“Testing and Treatment Roadmap (NCCN Guidelines)” (Rick Stanton) [#6]

April 27, 2022
Brad Power

Meeting Summary

Advanced prostate cancer patient Rick Stanton walked through a one-page summary of the NCCN (National Comprehensive Cancer Network) guidelines for advanced prostate cancer and illustrated it with his and Brian McCloskey’s treatment journeys. The NCCN guidelines are the “standard of care” - the evidence-based protocol for deciding on treatments for patients with prostate cancer. Rick started his summary of the NCCN guidelines after the prostatectomy step since he is focusing on the journey for advanced prostate cancer. From there, most branches in the NCCN decision tree depend on whether the patient’s PSA (prostate specific antigen, a blood test result), is rising or not, and whether their cancer has spread outside the prostate (metastasized).

Rick and Brian’s PSA rose after their prostatectomy, so they switched from observation (“watchful waiting”) and had the next recommended treatment: radiation and drugs (Lupron and bicalutamide/Casodex) that suppress androgen, the hormone that feeds the cancer. This androgen suppression treatment worked for about a year for Rick and Brian. Brian was doing so well, he and his medical team decided to take a holiday from the androgen suppressing drugs, and then after several months his PSA started rising rapidly. Rick’s PSA started rising rapidly after about a year. At this point for both, with PSA rising, and now both with metastases (cancer in other places besides their prostate), the NCCN treatment recommendation is to try one of several drug options. Rick chose a next generation androgen blocker (darolutamide). After a few months, it was clear that this wasn’t working for him, so he joined a clinical trial that had two arms: one for chemotherapy (docetaxel) and another with that same chemotherapy plus two other drugs (a PD1 inhibitor and an adenosine inhibitor). Unfortunately, Rick got the clinical trial control arm with chemotherapy only, which he has stayed on until today. It has knocked down his PSA. Brian chose another androgen-suppressing drug (abiraterone), which he is on now. It is keeping his PSA at a very low level.

The first four or five rounds of decisions in the NCCN guidelines are largely not personalized. They depend on whether the cancer has metastasized and the PSA level. It is only in the very advanced stages of prostate cancer that personalization (decisions which draw on genomic tests) enters.

In an upcoming meeting Rick and Brian will continue to talk about ways to (a) enhance the NCCN guidelines to refine this overview of the whole journey - a roadmap for communication between patients and doctors, (b) bring more testing and associated personalization earlier in the decision process, (c) add data on the efficacy of the treatment options, and (d) add a roadmap for steps beyond the end of the current guidelines.

Requests

“Testing and Treatment Roadmap (NCCN Guidelines)” (Rick Stanton) [#6]

- Do you have any feedback on Rick’s presentation on the decision tree for advanced prostate cancer testing and treatments? How could we improve it?
- Do you have contacts at the NCCN whom we could contact to explore the possibility of collaboration?

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Meeting Transcript

Brad Power: Today we’re focusing on advanced prostate cancer testing and treatment decisions and a decision tree that Rick has been working on with help from Brian. We want to get your feedback on how we can make it better. Some of you may know about the NCCN (National Comprehensive Cancer Network) guidelines. These guidelines are the standard of care for treatment. They are updated every six months. The NCCN publishes guidelines for a variety of cancers, including for advanced prostate cancer. Given all the learning that we’ve been doing, we thought we might be able to help in advancing the guidelines, and we could use it to illustrate where Rick and Brian have been on their cancer journeys and the decisions they face.

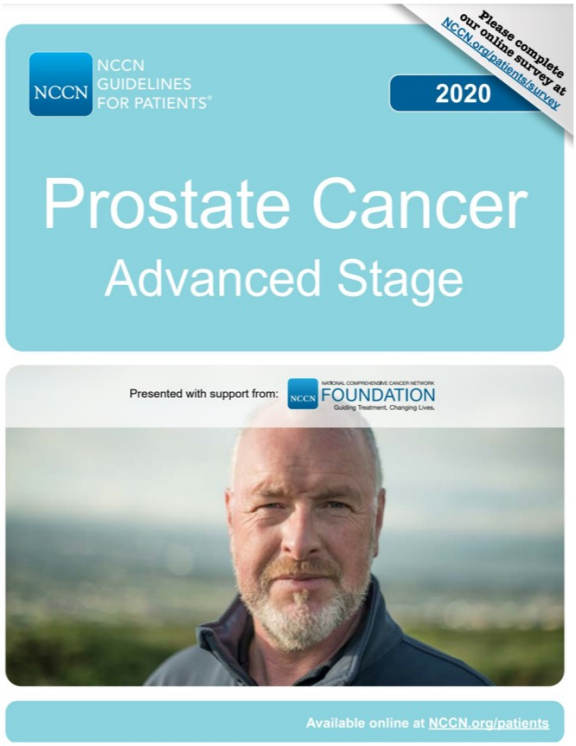
Rick Stanton: A little background: when I first got prostate cancer and had my prostate removed, I was at CIty of Hope. I talked to my medical oncologist, Dr. Lyou, who told me my next therapy steps were going to be completely in line with the NCCN guidelines. He said, “This is a comprehensive cancer center, and we do not follow ‘Wild West’ oncology guidelines.” I didn’t know what the NCCN guidelines were.

