REMINDER

ANNUAL PHCSG MEMBERSHIP $20

Join our Monthly meetings on the third Tuesday (Feb – Dec) 10am – 12:30pm

EFT Payments to:
Prostate Heidelberg CSG
BSB 083 256
Acct 583244292

Prostate Heidelberg

May 2021 Issue 206

For Education, Information and Support

Meeting Hall: Ivanhoe Uniting Church 19 Seddon Street, Ivanhoe
POB 241 Ivanhoe Victoria 3079
Email: prostateheidelberg@gmail.com
Website: www.prostateheidelberg.info

Next PHCSG Meeting – Tues 18 May (via Zoom)

10am – 12:30pm

Join Zoom Meeting: Copy link and paste into your browser
https://us02web.zoom.us/j/86101262643?pwd=YVpPeFNTWmpkaTZET1dPZmh6MDZWZz09

Meeting ID: 861 0126 2643
Passcode: 321305

PHCSG May

Sorry Folks. We have been unable to secure our meeting room this month due to Banyule reorganising the hall layout to accommodate COVID restrictions, so we will all need to zoom once again. We hope to announce alternative arrangements for June at our May Zoom meeting.

We have guest speaker Angela Mellerick, Nurse Unit Manager, Olivia Newton John Cancer and Wellness Centre to talk about her role, supporting people undergoing chemotherapy and other treatments.

Finally, if you've ever needed a list of PCa acronyms/terms, we have put together a glossary that you will be able to download from the web site soon.

In this month's newsletter we highlight:

If there is anything you want to talk through in relation to your treatment or wellbeing please don't hesitate to ring:

Max Shub	0413 777 342
Mike Waller	0438 616 240
Michael Meszaros	0407 837 538

Prostate Heidelberg Cancer Support Group

PHCSG provides information, education and support for those affected by Prostate Cancer. At our meetings we are committed to:

- showing respect to members, speakers and guests
- allowing members to speak without interruption
- respecting confidentiality

Full-on 'Kitchen Sink" for High-Risk Localized Prostate Cancer: Intensive Neoadjuvant Hormonal Therapy

Source:
Janet Farrar Worthington
https://www.pcf.org/c/full-on-kitchen-sink-for-high-risk-localized-prostate-cancer-intensive-neoadjuvant-hormonal-therapy/

Part One: Why the whole Kitchen Sink Now?

Which scenario would you prefer: "I've got high-risk prostate cancer. I sure hope it doesn't come back after surgery or radiation! Fingers crossed! My doctor and I are really hoping for the best!" or,

"I've got high-risk prostate cancer that has a chance of coming back after initial treatment. So, my doctor is going after it relentlessly, like Inspector Javert hunting Jean Valjean in *Les Mis*."

High-risk prostate cancer is formidable: it will spread if not treated and is more likely to recur after initial treatment. That's why doctors like Rana McKay, M.D., medical oncologist and PCF-funded Young Investigator at the University of California San Diego (UCSD) are now throwing the proverbial kitchen sink at high-risk prostate cancer *as soon as it is diagnosed*.

This marks a huge shift in medical thinking. Advanced prostate cancer treatment in the past has been like a methodical series of "if: then" statements in math, like, "If A, then B," or "C if and only if B." If cancer spreads beyond the prostate, then the traditional next step has been androgen deprivation therapy (ADT), shutting down testosterone and other male hormones that drive prostate cancer's growth. If the cancer becomes resistant to ADT, then other medications are added: chemotherapy and/or androgen-directed therapies (also called AR-signaling inhibitors).

Over the last few years, doctors have been compressing this time frame, giving these androgen-directed drugs at the time that ADT is initiated – based on studies such as STAMPEDE and LATITUDE, suggesting that the cancer, which evolves and mutates as it spreads, is more vulnerable to treatment sooner rather than later. Although these treatments can extend survival, they are not a cure.

What's different about this new, full-on, kitchen-sink approach? First, a high-intensity burst of hormonal suppression (ADT plus an androgen-directed drug, such as enzalutamide or abiraterone) is finite, given as neoadjuvant therapy for a few months before surgery and for up to a year afterward. Then it's over, and within a year, testosterone comes back.

Second: "We are going for a cure," says McKay.

Early results of exciting clinical trials, with more on the way, are highly encouraging. One Phase II trial still in progress, led at UCSD by McKay in collaboration with PCF-funded investigator Mary-Ellen Taplin, M.D., of the Dana-Farber Cancer Institute, grew out of a 2014 PCF Challenge Award study, led by Taplin. The investigators tested two combinations of drugs given for six months before surgery: abiraterone and prednisone plus leuprolide (Lupron), vs. abiraterone and prednisone, Lupron, and apalutamide. After surgery, "men were randomized to continue therapy for one year, or simply to be monitored." The initial results of this trial were presented at the American Society of Clinical Oncology meeting in 2020.

"We showed that about one out of five men who received intensive hormonal therapy up front demonstrated very residual amounts of tumor, or no tumor at all, in their prostatectomy specimen" when the surgically-removed tumor was thoroughly examined by a pathologist under the microscope. This "pathologic response," seen in the surgically removed tissue, "hasn't yet been proven in prostate cancer to be associated with long-term outcome," notes McKay. "But in several other tumor types – breast, bladder, rectal cancer, and others – evidence demonstrates that the pathologic response is associated with overall survival." In follow-up data from this and two other neoadjuvant studies, recently published in the *Journal of Urology*, McKay and colleagues showed that "of those patients who had no tumor or very little tumor left behind in their prostate, the rate of recurrence (the average follow-up time so far is 3.6 years) was significantly lower. In our cohort of 117 patients, only two patients who had a pathologic response and minimally residual disease had a recurrence, and no man died of prostate cancer. Our hope is that we will develop data to prove that a pathologic response is associated with long-term outcomes in prostate cancer."

(continued page 3)

Part Two: In some responders, at Prostatectomy, Cancer's Already Dead

What's the idea behind slamming high-risk prostate cancer with a battery of treatments: surgery plus a finite course of androgen deprivation therapy (ADT) plus an androgen-directed drug such as abiraterone, apalutamide or enzalutamide? Catching cancer when it's less prepared for battle.

Over time, prostate cancer acquires genomic alterations that help it to be more aggressive. Each tiny mutation gives the cancer extra protection, maybe starting out with the genetic equivalent of a bullet-proof vest or stronger helmet, then becoming much more sophisticated – imagine a fighter jet deploying decoy flares or chaff as missile countermeasures.

