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

Saturday, December 23,

NOVEMBER 2023 NEWSLETTER

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Phone: 619-890-8447 Web: http://ipcsg.org

Volume 16 Issue 11

Next Meeting Saturday, November18, 2023 IPCSG—10:00am—Noon PDT.

- **Sam Denmeade, M.D. - Bipolar Androgen Therapy (BAT)**
- Dr. Denmeade is the R. Dale Hughes Professor of Oncology and Urology at the Johns Hopkins University School of Medicine and Director of the Genitourinary Oncology Division for the Johns Hopkins Kimmel Cancer Center. His areas of clinical expertise include bladder cancer, kidney cancer, prostate cancer and testicular cancer. He will be discussing Bipolar Androgen Therapy (BAT), a new endocrinologic treatment for castration-resistant prostate cancer (CRPC). BAT is a treatment in which testosterone levels are oscillated between low and high levels in order to prevent the adaptation of prostate cancer cells to a low-androgen environment.
- As always, spouses/partners and caregivers are welcome and encouraged to attend!
- *After the meeting a light lunch will be served in the foyer outside the meeting room*
- **For links to further Reading: https://ipcsg.blogspot.com/ (includes member suggested links)**
- **If you have Comments, Ideas or Questions,** email Newsletter@ipcsg.org
- **For more information, please send email to bill@ipcsg.org or call Bill at (619) 591-8670 or Gene at (619) 890-8447**

Novel Therapeutic Strategies for Patients with Advanced Prostate Cancer

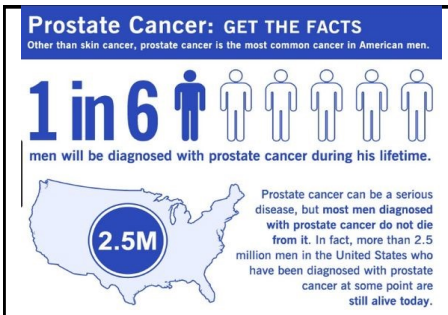
October 2023 IPCSG Presentation - Summary by Bill Lewis

Rana R. McKay, MD, is a board-certified medical oncologist who specializes in treating people with urogenital cancers, including bladder, kidney, prostate and testicular cancer. She leads a multi-disciplinary prostate cancer clinic focused on delivering advanced cancer care through a coordinated team approach.

Dr. McKay highlighted recent FDA-approved treatments for **advanced prostate cancer.** She also discussed current novel treatments that are in development and clinical trials of the next generation treatments for prostate cancer.

Hormone suppression, "ADT" (androgen deprivation therapy), has been the mainstay of prostate cancer treatment for many years – up until 2015, when it was reported that early use of chemotherapy pro-

(Continued on page 3)



Organization

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

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NEWSLETTER
Table of Contents

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.

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** Bill Lewis @ (619) 591-8670 "bill@ipcsg.org"; or **Director** Gene Van Vleet @ 619-890-8447.

From the Editor

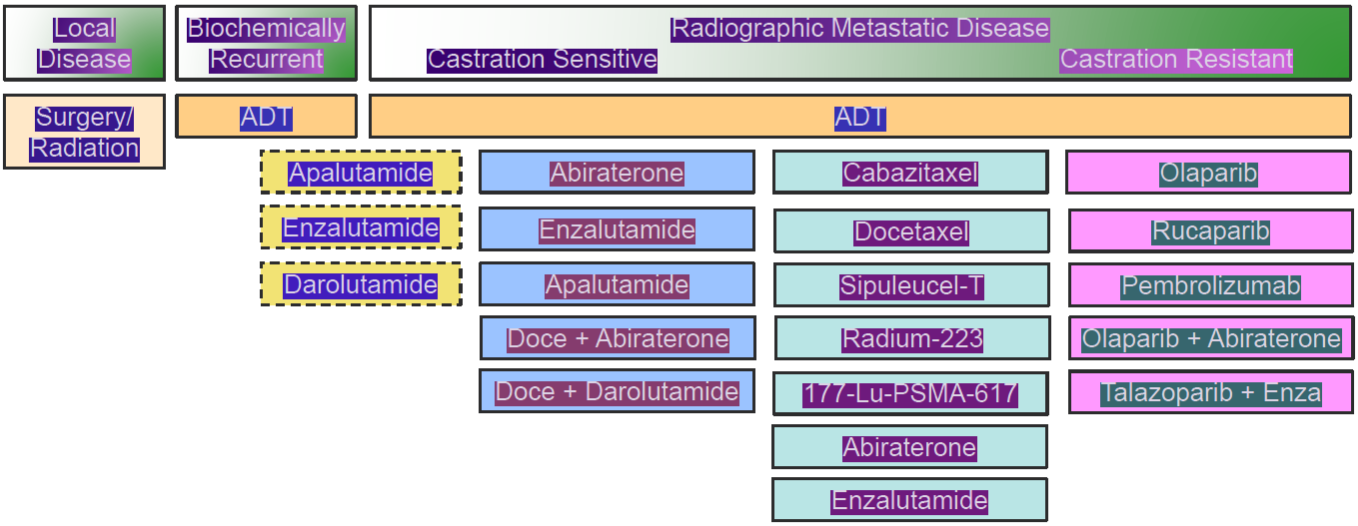
In this issue:

