Informed Prostate Cancer Support Group Inc.
"A 501 C 3 CORPORATION ID # 54-2141691"

June 2020 NEWSLETTER

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Wednesday, June 17,

Volume 13 Issue 06

Next Virtual Meeting: June 20th, starting at 10:00am PST Sharp:

Due to COVID-19, this meeting will only be available at https://ipcsg.org/live-stream
No in-person meetings at the Sanford Burnham Prebys Medical Discovery Institute will take place until further notice.

Dr. Irwin Goldstein, MD, Director of San Diego Sexual Medicine will speak about some of the negative side effects of prostate cancer treatment and their management, including urinary incontinence and erectile dysfunction. He will also discuss contemporary research in these areas.

Dr. Andrew Goldstein, PhD Professor-in-Residence of Urology, David Geffen School of Medicine at UCLA will explain how healthy prostate cells develop into cancer, and how prostate cancer cells develop resistance to therapy, which is critical for improving disease diagnosis and identifying new treatment options. He will present some of his laboratory's basic science research that will enable us to better understand prostate cancer biology and may lead to new approaches for therapy in the future.

- **For further Reading: https://ipcsg.blogspot.com/**
- **For Comments, Ideas and Questions,** email to Newsletter@ipcsg.org

May 2020 Informed Prostate Cancer Support Group Online Presentation

"ASCO GU" Updates in Prostate Cancer

Summary by Bill Lewis

Munveer Bhangoo, MD
Staff Physician / Medical Oncologist, Scripps MD Anderson Cancer Center
Clinical Focus: Genitourinary Malignancies
Research Interests: Clinical Genomics, Novel Therapeutics

Dr. Bhangoo's talk and this summary relate to presentations given at the recent ASCO (Amer. Soc. Clinical Oncology) GU (Genitourinary) conference in San Francisco, in January 2020.

1. Prostate Cancer Specific COVID-19 Updates – "Prostate Cancer in the COVID-19 Era."

A) No published evidence of increased risk of severity of coronavirus infection if on ADT (androgen deprivation therapy). No evidence of high risk of other viral infections such as influenza or hepatitis. Immune system not likely to be adversely impacted by ADT. However, chemotherapy can impair the immune system (i.e., causing neutropenia), and could increase the risk of greater coronavirus infection severity.

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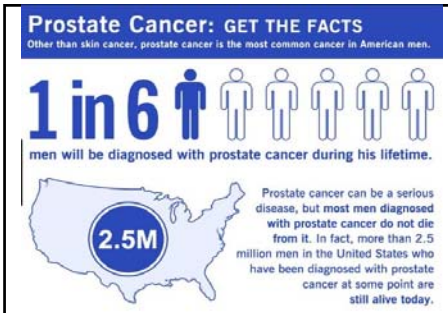

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a 501c3 non-profit organization - all positions are performed gratis

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NEWSLETTER
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PROSTATE CANCER—*2 WORDS, NOT A SENTENCE*

What We Are About

Our Group offers the complete spectrum of information on prevention and treatment. We provide a forum where you can get all your questions answered in one place by men that have lived through the experience. Prostate cancer is very personal. Our goal is to make you more aware of your options before you begin a treatment that has serious side effects that were not properly explained. Impotence, incontinence, and a high rate of recurrence are very common side effects and may be for life. Men who are newly diagnosed with PCa are often overwhelmed by the frightening magnitude of their condition. Networking with our members will help identify what options are best suited for your life style.

Be your own health manager!!

Meeting Video DVD's

DVD's of our meetings are usually available in our library for $10ea. Refer to the index available in the library. They can also be purchased through our website: http://ipcsg.org Click on the 'Purchase DVDs" tab. However since this meeting was not recorded at the speakers request, only the slides will be available for download.

The DVD of each meeting is available by the next meeting date.

From the Editor

Facilities for the meeting are not available due to the COVID-19 epidemic, so it is cancelled until further notice. We will continue to post and distribute the newsletter in the interim. Our speaker this month may be streamed and broadcast via the group web site. Alternate web based meeting approaches such as zoom have been suggested and we will notify you via the newsletter and web site if such becomes available.

