# Tumor Regression in a Case of Stage IV Colon Carcinoma Achieved by a Novel Nutritional Therapy

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#### Abstract

**Objectives:** Colon adenocarcinoma is a cancer of high incidence in humans. The goal of this study was to determine the possible clinical benefit of the molasses-based MSQ 15D dietary supplement in a case of stage IV colon adenocarcinoma.

**Design:** Single case study.

**Settings/Location:** Home.

**Interventions:** The regimen of dietary supplement was administered as follows:

MSQ 15D, 2tbsp TID.

Outcome measures: Clinical improvement and regression of metastatic disease.

**Conclusions:** Treatment with the MSQ 15D formula resulted in tumor regression and the reversal of clinical manifestations of the disease. This approach may provide an effective therapeutic modality for stage IV adenocarcinoma of the colon.

### Introduction

Colorectal cancer is the fourth most prevalent cancer and the second leading cause of cancer-related death in the United States<sup>1</sup>. Risk factors include smoking, alcohol consumption, obesity, and a high-sugar, fat-rich, low-fiber diet. The etiology of colorectal cancer is unknown, but both genetic and life-style factors are suspected. The five-year survival rate is around 60%, primarily due to screening programs aimed at detecting early stage disease. Standard treatment options involve surgery, radiation and various chemotherapy protocols that are highly toxic<sup>2</sup>. Despite all therapeutic advances, the overall mortality rate remains high; disease progression can be rapid, and the development of multi-drug resistance poses a major obstacle. For these reasons, new therapeutic modalities are widely sought and anticipated.

In a previous paper, we reported a case study on using a novel, diet-based method for the therapy of an acute myelogenous leukemia (AML) patient<sup>3</sup>. AML is a lethal cancer with a very low 5-year survival rate<sup>4,5</sup>. The nutritional therapy utilized was based on our analysis of common dietary deficiencies present in cancer patients<sup>6</sup> as well as an analysis of the

etiology of cancer, in which the emergence of the cancer stem cell is linked to infectious causes<sup>7</sup>. These analyses led to the hypothesis that all cancers share a common initiation pathway, and therefore it may be possible to design a common therapeutic approach<sup>8</sup>. Here, we report that this nutritional therapy resulted in tumor regression in a case of metastatic colon cancer.

## Case report

A 54-year-old male observed change in his bowel function in September 2002. He experienced constipation and difficulties in emptying, as well as pain in the left side of his pelvis. Ultrasonographic exam detected a pathological mass in the descendent colon. Colonoscopy revealed a tumor that nearly occluded the lumen of the colon at the junction of the sigmoid and descendent colon. Upon tumor biopsy, histology confirmed adenocarcinoma stage III, T3N1MX, Dukes C1. Segmental resection of the colon was performed at the end of October 2002. In November 2002, 12 cycles of adjuvant chemotherapy were initiated using the DeGramont protocol. This therapy continued until April 2003.

In April 2004, the patient presented with a sudden rise in blood pressure. Abdominal computed tomography scan and renal scintigraphy diagnosed hydronephrosis carotis verte. Subsequently, a laparoscopic left-sided nephrectomy was performed. In the middle of August 2004, elevated CEA (4.2ng/ml) was detected, although abdominal and chest computed tomography scans found no abnormalities.

In mid-July 2005, a PET-CT scan detected a 10mm diameter focal FDG accumulation at the level of the 12<sup>th</sup> thoracic vertebra. Several 8-30mm diameter focal FDG accumulations were also detected paraaortically between the lumbar 2-5 vertebrae. About 2/3<sup>rd</sup> of the oral section of the ascendant colon showed intense FDG accumulation, while the rest of the intestines showed normal accumulation. These observations indicated recurrent, disseminated disease with retroperitoneal involvement.

In early August 2005, a colonoscopy discovered numerous pea-sized thickenings of the mucosal surface at a height of 30cm in the colon. A diagnosis of diverticulosis was made. Subsequent computed tomography scan detected the in 10<sup>th</sup> segment of the lung a subpleural focus 9mm in diameter that was a metastasis. Wall thickness of the rectal and a 13cm long section of the sigmoid colon varied from 8-11mm, suggesting a pathological mass.