Bill Lewis managed to summarize Dr. McKay's stimulating presentation from the last meeting. For further articles see the blog at https://ipcsg.blogspot.com/ . Some apparently important optimistic items of interest this month:

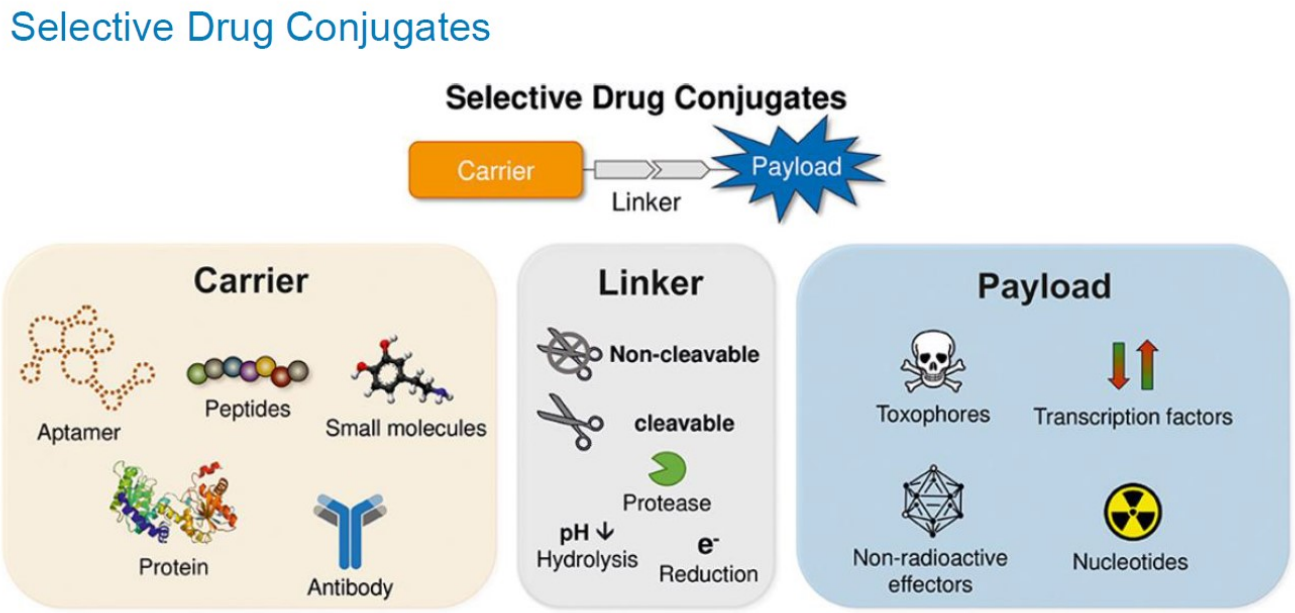
1. *Personalized dosing improves outcomes for men with metastatic castration-resistant prostate cancer—tailoring Lu177*
2. *.Novartis confirms unconstrained supply for Pluvicto® and continues to significantly expand the number of treatment centers—shortage over.*
3. "Mind-Blowing" Cancer Discovery – Common Chemotherapy Drugs Don't Work Like Doctors Thought—may lead to breakthrough in Chemo effectiveness.
4. *Docetaxel radiosensitizes castration-resistant prostate cancer by downregulating CAV-1: International Journal of Radiation Biology—using chemo with radiation may improve effectiveness of both.*
5. Real-world evidence of triplet therapy in metastatic hormone-sensitive prostate cancer - an Austrian multicenter study | medRxiv—ADT + CHEMO + X OR Z improves odds.