Join the IPCSG TEAM

If you consider the IPCSG to be valuable in your cancer journey, realize that we need people to step up and HELP. Call **President** Lyle LaRosh @ 619-892-3888; or **Director** Gene Van Vleet @ 619-890-8447.

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B) No published data on the impact of COVID-19 on prostate cancer survivors. However, other co-existing medical conditions (e.g., hypertension, diabetes, respiratory disease, or heart disease) can increase the risk of severe disease, regardless of prostate cancer diagnosis.

C) A number of NCCN (National Comprehensive Cancer Network) guidelines have been issued during the pandemic. To Dr. Bhangoo, they seem "weighted" toward the experiences of doctors in the East where the surge of cases has been much higher than here in San Diego. The guidelines de-emphasized the importance of routine localized prostate cancer care during the pandemic, suggesting that minimal harm is expected with delays in care or treatment of 3-6 months especially when weighed against the risk of mortality of COVID-19. They strongly recommended telehealth vs. in-person visits, indicating that very low, low, favorable -intermediate risk disease should not undergo further staging / active surveillance / treatment until deemed safe, and that for non-metastatic disease, ADT initiation should be avoided for patients with PSA doubling time >9 months. After ADT initiation, offsite PSA/testosterone monitoring and telehealth visits should be used to avoid clinic exposure.

D) Additional NCCN guidelines:

•Asymptomatic unfavorable intermediate risk, high risk, very high risk prostate cancer patients can defer further staging and radical treatment until deemed safe.

•Neoadjuvant (first-step) ADT should be considered in unfavorable intermediate risk, high risk patients planning to receive RT (radiation therapy). This may be safely given for up to 4-6 months.

•Data from Johns Hopkins suggest delaying surgical treatment in unfavorable intermediate risk, high risk patients upwards of 6 months (since initial biopsy) will not negatively impact outcome.

•Consider use of 3-, 4-, 6-month ADT formulations vs. 1-month injections.

•The shortest safe external beam RT (EBRT) regimen should be used per NCCN guidelines.

•Consider deferring repeat imaging over time if PSA is declining and in the absence of symptoms, until risk of COVID-19 has resolved.

•Educate patients receiving chronic steroids (e.g., prednisone) that they may need stress-dose steroids if they become sick (i.e., are in threat of adrenal crisis).

•For patients with advanced disease: Consider growth factor support and consider a non-myelosuppressive regimen (i.e., treatment that does not stop or slow the growth of blood-forming cells in the bone marrow) when alternatives exist.

E) An enzyme called TMPRSS2 (a Type II transmembrane serine protease) is implicated in the spread of several viruses, e.g., influenza A, SARS-CoV, and MERS-CoV. Recent reports show that SARS-CoV-2 (the COVID-19 virus) also obtains cell entry through the action of this enzyme. TMPRSS2 is highly expressed in both localized and metastatic prostate cancers. Androgen-dependent regulation of TMPRSS2 expression (i.e., testosterone increases the population of TMPRSS2), which also occurs in the lungs, may explain the increased susceptibility of men to develop SARS. In a region in Italy with a lot of Covid-19, among 9000+ patients with confirmed disease in 68 hospitals, males developed more severe complications, were more frequently hospitalized, and had worse clinical outcomes than females. However, the overall incidence of the disease among the men of the region was only 0.2% of non-cancer men, and 0.3% of those with prostate cancer. Severity of disease was higher in men with any type of cancer, but no worse if the cancer was prostate cancer. The same study authors also found that among the few men on ADT, that being on ADT (androgen deprivation therapy) seemed to have some protective effect against the virus. The US Veterans Administration has begun research on monthly Firmagon injections in relation to COVID-19 susceptibility and severity.

2. New drug approvals: Lynparza has been granted priority review for HRR-mutated (see below) mCRPC (metastatic castrate-resistant prostate cancer), but the decision has not yet been issued. Rubraca (rucaparib) has been approved as monotherapy treatment for patients with BRCA1/2-mutant MCRPCa who have been treated with advanced ADT (Zytiga, Xtandi or the like) and a taxane-based chemotherapy. This is based on phase 2

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studies, so may be affected by the future outcomes of phase 3 studies.

