Complementary Approach to Breast Cancer – A Case with Multiple Liver Metastases is Free from Disease.

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By Professor Jurasunas

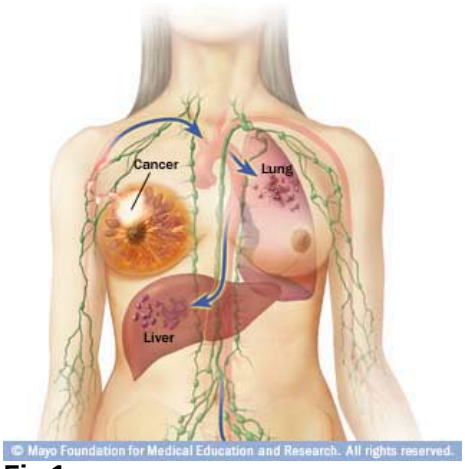

Fig.1

Abstract

Breast cancer remains the #1 killer among women with over 250,000 patients yearly diagnosed in U.S.(1) The incidence of this disease and the resulting deaths continue to increase in Western developing countries. Too many cases are diagnosed with metastases condition while evidence has shown that primary cancer began releasing cancer cells into the circulation at an early stage. Chemotherapy (gold standard), palliative therapy, radiation, surgery have not achieved the expectation to reduce breast cancer mortality and recurrence is still too high. 15% to 35% of breast cancer patients do not respond to chemotherapy but continue to receive treatment from which they do not benefit.

There is an urgent need to develop new effective ways to diagnose and treat cancer. New biomarkers testing may not only indicated in high risk patients but may further serve as diagnostic, prognostic and follow up during treatment (2). Complementary or integrative oncology is now attracting more interest among progressive medical doctors and oncologists. Many new lines of research have shown that integrative dietary agents have demonstrated efficacy in the prevention and treatment of cancer(3). As well as a support to chemotherapy agents, being safe and offering increased effectiveness.

Therefore this new important concept now indicates that a tumor can no longer be viewed simply as an uncontrolled proliferative mass, but rather as a cellular community interacting with the microenvironment. (4) Therefore a comprehensive treatment may more effectively target the disease according to this new concept, superseding the outmoded paradigm of using toxic therapy directed at killing the tumor.

Introduction

What is Cancer?

Cancer may be attributed to an accumulation of abnormal cells, which divide, without control, evade apoptosis, accumulate mutations and are able to grow, invade tissues and penetrate blood circulation. Cancer cell survival is totally dependent on defective P53 which is one of the main hallmarks of cancer (5). More than 50% of all cancer harbors a P53 mutation or inactive P53 genetic expression (6).

Immune defense is also associated with tumor growth and cancer cell invasion through blood circulation. Inhibition of apoptosis and immune suppression are two main keys responsible for tumor growth and cancer invasion. Indeed the most dangerous aspect of breast cancer is its ability to spread to distant sites while many cases of breast cancer are diagnosed only at the primary tumor,even with metastases to liver or bone.

