A LINK BETWEEN PROSTATE CANCER AND THYROXIN LEVEL: A PERSONAL STORY

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Prostate symptoms that occurred at the age when family history would have predicted clinical diagnosis of cancer were reversed by an hyperthyroid incident induced by a series of medical missed diagnoses. Subsequently, two incidents with prostate cancer symptoms occurred with reduction of thyroxin level and alleviated by raising thyroxin level.

INTRODUCTION

My experience with variations in thyroxin level, resulting from treatments for hyperthyroidism have demonstrated that this level has affected my prostate cancer, to which I am predisposed by genetics.There are now three separate incidents in my medical history in which artificial reductions in my natural high level of thyroxin, resulting from treatments for hyperthyroidism, jeopardized my life by the flaring up of prostate symptoms, which abated when thyroxin levels were again raised.

While I present my experiences in the hope of helping others, I realize that my story is only "anecdotal" in its meaning to medical science. This story is vital to me however, and I hope

to at least gain some control over my own life and my own health

PERSONAL INFORMATION

Born: December 13,1924

Occupation: design engineer, aircraft engines and gas turbines, in retirement since 1989. I continue research into engine dynamics.

My work has demonstrated my natural inclination to connect seemingly disparate puzzlers into a hunch that snowballs, often over many years, into a thesis that can be advanced before cognizant experts.

Family: Three brothers,

-- William (1919 - 1983), had surgery to remove cancerous prostate at age 57 and died of a heart attack at 63

-- John (1921 - 1978), suffered hyperthyroidism, died at 57 of car accident, no evidence of prostate cancer.

-- Peter (1927 - 1991), died of prostate cancer at 63.

RECORD OF HEALTH

I have enjoyed a sturdy constitution all my life.

In 1956, after an unusually debilitating bout with the Asiatic flu, I experienced weakness in the mornings, which usually disappeared later in the day, and the doctor identified skipping heart beats. I learned to avoid this mode by jogging each morning.

In 1969 resort to exercise failed. Even while shoveling two feet of fresh snow every weekend, my heart symptoms worsened. I was diagnosed with hyperthyroidism and placed on PTU.

From 1969 to 1984 I continued on this medication and learned to maintain a healthy balance. Usually the problem diminished or disappeared in summer and cramps in legs would signal that I needed less PTU. The morning jogs, which extended to two miles as new joggers emerged, gave me a good indication of strength.

1984 Incident. In the summer of 1984 I was under heavy pressure at work and my physical strength was weakening. I went for a physical examination, with Dr. Kilty of Community Health Plan (CHP). He found nothing wrong and recommended that I increase the intake of PTU, to lower thyroxin level. His recommendation contradicted my usual seasonal pattern; in the summer I would have needed almost no medication. I was encouraged by his diagnosis, and thought that only a respite from the pressures of work was needed. So in September, my wife and I embarked on a trip to London and Paris.

My strength continue to wane on that trip,and I also developed a urinary problem and experienced visible bleeding on urination one morning. On return, I immediately visited Dr. Kilty, who happened to be at his office. He placed me on a sulfa antibiotic, and ordered me to return in a week. Unfortunately, I was not to last the full week. My family seeing that I was not capable of almost any activity, and could only sleep, brought my to CHP under emergency. It was then that Dr. Charles Trout took charge and with much urgency ordered me to Ellis Hospital.

The basic problem was that I had pernicious anemia; my blood count was dangerously low. I continue injections of Vitamin B-12.

There were also some puzzlers, for which I will offer explanations after discussing further incidents:

--There was much concern about some indication in my eyes that might be a sign of pericarditis.

-- I was treated only for an infected prostate, but numerous attempts to grow cultures of the infection came out negative.

On leaving the hospital, I continued for two weeks or so with sporadic fevers, which turned into night sweats and disappeared over several months. My urinary problem also persisted for that time.

In the meantime, I was ordered off the PTU - the doctors suspected a toxic reaction from this drug that I had by this time taken for over fifteen years, rather than from the newly prescribed sulfa drug - and relapsed heavily into hyperthyroidism.

In December, I was treated with a mild dose of nuclear thyroid killer. My health was restored, but with the coming of the following winter I was given a second dose of the killer. I began taking Synthroid, and as with the PTU, I was able to manage the dosage. In two years my heart returned to normal, contradicting the prognosis of the doctor who guided the transition to Synthroid. I continued to seek a thyroxin level to which I was accustomed and at which I was able to jog strongly while avoiding the cramps and twinges, of which I first became aware on taking PTU and which returned seriously during the transition to Synthroid.

1991 Diagnosis of Prostate Cancer From recommendation of Dr. Susan Trepeta of Albany Medical Center (AMC), I began to think that perhaps a reduction from the level of thyroxin to which I was accustomed may actually be good, so I began taking less Synthroid and looked to control the level at the lower bound, when cramps returned.