3. Impact of Genetic and Genomic Testing in Prostate Cancer: Across the "landscape" of prostate cancer patient genomics, more than 70% of mCRPC patients have "actionable" genetic variations/defects. Some 60% have AR (androgen receptor) alterations, 40-60% have PI3K-AKT-mTOR alterations, 25% have DDR (DNA repair defects such as BRCA1 and BRCA2, which are the most common among many variations) and/or Cell Cycle Regulator (RB1/CDK) defects. A large variety of other defects are seen in smaller portions of the population.

In 2015, it was reported that, based on biopsies of mCRPC patients, 23% had DNA repair defects. In the PROFOUND study of over 4000 men, there were "alterations" in homologous recombination (a method of DNA repair; often abbreviated "HHR") genes in 28% of the men. Germline (inherited) mutations are much more commonly found in metastatic prostate cancer patients than in the general population, or even in "early" prostate cancer patients: 12% vs. 5% vs. 3%.

About 30-40% of men with germline mutations have no family history of cancer, but if a man develops metastatic PCa (prostate cancer), this should be considered a "whistle-blower" indication of a possible inherited cancer predisposition for the family. So the NCCN guidelines suggest that tumor genetic testing be considered.

The effects of gene defects on the prognosis for a PCa patient are not yet clear in the case of somatic mutations (i.e., those not inherited), but for some inherited defects such as BRCA2, the prognosis is clearly worse. Thus, the time from continuous ADT to the development of mCRPC tends to be shorter for those with inherited BRCA2 mutation, indicating they have "more aggressive" disease.

The concept of "Synthetic Lethality" is that if a patient has, for example, the BRCA2 mutation, this means that an important pathway of DNA repair is not available. If a drug is administered that blocks a significant alternate repair pathway, the cell is very much inhibited in its DNA repair capability by this "double whammy," and the cancer cell is much more likely to die. This is the concept behind "PARP" inhibitors. Four new drugs are being actively studied: Olaparib, Rucaparib, Talazoparib, and Niraparib. They seem to operate in the same way, and mainly differ in side effects, and in the particular populations studied so far. Best results have been obtained with BRCA2 patients, with less benefit seen with several of the other possible mutations that a patient could have.

At least with HHR gene alterations, essentially equivalent findings are obtained whether the primary tumor or a metastatic lesion is biopsied. However, the overall success rate in analyzing the DNA from tumor tissue samples is only about 70% (based on the PROFOUND and TRITON2 trial reports). Circulating tumor DNA in the blood may be analyzed for genetic alterations, which is especially helpful for metastases to the bones, from which tumor DNA is difficult to extract. However, the accuracy is limited by the fraction of DNA coming from tumors, which is understandably low in patients with a low "burden" of disease.

Germline testing (for inherited genetic mutations) is cheap and readily accessible (typically through a saliva test), but reportedly misses identifying 10-15% of patients who have mutations that qualify them for treatment with a PARP inhibitor. Somatic (tumor biopsy) testing typically costs $4-5,000, but detects most DNA repair defects. It doesn't definitively indicate whether the defects were inherited, so separate germline testing is advisable for family information if DNA repair defects that could be inherited are found.

Here are reasons that tumor profiling (tumor biopsy testing) may not identify some germline (inheritable) mutations: 1) Advanced disease may include large DNA segment deletions or chromosomal loss that can mask a point germline mutation. 2) Some tumor testing protocols deliberately filter out germline variants (so you would have to test separately for those). 3) The test you choose may not analyze for all disease-relevant DNA variations. 4) Similarly, some genes associated with inherited "cancer predisposition" may not be included in the panel of tests run by a particular testing company.

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In conclusion, many drugs are emerging for "precision medicine," to treat patients with various DNA mutations, including BRCA1/2 and other HHR (homologous recombination) genes, "PTEN" loss, CDK12, and dMMR/MSI-H genes. Drugs under study include Olaparib, Rucaparib, Niraparib, Talazoparib, Velaparib, checkpoint inhibitors, PARP inhibitors, Pembrolizumab, and PD1/PDL-1 therapy. Expect to hear more about these in the near future!

