


Solving the Puzzle

Prostate Forum

Hormonal Therapy, I

Volume 9 Number 5

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Puzzle Points

-  Clinical trials show that hormonal therapy can be an extremely effective treatment for prostate cancer at various disease stages.
-  Hormone resistance is the result of a mutation, a change in the genetic material in the cell, which allows the prostate cancer cell to grow at very low testosterone levels.
-  Drs. Scardino and Scher at Memorial Sloan Kettering in New York City found that half of their patients were still responding to hormonal therapy at the 10-year mark.
-  Time and again, hormonal therapy clinical trials have reported shrinkage of measurable prostate cancer metastases.
-  I find it very puzzling that most urologists don't measure testosterone in their patients and that nearly all fail to measure dihydrotestosterone.

THE DANGERFIELD OF PC TREATMENT

It is an unfortunate fact that in some circles, just like Rodney Dangerfield, hormonal therapy gets no respect. However, the clinical trials show that hormonal therapy can be an extremely effective treatment for prostate cancer at various disease stages. Yet there are more misconceptions about hormonal therapy than any other area of prostate treatment. For the most part, these misconceptions paint a pessimistic picture of hormonal therapy's effectiveness and often lead to an unfounded sense of hopelessness in many patients. These misconceptions often lead patients to avoid effective methods of controlling their diseases. But *why* are there so many misunderstandings about hormonal therapy? Well, I think the problem has its root in the fact that the pace of prostate cancer research has been so overwhelming that it is impossible for any one physician to keep up with everything published on the disease. Physicians tend to read only those prostate cancer papers that directly relate to their own specialty. In other words, surgeons tend to read about advances in surgery, radiation therapists about radiation, and medical oncologists about chemotherapy. Unfortunately, while each of the above specialties administers hormonal therapy, none make it a central focus of their practice.

In the next two issues of *Prostate Forum* we aim to provide an accurate and updated view of hormonal therapy.

Myth #1

Responses Only Last 18 months

This is one of the more persistent myths in the field and I can't understand how it gained such wide circulation among patients and physicians. As far as I can tell, the idea dates from a paper published in 1989 by David Crawford. Crawford conducted a large, randomized controlled trial comparing Lupron alone to Lupron + Flutamide (Eulexin). The patients on this trial had been diagnosed with prostate cancer prior to the advent of PSA screening and therefore had more advanced prostate cancer than generally seen today. Dr. Crawford and his colleagues classified patients according to whether they had advanced, moderate, or minimal disease according to the standards of that time. Those with advanced disease had widespread bone metastases and suffered from significant symptoms. On average, these patients became resistant to hormonal therapy after just over 8 months. Those with moderate disease had cancers that spread throughout the skeleton, but did not have any symptoms. These patients became resistant to hormonal therapy after an average of 18 months. But you may very well not have an average result. While you may do worse, you may also do much better. This is one of the reasons why the average number is of so little value to patients.

Patients with widespread bone metastases need to know that there's a chance their cancers may continue to respond to hormonal therapy even after the oft-cited 18 month mark.

I think the common assumption that hormonal therapy lasts 18 months comes from the results seen in the patients with what was then considered moderate disease—or widespread bone metastases without symptoms. But there are many reasons why it is inappropriate to generally cite this statistic.

First, even in 1989, 18 months was just the average. In Crawford's study, half the patients continued to respond after 18 months and a significant percentage were responding at 5 years. I think it's important for patients with widespread bone metastases to know that there's a chance their cancers may continue to respond to hormonal therapy even after the oft-cited 18 month mark. Still, time and again I see men arrange their financial affairs with the assumption that they won't live another 5 years only to find themselves impoverished when they're lucky enough to beat the 18-month figure. But there is no reason you *have* to have the average result!

Some physicians even cite the 18 month figure to men who only have lymph node metastases or even simply a rising PSA post radical prostatectomy or radiation therapy.