We are advocating for advanced testing and personalized medicine that help direct patient care. I wanted to know the value of advanced testing. What are these guidelines? Are they good enough? Where do they end?

“Testing and Treatment Roadmap (NCCN Guidelines)” (Rick Stanton) [#6]

Interpreted NCCN prostate cancer decision tree
(physician and patient guidelines)
April 2022
Rick Stanton

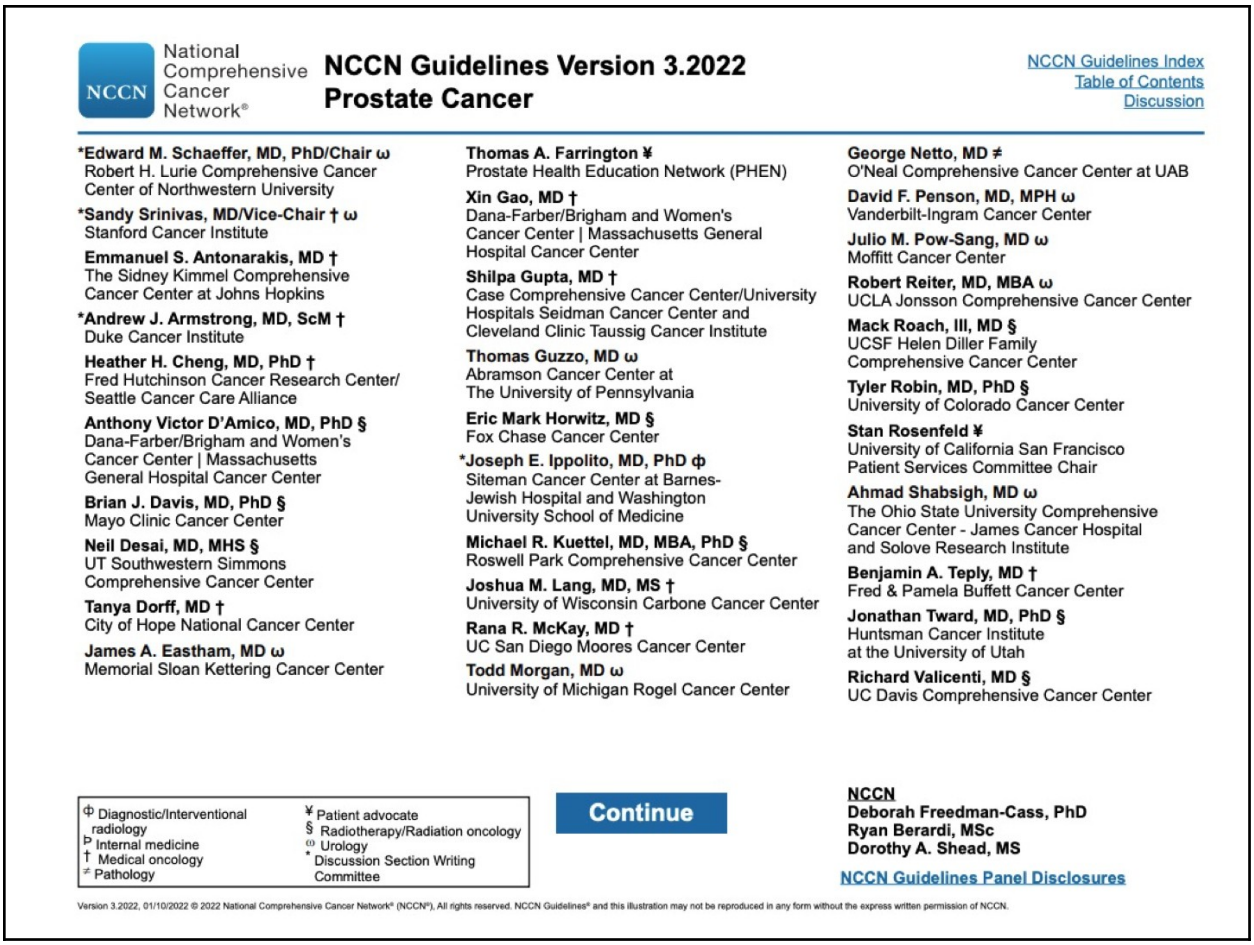
All suggestions welcome!
Evolution is happening fast!



https://www.nccn.org/professionals/physician_gls/pdf/prostate.pdf
https://www.nccn.org/patients/guidelines/content/PDF/prostate-advanced-patient.pdf

Rick Stanton: This image says 2020, but the guidelines I will be discussing are from January 2022. This is my interpretation of about a 60-page document. There are two documents. One is for physicians and one is for advanced patients.