My younger brother, Peter, died of prostate cancer in the spring of 1991. I had heard of a new blood test (PSA) available at AMC Dr. Trepeta agreed that I should receive the test, and the net result that I was diagnosed by Dr. Barada with stage D1 prostate cancer.

Realizing that I might be only clutching at a straw, after all medical science offered very little, I thought that I should increase my thyroxin level. My hunch came from the family history. Although I was aware that my brother John may have died too soon to confirm an avoidance of the family susceptibility to prostate cancer, I had nothing to lose.

Significantly, I gingerly tried to broach my hunch on thyroxin level in a letter to Dr. Kantoff of Dana-Farber, who was then interested in the history of my family, of which the pertinent addendum is copied in the Appendix. Here, I recorded my experiences with variations in this level.

In July, 1992 I began treatment by Dr. Barada through Lupron Depot injections and Eulexin caps, and have continued to this day.

1996 Incident Dr. Trepeta cooperated in assuring that I could continue the dosage of Synthroid that I had been taking, 300 mg. daily. But when she left AMC, I was transferred to the care of Dr. Caramore who warned of dire consequences from this dosage - as if I were not already in severe jeopardy from continuing prostate cancer - and chopped the dosage.

I tried to appeal to Drs. Caramore and Barada through my letter of October 30, 1995, addressed to Dr. Caramore with a copy sent to Dr. Barada. But with renewal of the warning from Dr. Caramore and with no encouragement from Dr. Barada, I felt reassured and began reducing the thyroxin level, expecting to avoid only the threshold of cramps and twinges. While waiting for onset of these signs, I began almost eliminating Synthroid entirely, But it was not these sympoms that brought me back the reality that after the medical destruction of my thyroid I was to remain dependent on Synthroid.

About March 1 I suddenly found a small blood stain and lapsed into the burning pain and need for very frequent urination. Immediately, I began raising thyroxin level and experienced a slow improvement, which continues today.

A few days after this lapse, I saw Dr. Barada in what was a scheduled routine visit. He suspected an infection, and prescribed a sulfa-based antibiotic , which he seemed confident would quickly provide relief. Based on the 1984 incident, where the antibiotic was tried with no success,, I felt skeptical and did not begin the prescription immediately,

In about a week, after I had begun improving, to my mind because of the restoration of thyroxin, I decided to be a compliant patient, as my loved ones expected, and began the prescribed medication. Doggedly, I persisted with the treatment, as the drug clouded my mind, and did not surrender until only one day was left.

Finally, I was to learn what I should have checked sooner, the lab tests did not indicate an infection. The sulfa drug did not help; it only made me sick. However, this sickness explained previous puzzlers from when sulfa drugs were perscribed. .

EUREKA!

This reaction to the drug led me to recall my first incident with prostate problems, that of 1984 and to relate it as further evidence of my hunch on the effect of thyroxin level.

I now realize that the puzzling symptoms that almost sent me into treatment for pericarditis were allergic reactions to the sulfa drug. My eyes were affected. Fortunately, these symptoms alone were insufficient for a positive diagnosis, and I was spared that treatment, but I was exposed to a prolonged bout of hyperthyroidism.

During the illness the hyperthyroidism was left untreated because doctors suspected a toxic reaction from PTU, and I emerged with a skeletal body. No infection was determined, despite numerous tests and my prostate symptoms did not vanish, until about two months after the hospitalization. I now believe that it was prostate cancer that was reversed by the effect of severe hyperthyroidism, that an opportunity for early detection was lost From what I have learned about the role of my heredity, clinical onset of prostate cancer at that time would have been inevitable.

For clinical acceptance, medical science would require a more comprehensive proof than I have accepted. For my health, I need only evidence to guide my own choices, the results of which I constantly monitor. My philosophy is to follow a path that leads to steady improvement in the prostate cancer without entering a regime of symptomatic hyperthyroidism. Admittedly, the margins guiding me are narrow, and I fear that following perfunctory guidance of specialists may yet again produce a relapse.

CURRENT STATUS

My prostate symptoms continue to diminish, and now are often hardly noticeable. I continue morning physical workouts, now doing forty five minutes on a *NordicTrack's WalkFit*™ every morning.

In January Dr. Barada expressed satisfaction with my condition, and credited his choice of treatment, having earlier discounted my sense of the link between thyroid and the

progression of the cancer. He seems to ignore my sinking condition only two years ago.