Question: Should I get genomic testing on becoming castrate resistant? Almost every patient with recurrent or metastatic disease should be getting genetic testing. If biopsy tissue is available, Dr. Bhangoo favors getting it tested. Turnaround time is faster than with germline testing, which at Scripps is done through a genetic counseling office and takes several months. Companies that do somatic (biopsy tissue) testing include Guardant Health, Foundation Medicine, and Caris Life Sciences. If tissue is not available, he would get the somatic testing done on a blood sample. [Note: other test companies are GenomeDx Biosciences, Intermountain Healthcare, Genomic Health, Trovagene, Varientyx, Invitae, Paradigm, and many others. See https://www.curematch.com/blog-posts/a-patients-guide-to-tumor-profiling/]

Can you discuss more about seeking prostate cancer care during the COVID-19 pandemic? Low- and favorable-intermediate-risk patients can delay treatment up to six months. For intermediate and high-risk patients, NCCN guidelines suggest weighing the pros and cons. Scripps is actively treating these patients. See also the NCCN guidelines above.

Dr. Bhangoo included slides from one other talk from the ASCO GU conference in the set that he sent to us, but there wasn't time available during the online presentation to discuss them. The talk was an introduction to Targeted Therapy in the context of Low-volume Metastatic Prostate Cancer, by Dr. Michael Morris, Prostate Cancer Section Head at the Memorial Sloan Kettering Cancer Center.

In this talk, Dr. Morris pointed out the imprecision of current definitions of low volume vs. high volume metastatic disease. He bluntly stated "our [doctors'] terminology is confusing, misleading and inconsistent." The inconsistency is in the way the quantity and location of metastases is figured into the designation of low vs. high volume disease. So you need to get past the simple label, and understand how much disease is where in your particular case.

One kind of targeted therapy is the use of drugs that target the androgen receptor. Dr. Morris referenced four studies that all showed a (similar) benefit in overall survival from adding Zytiga (abiratirone), Xtandi (enzalutamide) or Erleada (apalutamide) to standard ADT (Lupron, Firmagon, or the like). These studies are the Stampede, Latitude, Enzamet and Titan trials.

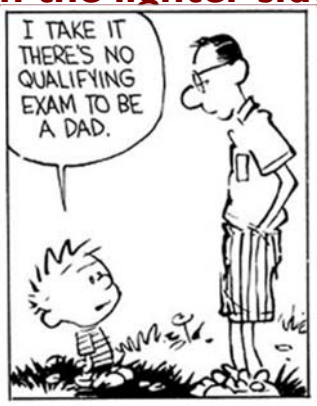
An intriguing report (Parker. Lancet, 2018) has been made that radiation therapy to the primary tumor in "low volume" metastatic disease improved overall survival. Three studies are working to confirm this: PEACE-1 (NCT01957436), SWOG 1802 ((NCT03678025) and G-RAMPP (NC02454543).

[Note that no mention seems to be made by Dr. Morris in connection with "treating the primary" that this has also been done with cryotherapy, nanoknife and various other alternatives to external beam radiation therapy, with the theory that the killed cancer cells in the primary tumor lead to an activation of the immune system -- which can then better attack remote tumors.]

There are also five trials testing radiation therapy to metastatic sites, while treating the primary tumor with systemic therapy, which are listed in the slides.

Dr. Morris concluded, "much more thought to and study of this field is warranted."

On the lighter side



Articles of Interest medpagetoday.com

Consider Genetic Testing in All Metastatic Prostate Cancers

by Charles Bankhead, Senior Editor, MedPage Today June 10, 2020

Oncology/Hematology > Prostate Cancer

— Expert panel recommends testing for men with family history, selective testing for others

All men with metastatic prostate cancer should consider testing for germline mutations, as should men with a family history of cancer suggesting hereditary cancer predisposition, an international consensus panel recommended.

The experts supported use of a large gene panel for germline testing, with priority given to *BRCA1/2* and DNA mismatch-repair (MMR) genes. Testing for additional genes should be guided by personal or family history. The panel also recommended testing for somatic mutations by means of next-generation sequencing and confirmatory germline testing for somatic mutations with priority to *BRCA2*. The recommendations apply to metastatic castration-resistant prostate cancer (CRPC) and non-CRPC.

For men with nonmetastatic disease considering active surveillance, the expert panel recommended prioritizing *BRCA2* and relying on family or personal history to guide testing for additional genes. For men without prostate cancer but with a family history of cancer, priority should be given to testing for *BRCA2* and *HOXB13*. Testing for *BRCA1*, *ATM*, and DNA MMR may be considered. The panel suggested multiple other clinical scenarios for selected testing.

