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Research Article

Tumor Therapy with *Amanita phalloides* (Death Cap):Long Term Stabilization of Prostate Cancers

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Abstract

Molecular events that cause tumor formation up regulate a number of HOX genes, called switch genes, coding for RNA polymerase II transcription factors. Thus, in tumor cells RNA polymerase II is more active than in other somatic cells. *Amanita phalloides* contains Amanitin, inhibiting RNA polymerase II. Partial inhibition with Amanitin influences tumor cell - but not normal cell - activity.

Objectives: To enlarge treatment spectrum, dilutions of *Amanita phalloides*, are applied to prostate cancer patients. Monitoring prostate specific antigen, different doses of Amanitin are used.

Results: Within a period of up to five years, prostate specific antigen values can be stabilized. Somatic investigations and imaging methods reveal remission in all three cases. No prostate cancer associated symptoms, nor liver damage occur. When pausing the therapy, PSA levels always increase, even after five years.

Conclusion: This new principle of tumor therapy shows high potential to provide a smooth medical treatment. Treatment should exceed the period of five years.

Keywords: *Amanita phalloides*; Tumor Therapy; Prostate Cancer; Amanita Therapy

Introduction

A tumor may develop within the prostate, a gland in the male reproductive system, in the same way as in many other tissues. Some prostate cancers are slow growing, some are fast and aggressive, and may metastasize to other parts of the body, frequently spreading into the lymph nodes. Whereas a slow growing prostate cancer might remain without symptoms, an aggressive tumor may cause difficulty in urinating, problems with sexual intercourse, including erectile dysfunction and pain.

Prostate cancer tends to develop in men over the age of fifty. It is one of the most prevalent types of cancer in men. About two thirds of tumors are slow growing, the remainder being more aggressive. As in most cancer cases a familial disposition is discussed. Diagnosis may be indicated by symptoms, physical examination, prostate specific antigen (PSA), or biopsy. Screening for prostate cancer in men over 50 is discussed [1].

In a genetic experimental study with tumor forming *Drosophila melanogaster*, four classes of genes involved in tumor formation could be distinguished. Proliferative genes break the cell cycle control by allowing replication immediately after mitosis. One single proliferative mutation is sufficient for tumor formation [2]. Mutations in oncogenes and tumor-suppressor genes are secondary events and add by destabilization of the differentiation pattern of the cells. In search for ubiquitous events - the central possible targets for therapeutic intervention - switch genes were identified [3]. All switch genes belong to the class of HOX genes, and code for RNA polymerase II (RNAP) transcription factors. Some HOX genes are over expressed in human tumor cells [4, 5]. Due to this over expression, the event that leads to tumor cell growth should lead to high activity of RNAP. In somatic cells of adults, RNAP is expected to be less active. Partial inhibition of this enzyme consequently causes inhibition of tumor cell activity, without severe effects on somatic cells. The drug in the extract of *Amanita phalloides*, Amanitin, blocks RNAP in all cells. Inhibition of about 50% of this molecule breaks tumor cell activity.

The immune system might recognize and lyse tumor cells; partial inhibition of tumor cell activity raises the possibility for stabilization of the disease. Amanita treatment already showed good results in prophylaxis or therapy of a number of tumor cases [6, 7]. To enlarge the possibilities of experimental medicine, here a pilot study of long term treatment of prostate cancers is described.

Patients, Method

Amanita phalloides (zert. Riede) dilutions are used. *Amanita phalloides* dilutions are applied since 300 years, the classical homoeopathic indication is fear of death.

Patient 1HB was born in 1940. Anamnesis finds no familial or other risk for tumor formation. In 2005 he suffers an Apoplexia. In September 2007 prostate cancer was diagnosed by routine analysis. In November 2007 prostatektomie occurs, PSA drops subsequently from 3 3ng/ml to 0, 1ng/ml. In December 2007, Amanita therapy starts (Figure 1). Body weight is 75 kg and remains constant throughout the therapy.

Patient 2JR was born in 1942. Anamnesis finds no familial or other risk for tumor formation. In 1998 a carcinoma of the mouth bottom is operated and chemotherapy occurred. In 2000 he obtains bypass operation. End of 2006 difficulties in urinating occur, subsequent analysis reveal diagnosis of prostate cancer in December 2006. Amanita therapy starts in July 2007 (Arrow in Figure 2). Body weight is 87 kg and remains constant over the period of Amanita therapy.