(Continued from page 1)

longed the lives of men with metastatic disease. Likewise, early use of Zytiga (abiraterone), Erleada (apalutamide) or Xtandi (enzalutamide) was soon shown to be helpful. It was also found the radiating the prostate when there were few metastases gave a survival benefit. By 2022, it was shown that adding Zytiga or Nubeqa (darolutamide) to chemotherapy (with continuing ADT, thus triple therapy) was also beneficial. Many new drugs have been developed, as she discussed, and are shown below. Numerous clinical trials showed the benefits of the new treatments: LATITUDE, STAMPEDE, TITAN, PEACE-1, CHAARTED, STAMPEDE, GETUG-15, HORRAD, ARCHES, ENZAMET, AND ARASENS.



The various stages of prostate cancer were discussed, both hormone-sensitive, with or without metastases, as well as hormone resistant ("castrate resistant") with or without metastases, and the kinds of



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treatments used at each stage.

Lots of new studies are coming along – see the video for discussion of ARANOTE, SWAO 1802, Keynote-991, CAPitello-281, PSMAddition, AMPLITUDE, TALAPRO-3 and Cyclone 3. The latter trial is especially interesting, in studying a new drug called abemaciclib – effective in breast cancer – which blocks key steps in cell division/growth of tumors.

Radioligand therapy was explained, and she updated information on 177Lu-PSMA-617 alone or in combination with other new drugs. Also, selective drug conjugates are expanding, where a carrier molecule is linked with "payload" that can be a radionucleotide, or a non-radioactive toxic agent, or a transcription (cell growth) factor.

PARP inhibitors such as Olaparib are especially used for men with genetic mutations such as BRCA or others. Various trials were discussed.

Survival was found to be improved for patients who went through clinical trials.

A variation of the CONTACT-02 trial, called Canopy, for mCRPC (metastatic castration-resistant PCa) patients is enrolling at UCSD, providing a new combination of drugs: Cabozantinib and Nivolumab (a PD-1 inhibitor).

ROR1 Targeting is another new strategy in PCa therapy. Receptor tyrosine kinase-like orphan receptor 1 (ROR1) is a cell-surface protein that mediates signaling from its ligand, Wnt5a, to drive physiologic embryonic stem cell proliferation. Targeting ROR1 appears to reduce the aggressiveness of the offspring of PCa stem cells and can lead to tumor shrinkage. Cirmtuzumab is a ROR1 Targeting Monoclonal Antibody that will be used with docetaxel in a study now enrolling at UCSD.

Bipolar Androgen Therapy (BAT) was discussed briefly, but will be treated next meeting in detail by Dr. Denmeade of Johns Hopkins.

Androgen receptor degraders are being developed, to get rid of the androgen receptor (which can undergo harmful changes during the course of PCa disease). ARV-766 is being used in a study at UCSD that is enrolling patients with mutations in their androgen receptors.

Another trial enrolling at UCSD is the COMRADE trial for mCRPC, which will combine Xofigo (Radium 223) with Olaparib. Only a few spots are left open.

There is a video from UCSD that is educational about germline testing.

Treatment with hormone blocking before surgery has been found very effective in preventing recurrence, in small trials to date.

At UCSD, the medical team is very multidisciplinary, with pathologists, radiologists, urologists, medical oncology, radiation oncology, nursing, cardio & endocrine specialists, and social workers, to provide personalized treatment strategies.

A "Register Now" QR code is in the video, for the Prostate Cancer Summit, February 10, 2024. Registrations are limited, so sign up soon.

If you would like to participate in a clinical trial at UCSD, but have Medicare Advantage elsewhere, be aware that California law requires that you be given access. Contact Dr. McKay.