The recommendations were published in the *Journal of Clinical Oncology*.

"Urologists who are on the front lines of the diagnosis of prostate cancer need to be familiar with these rapidly evolving genetic testing recommendations," consensus conference co-chair Leonard G. Gomella, MD, of Jefferson Health in Philadelphia, said in a statement. "This includes the proper ordering of prostate cancer gene panel testing and the utilization of appropriate genetic counseling."

Germline testing will be central to the decision to prescribe a PARP inhibitor, in light of the recent FDA approvals of rucaparib (Rubraca) and olaparib (Lynparza) for men with metastatic CRPC associated with a deleterious *BRCA* mutation or homologous recombination repair deficiency, according to conference co-chair Veda N. Giri, MD, at Jefferson Health's Sidney Kimmel Cancer Center.

"As it is not feasible that all men be referred to genetic counseling, it is imperative that providers offering genetic testing are aware of best practice recommendations so that they can help their patients make an informed decision," said Giri. "These recommendations will be helpful to urologists and oncologists when thinking of offering genetic testing to men with prostate cancer across the stage spectrum."

From 12%-17% of men with metastatic prostate cancer harbor germline mutations, primarily in DNA repair genes (*BRCA1/2*, *CHEK2*, *ATM*, *PALB2*, and DNA MMR genes), as do about 7% of men with localized prostate cancer. Increasingly, determination of a patient's mutation status has the potential to inform decision making about treatment options, referral to clinical trials, and active surveillance for men with localized disease.

As germline testing for prostate cancer has expanded, challenges have arisen with regard to expanded options for multigene panels, lack of information about optimal use of the panels, variability in testing guidelines, and a shortage of genetic services.

"There is a need for clarity on panel choice and priority genes to test in men with metastatic PCA [prostate cancer], nonmetastatic PCA, and men at high risk for PCA," Giri, Gomella, and colleagues noted.

Convened in October 2019, the Philadelphia Prostate Cancer Consensus Conference followed a 2017 conference to examine the role of genetic testing for hereditary prostate cancer. The 2017 meeting resulted in published recommendations for multigene testing and genetic consultation for prostate cancer risk. Participants in the 2019 conference took on the task of developing a "genetic implementation framework" for germline testing in prostate cancer.

In addition to the recommendations for testing in metastatic prostate cancer and men with a family history of cancer, the consensus panel recommended that men who are *BRCA2* carriers should begin prostate cancer screening at age 40 or 10 years before the youngest prostate cancer diagnosis in a family. Early onset of screening might also be considered for men who are carriers of *HOXB13*, *BRCA1*, and *ATM*.

The panel also endorsed collaborative evaluation models for healthcare and genetic service providers to address the shortage of genetic counselors. The framework covers pretest informed consent, posttest discussion, cascade testing, and technology-based approaches.

Charles Bankhead is senior editor for oncology and also covers urology, dermatology, and ophthalmology. He joined Medpage Today in 2007. Follow

nature.com

Testosterone therapy does not increase the risks of prostate cancer recurrence or death after definitive treatment for localized disease

Brent S. Rose

DOI: https://doi.org/10.1038/s41391-020-0241-3

Abstract

Background

The safety of testosterone therapy (TT) after definitive treatment for localized prostate cancer remains undefined. We analyzed the risks of biochemical recurrence and mortality in men receiving TT after treatment for localized prostate cancer.

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Methods

Cohort analysis using the national US Veterans Affairs Informatics and Computing Infrastructure. We identified 69,984 patients with localized prostate cancer diagnosed from 2001 to 2015 treated with surgery or radiation. We coded receipt of TT after treatment as a time-dependent covariate; used the National Death Index to identify cause of death; and defined biochemical recurrence as PSA > 0.2 ng/mL after surgery and nadir + 2 ng/mL after radiation. We analyzed recurrence and mortality using cumulative incidence curves, Fine–Gray competing risk regression, and Cox regression.