Is it more vulnerable, and easier to kill, early on? PCF-funded investigator Rana McKay, M.D., a medical oncologist at the University of California-San Diego (UCSD), and colleagues believe the answer is yes, and they're testing this idea in several clinical trials. One phase II study at UCSD still in progress, in collaboration with PCF-funded investigator Mary-Ellen Taplin, M.D., of the Dana Farber Cancer Institute involved 119 men with "unfavorable intermediate or high-risk disease. "More than 90 percent of the patients had high-risk disease, and all of them, from the get-go, had very aggressive tumors," says McKay. "Over one-third of patients had Gleason 9 or 10 disease, and about 60 percent of patients had stage 3 cancer," that had spread slightly beyond the prostate but with no evidence of distant metastases. Men in the trial received either neoadjuvant abiraterone and prednisone plus leuprolide (Lupron), vs. abiraterone and prednisone, Lupron, and apalutamide.

One major reason why McKay and colleagues are testing this approach with surgery rather than radiation is to study the pathologic response: looking at how much residual tumor is present in the surgical specimen that has been removed after treatment. Have they seen any changes? Not in all men, but in about 20 percent, there's a remarkable change: "The primary tumor was dead and necrotic." The pathologists "looked at every little sliver of the prostate," and found that these exceptional responders had either "less than 5 mm of tumor left behind, or no tumor left behind."

Just think about that for a minute: the surgeon removes the prostate, gives the tissue to the pathologist, who starts looking at it under the microscope and sees only corpses of cancer cells!

One patient who participated in this study is Pat Sheffler (https://www.pcf.org/c/sheffler-strong/), who was diagnosed at age 53 with stage 3 prostate cancer and had a PSA of 37. He received abiraterone and prednisone, Lupron, and apalutamide for six months before prostatectomy, and started to see results right away. In monthly blood tests before his surgery, his PSA levels dropped: "34, 27, 21, 10, 4, 2, and 0.2." At surgery, he had "very minimal remaining tumor," says McKay. Then he underwent one more year of hormone therapy after surgery. Two months after he stopped taking the trial medications, not only was his PSA undetectable, but his testosterone levels were coming back to normal. "My hope for Pat is that he's cured, that he can go on just being an amazing dad, husband, and advocate for prostate cancer awareness."

In another phase II study led by Taplin, published in the *Journal of Clinical Oncology*, McKay and colleagues at UCSD, Dana-Farber, Beth Israel Deaconess Medical Center, Johns Hopkins, and the University of Washington reported a complete pathologic response (no remaining live cancer cells in the prostate) or minimal residual disease in 30 percent of patients treated with neoadjuvant enzalutamide, Lupron, abiraterone and prednisone before prostatectomy.

But what about the men who were not exceptional responders to big-gun hormone therapy? The scientists have identified some key genetic changes in men who were non-responders, and they have some ideas about how to help these men, as well.

PLEASE NOTE:
Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist

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Part Three: Men Who Need a Different Approach

In several clinical trials, including this one, an intense blast of neoadjuvant androgen deprivation therapy (ADT) and androgen-directed treatment (medications such as abiraterone and enzalutamide) has shown promising results in some men – but not all men. Why is this?

PCF-funded investigator Rana McKay, M.D., medical oncologist at the University of California-San Diego (UCSD), in collaboration with PCF-funded investigator Mary-Ellen Taplin, M.D,. of Dana-Farber, and colleagues have found an explanation: Men who have not responded (who had a significant amount of tumor remaining after neoadjuvant treatment) in these clinical trials have certain genetic differences in their prostate cancer – loss of PTEN (a tumor suppressor gene, which is knocked out in as many as 20 percent of men with localized prostate cancer) or alterations in ERG (an oncogene that fuses with another gene, called TMPRSS2, in as many as half of all men with prostate cancer).

"Very few of the men who responded had PTEN loss," says McKay, "and ERG positivity was also associated with lack of response." But these men also seem to have something else that might render AR-blocking drugs unhelpful: lower AR expression, compared to other men. Basically, if a tumor does not seem to have a lot of androgen receptor activity, then a medicine that targets these receptors won't have much to work with.

This information is not discouraging, McKay hastens to add: it's helpful! It has taught the scientists that "the responders have a certain tumor profile, and non-responders have a certain profile. Similarly, responders had mutations in a gene called SPOP" (which is mutated in about 10 percent of primary prostate tumors).

Knowing this, McKay adds, could be an opportunity: a springboard for additional or different therapy – perhaps neoadjuvant chemotherapy, for example. Remember: you're still ahead of the game here. You don't have metastatic cancer, and many scientists believe that high-risk cancer, when it's localized, is still vulnerable enough to be cured, if it's hit hard with multiple weapons.

"This is an opportunity for us to develop and design a personalized treatment strategy for these men," says McKay. "It would be awesome if we could use somebody's own genomics to help design the best treatment for him – similar to what's being done in the breast cancer I-SPY trials, neoadjuvant studies with multiple treatment arms, some determined by biomarkers (specific genetic alterations that show up in a blood or tissue test).

Some men with high-risk prostate cancer might respond better to a PARP-inhibiting drug, such as olaparib and rucaparib. This is the focus of another study that will be starting soon, McKay says. "In men who have germline (inherited) alterations, such as a BRCA1 or BRCA2 mutation, we hypothesize that giving a PARP inhibitor in a neoadjuvant setting before prostatectomy might significantly improve pathologic response. We are finalizing the protocol for NEPTUNE, a biomarker-focused neoadjuvant trial testing PARP inhibitors in localized prostate cancer."

"It is really exciting to be part of this paradigm shift," says McKay. "We have the opportunity to improve outcomes for men with high-risk localized disease, and we're in the midst of trying to prove that through well-organized, thoughtful clinical trials.

"At the end of the day, the question is, how can we help our patients live longer and live better? That's really the big driver. The good thing about localized disease is that we can try to *cure* more men of prostate cancer – not just extend life for metastatic disease, but can we develop a pathway so they don't ever develop metastatic disease, and so they can be cured? That's what we're aiming to do." And, bonus: after the big blast of intense hormonal treatment, most men get their testosterone back. "Most patients actually recovered their testosterone fully within the first year of discontinuation of treatment."

The groundwork for these studies was laid by PCF funding over the last six years. "PCF has been a champion in revolutionizing the science and helping advance the science," says McKay. "PCF has been a huge catalyst in all of this."

Like all treatment decisions, you have to weigh how you feel about the potential benefits against the potential risks. No one can do that for you.

Calcium and vitamin D supplements not necessary for healthy adults, research finds

Source:
ABC Health Report
Olivia Willis
29 Nov 2019
https://www.abc.net.au/news/health/2019-11-29/calcium-vitamin-d-supplements-for-osteoporosis/11742866

Australians spend more than $2 billion on vitamin and dietary supplements every year — many of which are effectively useless.