To meet with Dr. McKay, call UCSD, or email her at rmckay@health.ucsd.edu

The video is available on YouTube at https://www.youtube.com/watch?v=5XukQhrhgqo

Items of Interest

Personalized dosing improves outcomes for men with metastatic castration-resistant prostate cancer

news-medical.net

Reviewed by Megan Craig, M.Sc.Jun 26 2023

By monitoring early-response biomarkers in men undergoing ^{177}Lu-PSMA prostate cancer treatment, physicians can personalize dosing intervals, significantly improving patient outcomes. In a study presented at the Society of Nuclear Medicine and Molecular Imaging 2023 Annual Meeting, early stratification with ^{177}Lu-SPECT/CT allowed men responding to treatment to take a "treatment holiday" and allowed those not responding the option to switch to another treatment.

Approved by the U.S. Food and Drug Administration in 2022, ^{177}Lu-PSMA is an effective treatment for metastatic castration-resistant prostate cancer. However, not all men respond equally to treatment, with some responding very well and others progressing early.

Currently, a standardized dosing interval is used for ^{177}Lu-PSMA treatment. However, monitoring early-response biomarkers to adjust treatment intervals may improve patient outcomes."

Andrew Nguyen, MBBS, FRACP, AANMS, senior staff specialist in the Department of Theranostics and Nuclear Medicine at St. Vincent's Hospital in Sydney, Australia

In the study, researchers sought to evaluate progression-free survival and overall survival of different dosing intervals. Study participants included 125 men who were treated in a clinical program with six weekly doses of ^{177}Lu-PSMA. The men were imaged with ^{177}Lu-SPECT/CT after each dose. After the second dose, researchers analyzed the men's prostate specific antigen (PSA) levels and the ^{177}Lu-SPECT response to determine ongoing management.

Patients were grouped by level of response. Those in Response Group 1 (35 percent of participants) had a marked reduction in PSA level and partial response on ^{177}Lu-SPECT and were advised to cease treatment until PSA levels rose. Response Group 2 (34 percent) saw stable or reduced PSA and stable disease on SPECT imaging; these men continued on their six week treatment plan until no longer clinically beneficial. In Response Group 3 (31 percent), men saw a rise in PSA levels and had progressive disease on SPECT imaging. These patients were offered the opportunity to try a different treatment.

PSA levels decreased by more than 50 percent in 60 percent of patients. Overall study participants had a median PSA progression-free survival of 6.1 months and a median overall survival of 16.8 months. Median PSA progression-free survival was 12.1 months, 6.1 months, and 2.6 months, for Response Groups 1, 2 and 3, respectively. The overall survival was 19.2 months for Response Group 1, 13.2 months for Response Group 2, and 11. 2 months for Response Group 3. Additionally, for those in Response Group 1 who had a "treatment holiday," the median treatment-free time was 6.1 months.

"Personalized dosing allowed one-third of the men in this study to have treatment breaks while still achieving the same progression-free and overall survival outcomes they would have if they received continuous treatment," noted Nguyen. "It also allowed another one-third of men who had early biomarkers of disease progression the opportunity to try a more effective potential therapy if one was available."

Patients at St. Vincent's Hospital will continue to be stratified by these early response biomarkers. Once validated in a prospective clinical trial, Nguyen hopes that this stratification strategy will become more widely available for patients.

Posted in: Men's Health News | Medical Research News

Novartis confirms unconstrained supply for Pluvicto® and continues to significantly expand the number of treatment centers

Oct 26, 2023

US FDA has classified drug shortage status as resolved[1]

Novartis capacity to produce Pluvicto will continue to grow with anticipated expansions to the manufacturing network in the US and globally

More than 200 centers are actively ordering doses of Pluvicto for patients in need, with plans to onboard approximately 130 more

East Hanover, October 26, 2023 — Novartis today announced that the US Food and Drug Administration has classified the Pluvicto® (lutetium Lu 177 vipivotide tetraxetan) drug shortage status as resolved. This determination is the result of efforts to significantly scale up production of Pluvicto that have more than doubled weekly production capacity since May. Novartis is committed to providing a consistent, reliable supply of Pluvicto and making this important medicine readily available to patients.