The second problem with the 18-month figure is that I see it quoted to men who do not have widespread bone metastases. Some physicians even cite the figure to men who only have lymph node metastases or even simply a rising PSA post radical prostatectomy or radiation therapy. Of course, these patients don't have nearly as extensive prostate cancers as those in the Crawford study and are likely to continue to respond to hormonal therapy for many years to come. Table 1 (see next page) lists our best guess of the *average* duration of hormonal therapy responses for patients with metastatic prostate cancer of various degrees of severity.

Why do patients with lymph node metastases do so much better if they've had their prostate gland removed? The best study to shed light on this issue is one from MD Anderson Cancer Hospital published in 1994 by Zagars, et al. In this study, patients with lymph node spread were placed on hormonal therapy and followed until they recurred. (Note that these men did not have their prostate glands removed.) Once a patient recurred, researchers recorded where hormone-resistant disease emerged. In more than half the cases, hormone-resistant disease first emerged in the prostate gland. I think this makes sense. Hormone-resist-

Table 1. Average Time to Hormonal Therapy Failure

(Point in time when half the patients have developed advancing disease)

	<i>Percent Still In Remission</i>
Widespread metastases and symptoms	8-9 months
Widespread metastases, no symptoms	18 months
Bone metastases, pelvis, lower spine	4-5 years
Lymph node metastases prostate in place	7-8 years
Lymph node metastases, prostate removed	50-95% @ 10 years <i>(Depends on the number of lymph nodes involved)</i>

In most studies of PSA screening, widespread metastatic disease is often identified in the first and sometimes the second year after initial diagnosis, but thereafter an overwhelming majority of patients have cancers that appear to be confined to the prostate gland. In fact, the worst situation you are likely to see with any frequency is patients with disease that has extended to the prostate capsule,

ance is the result of a mutation, a change in the genetic material in the cell, which allows the prostate cancer cell to grow at very low testosterone levels. In general, mutations are random events that occur once in every 1-10 million cells. Thus, all other things being equal, you would expect hormone resistance to emerge at locations where there are a large number of cancer cells. (At the time of diagnosis, patients with lymph node metastases still usually have the largest bulk of cancer in their prostate glands.)

If the prostate gland is an important source of hormone-resistant prostate cancer, then removing the prostate gland should improve hormonal therapy's results. And indeed, at the Mayo Clinic, Horst Zincke makes it a practice to remove the prostate glands in those patients with lymph node metastases. His results, shown in Table 1, represent a dramatic improvement in the duration of hormonal therapy response. A randomized controlled clinical trial published in 1999 by Edward Messing confirms the Mayo Clinic results.

Since 1989, PSA screening has revolutionized the field of prostate cancer diagnosis and treatment: we're diagnosing cancers earlier and earlier.

seminal vesicles, or pelvic lymph nodes. Even these patients can be treated successfully with hormonal therapy combined with aggressive external beam radiation therapy + radioactive seed implantation, called brachytherapy.

More often than not, a man considering hormonal therapy has a PSA increasing after surgery or radiation therapy and metastases too small to see with an CT or MRI scan.

What this means is that more often than not, a man considering hormonal therapy has a PSA increasing after surgery or radiation therapy and metastases too small to see with an CT or MRI scan. We have no published series that accurately reports response duration in these patients, but I suspect that they would do as well or better than those with documented lymph node metastases after surgical removal of the prostate gland. Indeed, one series has been presented in abstract form, but not published. Drs. Scardino and Scher reviewed their experiences at Memorial Sloan Kettering in

New York City and found that half of their patients were still responding at the 10-year mark. Based on my clinical experience, this result looks to be approximately correct.

My conclusion is that hormonal therapy is far more durable than generally thought. In fact, almost all men who recur after radical prostatectomy or radiation therapy will continue to respond to hormonal therapy after five years and about half will continue to respond after ten years.