Key points:

- Vitamin D and calcium supplements are often recommended to curb osteoporosis risk
- New review finds the vitamins have little value for people who are not vitamin deficient
- Calcium supplements may even cause harm, and have no place in modern medicine, experts say

Now, research has revealed it might be time to ditch two of our most popular vitamins.

A review recently published in the Medical Journal of Australia found calcium and vitamin D supplements, often recommended to older Australians to prevent osteoporosis, offer very little benefit to healthy adults.

In fact, calcium supplements may be doing more harm than good.

While the nutrients themselves are important, the researchers found calcium and vitamin D supplements did little to reduce fracture risk or improve bone density in the healthy older adult population.

The use of vitamin D as a "general tonic" in individuals who were not vitamin D deficient (or at risk of becoming deficient) was found to be largely fruitless.

"Just as we would not expect antibiotics given to individuals without an active infection to have beneficial effects, we should not expect supplements of calcium and vitamin D to benefit people who do not have demonstrable deficiency," the study authors wrote.

Supplements may cause harm

Calcium and vitamin D supplements are often administered together for the prevention and treatment of osteoporosis, which occurs when bones lose minerals, such as calcium, more quickly than the body can replace them.

Previous research into these supplements has produced conflicting results.

Is that really working?

But this latest review, which assessed the overall safety and effectiveness of supplements, suggests the supplementation of calcium has little place in modern medicine.

"When you give extra calcium to otherwise healthy people living in the community, it makes no material difference to the number of fractures that occur," lead author Ian Reid, professor of medicine and endocrinology at the University of Auckland, told the Health Report.

"And the main reason for giving extra calcium was a belief that that would make bones stronger."

According to the review article, calcium supplements can cause constipation, bloating and kidney stones, and may increase the risk of heart attack.

"Calcium supplements are frequently associated with gastrointestinal symptoms ... and they have also been reported to double the risk of hospital admissions related to abdominal symptoms," the authors wrote.

Vitamin D supplements, on the other hand, rarely cause adverse health outcomes. But there is evidence that very high levels of vitamin D can increase the risk of falls and fractures.

Either way, supplements were found to generally only have value in people with vitamin deficiencies, and not across the healthy older population — so talk to your doctor before starting or stopping any supplements.

When they should be used

Although the evidence for supplements in osteoporosis treatment is not strong, Professor Reid said there are some circumstances where they should still be used.

"Some of the new drugs that we are currently using in osteoporosis have only been assessed when calcium and vitamin D have been given at the same time, so I think we need to proceed cautiously," he said.

Favorable prognosis of patients who received adjuvant ADT after RT achieving undetectable levels of prostate-specific antigen in high-or very high-risk PCa

Source:
March 12, 2021
https://europepmc.org/article/MED/33711055

As the incidence of prostate cancer increases, there is a trend that the proportion of locally advanced prostate cancer also increases. Those who are considered to have high risk are defined by the National Comprehensive Care Network (NCCN) as having at least one of the following features: T3a, Gleason Group 4 or 5, and serum prostate-specific antigen (PSA) value of more than 20 ng/mL. Those with at least one of the following features, T3b-4, primary Gleason pattern 5, or >4 cores with Gleason Group 4 or 5, are defined as being at very high risk. Radiation therapy (RT) for these patients could be a therapeutic option among various modalities

Currently, RT with androgen deprivation therapy (ADT) is the standard treatment of choice for high-risk patients. Neoadjuvant ADT (N-ADT) and concurrent ADT (C-ADT) could improve the prognosis of intermediate and high-risk patients. Several randomized trials have illustrated the efficacy of adjuvant ADT (A-ADT) administered with RT in locally advanced prostate cancer. Additionally, long-term A-ADT could improve treatment outcomes in high-risk patients treated with RT. A randomized trial demonstrated that A-ADT for 34 months resulted in better treatment results than A-ADT for 4 months. However, the optimal duration of A-ADT is unclear in the setting of RT.

The treatment outcomes and related predictive factors in a single institute cohort of patients were investigated with high or very high-risk prostate cancer without regional lymph nodal involvement who received RT. The aim of this study was to evaluate the efficacy of long-term A-ADT and to determine other prognostic factors associated with patient- or treatment-related characteristics in the setting of RT.

A total of 197 patients with prostate cancer received RT, with a follow-up of ≥12 months. Biochemical failure was defined as PSA ≥nadir + 2 ng/mL after RT. The clinical outcomes were analyzed, including survival, failure patterns, and prognostic factors affecting outcomes.

Biochemical failure-free survival (BCFFS), clinical failure-free survival, distant metastasis-free survival, cancer-specific survival, and overall survival (OS) rates at 5 years were 91.1%, 95.4%, 96.9%, 99.5%, and 89.1%, respectively. Administration of long-term A-ADT significantly predicted favourable BCFFS (p = 0.027) and OS (p < 0.001) in multivariate analysis. Nadir PSA ≤0.001 ng/mL was an independent prognostic factor for BCFFS (p = 0.006) and OS (p = 0.021). The use of long-term A-ADT significantly affected nadir PSA ≤0.001 ng/mL (p < 0.001). The patients with A-ADT for 1 year or longer had better BCFFS or OS than those for less than 1 year or those without A-ADT (p < 0.001). The best prognosis was demonstrated in patients treated with long-term A-ADT and nadir PSA ≤0.001 ng/mL in BCFFS (p < 0.001).

Conclusion: The addition of long-term A-ADT over 1 year to RT demonstrated good treatment outcomes in patients with locally advanced prostate cancer. Achieving a nadir PSA value ≤0.001 ng/mL using combination therapy with RT and A-ADT is a powerful clinical predictor of treatment outcomes.

PLEASE NOTE:
Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist

Healthy Lifestyle May Offset Genetic Risk in PCa

Source:
Abstract Medscape Medical News
15 Aril 2021
https://mybestmedicine.com/health-news/healthy-lifestyle-may-offset-genetic-risk-in-prostate-cancer/+to+results+of+a+large+U.S.+study.&t=osx&ia=web

Adhering to a healthy lifestyle may offset the heightened risk of lethal prostate cancer (PCa) in men with adverse genetic risk factors, according to results of a large U.S. study.

"In men at the highest risk of PCa death, having the highest healthy lifestyle scores cut the risk of fatal disease in half," said study author Anna Plym, PhD, of Brigham and Women's Hospital and Harvard School of Public Health, both in Boston. She presented these findings at the American Association for Cancer Research (AACR) 2021 Annual Meeting.

Plym noted that genetic factors account for about 58% of variability in PCa risk, with common single-nucleotide polymorphisms (SNPs) accounting for a large proportion of PCa susceptibility.