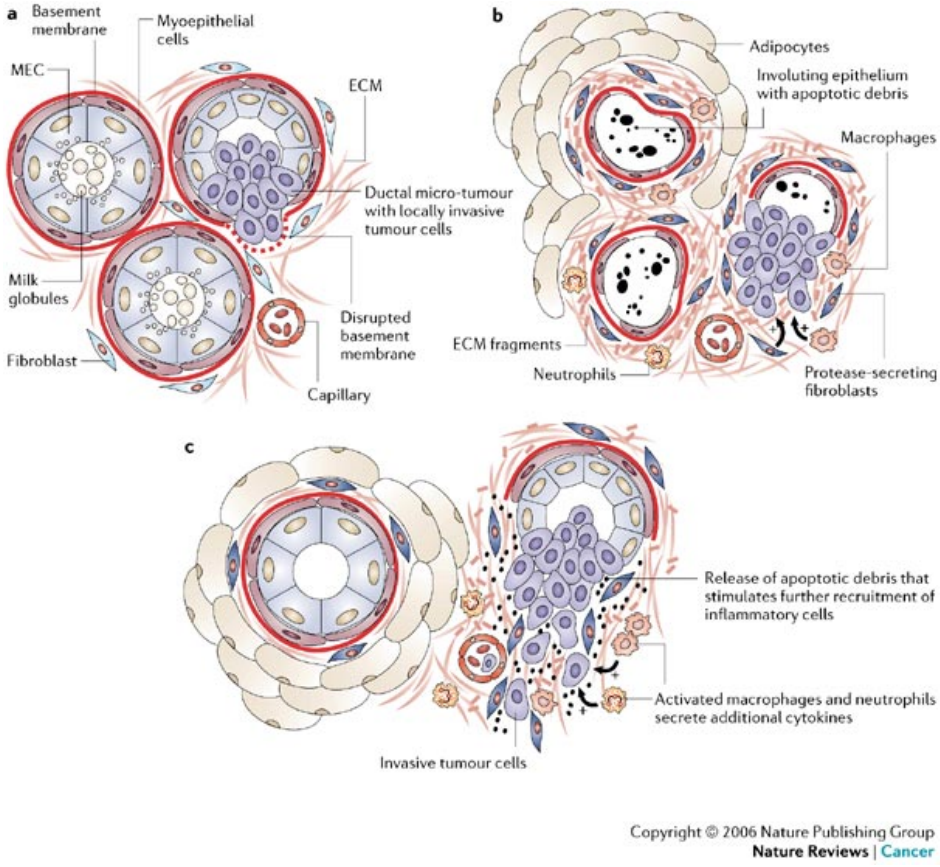

Fig.2

A new emerging theory implicates inflammation being involved in cancer (7) and links the tumor with the surrounding tissue that stimulates tumor growth and expansion (8).

The tumor microenvironment is increasingly recognized as a major regulator of carcinogenesis and has been implicated in both cancer progression and invasion (9).
The Inflammation process boosted by chemokines and cytokines, such as NFKB and components of the extracellular matrix (ECM), such macrophages, fibroblasts and mast cells that may influence negatively the structure of ECM through the production of proteases, MMP2, MMP14 that have shown elevated expression in situ, responding to invasive carcinoma transition (ICT). This process plays a key role in the destruction of the basement membrane. Macrophages respond to angiogenic signals from cancer cells to induce pro-angiogenic factors such as MM1-MMP12 (10).

High oxidative stress may also damage cell membranes including membrane polyunsaturated fatty acids, which disturbs the synthesis of prostaglandins, decreasing PGE1 and increasing PGE2 to excess, thereby promoting chronic inflammation.

PGE2 also has potent immunosuppressive effects, down-regulating T-cell and B-cell proliferation and the cytotoxic activity of NK cells (11) including activation of MMP2-MMP9, a critical step for angiogenesis and the degradation of the E.C.M: which is an independent prognostic indicator of primary breast carcinoma.

P53 Mutation

We know the P53 tumor suppressor gene is mutated in approximately 50% to 70% of all human cancers. In fact the research of new biomarkers in cancer leads to the study of P53. (12)

However P53 mutation may not have major positive effects on tumor growth by itself. Recent data has shown that in addition to loosing transcriptional function, mutant P53 gains independently of the loss of wild type function, gains new function know as Oncogenic function, termed “Gain-of-function” (GOF) that drives cell migration, invasion and metastases (13).