My new primary physician, Dr. Swicker of AMC found on physical examination in November, 1996 that I did not show physiological symptoms of hyperthyroidism, but later became concerned when he received the results of the blood test. Dr Swicker now wants me to ensure that my thyroxin level conforms to a preset clinical standard. I am concerned that a reduction in this level will mean a return to prostate symptoms, as it has twice in the past. He has declared that the only way I can get my prescription renewed is if I first cut my thyroxin level to a level he finds acceptable.

MY DILEMMA

After three serious encounters with deteriorating condition due to prostate cancer, I am determined to follow my own beliefs until convincing alternatives are offered.

Why, just because I had submitted to the destruction of my thyroid gland, should my thyroxin level be dictated by a chemical standard that is less than what I had lived with until i was almost sixty years old and which I have maintained almost steadily since my thyroid was killed off.? If this level were achieved naturally, it would not even be noticed. But now physicians have the mandate to manage my thyroid levels, seemingly without understanding the consequences I have suffered when thyroid levels have been arifically dropped below that which my body produced naturally before phrophylactic medical intervention.

I believe that my thyroxin level and my level of prostate problems are related. While this link is not recognized in the literature, the link in my body is clear to me. I want to stay well, and would like to participate in choices of treatments or nontreatments

I also suggest that this link may be useful as an indicator to researchers looking at ways to slow or reverse the progress of prostate cancer.

APPENDIX

Copied below is the addendum, below my signature, of the letter of January 13, 1992 to:

Dr. Phillip W. Kantoff
Dana-Farber Cancer Institute
44 Binney Street, Boston, MA 02115

I had been examined at Dana-Farber by Dr. Eric H. Rubin for a second opinion on november 25, 1991. In this letter I sought reassurance that the data sent by Dr Barada were complete and valid.

In this addendum, I tried to counter a concern by Dana-Farber's radiologists about the bone scan.

ABNORMAL AREA IN BONE SCAN

History of Sensations

A severe cramp in this area, and also in the right buttock was experienced on the first attempt to at exercise after initial treatment for hyperthyroidism in 1969. Since then, I became aware that reduction in thyroid level causes muscle cramps primarily in the right calf and nerve jabs in the fingers. By last summer both sensations had disappeared and I slowly began to reduce my Synthroid intake. I had brought down my evening pulse rate to 53, whereas previously in the best of times it would be 72. I was away over Labor Day weekend and was delayed through a period of about four days for which I was not prepared to maintain the anticipated dosage. { this is a fancy way to tell Dr. Kantoff that I had forgotten the pills at home} On return, while jogging in the morning I experienced a severe cramp on the left side only. This was the first cramp in this area since 1969. {this is the area questioned in the bone scan}

The post cramp feeling continued sporadically until recently.It would go away completely and then return as a faint pain. Within the last two weeks I have begun to take more Synthroid and have almost completely eliminated any unusual sensation.I had always felt that I was best when the thyroxin level was just below producing symptoms of hyperthyroidism and the latest experience reinforces this feeling.{the pain is long gone}

Radiologist's Opinion

The radiologist showed me that the X-ray clearly revealed a calcified tendon rather than an abnormal bone. He reaffirmed this observation through fluoroscopic examination and two relevant images. He confirmed that his observations are consistent with an area that would be prone to cramping.

4/08/1998

Dr Paul Rennie, researcher at my Alma Mater, the University of British Columbia, has referred me to the paper by you and your associates (Mol Cell Endocrinol 1995 Mar;109(1):105-11) on the possible link between thyroxin level and prostate cancer. I have developed arguments for this link from my personal experiences with hyperthyroidism and prostate cancer and am now enjoying good health using Synthriod to maintain an elevated level - as specified by physicians relying on chemical indicators - of thyroxin but avoiding symptoms of hyperthyroidism.

The note entitled *A Link between Prostate Cancer and Thyroxin Level: A Personal Story*, April 08,1998, copied below recounts my experiences as outlined in my search for a personal physician who would cooperate in my wish to secure a thyroxin level based on my physiology rather than an arbitrary chemical standard.

Dr. Rennie has indicated an interest in any correlation between PSA levels and episodes with hypo/hyperthyroidism. It would be difficult to show a correlation even with access to medical records. Nevertheless, I will relate only what I was told, which to my mind may not be accurate and complete. However, if scientific interest emerges, I should be pleased to request that pertinent records be released to interested physicians and/or scientists.

I did not become aware of the PSA test until sometime in 1990, and requested the test in the summer of 1991. The results revealed a highly elevated level, somewhere in the sixties. By early January Dana-Farber reported a confirmation of this level, although I had begun earlier to increase Synthroid intake.

I resisted starting immediately on Lupron, asserting the "later" philosophy while at the same time hoping that the elevation of thyroxin level would be shown effective, as I felt that it was beginning. Dr. Barada was clearly of the mind for "now" rather than "later."