"A recent study showed that a polygenic risk score (PRS) derived by combining information from 269 SNPs was highly predictive of PCa," Plym said. There was a 10 fold gradient in disease risk between the lowest and highest genetic risk deciles, and the pattern was consistent across ethnic groups. "In addition," Plym noted, "previous studies have suggested that a healthy lifestyle reduces lethal PCa risk." What remains unclear is whether the risk for both PCa development and the risk of progression to lethal disease can be offset by adherence to a healthy lifestyle.

To investigate, Plym and colleagues used the 269-SNP PRS to quantify the genetic risk of PCa in 10,443 men enrolled in the Health Professionals Follow-up Study. Men were divided into quartiles according to generic risk.

The investigators also classified the men using a validated lifestyle score. For this score, one point was given for each of the following: not currently smoking or having quit 10 or more years ago, body mass index under 30 kg/m2 , high vigorous physical activity, high intake of tomatoes and fatty fish, and low intake of processed meat. Men with 1-2 points were considered the least healthy, those with 3 points were moderately healthy, and those with 4-6 points were considered the healthiest.

The outcomes assessed were overall PCa and lethal PC (i.e., metastatic disease or PCa-specific death).

At a median follow-up of 18 years, 2,111 cases of PCa cancer were observed. After a median follow-up of 22 years, 238 lethal PCa events occurred.

Men in the highest genetic risk quartile were 5 times more likely to develop PCa (Hazard Ratio [HR], 5.39; 95% confidence interval [CI], 4.59-6.34) and 3 times more likely to develop lethal PCa (HR, 3.43; 95% CI, 2.29-5.14), vs. men in the lowest generic risk quartile.

Adherence to a healthy life- style did not decrease the overall risk of PCa (HR, 1.01; 95% CI, 0.84-1.22), nor did it affect men in the lower genetic risk quartiles.

However, healthy lifestyle did appear to affect men in the highest genetic risk quartile. Men with the highest healthy lifestyle scores had roughly half the risk of lethal PCa when compared with the men with the lowest lifestyle scores (3% vs. 6%).

Plym observed that the rate of lethal disease in men with the best lifestyle scores matched the rate for the study population as a whole (3%), suggesting that healthy lifestyle may counterbalance high genetic risk

She added that previous research has confirmed physical activity as a protective factor, but more study is needed to shed light on the relative benefit of the healthy lifestyle components.

In addition, further research is needed to explain why the benefit was limited to lethal PCa risk in men with the highest genetic risk.

Plym speculated that genetic variants contributing to a high PRS may also be the variants that have the strongest interaction with lifestyle factors. "For men with a genetic predisposition to PCa," she added, "these findings underscore the potential value of surveillance.

"Our findings add to current evidence suggesting that men with a high genetic risk may benefit from a targeted PCa screening program, aiming at detecting a potentially lethal PCa while it is still curable," she said.

Charles Swanton, MBPhD, of the Francis Crick Institute and UCL Cancer Institute in London, raised the possibility that competing risk issues could be at play.

"If a healthy lifestyle leads to longer life," he asked, "does that make it more likely that patients will live long enough to die from their PCa because they are not dying from cardiovascular disease, complications of diabetes, etc.? In that case, is the healthy lifestyle really affecting PCa at all?"

Plym responded that, among those in the highest genetic risk group with an unhealthy lifestyle, the increased risk for prostate cancer exceeded the risk for other illnesses.

Research Uncovers Additional Treatment Option in PCa

Source:
29 March 2021
Karolinska Institutet

https://medicalxpress.com/news/2021-03-uncovers-additional-treatment-option-prostate.html

The standard treatment for advanced metastatic prostate cancer (PCa) is androgen deprivation therapy (ADT). However, 1/3 of men will become resistant and develop castration-resistant PCa cancer (CRPC). A new study, by Karolinska Institutet and others, shows that estrogen receptor beta (β; ERβ) agonists together with ADT could be a useful treatment. ADT is based on the use of hormones to cause chemical

castration and is the usual treatment of metastatic PCa. And even if this is an efficient way to treat PCa in the short term, some will build up a resistance to ADT and develop fatal CRPC. For this reason, there is a clear need for alternative treatments. ERβ is a tumor suppressor and its role in PCa treatments and prevention has been investigated for more than 20 years. ERβ expression is lost as PCa progresses.

But a new study published in the Proceedings of the National Academy of Sciences (PNAS), by Karolinska Institutet, University of Houston, University of Texas MD Andersson Cancer Center and Barmherzige Schwestern Hospital shows that the nu- clear transport of epidermal growth factor receptor (EGFR) could be a target of ERβ agonist treatment. Immunochemical staining of sequential sections in Θssue arrays indicated that ERβ was expressed in both luminal and basal prostate epithelial cells. But the androgen receptor (AR) was only ex- pressed in luminal cells and not in basal cells. This is the reason why ADT can prevent the spread of AR-positive cancer cells but has no effect on basal cells. Increased EGFR nuclear

translocation seen with finasteride is markedly reduced by adding ERβ agonist, isoflavone, suggesting it may prevent onset of tyrosine kinase cancers. "This study provides further evidence that ERβ agonists may be a good medicine vs. certain forms of PCa," says Professor Jan-Åke Gustafsson at the Department of Biosciences and Nutration, KI. "This is a line of research that we intend to continue working with."

What is Androgen Deprivation Therapy (ADT)?

ADT (hormonal therapy) may be offered to men with prostate cancer to shut off testosterone (mainly produced by the testes:

- as a potentially curative treatment for a period before and after radiotherapy
- if they experience a rise in PSA after initial curative treatment
- where the cancer has spread outside the prostate at the time of diagnosis

Common ADT drugs re Lupron®, Zoladex®, Eligard®, Suprefact® and Firmagon®.

Unfortunately, for some men, a lower testosterone level can have a negative impact on quality of life.

The *APCR Prostate Cancer Centre* is an ADT Clinic to help men prevent or manage the side effects.

What should you expect?

You will see a team of specialists including a nurse, GP, and exercise Physiologist to receive:

- Education about ADT and potential side effects
- A comprehensive health assessment
- An individualized exercise program

APCR Prostate Cancer Centre

Level 8, 14-20 Blackwood Street
North Melbourne, Vic 3051

03 8373 7600
info@prostatecentre.org.au
prostatecentre.org.au

Mon- Fri 9am – 5pm

The centre bulk bills medical consultations and procedures.

A GP care plan is needed for psychology, pelvic floor physiotherapy and exercise physiology.