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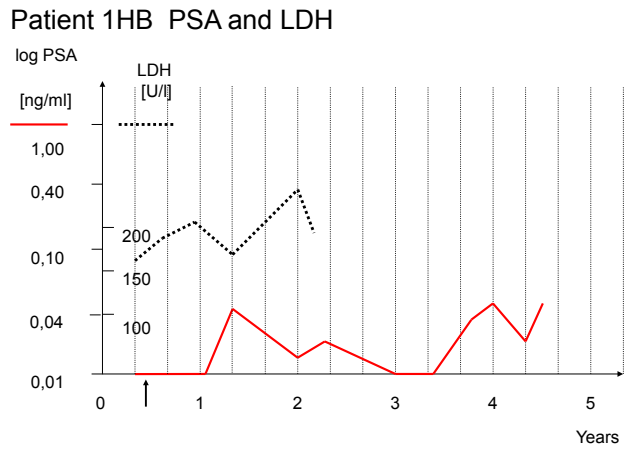

Figure 1: Stabilization of patient 1HB with pT3pN1M0R0, Gleason 7 (3+4). At time of first diagnosis (time 0) of prostate cancer PSA was at 3,3 ng/ml. Natural duplication time of PSA is two months and shows after one year, in a period when the patient pauses with Amanita. Amanita therapy started six months after prostatectomy (arrow), with 3 x 15 drops of *Amanita phalloides* D4 per day. At one year, a significant increase of PSA occurred. Increasing dosage of Amanita to 4 x 20 drops D4 per day stabilizes PSA again for another two years. Further increase of PSA required the increase of dosage to 3 x 15 drops D3 per day, followed by an uptake of 100 ml D2 within short time (in 4 x 10 drops per day) after four years. At this time, somatic investigations and sonography, diagnose complete remission. Due to an accident and hospitalization, the patient pauses again with the Amanita intake, followed by increase of PSA – the same exponential growth rate than after the first year. He suffers for a month from micturition problems, further analyses remained without findings, the dose is adapted to 3 x 15 drops D4 per day, retarding the duplication rate of PSA. A complete check up with sonography reveals a complete remission of the disease. Beginning of year 4 100 ml of D2 is applied in 4 x 10 drops per day, this reduces the PSA level again. Despite long survival, and the diagnosis of remission, tumor cells still remain, requiring further attention and treatment with Amanita. In case of natural duplicaition rate of PSA, the value would be at 300,000 ng/ml.

Patient 3BD was born in 1936. Anamnesis finds familial risk; his grandfather had suffered from colon carcinoma. He has hypertension and neuropathies. First diagnosis occurred in March 2007 by biopsy after micturition problems. No operation was possible due to infiltration to the bladder. X-ray irradiation with 77 G-ray occurs in summer 2007. Anti-hormones Tredantone and Proscar were applied several times. In October 2008, the patient decides to start Amanita therapy and stop anti-hormone treatment (arrow in Figure 3). Body weight is 82 kg and remains constant throughout the therapy.

In all patients, in addition to Amanita as the only tumor specific drug, the oral uptake of essential fatty acids and dermal application of Zinc salve is indicated. Essential fatty acids enhance the fluidity of cellular membranes, and decrease the risk of autoimmunity.

For monitoring of the therapy, the regular measurement of following parameter is arranged

1. Lactate dehydrogenase (LDH) in serum. This enzyme is present in all cells, the occurrence in serum shows lysis of cells. LDH levels increase in all events resulting in cell degradation, i.e. myocardial or kidney infarction, embolism, autoimmunity, anaemia, infectious diseases – or lysis of tumor cells.
2. Prostate Specific Antigen (PSA).
3. Cholesterol, differential blood count, liver transaminases to monitor general state of health.