“Testing and Treatment Roadmap (NCCN Guidelines)” (Rick Stanton) [#6]



National Comprehensive Cancer Network®

NCCN Guidelines Version 3.2022
Prostate Cancer

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NCCN Guidelines Panel Disclosures

Rick Stanton: A shout-out: I noticed (and I didn’t know this before I started laying out this decision tree) that Tanya Dorff, who is guiding me and Brian, is one of the authors, and so is Rana McKay, who is also guiding us. I look across this list of authors, and it’s just about as prestigious as it gets.

Rick Stanton: So here’s my interpretation of the NCCN guidelines for advanced prostate cancer.

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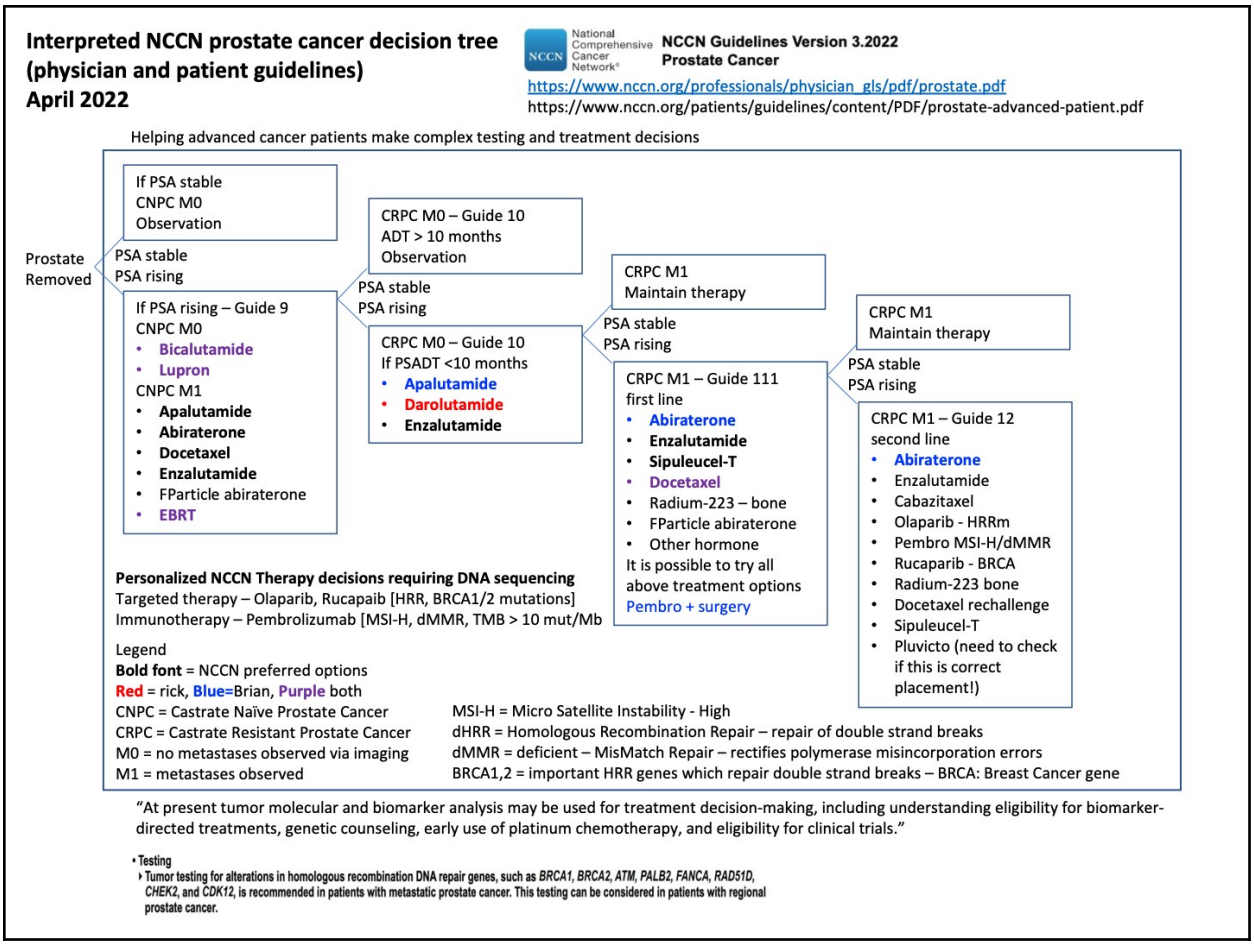

This is a decision tree. It’s boiling down 50 or 60 pages of therapeutic decisions provided in the NCCN guidelines into one page.

I start with “prostate removed” because it’s for advanced prostate cancer. There is more in the guidelines about when to take out a prostate.

If your PSA is stable after your prostate is removed, then you’re called “castration-naive prostate cancer” (CNPC) and M0 means that there is no evidence of metastases. (There are some acronyms here, which I have spelled out in the notes at the bottom.)

At this point – if there is no evidence of metastases and you are “castration naive”, which means you have not yet gone on androgen-deprivation therapies, and your PSA is stable – then you wait.