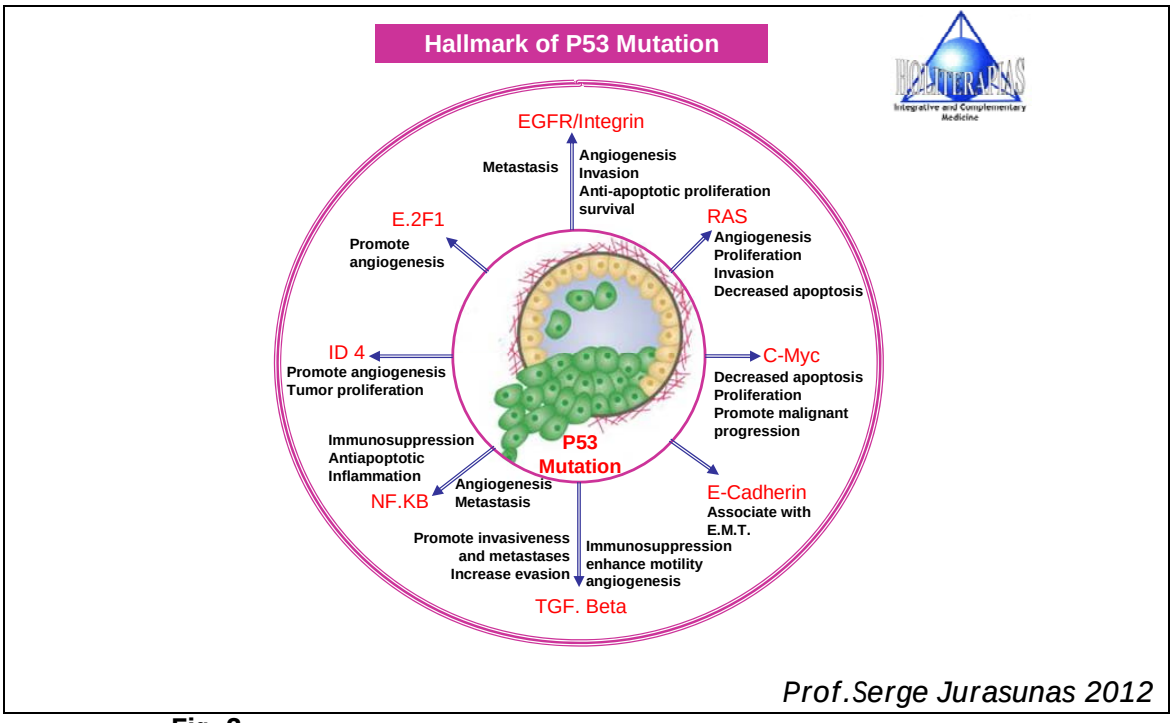

Fig. 3

Mutant P53 may activate a network (13) of specific transcription factors and other target genes such as E2F1, GOF, TGFB RAS, C-Myc, NF-KB, ID4, and E-Cadherin that all contribute to accelerate tumor progression, angiogenesis, mobility, extraversion and invasion. For instance less E-Cadherin (that sick cells together) is mostly associated with invasive breast cancer (14) one half of the invasive ductal carcinoma that developed distant metastases have aberrant E-Cadherin protein expression. Transcription factors such TGF-Beta, NF.KB and GOF have strong immunosuppressive effects, which stimulate angiogenesis and inhibit apoptosis (15-16).

Presentation of the Case

A 44 year old Caucasian female was diagnosed in October 2011 with a cancer of the left breast, stage III with an extensive process of multiple metastases. About 30 lesions, up to 1.5cm wide were localized in the left lobe and in segment VIII and V of the right lobe of the liver. The tumor of 3 cm spread inflammation around neighboring tissues making immediate surgery impossible. Many swollen lymph nodes were diagnosed on the left part of the neck suggesting metastasis invasion.

The hospital delayed surgery for 2 months, while starting a chemotherapy regimen. The patient then came to my clinic in January 2012, in poor physical condition and feeling the adverse effects of chemotherapy such as fatigue, loss of appetite, nausea, anxiety yet felt confident about what I could do for her.

Complementary Diagnosis

Our total approach offers a whole view and more information about the disease and is a complement to the international classification TNM including diagnostic, prognostic and follow up of the treatment.

A) A complete molecular markers test

P53 Gene Expression – P53 protein level – BCL2 – BAX – Survivin – P21 – V.G.E.F.

In a stage III breast cancer with a tumor of 3cm wide and metastases, usually the angiogenic mechanism is strongly active associated with overexpressed BCL2, increasing resistance to chemotherapy (www.sergejurasunas.com – Molecular Marker Tests).

B) Alternative blood diagnosis to regular blood parameters.

1 – Live Blood Microscopy Analysis
2 – Oxidative Dried Blood Layer Testing

These 2 tests were defined by Robert Bradford as the Peripheral Blood Analysis (17) that we have been using in our clinic for over 34 years.

The Live Blood Microscopy analysis offers a direct view on whole blood, where you can observe conditions such as blood viscosity, micro-clots, and micro-plaques in real time including excess toxins from poor liver function and platelet aggregation. It monitors the effect of wrong dietary style, oxidative stress, and an intoxicated colon.