New type of treatment could reawaken immune response against prostate cancer

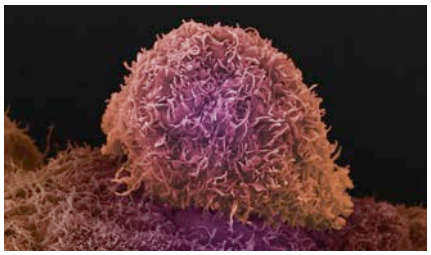

Image: A single prostate cancer cell. Credit: Anne Weston, Francis Crick Institute, CC BY-NC

Source: https://www.icr.ac.uk/news-archive%2Fnew-type-of-treatment-could-reawaken-immune-response-against-prostate-cancer?utm_medium=email&utm_source=rasa_io&PostID=29954105&MessageRunDetailID=517088583

Targeting a molecule on the surface of immune cells could offer an exciting new way to treat prostate cancer by reawakening the immune response against it.

A team at The Institute of Cancer Research, London, and The Royal Marsden NHS Foundation Trust, found that patients whose immune cells within tumours displayed a molecule called CD38 on their surface lived less long than those without.

The researchers found that the CD38 protein molecule seems to suppress the immune response and is a sign that prostate cancer is successfully hiding from the immune system.

Targeting CD38

Their study suggests that therapies which target CD38 – such as the multiple myeloma drug daratumumab – could hold promise against prostate cancer too, by reawakening the anti-cancer immune response.

As a result of the new findings, researchers at The Institute of Cancer Research (ICR) and The Royal Marsden are now running clinical trials to test out if targeting this CD38 pathway in people with prostate cancer can benefit them.

They also believe that testing for CD38 could pick out patients with a poor prognosis and could help assess the likelihood that they will respond to certain treatments.

The study is published in the journal *European Urology* and was funded by Prostate Cancer UK, Movember, Prostate Cancer Foundation, Cancer Research UK and Sanofi-Aventis.

The researchers studied prostate tumour samples to find out how often CD38 was present on different immune cells, whether its presence had influenced how quickly their cancer progressed and whether it made their cancer more likely to evolve and develop resistance to treatment.

Linked to worse survival outcomes

The team found that having a higher density of immune cells displaying CD38 was linked to worse survival outcomes for people with prostate cancer. A density of more than 1.5 of CD38 immune cells per mm2 in these biopsies from advanced prostate cancer was associated with a more than doubled risk of dying.

Researchers also found that there was an increase in the density of immune cells displaying CD38 in tumours as prostate cancer progressed to become resistant to hormone therapy.

They showed that CD38 is mainly present on specific types of immune cells known as B-cells, which are responsible for producing various molecules which turn the level of the immune response up and down.

Supporting the 'anti-cancer' immune response

Some immune cells can move from the blood into tumours to help recognise cancer cells as abnormal and destroy them – supporting the body's 'anti-cancer' immune response. However, researchers found that immune B-cells displaying CD38 on their surface may stop anti-cancer T-cells from functioning, suppressing the anti-cancer immune response and increasing the chances that the disease will progress.

Researchers think that CD38 levels could therefore identify patients who could benefit from treatments that target this molecule's function.

Clinical trials are now under way to translate these findings and reactivate the anti-cancer immune response in prostate cancer.

Fighting cancer's cloaking strategy

Study leader Professor Johann de Bono, Professor of Experimental Cancer Medicine at The Institute of Cancer Research, London, and Consultant Medical Oncologist at The Royal Marsden NHS Foundation Trust, said:

"We believe that CD38 on the surface of immune cells is acting to dampen down the immune response. We have shown that the presence of this protein on immune cells within prostate tumours is a sign of worse survival outcomes and exhausted anti-cancer immune responses. It is now clear that CD38 has a role in prostate cancer's growth and spread – suggesting that targeting it with drugs, which already exist and are used in other cancers, could be a promising new approach to treatment.

"Our findings suggest that we can target immune cells displaying CD38 proteins on their surface to reawaken the immune system and fight cancer's 'cloaking' strategy. I'm already leading a clinical trial in this area, which is a first in prostate cancer."

Professor Paul Workman, Chief Executive of The Institute of Cancer Research, London, said:

"As cancers develop, they often evolve the ability to evade the immune system so they can keep growing and spreading without being attacked. This new study suggests that in prostate cancer, tumours can supress the immune system via the CD38 molecule on the surface of immune cells. The findings are exciting and open up a whole new potential approach to treating prostate cancer using immunotherapy – an approach that is now being tested in clinical trials which have the potential to show real benefit for patients."

Source:
Sunday, April 19, 2020
https://wchh.onlinelibrary.wiley.com/doi/epdf/10.1002/tre.351

Penile rehabilitation after radical prostatectomy

Erectile dysfunction remains a common complication after radical prostatectomy. Natural recovery of erectogenic stimulus may take years after nerve-sparing surgery, during which time the penis undergoes physiological and structural changes that are harmful to erectile function. The goals of penile rehabilitation are to prevent these changes and thereby produce a faster and better return of erections and sexual function. In this article, the authors discuss current approaches to penile rehabilitation after prostatectomy.

In spite of advances in surgical technique over the previous decades, erectile and sexual dysfunction remain common complications of radical prostatectomy. In contemporary series, over half of men with normal preoperative sexual function will have persistent baseline sexual function, and technical factors of the nerve-sparing technique. Even under optimal circumstances, there appears to be a near-universal period without erections that may last for two years or longer.

In addition to erectile dysfunction, up to 70 per cent of men may also suffer from loss of penile length after prostatectomy and often have additional sexual complaints including low libido, dysorgasmia and climacturia.

The concept of penile rehabilitation has been around for decades and aims to increase the rate of eventual return of erections, decrease the time to recovery, and ultimately optimise the quality of erectile and sexual function. According to recent surveys, the majority of urologists offer some sort of penile rehabilitation after prostatectomy. Where there is extensive pre-clinical evidence supporting penile rehabilitation, clinical evidence is lacking and there is no consensus on the optimum rehabilitation regimen.

ARGUMENT FOR REHABILITATION

The primary argument for penile rehabilitation is to avoid the adverse structural and physiological changes that occur in the penis after prostatectomy. These changes include loss of corporal smooth muscle, impairments in veno-occlusive function, and ultimately penile fibrosis. They are hypoxia induced, and time dependent so that the potential for erectile function decreases with time after prostatectomy. Any rehabilitation, the reasoning goes, would be better than allowing uninhibited deterioration of penile tissue. An ideal penile rehabilitation protocol would intervene early postoperatively to prevent deterioration of penile tissues.

