaluable                   | 1 (1.5)                                   | 1 (1.6)                          |
| **Overall Efficacy Population** |                                           | 63 (54.8; 45.2 to 64.1)          |

**NOTE.** Data presented as No. (%) unless otherwise indicated. Visit cutoff date: December 23, 2019. Abbreviations: IRR, independent radiology review; ORR, objective response rate; PSA, prostate-specific antigen.

*Per modified RECIST/Prostate Cancer Clinical Trials Working Group 3 criteria.
samples received (66 of 115 patients [57.4%]), alteration zygosity could only be determined in 45 patients (39.1%) based on the availability of sufficiently high-quality next-generation sequencing data from tissue for copy-number analysis. Importantly, despite the relatively small numbers, confirmed PSA responses were observed in all molecular and clinical subgroups examined.

Our results are consistent with those from other studies demonstrating the clinical activity of PARP inhibitors (olaparib, niraparib, and talazoparib) in patients with mCRPC and a BRCA alteration who received prior AR-directed therapy. For example, in the PROfound study, patients with a BRCA alteration and prior AR-directed therapy showed improved rPFS with olaparib versus abiraterone acetate or enzalutamide (median, 9.8 vs 3.0 months; hazard ratio, 0.22; 95% CI, 0.15 to 0.32). Although there are important differences in the study designs for these trials (eg, differences in the method of determining
genomic alteration, type of alterations eligible for enrollment, assessment of response by RECIST, PSA decrease, or a composite that includes changes in circulating tumor cell count), these studies reinforce the potential benefit of PARP inhibitors in patients with mCRPC associated with a BRCA alteration.

The safety profile of rucaparib in patients with mCRPC was consistent with that observed in prior studies conducted in patients with ovarian cancer and other solid tumor types, as well as in studies of men with mCRPC who received other PARP inhibitors, with asthenia/fatigue, GI adverse effects, and myelosuppression among the most common TEAEs reported. Similar to studies of rucaparib in ovarian cancer, elevations in ALT, AST, and creatinine were commonly reported; however, these laboratory abnormalities were not associated with liver or kidney toxicity. Elevated creatinine has been observed with multiple PARP inhibitors and is thought to be due to inhibition of renal transporters (eg, MATE-1, MATE2-K, OCT2) rather than a direct impact on renal function.

Although there have been reports of fatal pneumonitis with other PARP inhibitors, interstitial lung disease has not been identified as a potential risk from rucaparib treatment when evaluated across studies in multiple tumor types; the majority of cases had an alternative etiology, and most resolved with continued rucaparib treatment or after dose interruption with negative rechallenge.

A strength of our analysis is the inclusion of both IRR- and investigator-assessed end points; a substantial proportion of patients achieved a confirmed radiographic response with rucaparib treatment via both assessment methods. Discordance in assessment by independent reviewers and investigators may be influenced by variations in lesion evaluation.

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selection or by historical/clinical information available to investigators but not the blinded IRR. One limitation of our analysis was that the DOR and rPFS results for the IRR-evaluable population were more heavily impacted by censoring. Per the protocol, radiographic assessments were to be continued until disease progression was observed by the investigator. If the investigator reported disease progression on a scan but the blinded IRR did not, the patient became censored in the IRR analysis. Additional limitations include the lack of a control arm and the immaturity of OS data. The phase III TRITON3 study (ClinicalTrials.gov identifier: NCT02975934) is ongoing to define the clinical benefit (eg, rPFS and OS) of rucaparib in an earlier disease setting among patients with mCRPC associated with a BRCA or ATM alteration who have progressed after one next-generation AR-directed therapy and who have not received taxane-based chemotherapy in the mCRPC setting. Rucaparib is being compared with physician’s choice of next-generation AR-directed therapy or docetaxel and will provide additional evidence of the effects of rucaparib treatment in men with mCRPC.

Altogether, results from the TRITON2 study demonstrate that rucaparib has meaningful antitumor activity and a manageable safety profile in patients with deleterious germline or somatic BRCA alteration, and support the use of rucaparib in this patient population.

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### TABLE 3. Most Commonly Reported TEAEs (N = 115)

| Individual TEAE (preferred terms) Occurring in ≥ 15% of Patients | Any Grade | Grade ≥ 3 |
|---------------------------------------------------------------|-----------|-----------|
| Asthenia/fatigue                                             | 71 (61.7) | 10 (8.7)  |
| Nausea                                                       | 60 (52.2) | 3 (2.6)   |
| Anemia/decreased hemoglobin                                  | 50 (43.5) | 29 (25.2) |
| ALT/AST increased                                            | 38 (33.0) | 6 (5.2)   |
| Decreased appetite                                           | 32 (27.8) | 2 (1.7)   |
| Constipation                                                 | 31 (27.0) | 1 (0.9)   |
| Thrombocytopenia/decreased platelets                         | 29 (25.2) | 11 (9.6)  |
| Vomiting                                                     | 25 (21.7) | 1 (0.9)   |
| Diarrhea                                                     | 23 (20.0) | 0         |
| Dizziness                                                    | 21 (18.3) | 0         |
| Blood creatinine increased                                   | 18 (15.7) | 1 (0.9)   |

NOTE. Data presented as No. (%). Visit cutoff date: September 13, 2019. TEAEs were graded according to National Cancer Institute-Common Terminology Criteria for Adverse Events version 4.03. There were no TEAEs of myelodysplastic syndrome or acute myeloid leukemia reported. Abbreviation: TEAE, treatment-emergent adverse event.
Rucaparib in mCRPC With a BRCA Gene Alteration

PRIOR PRESENTATION
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AUTHORS’ DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST AND DATA AVAILABILITY STATEMENT
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AUTHORS’ DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

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