Example:
Abnormal shaped red blood cells
Lipid plaques
Platelet aggregation
Bacterial invasion
WBC activation
Denatured WBC's and immune cells
RBC aggregation

The Oxidative Dried Blood Layer Test

This test is prepared from a number of dried blood layers collected on a clean microscope slide and the examination of the blood coagulation under a microscope after few minutes have allowed the fresh blood to dry, permitting the observation of a number of informative characteristics..

Chronic vs acute conditions

Inflammation
Oxidative stress level
Degenerative disease indication
Allergy
Psychological stress

Both tests are of paramount importance to monitor the patient's whole body condition, the stage of the disease, and the follow up of the treatment.

Our Complementary Approach to Cancer

The aim of the treatment is to target cancer in every direction as soon possible so as to obtain a better result with chemotherapy.

. Detox
- Boost immune system
- Increase apoptosis by targeting the molecular markers and transcription factors
- Reduce oxidative stress
- Inhibit angiogenesis
- Support the body with an appropriate nutrition.

The Treatment

1 – Enzyme Yeast Cells Preparation
From the beginning I explained that enzyme yeast cells is the bedrock of my method (18) and among various therapeutic application utilized, enzyme yeast cells increase detox, activate the immune system and increase cellular respiration (19).

Posology (dosage)– 60 ml per day divided in 3 dosages mixed in a glass of carrot and red beet juice.

2 – Biobran MGn3
A Biological Response Modifier made from modified arabynoxylan from rice bran cultived on shitake enzyme with anticancer effects. Biobran is a strong immunomodulator activating
T-cells, B-cells, macrophages and especially NK cells (20-21)

Posology – one sachet of 1gr, 3 times per day after meals

3 – Liquid Cartilage Extract (LCE)
Inhibition of the angiogenic factor plays a crucial role in tumor inhibition and LCE made from liquid molecules extracted from shark cartilage in frozen form has strong antiangiogenic properties by targeting VEGF and MMP's (22) and reduce solid tumor size – chemotherapy combined with VEGF inhibition have shown a much better result in killing cancer cells. LCE is absolutely free of toxic adverse effects to the contrary of Trastuzumab(Herceptin), associated with significant adverse reactions including mortality.

Posology – 1 vial of 14 ml each day before breakfast.

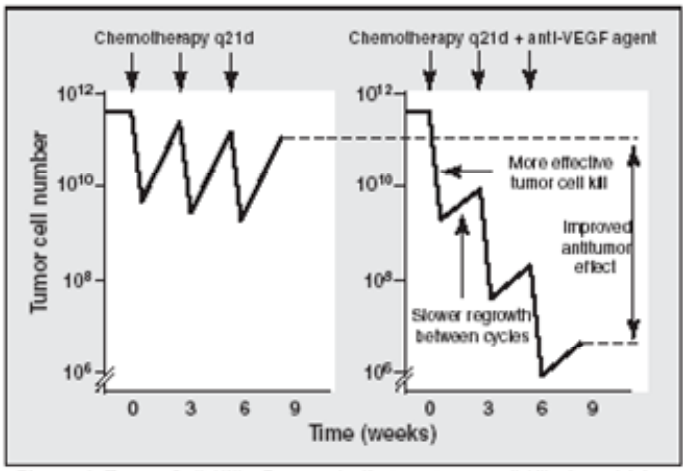

Figure 4: Tumor Cell Kill—Proposed effect on tumor cell kill of combining anti-VEGF agents with chemotherapy.

Fig. 4

4 – Oligopeptide
A short chain of amino acids that demonstrate efficiency to activate or reserve mutant P53 (23)

Posology – 4 tablets of 300 mg three times per day

5 – Curcuma
To target NK.KB, BCL2, P53 increase apoptosis – inhibit angiogenesis (24)

Posology – 1 capsule of 500 mg three times per day

6 – Venom Snake Therapy – Anticancer
This is an old therapy used in Europe to treat many diseases including cancer, depending on the composition and different type of snake venom.