Additional components of an ideal penile rehabilitation protocol are early counselling and continued discussion of erectile function. In the absence of formal counselling and discussion, men with good preoperative erectile function and strong desire to maintain it will often not pursue postoperative therapy and, even if they do, often discontinue therapy after a prostatectomy and the discouragingly long time before spontaneous recovery of erectile function. Unfortunately, by the time many men seek care for sexual issues after prostatectomy, significant and irreversible tissue damage has occurred. Therefore, we believe that discussion of the physiology of erectile dysfunction after prostatectomy and expectations for recovery is an important component of a rehabilitation programme.

ORAL THERAPIES WITH PDE5 INHIBITORS

Phosphodiesterase 5 (PDE5) inhibitors are the most commonly used form of rehabilitation therapy, with a strong scientific basis in pre-clinical studies. In animal models, there is consistent evidence that prolonged treatment with PDE5 inhibitors (ie Viagra (Sildenafil), Cialis (Tadafil), Levitra, Vardenafil)) is protective and minimises cavernosal damage. Unfortunately, for the most part these findings have not been replicated in clinical studies, which suggests, at most, a much more modest effect in humans.

A small prospective study of nightly sildenafil starting one month postoperatively showed 27 per cent success at one year (defined by a combined score of 8 or more on International Index of Erectile Function study found that on-demand vardenafil was as good as nightly vardenafil in producing erections sufficient for penetration. However, IIEF scores at one year were no different for on- postoperatively, but it is possible that further benefits may be observed later on. In a contemporary retrospective review, erectile function at three years influenced by factors such as patient or partner motivation and have not yet been confirmed by prospective studies.

We believe that some men may benefit from the use of PDE5 inhibitors and support their use, either nightly or on demand, as an alternative to no treatment. Emerging data on daily use of tadalafil suggest there supporting more than a modest benefit are lacking, and we recommend offering additional therapies to motivated patients.

INJECTION THERAPIES

The first clinical study on penile rehabilitation was published by Montorsi et al. in 1997. This study conducted in the pre-Viagra era, showed a 67 per cent return of spontaneous erections for patients receiving intracavernosal injections (ICI) with alprostadil three times weekly for 12 weeks compared to 20 per cent of men receiving no treatment (Figure 1). suggest a benefit to ICI, to our knowledge no further placebo-controlled trials have been reported. Based on these limited data, we believe ICI with a vasoactive agent is a promising tool that may help maintain cavernosal oxygenation and prevent structural deterioration of erectile function after prostatectomy.

About 75 per cent of physicians responding to an International Society for Sexual Medicine survey reported using ICI, although many

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urologists will offer it only after failure of PDE5 inhibitors. Major limitations to ICI relate to psychosexual aspects of injection in addition to presence of penile pain, particularly for regimens including alprostadil. Pain may occur either at the injection site or generalised penile pain may occur with erection, leading to a high rate of discontinuation.

A recent study reported safe use of alprostadil ICI in men starting one month after nerve-sparing prostatectomy. The alprostadil dose started low and was uptitrated until erections were sufficient for vaginal penetration. Overall the rate of penile pain was 11 per cent and men who persisted with injections may also be associated with decreased pain and better satisfaction.

Overall, we believe that ICI has significant potential in preventing hypoxia-induced changes that may diminish or prevent eventual return of erections. Intervention as early as one month after prostatectomy appears safe, and we support offering ICI early in the postoperative course. ICI may be associated with pain or psychosexual distress and may require significant patient counselling and discussion.

INTRAURETHRAL THERAPY

Intraurethral therapy is less commonly used than PDE5 inhibitors and ICIs in penile rehabilitation, but allows for direct administration of alprostadil to penile tissues without the psychosexual issues surrounding an injection. Recently, a multicentre randomised controlled trial compared nine months of therapy with intraurethral alprostadil with nightly sildenafil, starting one month postoperatively and followed by a wash-out period. While intraurethral alprostadil was very well tolerated, it did not show any benefit over sildenafil in terms intraurethral therapy appears harmless and may have some modest benefit for patients who are unwilling or unable to perform ICI.

VACUUM ERECTION DEVICES

Vacuum erection devices (VED) may be a safe and low-cost approach to penile rehabilitation. The device can be used on demand with a constriction ring for intercourse or used nightly with the idea of increasing blood flow to the penis and, possibly, preventing the collagen deposition and corporeal fibrosis.

Men randomised to use a VED with a goal of twice a week were no more likely to have return of were some benefits from using a VED. Men who used the VED for intercourse had benefits in terms of patient and spouse satisfaction. Additionally, only 14 per cent of patients who used the VED lost penile length compared to more than 60 per cent of those who did not.

Other experts have reported spontaneous return of erections in men using VED, but these studies are purely observational and not placebo controlled. Overall, VEDs are very well tolerated. We believe that VEDs may have a role in preventing loss of penile length and may be particularly useful in achieving sexual satisfaction for some couples early in the postoperative course. However, at this time, we do not see any evidence that the use of VEDs facilitates return of spontaneous erections.

EMERGING IDEAS IN PENILE REHABILITATION

Currently, the main focus of research on the cause of erectile dysfunction after prostatectomy has been on technical aspects of the nerve-sparing technique. Emerging data suggest that the prostate may have an endocrine role in the production and metabolism of androgens and that serum levels of dihydrotestosterone (DHT) and gonadotrophins may decrease after prostatectomy. In addition to damage to the cavernosal nerves, some of the effects of prostatectomy on erectile function could also be a result of loss of prostatic endocrine function. This theory is supported by similarities in structural changes to the penis after prostatectomy and after castration. Furthermore, men after prostatectomy commonly report decreases in libido and orgasmic dysfunction, which are generally associated with low androgen levels.

Testosterone therapy may improve erectile function in hypogonadal men who do radical prostatectomy. Testosterone supplementation in these men remains controversial, and we do not know of any studies examining the use of androgens to improve erectile or sexual function in men after prostatectomy. However, recent studies suggest that testosterone therapy may be safe in these men. In a small series of men with untreated, low-grade prostate cancer who underwent testosterone therapy, there was no evidence for an increased rate of disease progression. Additionally, elevated endrogenous testosterone and DHT levels do not seem to be associated with an increased risk of prostate cancer. Future research on the role of androgens in post-prostatectomy sexual function may lead to new concepts and therapies for penile rehabilitation.

SUMMARY

Penile rehabilitation after prostatectomy may help prevent structural changes to penile tissue and lead to faster and better recovery of erectile function. An effective approach to rehabilitation involves early intervention along with patient and partner counselling and discussion. Although clinical data are lacking, treatment with ICI may be an effective form of rehabilitation, but may be limited by penile pain and psychosexual issues. While efficacy has not been proven, many men may benefit from oral therapy with PDE5 inhibitors, intraurethral therapy, or the use of VEDs. Even in the absence of strong clinical evidence, we find rehabilitative techniques to be safe, and recommend that they be discussed with patients prior to undergoing prostatectomy.