Results

This cohort included 28,651 surgery patients and 41,333 radiation patients, of whom 469 (1.64%) and 543 (1.31%), respectively, received TT with a median follow-up of 6.95 years. Comparing testosterone users to nonusers, there were no between-group differences in biochemical recurrence, prostate cancer-specific mortality, or overall mortality after surgery [hazard ratios (HR): 1.07; HR: 0.72 ($p = 0.43$); and HR: 1.11 ($p = 0.43$), respectively] or radiation [HR: 1.07; HR: 1.02 ($p = 0.95$); and HR: 1.02 ($p = 0.86$), respectively]. Limitations included lack of detailed data on TT duration and serum testosterone concentrations.

Conclusions

In this multi-ethnic national cohort, TT did not increase the risks of biochemical recurrence or prostate cancer-specific or overall mortality after surgery or radiation. These data suggest that TT is safe in appropriate men after definitive treatment of localized prostate cancer.

nejm.org

Oral Relugolix for Androgen-Deprivation Therapy in Advanced Prostate Cancer

Bertrand Tombal

Abstract

Background

Injectable luteinizing hormone–releasing hormone agonists (e.g., leuprolide) are the standard agents for achieving androgen deprivation for prostate cancer despite the initial testosterone surge and delay in therapeutic effect. The efficacy and safety of relugolix, an oral gonadotropin-releasing hormone antagonist, as compared with those of leuprolide are not known.

Methods

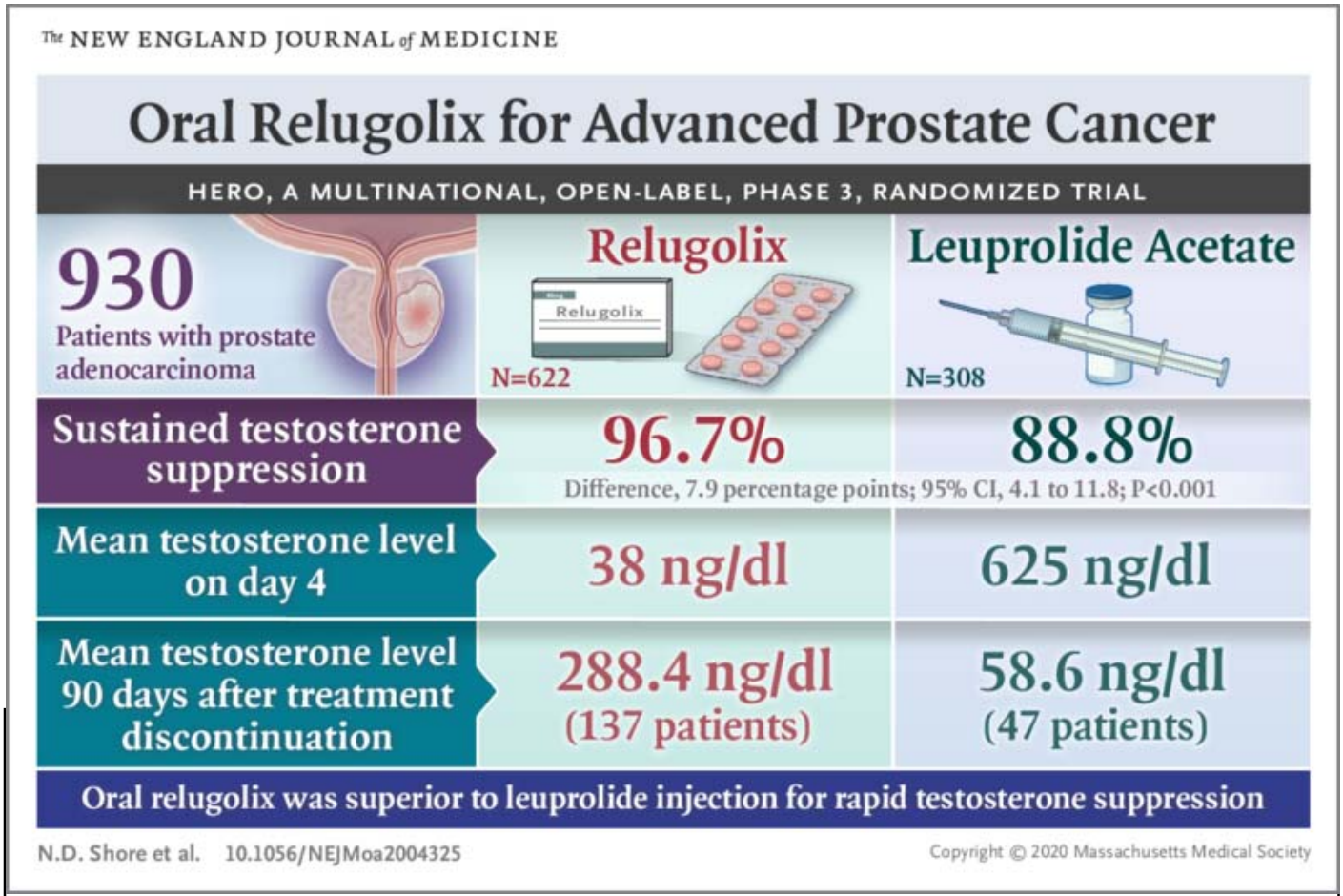
In this phase 3 trial, we randomly assigned patients with advanced prostate cancer, in a 2:1 ratio, to receive relugolix (120 mg orally once daily) or leuprolide (injections every 3 months) for 48 weeks. The primary end point was sustained testosterone suppression to castrate levels (<50 ng per deciliter) through 48 weeks. Secondary end points included noninferiority with respect to the primary end point, castrate levels of testosterone on day 4, and profound castrate levels (<20 ng per deciliter) on day 15. Testosterone recovery was evaluated in a subgroup of patients.