Brian and I were both unstable. When my prostate was removed my PSA was 0.6. 0.2 and above is the definition of recurrence. Right after I had my prostate removed I was not stable. My PSA was going up. I fell into this next category: my prostate is removed and my PSA is rising. So now, what do we do about it?

This is Guide 9 in the NCCN guidelines. There are two choices here. Again this is castration naive prostate cancer and M0 (no evidence of direct concentrated metastases on the imaging), but I did have a rising PSA. M1 means that there is evidence of metastases.

The guidelines say, and this is what Dr. Shen (UCLA) told me, put me on apalutamide.

The bold is the NCCN “preferred” options.

Within these options there is a lot of bold.

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In this depiction, both Brian and I shared the first step, which is shown in purple.
In the second step, we deviated. I’m in red.
Apalutamide is an androgen-deprivation therapy.
Lupron is another. Lupron is a shot. Darlutamide is an expensive pill (~$18K per month - Bayer - the cost for me was totally covered by a combination of Cigna insurance and Bayer patient assistance program - THANK YOU!!). Lupron is meant to knock down the androgen feeding the cancer, and Darlutamide is a pill that blocks the adrogen receptor on the tumor cells.
This should look very familiar to many advanced prostate cancer patients.

Saed Sayad: Do you have any statistical information about the success or failure rate at each stage?

Brian McCloskey: I was surfing yesterday with one of my dear friends, Ryon Graf, of Foundation Medicine. In between catching waves we were talking about how to improve the NCCN guidelines. He has his hands on tons and tons of data. And that’s one of the directions we want to go. We discussed bringing more data to the guidelines. I’m getting to the punch line here. This is such a rudimentary guide, and we think there are opportunities for us – I’ll keep it high level here – to bring more data into this, and response data is part of what we would love to add. Ryon could help us tremendously in that effort by looking at real world evidence to determine what the response rates are for each of these different scenarios.

Saed Sayad: And also if we can find any genotypes for success or failure?

Brian McCloskey: He’s the guy. He has a lot of information. We need to peel the onion on that to understand exactly what genomic information they do have. But, at a minimum, it’s going to be a great place for us to start.

Rick Stanton: As we go across this chart, from left to right, you’ll see that the rightmost is the second line therapies, and within the NCCN guidelines that’s where genomic indications first come into the decision-making. It doesn’t kick in until you are castrate resistant and metastatic and at the second line. For example, taking pembro, which is a PDL1 blocker, is based on genomic information. We’ll get there.
Both Brian and I got external beam radiation therapy (EBRT) as our next step, called “salvage” radiation (radiation given after a prostatectomy), and I also had SBRT (Stereotactic Body Radiation Therapy). We were hopeful that this would clean things up and confine this to the pelvis area.
But it didn’t for us.
After we left this box, we were told, “You have an incurable disease. You will manage this until the end.”
For me bicalutamide and Lupron lasted for about a year before my PSA started rising.
Brian: How was that for you?

Brian McCloskey: It was about the same for me. It was a little hard to tell because it overlapped with the radiation. Which was providing the benefit? It was a little muddy.

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Rick Stanton: After a year, after the radiation, my PSA started going up fast. It was clear that the bicalutamide was not controlling my disease anymore.
And then we go to the next box. This is Guide 10. Now I’m castrate resistant because I’ve been exposed to androgen deprivation therapy. My PSA doubling time was less than ten months. It was like three weeks. This was very scary. It was very aggressive.
These are the three preferred options: apalutamide, darolutamide, and enzalutamide.
I was told that darolutamide was the newest generation androgen blocker and with the least side effects. So I went on darolutamide. If I had been stable, I would have just been in observation. But the decision was that I needed to go to the next step, which was darolutamide, which unfortunately didn’t work very well for me. I only stayed on darolutamide for four months, and my PSA was higher than when I started. I didn’t get a benefit.
Brian, I’ll let you weigh in on your apalutamide experience.

Brian McCloskey: I got about 15 months of benefit. Then I decided with my medical oncology team that I was going to take a holiday from all androgen deprivation. When you’re on apalutamide, you stay on Lupron. I took a complete holiday from both. Unfortunately, within about three months my PSA went from undetectable when I ended apalutamide, it began to skyrocket, to a doubling time of two- to three-weeks, so very aggressive.