KEY POINTS

- Erectile dysfunction, loss of penile length and orgasmic dysfunction remain common problems after nerve-sparing radical prostatectomy
- Recovery of spontaneous erections may take two years or longer. Penile rehabilitation aims to prevent hypoxia-induced deterioration of penile tissues during this period
- Any rehabilitation therapy is preferable to no treatment. We believe that discussion of the physiology of erectile dysfunction after prostatectomy and expectations for recovery is an important component of a rehabilitation programme
- Oral phosphodiesterase 5 inhibitors, used either nightly or on-demand, may offer modest benefits at best. Use of intraurethral alprostadil therapy may not offer additional benefits. We recommend offering additional therapies to motivated patients
- Intracavernosal injections (ICI) may prevent deterioration of penile tissues and encourage faster or better return of erections. Use of ICI as early as the first month after prostatectomy appears safe, but may be associated with pain or psychosexual distress for some patients
- Vacuum erection devices may prevent loss of penile length and allow for intercourse and sexual satisfaction early in the postoperative course. Data supporting a benefit in terms of assisting recovery of spontaneous erections are lacking
- Future research on the role of androgens in post-prostatectomy sexual function may lead to new concepts and therapies for penile rehabilitation

Prostate Heidelberg Cancer Support Group Meetings

While we are having to distance ourselves and are unable to hold face-to-face group meetings we are engaging speakers via video conferencing.

Guest Speaker

Tuesday 19 May 2021

Angela Mellerick, Nurse Unit Manager, Ambulatory Cancer Services, Olivia Newton John Cancer and Wellness Centre will talk about the role of the services in supporting people undergoing chemotherapy and other treatments to deal with the challenges of these treatments in ways that minimise the need for inpatient of emergency admissions and attendant disruption to patients' lives.

Prostate Cancer Trial Results

Lutetium-177-PSMA-617 in low-volume hormone sensitive metastatic prostate cancer, a prospective pilot study

DOI: 10.1158/1078-0432.CCR-20-4298

Abstract

Background: ^{177}Lu-PSMA-617 radioligand-therapy is a novel treatment for metastatic castration-resistant prostate cancer (mCRPC), which could also be applied to patients with metastatic hormone-sensitive prostate cancer (mHSPC) with PSMA expression. In this prospective study (NCT03828838), we analyzed toxicity, radiation doses and treatment effect of ^{177}Lu-PSMA in low-volume mHSPC patients.

Patients and methods: Ten progressive mHSPC patients following local treatment, with a maximum of ten metastatic lesions on ^{68}Ga-PSMA-11 PET (PSMA-PET) and serum-PSA doubling time <6 months received two cycles of ^{177}Lu-PSMA. Whole-body SPECT/CT and blood dosimetry was performed to calculate doses to the tumors and organs at risk (OAR). Adverse events (AE), laboratory(-toxicity) and quality of life were monitored until week 24 after cycle two; the end of study (EOS). All patients underwent PSMA-PET at screening, eight weeks after cycle one, 12 weeks after cycle two and at EOS.

Results: All patients received two cycles of ^{177}Lu-PSMA without complications. No treatment related grade III-IV adverse events were observed. According to dosimetry, none of the OAR reached threshold doses for radiation related toxicity. Moreover, all target lesions received higher radiation dose than the OAR. All ten patients showed altered PSA kinetics, postponed androgen-deprivation therapy and maintained good quality of life. Half of the patients showed a PSA response of more than 50%. One patient had a complete response on PSMA-PET-imaging until EOS and two others had only minimal residual disease.

Conclusions: ^{177}Lu-PSMA appeared to be a feasible and safe treatment modality in patients with low-volume metastatic hormone-sensitive prostate cancer patients.

Enzalutamide in men with MHSPC: focus on the Arches & Enzamet trials

https://www.researchreview.com.au/getmedia/90ad28fd-0f67-4d1c-b4e2-ad5464079254/Educational_Series_Metastatic_hormone_sensitive_prostate_cancer_the_ARCHES_and_ENZAMET_trials.pdf.aspx?ext=.pdf

This review covers current treatments in metastatic hormone-sensitive prostate cancer, Enzalutamide background and summaries of the ARCHES and ENZAMET trials.

The review includes guiding commentary from Professor Ian Davis, medical oncologist and Professor of Medicine and Head of the Eastern Health Clinical School, Monash University and Eastern Health, in Melbourne.

Learn to be your own researcher to make the best treatment decisions, by being proactive and an advocate for your own health

Internet Resources

Members have found the following websites useful

Prostate Cancer Foundation of Australia for guides & help
https://www.pcfa.org.au
https://onlinecommunity.pcfa.org.au/

Australian Cancer Trials Information on clinical trials
https://www.australiancancertrials.gov.au

USA Prostate Cancer Foundation (Guide) PDF guide for men newly diagnosed with PC
https://www.pcf.org/guide/

Us TOO International PCa Education (USA) USA PC support groups' information & newsletter
https://www.ustoo.org

Cancer Council Victoria for general support services
https://www.cancervic.org.au

ExMed Cancer Program Melbourne based 'best practice' exercise medicine program
https://www.exmedcancer.org.au

ProstMate (PCFA) A companion to record PC results

Beyond Blue for help with depression and anxiety
HELPLINE 1300 22 4636

Continence Foundation of Australia for assistance with incontinence aids
HELPLINE 1800 33 0066

PCRI Prostate Digest (USA) Prostate Cancer Research Institute supporting research and disseminating information to educate and empower patients, families and the medical community
https://pcri.org/insights-newsletter

PAACT Newsletter (USA) Patient Advocates for Advanced Cancer Treatments
http://paact.help/newsletter-signup/

A Touchy Subject
https://www.youtube.com/channel/UCdyuxGuAuCWJbe-kZvwVSzQ

The internet is a good source for research but it should not be trusted to give you answers for your personal care. Always speak to your doctor to clarify any medical advice.

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Max Shub Co-Facilitator
Peter Anderson Treasurer
Spiros Haldas Library
David Bellair Web Site
Michael Meszaros Welfare Officer
Sue Lawes Secretary/Newsletter

PHCSG Meetings 2021 10am – 12:30pm

Tues 16 Feb
Tues 16 March
Tues 20 April
Tues 18 May
Tues 15 June
Tues 20 July
Tues 17 August
Tues 21 September
Tues 19 October
Tues 16 November
Tues 14 December (including Xmas lunch)

Please note that all face-to-face meetings have been cancelled until further notice. Please check your email regularly for updates from the PHSCG Committee.