Specific of breast cancer
Horvi 33
Horvi 300
Horvitrigon
Horvi x 44

1 ampoule of 1ml i.m. of each per week

To increase efficiency each ampoule can be mixed with some modern homeopathic remedies from Heel (Germany). Example: Glyoxal – Coenzyme Q10 – Ubichinon.

Additionally in case of a solid tumor 2 ampoules of Horvi 33 and Horvi 300 can be injected directly S.C. around tumor to achieve a better result.

Finally a tailored, aggressive anticancer diet that emphasizes plenty of fresh vegetables and fruits, whole grains, vegetable juices, increasing the detox process by combating constipation, as well as increasing liver and kidney function.

Suggested vegetables: Orange and yellow peppers, radish, leeks, tomatoes, red beet, cauliflower, broccoli, asparagus, egg lent, onion, garlic, and artichoke *.

* "Food That Fights Cancer" by R. Beliveau Biochemist and D. Gingras – Montreal – Canada
Healing Cuisine – Diet for Prevention and Recovery from Breast Cancer – www.healingcuisine.com

Hot Bath Therapy (Super Growth Energy Stone)

The Energy Sand Bath (ESB) is important during the course of the disease since not only does it stimulate detoxification of heavy metals, lipids, toxins but increases energy level and disrupts cancer cells. Take an ESB 3 or 4 times per week or even more. In case of solid resistant tumor together with the ESB the application of the ceramic sand balls (C.S.B.) directly on tumor decreases inflammation and contributes to reduction in tumor size. Information of how to take this bath is also available in the reference (25).

Initially the patient followed the treatment during 2 months, although the first molecular markers test was not done before the patient started the treatment, yet we are going to observe a significant modification between the first and the second test.

Live blood analysis

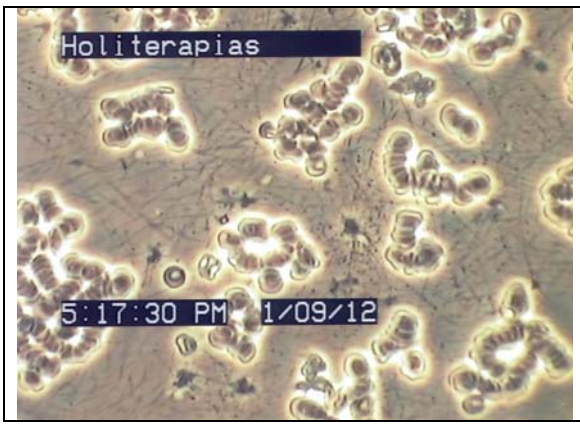

Fig .5

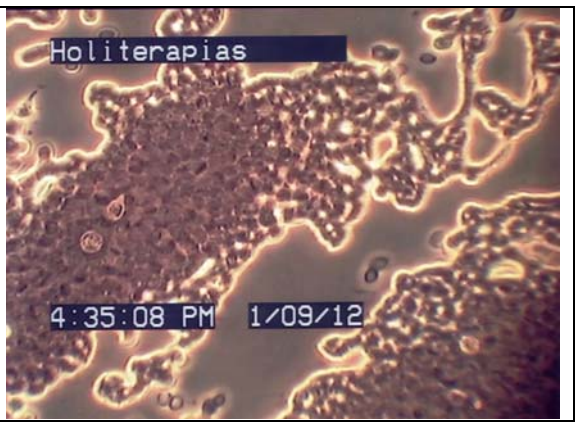

Fig. 6

The figure 1 shown denatures RBC's in rouleau and in between blood clots from disturbed homeostasis.

The figure 2 shown strong RBC's aggregation often observed in degenerative disease such cancer.

Both figures indicate blood viscosity decreasing blood circulation and oxygen delivery that stimulate hypoxia in breast cancer tissue which in turn stimulate the production of VEGF to induce angiogenesis (as demonstrate by the VEGF overexpression).