Rick Stanton: This was not good for me. At least you got a little time. Darolutamide wasn’t working for me.
I had some PSMA (Prostate Specific Membrane Antigen) scans, which check for metastases. My PSA was skyrocketing, so something was going on. I had gone from M0 (no detectable metastases from a scan) to a PSMA scan showing 4 or 5 lymph nodes that lit up.
So now I graduated to “nodal positive”, M1. This was a scary time for me. I felt as a cancer patient I was falling, and I needed a parachute. This transition didn’t seem proactive. It seemed reactive. My oncologist was surprised that I didn’t respond very well to darolutamide. What do we do? It shows from my journey, I went onto docetaxel. My medical oncologist told me, “You’re falling fast, and we need to hit you with something that is going to catch it right now. Your best bet is docetaxel.” I also was able to sign up on a clinical trial, as advocated by my medical oncologist team, led by John Shen at UCLA, that included docetaxel, a PD1 inhibitor, and an adenosine inhibitor. This clinical trial was being run by Arcus Biosciences. Some of you may recall that I worked at Amgen for 17 years, and I worked with Terry Rosen, who is the CEO of Arcus. I actually reported to him at Amgen. I know the science. I know his rigor. I suggested this trial, and Dr. Shen agreed - my care at UCLA transitioned to Dr. Sandy Liu who ran the Arcus clinical trial at UCLA.
Unfortunately, I got the docetaxel-only arm. That was a bummer, to say the least. I was hoping to get the docetaxel and immunotherapy arm, which would have a synergy. But those cards didn’t fall my way.
Other options as shown on the chart are: (Abiraterone, Enzalutamide, Sipuleucel-T, Docetaxel, Radium-223 – bone, FParticle abiraterone, Other hormone. It is possible to try all above treatment options Pembro + surgery.)

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I didn’t have any evidence of bone metastases, so radiation (radium-223) treatment wasn’t indicated.
I am on docetaxel currently. I’ve just finished seven rounds. It has held my disease progression in check. I wish it would have shrunk more, but at least it is stable. Right now, stable is wonderful.
Brian, what is your story?

Brian McCloskey: If you go back to the prior box. Rick was on darolutamide, and I was on apalutamide. I took a holiday beginning in March of 2020, and within 3 months I went from a PSA of 0.02 to 0.77. So just blazing fast. There was no evidence of disease when my PSA was at the 0.02 level in March, and within three months I had six metastatic lesions identified by whole body MRI, PSMA PET, traditional CT scans, etc. We decided that we would do surgery. My mets were entirely in soft tissue. I have no bone mets, and never have had bone mets (which are typical for prostate cancer metastases.) They were in my peritoneum (the membrane covering the abdomen), underneath the belly button area. Prior to surgery my PSA had gone from 0.77 in June to something like 2.05 in August of 2020 when I had my surgery. After surgery my PSA dropped from 2 to 1. We knew we didn’t get all of it. They removed 6 lesions, but there weren’t clean margins. And I also knew that I had some caking in the peritoneum that they couldn’t surgically remove. I knew we were going to have to have some kind of systemic therapy.
If you look at the box with CRPC M1 from NCCN Guideline 111, my systemic therapy choice was docetaxel, similar to Rick, but they added pembrolizumab, because my DNA suggested that I had one targetable mutation (PBRM1), and we thought that we could hit it with Keytruda (pembrolizumab) and with docetaxel. I did 6 rounds of docetaxel, and finished the docetaxel in January of 2021 and continued with pembro until October of 2021. The reason I stopped was because my PSA began to rise.
That gets us into the next box, NCCN Guideline 12.
After my PSA began to rise, we decided to go onto abiraterone.
I started in November of 2021, and I am currently on abiraterone. I’ve got 5 or 6 months under my belt. My PSA has gone from a 0.91 to 0.45. I’m going to get some labs today. We’ll see if I’m still responsive and stable.
After abiraterone, what do we do next?
You can see on the chart that there is still a family of drugs that we could go after. There is additional chemo. One oncologist recommended that I try darolutamide, even though there is evidence that would suggest that the transition across second line hormone therapies is not terribly effective. Another option is Pluvicto, a radioligand which recently got FDA approval. And there are others.

Brad Power: Clustering the treatments in broad strokes: after the prostatectomy and radiation, you hit it next with androgen deprivation, in the first and second boxes. Then chemo (docetaxel). Then there are immunotherapies (PD1, PDL1). Pluvicto is a different treatment methodology, using radiation to attack unique prostate-specific antigens. Some of these drugs are similar in their attack pathway?

Rick Stanton: That’s correct. Prostate cancer is a hormone-driven cancer. So shutting off androgen is the first step. When that is running out, there are still some other treatment options, like chemo, which kills all fast-dividing cells.
I don’t know where Pluvicto fits. It’s not in the NCCN guidelines yet. It was recently approved. It is a tremendous hope for me and Brian. This is a radioligand (a radioactive biochemical substance) that targets and attaches to PSMA, a surface marker antigen unique to prostate cancer cells, to deliver the kill.

Saed Sayad: This is a very useful decision tree. But it seems branching only depends on the PSA.

Rick Stanton: That is correct. I tried to faithfully lay out the NCCN guidelines. This is only PSA-based decision-making.

Saed Sayad: It’s good as a base. But it leaves a lot of room for improvement.

Brian McCloskey: There are actually two decision-points. One is if you are metastatic or not, and the other is PSA. It’s two-dimensional.