Myth #2: *Hormonal Therapy Doesn't Kill Prostate Cancer Cells*

Over the last several years, a growing number of patients tell me they've been told that hormonal therapy doesn't kill prostate cancer cells, but just stops cancer growth and artificially lowers the PSA test, thereby fooling us into thinking cancer cells have actually died. I find this myth very strange. Time and again, hormonal therapy clinical trials have reported shrinkage of measurable prostate cancer metastases. Depending on the clinical trial, up to 30% of patients enter complete remission, which means that all detectable prostate cancer has disappeared! On the other hand, it is certainly true that some patients do not respond to hormonal therapy and among those patients, hormonal therapy hasn't killed a significant number of prostate cancer cells.

The PSA test can be deceptive during hormonal therapy.

It is also true that the PSA test can be deceptive during hormonal therapy. With the drop in testosterone that follows Lupron, Zoladex, Eligard or Trelstar administration, PSA values often decline to below 0.05 ng/ml by the third month. If you look carefully at the extent of the cancer at that point, you may see little or no change in the size of the cancer in the prostate gland, lymph nodes, or other sites. Instead, the size of the cancer at these sites gradually decreases over a period of many months,

often taking 9-12 months to reach maximum shrinkage. However, it is true that a rapid and dramatic fall in the PSA is a good thing and indicates that the patient is a good candidate for subsequent, equally dramatic cancer shrinkage.

How Cancer Cells Adapt To Low Testosterone Levels

The concept behind hormonal therapy is that prostate cancer cells depend on the male sex hormone, testosterone, to both grow and survive. Actually, prostate cancer cells respond to both testosterone and dihydrotestosterone and these hormones are called androgens. Testosterone and dihydrotestosterone freely cross the outside membrane of the cell and enter the fluid inside, called the cytosol. In this fluid, there's a small protein called the androgen receptor that will bind to either of the two androgens. The receptor and the androgen to which it is bound then move into the nucleus of the cell where they bind to the genes that control growth and survival of the prostate cells. If the receptor-and-androgen combination is not available to bind to these genes, the prostate cells first stop growing and then gradually die.

What happens to testosterone and dihydrotestosterone in men on hormonal therapy? Prior to hormonal therapy, testosterone levels usually range from 400 to 800. Within 4-8 weeks after Lupron (or equivalent) administration testosterone levels fall to 10-30. Similar changes occur following surgical castration. Where does this testosterone come from? The adrenal gland generates chemical precursors of testosterone, such as androstenedione, and releases these into the blood. Many tissues in the body, including prostate cancer cells, have the capacity to convert these precursors into testosterone. Thus, standard forms of hormonal therapy *reduce* but *not eliminate* testosterone.

Prostate cancer cells respond to both testosterone and dihydrotestosterone.

What about dihydrotestosterone? I can find no publications that have measured dihydrotestosterone after medical or surgical castration. But in our clinic we find that while some may experience a decline in dihydrotestosterone of greater than 80%, many don't experience a decline at all.

Prostate cancer doesn't become independent of testosterone, but rather becomes so efficient at using testosterone that the small amount remaining after medical or surgical castration is sufficient to support growth.

When you look at the prostate cancer that grows despite this dramatic suppression of testosterone, you will still find the androgen receptor present and fully functioning. When these cancer cells are removed from the patient and tested in the lab, you nearly always find that testosterone is still needed for growth. But it may only take 1/10 to 1,000 the normal testosterone level to fully support cancer growth! Thus, prostate cancer doesn't become independent of testosterone, but rather becomes so efficient at using testosterone that the small amount remaining after medical or surgical castration is sufficient to support growth.

How is this accomplished? One of the more common mechanisms involved is for the cancer cell to make a lot more androgen receptor. This is like

using a large sail in a light wind. By increasing the amount of androgen receptor, the cancer cell makes it more likely that it will be able to bind enough testosterone to support growth. The other common change is that phosphate is added to the androgen receptor and this chemical change makes the receptor much more efficient so that fewer receptors and testosterone are needed to fully support cancer-cell growth.