"We have been working hard to increase the capacity and improve the reliability of the supply of our radioligand therapies to ensure patients have access to this therapy and to prepare for future growth as more patients may become eligible for this treatment," said Victor Bulto, President, Novartis US. "Radioligand therapies have the potential to shift the standard of care in oncology and we are excited about the possibilities of our broad RLT pipeline for patients. With substantial experience in developing a reliable supply chain and delivery infrastructure, we are well positioned to expand access to these therapies for years to come."

Following FDA approval for commercial production of Pluvicto at the Novartis RLT manufacturing facility in Millburn, New Jersey earlier this year, supply is now unconstrained. Having doubled weekly production, Novartis currently has more than sufficient supply to treat patients within two weeks of diagnosis, which is important for these patients with advanced disease who may need treatment quickly. Pluvicto supply availability should continue to increase into 2024 as Novartis builds additional capacity by expanding production lines at the Millburn, New Jersey site, as well as the new state-of-the-art RLT facility in Indianapolis, Indiana. This site has started clinical production of Pluvicto and, pending FDA approval, will also manufacture commercial doses.

Novartis is expanding access to its RLTs in the US by partnering with treatment sites to add more central locations for patients. As of October, there are more than 200 facilities in the US certified to administer the company's RLTs. Novartis plans to onboard approximately 130 more certified facilities, strengthening access for more eligible patients across the country.

The company's RLT manufacturing facility in Ivrea, Italy will continue to supply patients in and outside the US while the facility in Zaragoza, Spain will solely supply patients outside of the US, and Novartis has a vision to add more RLT manufacturing locations around the world. With four active facilities by 2024 and an anticipated combined annual capacity target of at least 250,000 RLT doses in 2024 and beyond, Novartis is committed to providing adequate supply for current and future demand as ongoing clinical trials may present the potential to bring Pluvicto to more patients.

"Mind-Blowing" Cancer Discovery – Common Chemotherapy Drugs Don't Work Like Doctors Thought

scitechdaily.com University of Wisconsin-Madison

New research challenges the traditional belief about how microtubule poisons, a class of cancer drugs, work. *[docetaxel is one]* Instead of stopping cancer cells from dividing, these drugs alter the process, sometimes causing new cancer cells to die. The findings provide insights into why certain drug discovery attempts were unproductive and suggest a need to focus on disrupting the cell division process differently.

The research uncovers the likely reason why certain chemotherapies are effective for many patients. Importantly, they also shed light on why endeavors to discover new chemotherapy drugs based solely on stopping cellular division have been so disappointing.

Recent research from the University of Wisconsin–Madison indicates that chemotherapy may not be reaching its full potential, in part because researchers and doctors have long misunderstood how some of the most common cancer drugs actually ward off tumors.

Historical Understanding

For decades, researchers have believed that a class of drugs called microtubule poisons treat cancerous tumors by halting mitosis, or the division of cells. Now, a team of UW–Madison scientists has found that in patients, microtu-

(Continued from page 6)

bule poisons don't actually stop cancer cells from dividing. Instead, these drugs alter mitosis — sometimes enough to cause new cancer cells to die and the disease to regress.

Cancers grow and spread because cancerous cells divide and multiply indefinitely, unlike normal cells which are limited in the number of times they can split into new cells. The assumption that microtubule poisons stop cancer cells from dividing is based on lab studies demonstrating just that.

Details of the Study

The new study was led by Beth Weaver, a professor in the departments of oncology and cell and regenerative biology, in collaboration with Mark Burkard in the departments of oncology and medicine. Published in the journal *PLOS Biology* and supported in part by the National Institutes of Health, the study broadens previous findings the group made about a specific microtubule poison called paclitaxel. Sometimes prescribed under the brand name Taxol, paclitaxel is used to treat common malignancies including those originating in the ovaries and lungs.

"This was sort of mind-blowing," Weaver says about the previous research. "For decades, we all thought that the way paclitaxel works in patient tumors is by arresting them in mitosis. This is what I was taught as a graduate student. We all 'knew' this. In cells in a dish, labs all over the world have shown this. The problem was we were all using it at concentrations higher than those that actually get into the tumor."

Weaver and her colleagues wanted to know if other microtubule poisons work the same way as paclitaxel — not by stopping mitosis but by messing it up.