Results

A total of 622 patients received relugolix and 308 received leuprolide. Of men who received relugolix, 96.7% (95% confidence interval [CI], 94.9 to 97.9) maintained castration through 48 weeks, as compared with 88.8% (95% CI, 84.6 to 91.8) of men receiving leuprolide. The difference of 7.9 percentage points (95% CI, 4.1 to 11.8) showed noninferiority and superiority of relugolix (P<0.001 for superiority). All other key secondary end points showed superiority of relugolix over leuprolide (P<0.001). The percentage of patients with castrate levels of testosterone on day 4 was 56.0% with relugolix and 0% with leuprolide. In the subgroup of 184 patients followed for testosterone recovery, the mean testosterone levels 90 days after treatment discontinuation were 288.4 ng per deciliter in the relugolix group and 58.6 ng per deciliter in the leuprolide group. Among all the patients, the incidence of major adverse cardiovascular events was 2.9% in the relugolix group and 6.2% in the leuprolide group (hazard ratio, 0.46; 95% CI, 0.24 to 0.88).

Conclusions

In this trial involving men with advanced prostate cancer, relugolix achieved rapid, sustained suppression of testosterone levels that was superior to that with leuprolide, with a 54% lower risk of major adverse cardiovascular events. (Funded by Myovant



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Sciences; HERO ClinicalTrials.gov number, NCT03085095. opens in new tab.)

nejm.org

Olaparib for Metastatic Castration-Resistant Prostate Cancer

Maha Hussain

Abstract

Background

Multiple loss-of-function alterations in genes that are involved in DNA repair, including homologous recombination repair, are associated with response to poly(adenosine diphosphate–ribose) polymerase (PARP) inhibition in patients with prostate and other cancers.

Methods

We conducted a randomized, open-label, phase 3 trial evaluating the PARP inhibitor olaparib in men with metastatic castration-resistant prostate cancer who had disease progression while receiving a new hormonal agent (e.g., enzalutamide or abiraterone). All the men had a qualifying alteration in prespecified genes with a direct or indirect role in homologous recombination repair. Cohort A (245 patients) had at least one alteration in *BRCA1*, *BRCA2*, or *ATM*; cohort B (142 patients) had alterations in any of 12 other prespecified genes, prospectively and centrally determined from tumor tissue. Patients were randomly assigned (in a 2:1 ratio) to receive olaparib or the physician's choice of enzalutamide or abiraterone (control). The primary end point was imaging-based progression-free survival in cohort A according to blinded independent central review.

Results

In cohort A, imaging-based progression-free survival was significantly longer in the olaparib group

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than in the control group (median, 7.4 months vs. 3.6 months; hazard ratio for progression or death, 0.34; 95% confidence interval, 0.25 to 0.47; P<0.001); a significant benefit was also observed with respect to the confirmed objective response rate and the time to pain progression. The median overall survival in cohort A was 18.5 months in the olaparib group and 15.1 months in the control group; 81% of the patients in the control group who had progression crossed over to receive olaparib. A significant benefit for olaparib was also seen for imaging-based progression-free survival in the overall population (cohorts A and B). Anemia and nausea were the main toxic effects in patients who received olaparib.