Treating Prostate Cancer After Lupron Fails

Frequently men come to my clinic after having failed treatment with Lupron or similar drugs, like Eligard, Zoladex, and Trelstar. These drugs aren't perfect and they don't suppress testosterone in every man. The first step in treating a man who's failed Lupron treatment is to measure their testosterone and dihydrotestosterone blood levels. You would be surprised how often we find that there's enough of either testosterone or dihydrotestosterone to fully explain why the treatment has failed. I find it very puzzling that most urologists don't measure testosterone in their patients and that nearly all fail to measure dihydrotestosterone. If the goal of treatment is to lower androgen levels, it seems obvious to me that you would want to measure androgen levels to make sure the drugs are working. When you go on a diet, you measure your weight. When you depress the gas pedal on your car, you look at the speedometer. So, when you are trying to treat cancer by suppressing testosterone, you check to see that the drugs are doing what they should. This is not rocket science. If Lupron doesn't do the job, often switching to one of the competing products *will* get the job done. Surgical castration is another option. The final option is to prevent the remaining testosterone from binding to the androgen receptor.

In the previous section, we discussed how a prostate cancer that progresses after Lupron is still dependent on testosterone and outlined the various mechanisms the cancer cell uses to grow despite low testosterone levels. It turns out that in nearly every paper published on this subject, researchers added Casodex and subsequently showed that it

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prevented low levels of testosterone from supporting cancer growth. The concentrations of Casodex used are similar to those obtained in patients who received 150–250 mg of oral Casodex per day. I can find only one clinical trial that has tested this. Brown, R.S., et al biopsied the metastatic cancer masses that continued to grow despite medical or surgical castration and tested for androgen receptor content. In those patients where the cancer appeared to be making an abnormally large amount of androgen receptor, Casodex caused a response in 80% of the patients.

Since I opened my clinic—the American

Since I opened my clinic in 2002, I've made it a practice to measure dihydrotestosterone levels in each patient we see. And I have to tell you that medical castration, while effective at reducing testosterone from the normal range of 300-800 ng/dL to below 30 ng/dL, often leaves dihydrotestosterone levels within the normal range (30-80 ng/dL).

Institute for Diseases of the Prostate—in 2002, I've made it a practice to measure dihydrotestosterone levels in each patient we see. And I have to tell you that medical castration, while effective at reducing testosterone from the normal range of 300-800 ng/dL to below 30 ng/dL, often leaves dihydrotestosterone levels within the normal range (30-80 ng/dL). And dihydrotestosterone is ten times more powerful than testosterone at stimulating prostate growth, so a dihydrotestosterone of 30 ng/dL is potentially as powerful as a testosterone of 300. Dihydrotestosterone formation can be blocked in most patients with either Proscar or Avodart, with Avodart being more consistently effective. I've found this can aid in inducing remission in patients who've failed Lupron. Luckily, Proscar and Avodart don't cause any additional side effects in men on hormonal therapy. But again, we have to measure dihydrotestosterone levels to see if Proscar or Avodart are in fact suppressing dihydrotestosterone.

Intermittent Hormonal Therapy

All of the above considerations play a role in my current approach to hormonal therapy, which is based on intense intermittent hormonal therapy (IHT). My approach integrates all I've learned about how hormone-resistance develops and aims to minimize treatment side effects. There seems to be no controversy about the fact that IHT results in a higher quality of life for men with prostate cancer compared with continuous treatment. And truth be told, I'm concerned with continuous hormonal therapy's impact on the other diseases common in men of this age group. Androgen deprivation causes a decrease in arterial compliance and an increase in insulin resistance. The change in arterial compliance fosters the development and progression of systolic hypertension and a widened pulse pressure. The increase in insulin resistance causes weight gain and exacerbates hyperlipidemia. I think it very likely that a long-term study of men on continuous hormonal therapy will show it increases heart attack, stroke, and diabetes mellitus.

While IHT improves a man's quality of life and very likely reduces potentially life-threatening complications of hormonal therapy, doubts persisted about whether it is as effective as continuous hormonal therapy in controlling prostate cancer. Existing lab models consistently show that IHT delays the onset of hormone resistance and improves survival. At present, only one small randomized controlled trial compares IHT with continuous treatment (de Leval, et al). At the three-year mark, only 7% of those on IHT were hormone-resistant compared with 38.9% of those on continuous treatment ($p=0.0052$). My own conclusion is that IHT is preferable because it is certainly less toxic and at least as effective as continuous treatment.