Implications for Future Research

The question has significant implications for scientists searching for new cancer treatments. That's because drug discovery efforts often hinge on identifying, reproducing, and improving upon the mechanisms believed to be responsible for a compound's therapeutic effect.

While microtubule poisons are no panacea, they are effective for many patients, and researchers have long sought to develop other therapies that mimic what they believe the drugs do. These efforts are ongoing even though past attempts to identify new compounds that treat cancer by stopping cell division have reached frustrating dead ends.

"There's still a lot of the scientific community that's investigating mitotic arrest as a mechanism to kill tumors," Weaver says. "We wanted to know — does that matter for patients?"

With Burkard, the team studied tumor samples taken from breast cancer patients who received standard anti-microtubule chemotherapy at the UW Carbone Cancer Center.

They measured how much of the drugs made it into the tumors and studied how the tumor cells responded. They found that while the cells continued to divide after being exposed to the drug, they did so abnormally. This abnormal division can lead to tumor cell death.

Normally, a cell's chromosomes are duplicated before the two identical sets migrate to opposite ends of the cell mitosis in a process called chromosomal segregation. One set of chromosomes is sorted into each of two new cells.

This migration occurs because the chromosomes are attached to a cellular machine known as the mitotic spindle. Spindles are made from cellular building blocks called microtubules. Normal spindles have two ends, known as spindle poles.

Weaver and her colleagues found that paclitaxel and other microtubule poisons cause abnormalities that lead cells to form three, four, or sometimes five poles during mitosis even as they continue to make just one copy of chromosomes. These poles then attract the two complete sets of chromosomes in more than two directions, scrambling the genome.

"So, after mitosis, you have daughter cells that are no longer genetically identical and have lost chromosomes," Weaver says. "We calculated that if a cell loses at least 20% of its DNA content, it is very likely going to die."

These findings reveal the likely reason why microtubule poisons are effective for many patients. Importantly, they also help explain why attempts to find new chemo drugs based solely on stopping mitosis have been so disappointing, Weaver says.

(Continued on page 8)

"We've been barking up the wrong tree," she says. "We need to refocus our efforts on screwing up mitosis — on making chromosomal segregation worse."

Reference: "Diverse microtubule-targeted anticancer agents kill cells by inducing chromosome missegregation on multipolar spindles" by Amber S. Zhou, John B. Tucker, Christina M. Scribano, Andrew R. Lynch, Caleb L. Carlsen, Sophia T. Pop-Vicas, Srishrika M. Pattaswamy, Mark E. Burkard and Beth A. Weaver, 26 October 2023, *PLOS Biology*.
DOI: 10.1371/journal.pbio.3002339

Docetaxel radiosensitizes castration-resistant prostate cancer by downregulating CAV-1: International Journal of Radiation Biology: Vol 0, No 0

Nrusingh C. Biswal

Abstract

Purpose: Docetaxel (DXL), a noted radiosensitizer, is one of the few chemotherapy drugs approved for castration-resistant prostate cancer (CRPC), though only a fraction of CRPCs respond to it. CAV-1, a critical regulator of radioresistance, has been known to modulate DXL and radiation effects. Combining DXL with radiotherapy may create a synergistic anticancer effect through CAV-1 and improve CRPC patients' response to therapy. Here, we investigate the effectiveness and molecular characteristics of DXL and radiation combination therapy in vitro.

Materials and methods: We used live/dead assays to determine the IC_{50} of DXL for PC3, DU-145, and TRAMP-C1 cells. Colony formation assay was used to determine the radioresponse of the same cells treated with radiation with/without IC_{50} DXL (4, 8, and 12 Gy). We performed gene expression analysis on public transcriptomic data collected from human-derived prostate cancer cell lines (C4-2, PC3, DU-145, and LNCaP) treated with DXL for 8, 16, and 72 hours. Cell cycle arrest and protein expression were assessed using flow cytometry and western blot, respectively.

Results: Compared to radiation alone, combination therapy with DXL significantly increased CRPC death in PC3 (1.48-fold, $p < .0001$), DU-145 (1.64-fold, $p < .05$), and TRAMP-C1 (1.13-fold, $p < .05$) at 4 Gy of radiation. Gene expression of CRPC treated with DXL revealed downregulated genes related to cell cycle regulation and upregulated genes related to immune activation and oxidative stress. Confirming the results, G2/M cell cycle arrest was significantly increased after treatment with DXL and radiation. CAV-1 protein expression was decreased after DXL treatment in a dose-dependent manner; furthermore, CAV-1 copy number was strongly associated with poor response to therapy in CRPC patients.