Conclusions

In men with metastatic castration-resistant prostate cancer who had disease progression while receiving enzalutamide or abiraterone and who had alterations in genes with a role in homologous recombination repair, olaparib was associated with longer progression-free survival and better measures of response and patient-reported end points than either enzalutamide or abiraterone. (Funded by AstraZeneca and Merck Sharp & Dohme; PROfound ClinicalTrials.gov number, NCT02987543. opens in new tab.)

More on the Lighter Side

"I TOLD you, I saved a ton by getting your meds from an online pharmacy in the Amazon. Now turn around."

"I can never understand your doctor's handwriting. What are your symptoms?"

"It was your medications side effects that caused those special effects."

NETWORKING

Please help us in our outreach efforts. Our speakers bureau consisting of Lyle LaRosh, and Gene Van Vleet are available to speak to organizations of which you might be a member. Contact Gene 619-890-8447 or gene@ipcsg.org to coordinate.

Member and Director, John Tassi is the webmaster of our website and welcomes any suggestions to make our website simple and easy to navigate. Check out the Personal Experiences page and send us your story. Go to: https://ipcsg.org/personal-experience

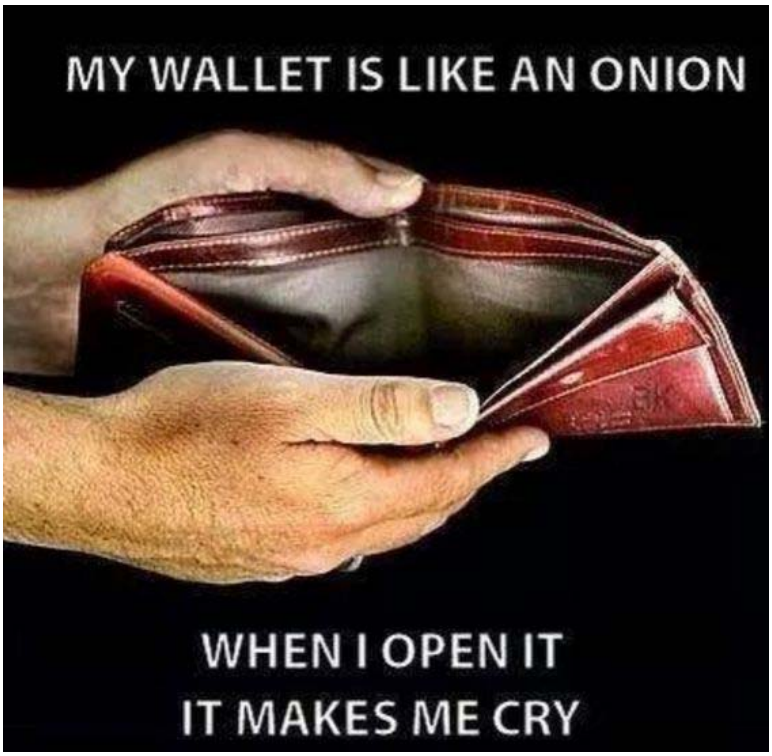
Our brochure provides the group philosophy and explains our goals. Copies may be obtained by mail or email on request. Please pass them along to friends and contacts.

Ads about our Group are in the Union Tribune the week prior to a meeting. Watch for them.

FINANCES

We want to thank those of you who have made special donations to IPCSG. Remember that your gifts are tax deductible because we are a 501(c)(3) non-profit organization.

We again are reminding our members and friends to consider giving a large financial contribution to the IPCSG. This can include estate giving as well as giving in memory of a loved one. You can also have a distribution from your IRA made to our account. We need your support. We will, in turn, make contributions from our group to Prostate Cancer researchers and other groups as appropriate for a non-profit organization. Our group ID number is 54-2141691. Corporate donors are welcome!



While our monthly meetings are suspended, we still have continuing needs, but no monthly collection. If you have the internet you can contribute easily by going to our website, http://ipcsg.org and clicking on "Donate" Follow the instructions on that page. OR just mail a check to: IPCSG, P. O. Box 420142, San Diego CA 92142