Rick Stanton: In the left 3 boxes, there is no personalized medicine going on. Maybe you could say it was personalized if you had metastases to the bone or not. But otherwise, largely, in my interpretation as a patient figuring out what to do next, the standard of care in the left 3 boxes has nothing to do with individual patient personalization. It is based on population statistics, which is fine if it works. Lupron is not cheap, but bicalutamide is cheap. So it’s easy to get reimbursed. After you graduate from these 3 boxes, you start to get into guidelines that bring some personalized decisions. I’ve had my tumor sequenced, and so has Brian. Sequencing is recommended by the guidelines.
For this rightmost fourth box, you could do abiraterone. That is independent of any personalization.
But if we get into olaparib, tumor testing for homologous recombination repair genes could guide whether this treatment might be indicated. For example, I have a CDK12 mutation, one of these genes. Testing for homologous combination repair genes is included in the guidelines, and here are the genes which could determine if you might be a candidate for olaparib, which is a PARP inhibitor.
I don’t want to get into every detail, but if you’re microsatellite instability high, you might have a lot of mutations, which might attract your CD8 T cells for the kill. They might be immunosuppressed by PDL1, so you might be a candidate for a PD1/PDL1 inhibitor (pembrolizumab).
Now we’re talking about some personal guidance.
If you have a mutation in a BRCA gene (which stands for breast cancer), it is an important one of these homologous recombination repair genes, which repairs double stranded breaks, then you are a candidate for rucaparib. We are now getting into decisions based on your personal genomics, which is a good thing.

Pluvicto could also be considered personalized, since it depends on being PSMA positive, although population statistics argue that PSMA is highly expressed. You can verify this from a PSMA scan. If your PSMA scan lights up like mine does with lymph nodes, then there’s no question you are PSMA positive, and this would be a good choice.
I wish I would have known this when I was at the front end of my diagnosis. I had no idea of what was coming ahead or what the strategy was when my doctor put me on my first treatment. “We will put you on this stuff, and we will see how long it goes. And hopefully it goes for a long time.”
I hope this has brought some light in that regard.

Brad Power: Let me ask the four prostate cancer patients who are on this call for their reactions. Steve Abbott? That is the intended audience.
Was this useful to have it laid out for you?

Steve Abbott: I would agree. It would be very useful. I found this whole tale frighteningly similar to my journey as well. I’m pretty much where the guys are. For someone like Mike on the call, who is not quite where we are yet, I would think it would be very, very helpful.

Mike Yancey: This is very, very helpful to me because I wasn’t aware of all these boxes of treatments based on being metastatic or not, and whether your PSA is stable or rising. In my case, even though my diagnosis was immediately in the M1 (metastatic) area, with PSA rising, the Guide 111 box, with docetaxel and Lupron, which knocked the PSA down significantly. It seems to be holding at this time. That’s why I am pushing to make some changes to get sequenced, etc., to be doing some planning for what the next step might be. So this is very helpful.

Jan Sobieralski: I am castration sensitive, so it (androgen deprivation therapies) has worked very well for me. I would like to see the Pluvicto pushed up earlier in the treatment options. The research I have done shows that Pluvicto is much more effective in the earlier stages of your treatment than the later stages. This is according to Dr. Moyad from the Prostate Cancer Research Institute in California. He has treated a lot of patients overseas, like in Australia and Germany, over the last few years. They found that people who are treated earlier have come out much better, almost full remission. Whereas people who were castration sensitive did not respond well: 30% did not respond, 30% responded well, and 30% were neutral.

Stacy Hurt: Rick and Brian, thank you for sharing this. I am not familiar with prostate cancer, so this is great to learn.
You bring up a really interesting point, that when you were starting treatment, the oncologist didn’t lay out the NCCN guidelines for you. You went and sought out this information as informed advocates for yourselves to find out what the protocol was.
I do a lot of broad level advocacy work, and it is making me wonder, as a stage IV colon cancer survivor. My oncologist never laid this out for me.

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I look at process and workflow. How useful would it be for our oncologists to lay this out for us and say, this is step by step? When you are told you have cancer, it’s all out of your control, and it would be so nice to have some level of control, or information, or knowing what to expect.
I would like to get your or anybody’s feedback on that.
Is it too much information to know at the beginning? Or should we give patients the option of saying, “this is the pathway we will be following”?

Brian McCloskey: This touches on a lot of things. We spoke to Steve Abbott over a week ago about the need to improve communication between patients and doctors. This is a fundamental tool to help that communication between the patient and the doctor.
I would go one step further. An informed patient can really help his or her doctor. Rick and I have gone off and worked with life sciences companies to do tests, for example RNA seq analysis. And that RNA seq analysis has helped us to identify potential targets. And when we identify those potential targets, that can come into play in the treatment options that we have. The other thing is that with testing we can determine whether or not a family of drugs will be useful for us. This is what we are probing on.
For example, there is a test for AR-V7, which is from Epic Sciences, and there are others that can test for AR (androgen receptor, the driving receptor of prostate cancer) amplification. This class of tests can help inform a patient and doctor whether or not the patient will respond to any of the second line hormone therapies in the second box, Guide 10, on this chart. If we tested negative for AR-V7, we could potentially write those off and move on to another, more responsive treatment. There are others, but that’s just one example.
We think this is an excellent tool to facilitate the communication between the patient and the doctor.