Oxidative dried blood layer test

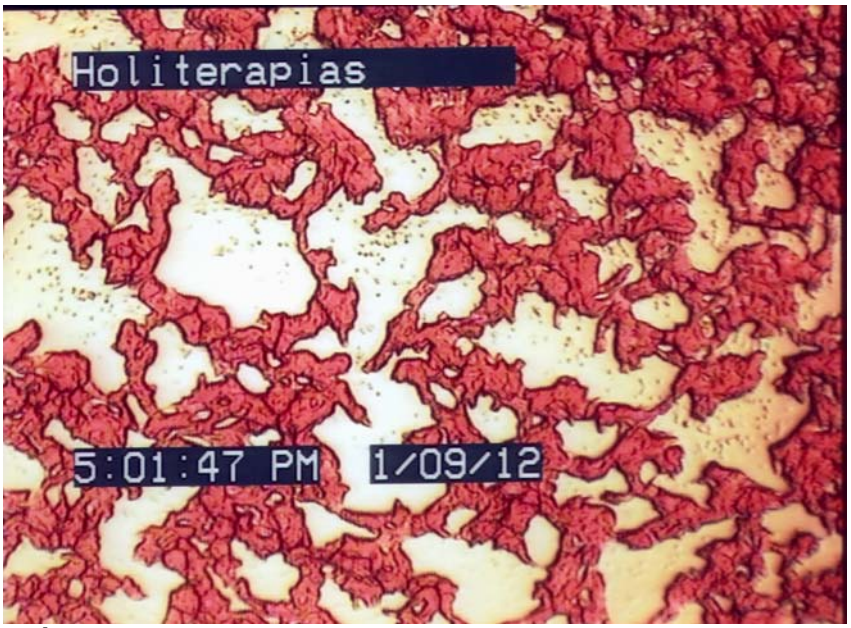

Fig. 7

The figure shown a major inflammatory process and cancer stage 3 with dissemination of large PPP's of 30 microns or larger. This is the 4th layer representing outside organ. (in this case breast area, chest, peripheral lymphatic system). This is an indication of persistent oxidative stress which may promote angiogenesis and cancer cells resistance in breast cancer. Indeed we found as demonstrate by the molecular markers test a very high VEGF activity.

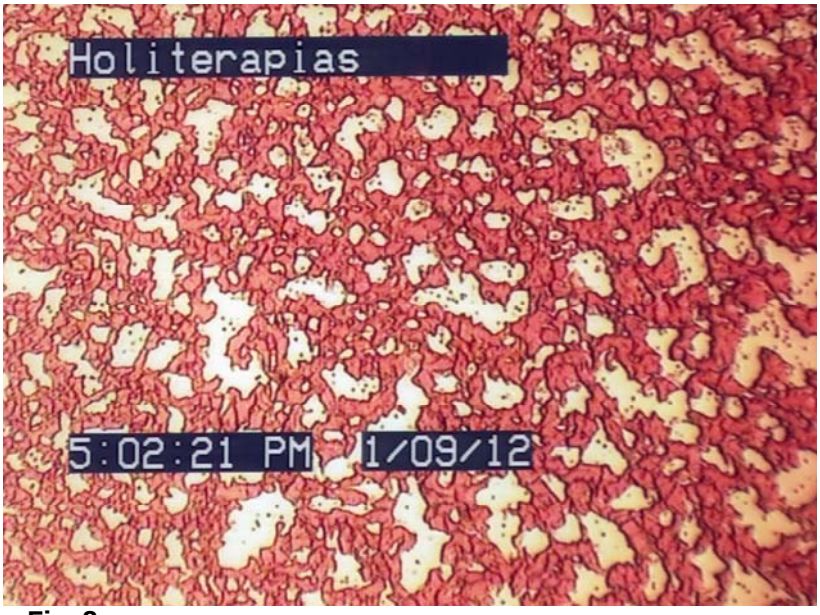

Fig. 8

The figure which represent the 6th layer of the drops of blood corresponding to internal organs (intestine, liver, etc.) we observe a total dissemination of smaller PPP's from 2 to 20 microns on the whole layer indication of a major inflammation and severe liver condition.