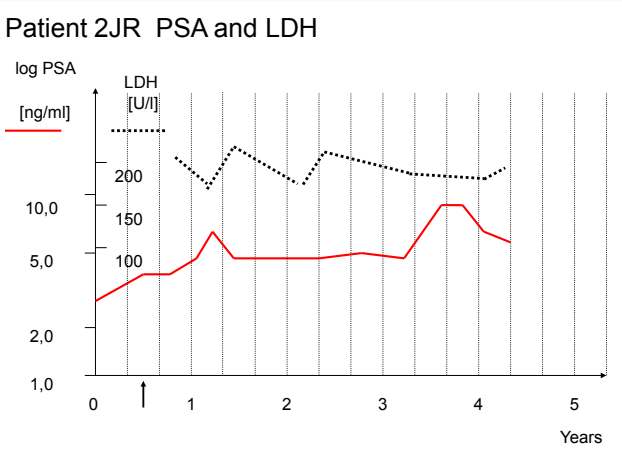

Figure 2: Stabilization of patient 2JR with pT2pNXM0R0, Gleason 2. At time of first diagnosis of prostate cancer PSA was at 3,49 ng/ml, Amanita therapy started six months later with 3 x 5 drops of *Amanita phalloides* D4 per day (arrow). Natural duplication time of PSA is initially 23 months. Duplication time of PSA appears to be 5 months after one year and after 3,2 years, in periods when the patient pauses with Amanita and suffers from prostatitis. After one year, a significant increase of PSA occurred. Increasing dosage of Amanita to 10 drops D2 per day stabilizes PSA again within a month. For another two years, 4 drops of *Amanita phalloides* D4 per day stabilize PSA values. After three years, PSA increases after a prostatitis again, antibiotics were applied. The uptake of 100 ml D2 in 4 x 10 drops per day stabilizes PSA values again within two months. Further dosage is 2 x 5 drops of D2 per day. MRT reveals non malignant enlargement of the prostate, no lymph node or other abnormalities. However, PSA values indicate the occurrence of remaining tumor cells, requiring further Amanita therapy. In case of natural duplication rate of PSA in 23 months, the value would be at 15 ng/ml.

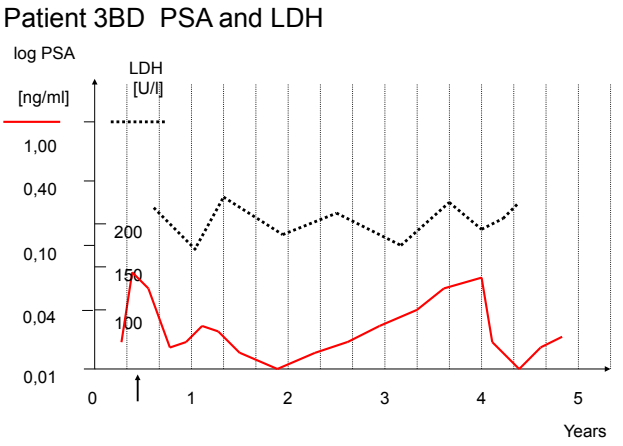

Figure 3: Stabilization of patient 3BD with pT4pN1M0R0, Gleason 9 (4+5). At time of first diagnosis (time 0) of prostate cancer PSA was at 13, 0 ng/ml. Natural duplication time of PSA is initially 1 month. Operation is impossible, infiltration of the right seminal ventricle and bladder were diagnosed. Therapy occurred with Tredantone and Proscar, in addition with Zometa. The initially fast growing tumor cells were halted with Proscar four months after first diagnosis. Amanita therapy started six months after first diagnosis (arrow), the patient decided to stop anti hormone treatment. Initially the patient takes 50 ml *Amanita phalloides* D2 in 2 x 10 drops per day. Further dosage of 3 x 15 drops of *Amanita phalloides* D4 per day was sufficient for stabilization for two years. Increasing PSA required a dosage of 3 x 15 drops of D3 per day. Further increase after nearly four years required the uptake of 100 ml D2 in 4 x 10 drops per day. This dosage reduces the PSA to a low amount. Further dosage is 10 drops of D2 per day. After a surgery of the knee, the patient pauses, resulting in increase of the PSA. This increase is slower than the initial exponential growth rate. At this time, magnetic resonance imaging diagnoses complete remission. In case of natural duplication rate of PSA, the value would be over 500,000 ng/ml.

A PSA value of 10ng/ml is defined by academic medicine as intervention point. Below this value, watchful waiting is indicated. Thus, all three patients from beginning to end of this case study treatment remain below this point of interference. Thus, no indication for other therapies exists. All patients were informed of all possibilities of treatment, like anti hormones, all decided against it.