In June 1992, Dr. Barada displayed a computer-generated graph of his projection with time for my PSA increase. The curve was constructed from two points, the original PSA and a higher one, purported as the latest number. His asymptotic curve indicated very little time left. Faced with this pressure, I succumbed and went the Lupron route, despite being suspicious of the actual numbers.

I continued to take more Synthroid than my internist, Dr. Caramore of Albany Medical Center, considered prudent from chemical blood tests – physiology was irrelevant to him. I managed to defy his admonitions for several years, after all I had lived with hyperthyroidism for many years and felt that I could avoid the worst consequences by vigilance against serious symptoms, which I had learned to sense. Dr. Barada dismissed my observations on the thyroxin link to the progression of my cancer, claiming contrary evidence.

However, Dr. Barada soon, probably within two months from initiation, pronounced success of his therapy: my PSA dropped to almost zero and he was pleased with what he was able to feel. His first remark was that he would look into reducing therapeutic agents, Lupron and Eulexin,but this suggestion never came up again.

The pressure from the internist continued, with dire foreboding. Dr. Caramore, in his last letter of November 05,1995, offered to reconsider his strong opposition to my dose of Synthroid if I were to "find contrary information based on scientific fact." Of course there was no way to convince him, but it is interesting now to recall that he professed willingness to submerge his fear of deviation from dogma, in the control of

thyroxin replacement, if there were proven a countervailing benefit. I was feeling well and succumbed to the overwhelming expertise aligned in deprecation of my perceptions, only to fall into the relapse of 1996.

During the relapse, Dr. Barada and his nurse seemed not so forthcoming with PSA level. It was not until January, 1998 that Dr. Barada was to mention it again, this time almost "not measurable." In July, it was 0.00 and he was pleased with his examination. He recommended staying with "what works," which I interpreted to include continuation of the present dose of Synthroid.

ABSTRACT OF PAPER ON PROSTATE CANCER/THYROXIN LINK

Mol Cell Endocrinol 1995 Mar;109(1):105-11

Triiodothyronine modulates growth, secretory function and androgen receptor concentration in the prostatic carcinoma cell line LNCaP.

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Department of Developmental Biology, Catholic University of Leuven, Belgium.

There is increasing evidence that the course of prostatic carcinoma is determined by a complex interplay between genetic events, paracrine interactions, and hormonal and dietary factors. These latter factors include several ligands of the nuclear receptor family such as androgens, vitamin D3 and retinoids. To test whether thyroid hormones also influence the growth and differentiated function of prostatic carcinoma cells, LNCaP cells were treated with or without triiodothyronine (T3) in the absence or in the presence of other regulatory factors. Exposure of LNCaP cells to T3 for 6 days in the absence of androgens caused a dose-dependent increase in [3H]-thymidine incorporation with a maximal stimulation of 2.5-fold at 10(-9) M T3. Secretion of prostate-specific antigen (PSA) was also stimulated 2-3-fold. The observed effects may well be mediated by a nuclear T3 receptor as evidenced by displaceable T3 binding studies. Combined treatment of LNCaP cells with androgens and T3 revealed intriguing interactions between these two pathways. Below and up to 10(-10) M of the synthetic androgen R1881, the concentration that evokes optimal proliferative responses, T3 stimulated [3H] thymidine incorporation. At higher concentrations of androgens, T3 displayed antiproliferative effects. No androgen-dependent effects on T3 receptor levels were observed. Conversely, T3 increased androgen receptor levels up to twofold. Androgen as well as T3 stimulation of proliferation was abolished by high concentrations of the retinoid 9-cis-retinoic acid. These data add T3 to the list of factors that influence growth and differentiation of prostatic tumor cells and contribute to our understanding of the intricate pathways that ultimately determine the course of prostatic carcinoma.

E-MAIL COMMENT BY PROFESSOR VERHOEVEN

Dear Mr Klompas,

Thank you for sending me your interesting but painful story. I want to stress that our studies have only been performed on prostate tumor cells cultured in vitro (and never on patients) and that under these conditions we observed both stimulatory and inhibitory effects of thyroid hormone depending on the presence or absence as well as on the concentration of other hormones such as androgens. The main conclusion at this point is that tumor growth is a very complex phenomenon that may be modulated by numerous factors but not always in a clearly predictable way. I was contacted by Dr A Hercbergs from Cleveland Clinic Foundation who wrote a paper on clinical observations on the influence of thyroid hormone on advanced tumors (In Vivo 10: 245-248; 1996). His data rather suggest that low levels of thyroid hormone might be advantageous.

No definitive conclusions are possible at the present time. What is sure, however, is that too much thyroid hormone as well as insufficient thyroid hormone may be very dangerous for

your health. So please take care of yourself and stay under close supervision of your physician. I wish you all the best.

Prof. G. Verhoeven