Conclusions: Our results suggest that DXL sensitizes CRPC cells to radiation by downregulating CAV-1. DXL + radiation combination therapy may be effective at treating CRPC, especially subtypes associated with high CAV-1 expression, and should be studied further.

Real-world evidence of triplet therapy in metastatic hormone-sensitive prostate cancer - an Austrian multicenter study | medRxiv

medrxiv.org

Mona Kafka

Abstract

Introduction Two randomized trials demonstrated a survival benefit of triplet therapy (androgen deprivation therapy [ADT]) plus androgen receptor pathway inhibitor [ARPI] plus docetaxel) over doublet therapy (ADT plus docetaxel) changing treatment strategies in metastatic hormone-sensitive prostate cancer (mHSPC).

Patients and methods We conducted the first real-world analysis including 97 mHSPC patients from sixteen Austrian medical centers. 79.4% of patients received abiraterone, 17.5% darolutamide, 2.1% apalutamide and 1% enzalutamide. Baseline characteristics and clinical parameters during triplet therapy were documented. Mann-Whitney-U-Test for continuous or X^2-test for categorical variables was used. Variables on progression were tested using logistic regression analysis and tabulated as hazard ratios (HR), 95% confidence interval (CI).

Results 83.5% of patients with synchronous and 16.5% with metachronous disease were included, with 83.5% high-volume disease diagnosed by conventional imaging (48.9%) or PSMA PET-CT (51.1%).

While docetaxel and ARPI were administered consistent with pivotal trials, prednisolone, prophylactic gCSF and osteoprotective agents were not applied guideline conform in 32.5%, 37% and 24.3% of patients, respectively.

Importantly, a non-simultaneous onset of chemotherapy and ARPI, performed in 44.8% of patients, was significantly associated with worse treatment response (p=0.015, HR 0.245).

Starting ARPI before chemotherapy was associated with significant higher probability for progression (p=0.023, HR 15.781) than vice versa. Strikingly, 15.6% (abiraterone) and 25.5% (darolutamide) low-volume patients as well as 14.4% (abiraterone) and 17.6% (darolutamide) metachronous patients received triplet therapy.

Adverse events (AE) occurred in 61.9% with grade 3-5 in 15% of patient without age-related differences. All patients achieved a PSA decline of 99% and imaging response was confirmed in 88% of abiraterone and 75% of darolutamide patients.

Conclusions Triplet therapy arrived in clinical practice primarily for synchronous high-volume mHSPC. Regardless of selected therapy regimen, treatment is highly effective and tolerable. Preferably therapy should start simultaneously, if not possible chemotherapy should be started first.

Take Home Massages Triplet therapy consisting of ADT plus ARPI (abiraterone or darolutamide) plus docetaxel is an effective and mostly well tolerable treatment option for mHSPC patients also in the real-world setting especially for synchronous, high-volume patients.

On the Lighter

"Before you go inside, give me a chance to explain the mess..."

NETWORKING

Please help us in our outreach efforts. Our speakers bureau consisting of Gene Van Vleet and Bill Lewis is available to speak to organizations of which you might be a member. Contact Gene 619-890-8447 or gene@ipcsg.org or Bill 619-591-8670 (bill@ipcsg.org) to coordinate.

Member 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

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!

DIRECTIONS TO MEETINGS

Directions to Sanford-Burnham-Prebys Auditorium
10905 Road to the Cure,
San Diego, CA 92121

- Take I-5 (north or south) to the Genesee exit (west).
- Follow Genesee up the hill, staying right.
- Genesee rounds right onto North Torrey Pines Road.
- **Do not turn into the Sanford-Burnham-Prebys Medical Discovery Institute or Fishman Auditorium**
- Turn right on Science Park Road. Watch for our sign here.
- Turn Left on Torreyana Road. Watch for our sign here.
- Turn Right on Road to the Cure (formerly Altman Row). Watch for our sign here.