Result of the 1st Molecular Marker Test - 2/27/2012	
P53 gene expression – 200 units/ul of plasma Reference range – 10-50 units	
P53 normal protein – N.D. Reference range – 0.10-1.00 units	
P53 mutated protein – N.D. Reference range – N.D. units	
BCL2 gene expression – **8.000 units/ul of plasma** Reference range – 10 units	
BAX gene expression – 167 units/ul of plasma Reference range – 10-100 units	Ratio: 0.02
Survivin gene expression – **171 units/ul of plasma**	
P21 gene expression – 139 units/ul of plasma	Ratio: 0.8
VEGF gene expression – **2.353 units/ml of plasma** Reference range – 10-100 units	

Comment

It would appear that the tumor suppressor P53 was only active to some extent, not been able to produce normal protein, but we had no mutation. It could be the result of the treatment. However the current level of its activity was not enough to control the expression of the BCL2 gene expression which was very high and in all probability a major factor leading to disease progression. The BCL2/BAX ratio was very low 0.02 representing a bad prognosis. The Survivin/P21 ratio was 0.8 which needed to be improved. VEGF was very high, an indication of a very strong angiogenic activity leading to the growth of the tumor. Both the BCL2/BAX ratio (0.02), the high VEGF expression, and lack of P53 protein level to induce apoptosis, had shown a high resistance by the cancer cells to chemotherapy agents. Survivin was active to some extent but partially controlled by P21 although low in comparison to Survivin. The ratio Survivin/P21 was 0.8

At the second and third consultation, the patient significantly improved from her physical condition and the swollen lymph nodes disappear. We reduced the size of the tumor demonstrating that our complementary approach was well tolerated by the patient, increasing chemotherapy effectiveness – clinical experimentation such at the MD Anderson Clinic Center, University of Texas, shown that a combination of anti-VEGF agents as LCE together with chemotherapy was more efficient that chemotherapy alone.

Finally after obtaining a positive result and tumor reduction, the patient was operated on for a total mastectomy in July 2012.

The result of the cytology had shown 6 out of 8 ganglions isolated, were infected with metastases. After surgery the patient quickly recuperated and continued with our treatment and subsequently took chemotherapy. She developed some anemia and low WBC's which were quickly balanced by an addition of 2 injections of umbilical cord extract and extra red beet juice, together with some fermented chlorella rich in iron and vitamin C.

Result of the 2nd Molecular Marker Test – 5/25/2012	
P53 gene expression – 427 units/ul of plasma Reference range – 10-50 units	
P53 normal protein – 0.4 units/ul of plasma Reference range – 0.10-1.00 units	
P53 mutated protein – N.D. Reference range – N.D. units	
BCL2 gene expression – **796 units/ul of plasma** Reference range – 10 units	
BAX gene expression – 1.543 units/ul of plasma Reference range – 10-100 units	Ratio: 0.1
Survivin gene expression – **900 units/ul of plasma**	
P21 gene expression – 738 units/ul of plasma	Ratio: 0.8
VEGF gene expression – N.D. Reference range – 10-100 units	

2nd test done during chemotherapy regimen

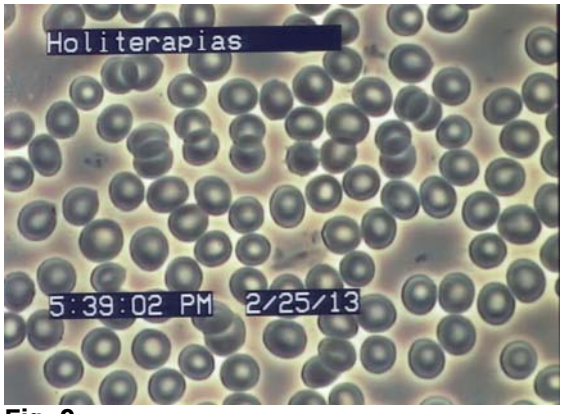

Fig. 9

Live blood analysis

We observe a total modification of the blood status.
Normal shaped RBC's clean blood plasma.
Good negative charge.
Balanced nutritional and antioxidant status.

Fig.10

Oxidative dried blood layer test

The figure shown a total change and a 100% elimination of the PPP's corresponding to ROS activity and metabolic dysfunction.

Both tests are in favor of our treatment which as demonstrate that have increased the efficiency of chemotherapy.