Results

Diagnosis of Prostata-CA in patient 1HB occurred in a prophylaxis screening. Immediate operation occurred, despite a PSA of only 3, 3ng/ml. Pathology defined a malignant tumor (Figure 1). Remaining tumor cells were estimated to be few therefore a dose of 3 x 15 drops of *Amanita phalloides* D4 per day was applied. Additionally the patient takes lecithin 1 teaspoon per day, and olive oil 30 ml per day. Within the first year, LDH showed an increase, suggesting that cells lysed. PSA values are initially below the lower measurement range. In a pause of the Amanita therapy, the PSA starts to rise. Continuing Amanita uptake retards the PSA increase. Higher doses are applied to reduce PSA values. Reducing PSA values are associated with increasing levels of LDH, suggesting lysis of cells. After three years, the blood pressure of the patient is at 100/70 due to additional medication of diuretic. Stopping the intake solves the problem. Somatic investigations reveal complete remission after 3, 5 years. Reassuring the statement of complete remission is done after 4, 5 years. However, breaking the Amanita therapy leads to increasing PSA values, thus the actual dosage is 2 x 10 drops of D2 per day in searching the minimal daily dose for further stabilization.

Diagnosis of Prostata-CA in patient 2JR occurred after micturition problems. Besides the Amanita therapy, he takes initially some globules of Cactus D4 to stabilize the cardiac function. In dietary composition, he reduces animal fat and replaces it by plant oil. Throughout the therapy, cholesterol level declines from initially 297 mg/dl to 210 mg/dl. Initially within the first year, he suffers from rheumatic disorders, like pain in the finger joints, memory failing, and inflammations. Whereas the rheumatic disorders decrease, the disposition to infections increases. He suffers from prostatits after one year (Figure 2), with an increase of PSA and occurrence of blood in stool. With an increase of the daily dose of Amanita, and antibiotic intake, the symptoms disappear within short time. He additionally takes Crataegus now to strengthen the cardiac functions. Prostatits follows after 3, 5 years, and a high dose of Amanita reduces again the PSA values. Blood in ejaculate occurs. After 4 years, he suffers from a severe general infection and is hospitalized with high blood pressure. All examinations remain without result. He pauses with Amanita for a month, and restarts with 2 x 5 drops of D2 per day. Somatic investigations reveal remission of the disease; however the prostate is still elongated. No somatic symptoms remain.

Diagnosis of prostate cancer in Patient 3RD occurred after urination problems. He obtained X-ray irradiation, but no chemotherapy. He suffers from anti hormone treatment and starts with *Amanita phalloides* D2 in a two months overlap with anti hormone therapy (Figure 3). In addition he takes lecithin and plant oils. Initially, 50 ml of D2 are sufficient to cease PSA values. With variant doses, the disease is stabilized. However, even after five years a pause of the Amanita treatment leads to increasing PSA levels, tumor cells are still active. Magnetic resonance imaging diagnoses complete remission.

Discussion

Here three cases of long term management of prostate cancer with Amanita are presented. During the periods of therapy, after times with pauses or insufficient uptake of Amanita, PSA values increase. A higher dose of drug is applied subsequently. Each time, this management lowers the PSA level again, concomitant with the increase of LDH. Lysis of cells increase LDH levels in serum, thus higher doses of Amanita appear to lyse tumor cells, lowering the level of PSA. In that, the disease can be stabilized over years without severe side effects. In all cases, remission is diagnosed by routine check-up at the end of the period.

Overall survival rates with cancer lie near the five years postoperative survival rates. Thus in most regimens, patients are defined as being healthy after five years; new upcoming tumors might presumably be as frequent as relapses. In all cases presented here, a pause of the therapy within the fourth year leads to an increase of the monitoring marker PSA, meaning, tumor cells still persist. Thus, therapy is recommended to extend the period of five years.

In all cases, initially very low dose, some drops of D4 per day, are sufficient to stabilize PSA values. The first years, fear of the tumor lead to voluntary uptake of Amanita. After four years, all three patients start to feel healthy and stop Amanita uptake arbitrarily. The patients might disregard the disease. PSA values rise, and require an uptake of 100 ml D2 within two months to stabilize at low serum level again. The long term necessity of Amanita uptake without physical symptoms requires a convincing leadership. Further analysis will reveal more aspects of this new possibility.

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