Dr. Robert Leibowitz reports excellent results with thirteen months of Lupron, Casodex and

My own conclusion is that IHT is preferable because it is certainly less toxic and at least as effective as continuous treatment.

Proscar, followed by Proscar maintenance. In fact, the response rate Leibowitz reported is higher than that of any other androgen ablation program used to treat prostate cancer. And I myself have noted at the clinic that men on Lupron or Zoladex in combination with Casodex and Proscar experience a much more rapid decline in PSA, as well as a higher frequency of undetectable PSAs, suggesting the response is both more rapid and more complete. However, until someone does a randomized controlled clinical trial, we can never be sure that this more aggressive form of treatment favorably influences survival.

There are also added advantages to shortening hormonal therapy's duration. Men who are on an

Men who are on an LHRH agonist for two years are at substantial risk of never recovering normal function of their testes.

LHRH agonist for two years are at substantial risk of never recovering normal function of their testes. In contrast, men who take hormonal therapy for only twelve months are more likely to return to normal testicular function. Thus, ironically, more intense hormonal therapy for twelve months is more likely to result in restoration of normal testicular function in the future than less intense treatment that lasts two or more years. Also, androgen ablation subjects men to significant side effects. Men rendered hypogonadal in the treatment of prostate cancer commonly develop insulin resistance as well as increased rigidity of their blood vessels. These changes lead to weight gain, an increase in serum lipids, and systolic hypertension. The loss of androgen and estrogen in the brain result in impaired recent memory and cognitive performance as well as in depression and fatigue. These side effects can take a serious toll if treatment is extended beyond one year. For these reasons, I think that continuous hormonal therapy carries with it an unacceptable risk of cardiovascular disease as well as the obvious emotional and intellectual costs.

Many men labeled hormone resistant really just have cancers that are hypersensitive to androgen.

That is, their prostate cancer has learned to proliferate maximally at testosterone levels of $1/10^{\text{th}}$ to $1/1000^{\text{th}}$ of that normally present. Mechanisms involved can include increased expression of the androgen receptor, as well as a variety of mechanisms that enhance androgen receptor activation. Interestingly, in all of the laboratory papers on this process, Casodex at levels obtained in patients after 150-250 mg a day effectively suppress tumor growth. For this reason, I recommend men start on complete androgen blockade composed of Zoladex or Lupron with 150 mg of Casodex a day. By using high dose Casodex, we treat the most common mechanisms of hormone resistance ahead of time.

We've also found that many men on an LHRH agonist such as Lupron or Zolodex often exhibit male-pattern baldness. We often find significant dihydrotestosterone levels in these men. Adding Proscar or Avodart frequently reduces dihydrotestosterone levels to undetectable while reversing male-pattern baldness as well as improving PSA suppression. I think these observations help explain why Dr. Leibowitz obtained such a high response rate with triple hormonal blockade.

Keep in mind also that Casodex can switch from an antagonist of the androgen receptor to an agonist.

Many men labeled hormone resistant really just have cancers that are hypersensitive to androgen.

This is signaled by prostate cancer progression during hormonal therapy with this agent. For this reason, a man's PSA must be followed carefully and if the results indicate cancer progression, Casodex discontinued and second line hormonal therapy considered.

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Corrections

In our last issue we referred to a supplement called Ambertose. It is actually called Ambrotose. We apologize for any confusion this error may have caused.

In Volume 9 #3 many were confused about the correct Vitamin D recommendation. To clarify: 2000 IU of Vitamin D is the best place to start if you are not under care of a doctor who is following your 25-hydroxyvitamin D levels. At my clinic, we now start patients with 4000 IU and follow 25-hydroxyvitamin D levels quite closely. Some men have Vitamin D levels initially so low they need to stay on 4000 and often need 2-4 weeks at 10,000 IU daily. In all cases, your best bet is to get your 25-hydroxyvitamin D level evaluated and to talk with your own doctor about an appropriate dose.

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