Mike Yancey: I would add to Stacey’s question. If I had been given this on day one, it would have been overload. I would not have been able to understand it. It would only be after I had some preliminary education for it to be useful to me. It’s going to be very useful, but it’s going to take several sessions of education to get where you need to be.

Saed Sayad: You didn’t need to have the whole decision tree. Based on your situation, you could have chosen just one piece. For example, if you started from the root node, then you just need to know about the first two nodes after this. This is going to be a great tool that you need to build on top of this. Many of these branches are going to be expanded.

Brad Power: There can be personalization in treatment, and there can be personalization in education. Some people are going to be ready for this, and some people won’t, and some will be ready at different times. They may want to hear it in different ways.

Pradeep Mangalath: Brian and Rick, this is very informative. I agree with Stacy that it’s very important to communicate with the patient. My wife was diagnosed with breast cancer, and we didn’t have the NCCN guidelines or knowledge. We asked a lot of questions to our oncologist to get a full sense of what the treatment roadmap might look like. But I think that’s rare.

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My question for Brian and Rick is, “how frequently did you have your PSA measured over the course of your roadmap?” My guess is that would inform your progression based on a single marker, which is PSA.

Rick Stanton: When I was on the left side, I was typically being measured every month. I also had PSMA tests, which I pushed for and paid for. It’s expensive. $3,000 is expensive, but my life is worth more. I was lucky to have it. The longest break I had was 2 months. The shortest is 3 weeks. I’m measured now every 3 weeks. I am on an Arcus clinical trial, so I get scanned every 3 months. My last scan was OK. My decision about when I’m no longer responding to docetaxel is a combination of how I feel, PSA, and the scans.

Pradeep Mangalath: If a patient was in the top boxes (observation or maintenance therapy), would it be typical to come into the clinic every month to get your PSA measured?

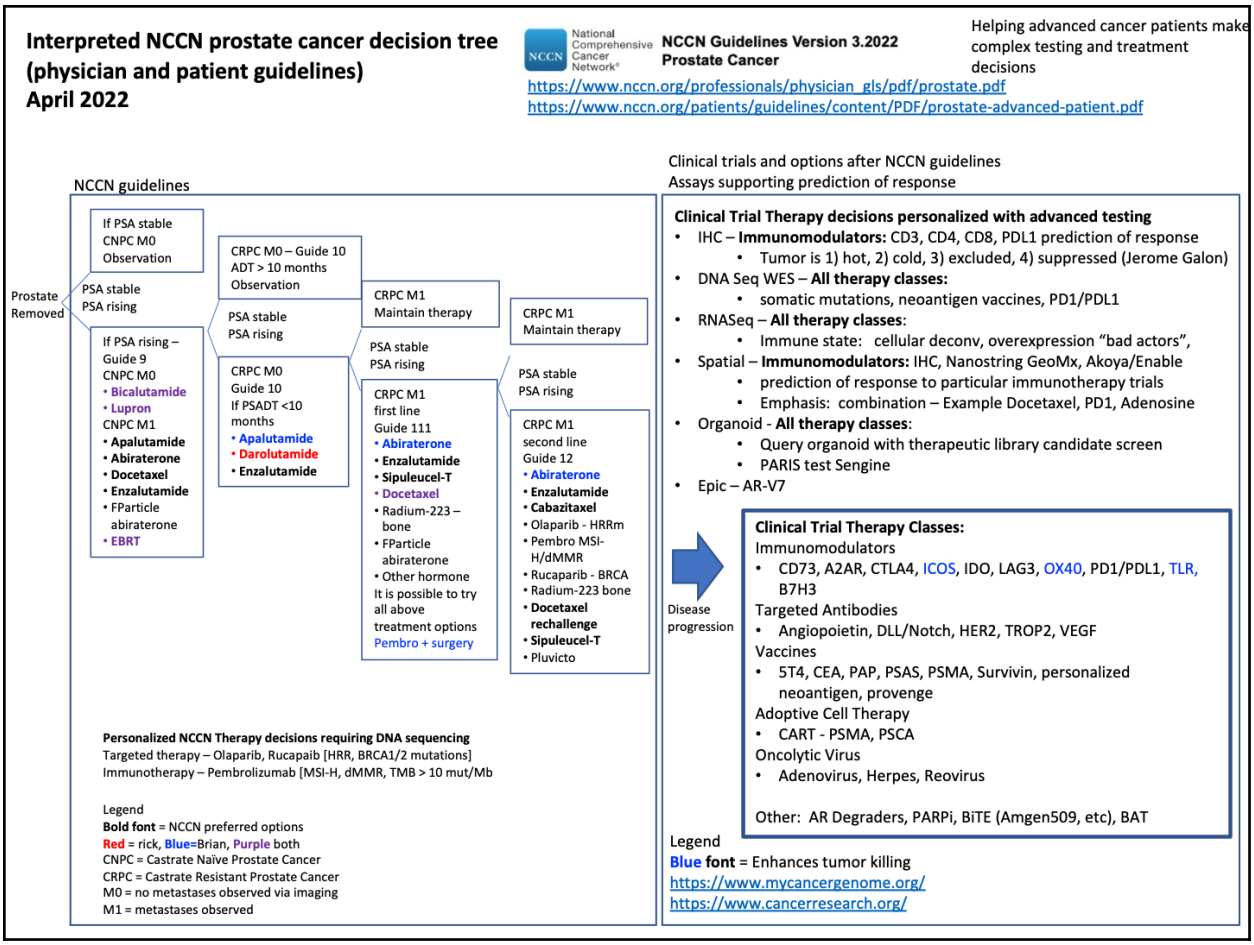
Rick Stanton: 2 months was my longest between tests. If your PSA is stable or going down, you get a little longer leash.