Comments

The current pattern has become more anticancer compared with the previous pattern. We have substantially activated the P53 tumor suppressor gene that produces normal protein, however only to some extent. BCL2 was considerably decreasing while BAX was highly active with a ratio of 0.19. However Survivin gene expression is now much increasing (900 units x 171 units), comparate to the first test and probably linked with many circulating cancer cells having high Survivin activity after surgery. In some study done by Yie et all Survivin expression was examined in 167 cases of breast cancer and the result suggest that apoptosis inhibition by Survivin alone or in cooperation with BCL2 is a significant prognostic parameter of worse outcome in breast carcinoma. However P21 gene expression also increase meaning the alternative chanel to apoptosis is activate and many cancer cells with active Survivin is self-destructed making the ratio same 0.8, while VEGF activity was totally eliminated. This new genetic profile was in favor of cancer cell self-destruction by chemotherapy, however BCL2 and Survivin needed to be decreased. Overall our treatment had positive effects to target BCL2 and VEGF both highly expressed, yet both had biomarkers with a predictive effect, strong resistance and opposition to apoptosis.

Gradually the number of lesions had decreased as shown by scan. By the middle 2013 the patient was totally free from liver lesions which are in favor of our treatment.

The patient continues our treatment except for the snake venom injection which was unnecessary. A third complete molecular markers test done on 4/8/2013 showed excellent results.

P53 gene expression – 874 units/ul of plasma Reference range – 10-50 units	
P53 normal protein – N.D. Reference range – 0.10-1.00 units	
BCL2 gene expression – 260 units/ul of plasma Reference range – 10 units	
BAX gene expression – 202 units/ul of plasma Reference range – 10-100 units	Ratio: 0.8
Survivin gene expression – 101 units/ul of plasma	
P21 gene expression – 738 units/ul of plasma	Ratio: more than 1

Comment

The tests had shown anti-tumor genetic activities associated with the treatment program. The oncogenes BCL2 and Survivin are now under the control of the anti-tumor genes P53, BAX and P21. P53 tumor suppression gene is active from 200 units/ul of plasma at the beginning to 874 units/ul of plasma although it now produces only trace of normal protein. The BCL2 gene expression has decreased (substantially from 8.000 units/ul of plasma to 260 units and BAX/BCL2 ratio increased from 0.1 to 0.8 in the same period of time. Survivin decreased from 900 units/ul of plasma to 101 units/ul of plasma. P21 gene expression increased to 176 units/ul of plasma with a ratio of more than 1. Further reduction of BCL2 and Survivin are necessary to sustain the state of remission through bioactive dietary supplementation and a 4th test should be done shortly and could be included in the complete article available on my internet site.

Result of the 4th Molecular Marker Test

The test done in 12/5/2013 about 8 months after the last test shown a beginning of a pro-tumor activity with the first time mutation of P53 and very high level of mutated protein (101.6 units/ml of plasma). The ratio-Bax/BCL2 remains almost the same, however Survivin gene expression is not detected (101 units/ml of plasma – 4/8/2013) and P21 gene expression is actived therefore the ratio. P21/Survivin is high 2.9 showing here a pro-tumor activity. However it also shown how is important to have the molecular marker test done at different interval in order to check any risk of cancer recurrence which may be her case but can be anticipated and prevented.

A heavy stress, infection, inflammation may be associate with the mutation of P53 and we are confident that our applied treatment will reactivate P53 in normal wild type function.

Conclusion

In this selected case we have clearly demonstrated that a complementary approach together with mainstream chemotherapy is quite efficient, safe, does not interfere with chemotherapy, increases the quality of life of the patient and increases the chance of remission by increasing chemotherapy effectiveness. We demonstrated how pro-apoptotic and anti-apoptotic genes can be targeted with bioactive dietary agents to contribute to increased apoptosis and cancer cell self-destruction. While molecular markers still remain in the field of research, we feel that by translating basic knowledge into clinical practice we have one very important step to better diagnosis and improving cancer cure which is badly need.

Fig.11 - *The patient with her daughter*

Among our many papers: "Integrative Cancer: How to Improve the Present Situation and Open New Doors in the Field of Cancer" is recommended and available in my website. Because of lack of space the complete article with full illustration of peripheral blood analysis before and after the treatment, is now available on my website, include additional information, paper on integrative cancer, etc. www.sergejurasunas.com .

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