2021 PHCSG Articles

If you have any feedback or wish to include articles on specific aspects of Prostate Cancer please contact Sue at:

prostateheidelberg@gmail.com

January 2021

- Exercise Infographic
- Sexual Dysfunction & Shared Decision Making
- FDA Approves first Oral Hormone Therapy
- Prolonged ADT Reduces Cardio Fitness
- Reducing the Burden of Out-of-Pocket Expenses
- BAT Sensitizes CRPCa to Subsequent Therapy
- Targeting Bone Mets with Radiation in Oligorecurrent Men

Prostate Cancer Trials

- PEACE V:STORM
- UpFront PSMA Phase II
- NINJA

February 2021

- Advantages of Coffee
- Our Biological Clock
- Statins tied to Better Outcomes
- What's New in Inflammation
- New PC Management Techniques
- About the Patch Trial
- Eating a Colourful Diet
- Dose Painting
- Advancement in Focal Therapy

Prostate Cancer Trials

- Enza-P
- DASL-HiCaP Trial
- Lu-177-PSMA-617
- Adding Apalutamide to Radiotherapy & LHRH Agonist

March 2021

- Challenging Your Private Health Provider
- How Research is Prioritised – Norman Swan podcast
- Metastatic PCa – Don't Accept Complacency
- An mRNA Vaccine for Cancer
- Life After Treatment – Wellness Program
- Focal Therapy – If It Sounds Too Good to be True
- Immune Checkpoints on CTCs

April 2021

- Study finds cancer cells evade chemo by going dormant
- High Risk Localised PCa: Changing the rules
- Automated Pathological Assessment of PCa Biopsy Slides
- Final Results from TITAN Study
- SBRT for High Risk Patients
- Benefit of taking 1year of ADT after radiation for high risk PCa
- Novel Radiopharmaceutical beats Cabazitaxel in MCRPC
- Novatis announces phase III positive results
- Estrogen – Our Sister Hormone

Prostate Cancer Trials

- Enzalutamide With Lu PSMA-617 Versus Enzalutamide Alone
- Darolutamide Augments Standard Therapy for Localised Very High-Risk Cancer

May 2021

- Full on Kitchen Sink for High Risk Localized PCa
- Calcium & Vitamin D Supplements
- Favourable prognosis with adjuvant ADT after RT
- Healthy Lifestyle may offset Genetic Risk
- Additional Treatment Option
- New Type of Treatment could reawaken Immune Response
- Penile Rehabilitation
- Prostate Cancer Trial Results

2020 PHCSG Articles

If you have any feedback or wish to include articles on specific aspects of Prostate Cancer please contact Sue at:

prostateheidelberg@gmail.com

March 2020
- PCFA Consumer Advisory- Coronavirus and Cancer

April 2020
- Telehealth & Delayed Hospital Treatments due to COVID-19
- Fexapotide Triflutate (FT) injection – a new kind of focal treatment to extend time on active surveillance

Prostate Cancer Trials
- DASL-HiCaP Trial
- Evaluation of a mainstream model of genetic testing for men with prostate cancer

May 2020
- ADT May Offer Some Protection From COVID-19 in Men with Prostate Cancer
- TULSA – Novel MRI-guided ultrasound treatment destroys prostate cancer
- Whack-a-Mole A Treatment of Oligometastasis
- Long-term adjuvant ADT improves results of brachy boost therapy in unfavorable-risk prostate cancer patients
- Harnessing the immune system to control prostate cancer spread to the bone

Prostate Cancer Trials
- A study to see whether PET scans using a chemical called Exendin can detect metastatic PC
- Evaluation of a mainstream model of genetic testing for men with prostate cancer

June 2020
- Evaluating the Outcomes of AS in Gleason Grade 2 Prostate Cancer
- Advancing precision medicine for metastatic prostate cancer
- Impact of Primary Prostate Cancer Treatment with Subsequent Metastatic Disease
- Comparative Analysis & Survival Outcomes in a Real-World Practice Setting
- Fexapotide Triflutate (FT) injection – a new kind of focal treatment to extend time on AS

Prostate Cancer Trials
- Impact of 18F-DCFPyL PET scanning in patients undergoing post-prostatectomy Radiotherapy

July 2020
- Testosterone Therapy does not Increase the Risks of PCR or Death after Definitive Treatment for Localised Disease
- Association of Pre-Salvage Radiotherapy PSA Levels after Prostatectomy with Outcomes of Long-term Antiandrogen Therapy in Men with Prostate Cancer
- Testosterone Replacement in the treatment of Advanced Prostate Cancer
- Memorial Sloan Kettering Cancer Center PCa nomograms Prediction Tools

August 2020
- Advanced Prostate Cancer Algorithm
- Blood Test Predicts Response to PC Treatment (liquid biopsy)
- The Perils and Pitfalls of Treating PSA in PCa
- Reprogramming Immune Cells could Switch Defence into Attack in PCa
- Maintenance of Sexual Activity Following ADT

September 2020
- ProtecT Trial showing patient outcomes after AM, RP & EBRT
- Changes in Penile Length after RP
- Active Surveillance for PC – is it right for you?
- The final part of The Perils and Pitfalls of "Treating PSA" in Advanced Prostate Cancer
- Managing Erectile Dysfunction – A Patient Guide

Prostate Cancer Trials
- Efficacy and Safety of Pembrolizumab (MK-3475) Plus Enzalutamide Plus Androgen Deprivation Therapy (ADT) Versus Placebo Plus Enzalutamide Plus ADT in Participants with (mHSPC)
- Navigate: An online treatment decision aid

October 2020
- World Osteoporosis Day
- Lifestyle Factors and Chronic Disease
- Hormone Therapy for PC
- Early ADT for Recurrent PC Challenged
- Unexpected aPC weakness can be targeted by drugs
- Hijacking an Epigenetic Program
- New PC Research: Immunotherapy; Gut Microbiome
- Veyonda New Research on Survival Rates

Prostate Cancer Trials
- MIndonline - mindfulness

November 2020
- Life insurance & Genetic Testing
- World First Surgery in NZ
- Melatonin increases survival
- SBRT disease control
- Public vs Private Hospitals
- Early ADT for Recurrent PC challenged
- Enzamet trial results

Prostate Cancer Trials
- Randomised Phase 2 of sequential 177Lu-PSMA & Docetaxel
- Exercise for Heart Health

December 2020
- ACTA Trial Award
- Rethinking Metastasis
- ESMO Phase 1 AMG160
- Five Ways to Get it Right
- Immunotherapy Offers Hope
- SBRT Doubles Pain Response
- Elevated Streess Hormone Levels

Prostate Cancer Trials