Brian McCloskey: There is testing anxiety waiting for the results of each test. I will go get my labs today, and I’ve got a little bit of testing anxiety. Am I going to continue to decline, or am I going to start to see that it’s going to go up? For me, I always pushed for more data. “Take my blood every other week.” I just did a quick look. Over the last 5 years, I have had about 50 labs. It’s about just over once a month or so.
Rick is right: if your PSA is stable, doctors always want you to wait a little bit. That was my case just over a year-and-a-half ago, and I saw my PSA rise at an insane rate. I waited about 2 or 3 months at one point, where I had been so diligent. That was a mistake. That’s when I developed these 6 lesions.

Jeff Waldron: We were talking about how early a patient should be investigating the NCCN guidelines. Rick and Brian are seeing the top experts in southern California. You are also educating the providers. In a rural area, or not at an academic medical center, there may be a need to be aware of the NCCN guidelines earlier to help educate the provider. It may seem counterintuitive to the people on this call, but many may not be in the situation of being at an academic medical center.

Rick Stanton: I’m anxious to show this next slide because this is the end of the standards, but it’s not the end of your life.

“Testing and Treatment Roadmap (NCCN Guidelines)” (Rick Stanton) [#6]



Rick Stanton: What happens when you graduate from Guide 12? Where do you turn?
This was the emphasis on helping advanced prostate cancer patients make complex testing and treatment decisions. What do we do after this?
This is the first cut. It’s not mature.
On the left side you see the NCCN guidelines, and on the right side you see clinical trials, which is our hope.
I’m a huge fan of immunotherapy. I feel it’s our only hope for a cure.
Maybe Pluvicto is another possible cure. It’s on the right because it’s not in the guidelines yet, since it’s so new.
We could spend a whole session on this slide.
In clinical trials, there are classes of drugs that are being matured. Some of this information was obtained from cancerresearch.org.

- Immunomodulators: Some with the greatest hope are immunomodulators. Immunomodulators are proteins in the tumor molecular environment. Those in black are immunosuppressive, and in blue are tumoricidal. A common one is PDL1, Programmed Death Ligand 1. Brian is already on it.
- Targeted antibodies is another class. I don’t know that much about these. From my work at Amgen, I know what VEGF is, but why it makes sense for prostate cancer I have got to learn. Some of this I’m not an expert on.

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- Vaccines: We know about PSMA and perhaps personalized neoantigen vaccines. I can explain more on another day.
- Adoptive cell therapy: Probably everyone has heard of CAR-T.
- Oncolytic virus: I know about this in other cancers, but not so much in prostate cancer.
- Androgen receptor degraders

These are the clinical trial classes. They may not be complete, but it is a decent overview.
The most hope for me is in the immunomodulators.

Brad Power: What questions or requests would you have to react to this?

Rick Stanton: Out of all these classes, what do you pick if you’re at the end of these NCCN guidelines, and your disease is still progressing? You still want to live.
Here is the testing that this community will use to inform which class of therapy or clinical trial will you go on? You wouldn’t want to pick something wrong. We are way out of population statistics. We are in personalized medicine. What test to figure out what personalized therapy would guide that decision?
Immunohistochemistry and spatial analysis will inform immunomodulator therapy decisions. DNA and RNA sequencing will inform all classes of therapy decisions. Organoid studies - all therapy classes. I don’t know about Epic.
Did I get this communicated? You’re at the end of the guidelines. You’re out into clinical trial land. You need some testing to make that decision on which one makes sense.

Brian McCloskey: We need to set up where we’re going. We are integrating testing into the treatment decision process. We are currently working with a number of companies to get them to present at this forum. For example, we’re working with NanoString to get them to present. We’re working with a company called SEngine around organoids. Hopefully they will present soon. We can probably pick up some people from Epic to help us understand AR-V7.
This is where we are going to help educate us, and this entire group on how to better integrate these testing options into treatment decisions. And along with that we are working with Bob Gatenby, an evolutionary biologist from the Moffitt Center, to help us think about treatment decisions not as tactics, but strategically. And other people that will provide a lot more context for how we can improve these guidelines, and to drive the hope of personalized medicine.