In mid-September 2005, 5 cycles of chemotherapy were initiated using the FOLFIRI protocol. At the end of October 2005, a computed tomography scan detected a 7mm diameter focus subpleurally in the 9<sup>th</sup> segment of the right lung, and below that another 3mm size focus. These were pulmonary metastases. Progression of the previously described abdominal lymph node metastases was also observed.

Progression of the disease required a change in the chemotherapy protocol to the FOLFOX regime (6 cycles). Computed tomography scan at the end of February 2006 demonstrated further progression of the retroperitoneal metastases. Despite its lack of efficacy, continuing the same chemotherapy protocol was recommended. At that point, the patient decided to pursue an alternative solution, and in the mid-April 2006 began taking our MSQ 15D dietary composition, 2tbsp TID.

At the end of July 2006, the patient presented with a severe *Salmonella* infection after eating an infected egg and was hospitalized for 5 days. After completing a course of antibiotic therapy, he continued to have difficulties with food intake for another two weeks, experiencing a weight loss of 15kg during this episode. He was taking probiotics and received injections of Vitamins B6 and B12, Vitamin C and Vitamin E from his general practitioner. He was unable to take the MSQ 15D during this period.

In early August 2006, a computed tomography scan found no progression of the retroperitoneal lesions, while some of them were already undergoing calcinosis. There was no change in the status of the lungs. In mid-August 2006, the patient was admitted to the hospital and presented with difficulties with swallowing and vomiting. Oesophago-

gastro-duodenoscopy exam found an obstruction in the jejunum that was biopsied. Braun anastomosis was subsequently performed to open up the passage. The jejunal obstruction was possibly a consequence of the inflammation caused by the *Salmonella* infection.

Restoring passage through the anastomosis required reoperating, followed by a nearly month-long period of recovery. The patient began using the MSQ 15D again in early October 2006, and shortly afterwards regained 4kg of weight. In mid-October 2006, a computed tomography scan demonstrated no disease progression in the abdomen and calcification of some of the retroperitoneal lymph nodes. There was no change in the status of the lungs.

During November 2006, the patient's weight gain continued (6kg gain). His energy returned and he resumed working, initially with a light workload. He continued to recover through the rest of the year, with a weight gain of 7kg. In early January 2007 the patient was hospitalized with passage disturbance, and was released following day after his passage resumed.

In early February 2007, a computed tomography scan showed some calcified retroperitoneal lymph nodes. No tumor was detected in the abdomen or the chest. The patient was carrying a full workload at that time and pursuing normal activities. On March 10, 2007, a Friday night, the patient felt abdominal pain after consuming a heavy meal, and reported to the hospital. Ultrasonongraphic scan detected ileus, and the patient was admitted to the ICU. No surgical team was on call for the weekend in the small town hospital. By Monday, the patient was septic and expired. Autopsy revealed that the obstruction in the small intestine was caused by adhesions; no tumors were found in the abdomen.

## Discussion

Colorectal cancer is the second leading cause of cancer-related death in the United States<sup>1</sup>. Despite advances made in the treatment of colon cancer, ample opportunities

remain for therapeutic improvements. The overall mortality rate is high and the therapies are highly toxic. The patient of this case study had received standard therapies, however the lack of efficacy as well as treatment side effects led him to our dietary composition for the treatment of his disease.

This paper describes the use of a non-toxic, nutrition-based therapy for a case of stage IV colon adenocarcinoma. Nutrient deficiencies of plant-derived phenolic compounds, folate, vitamin B12 and other vitamins of the B class, essential lipids, iodine, and several minerals have been found to co-exist in and increase the incidence of a variety of cancers<sup>6</sup>. This correlation led us to reexamine the role of nutrition, unifying the perspective on cancer and recasting it as a single disease, potentially treatable by a single protocol. Based on this perspective, we hypothesized that supplementing deficient nutrients in cancer patients might reverse the course of their disease. In a previous case study with an AML patient, we demonstrated the therapeutic effect of this approach<sup>3</sup>.

Recently, we analyzed links between infections, inflammation and tumorigenesis, specifically on how chronic infections and tissue inflammation could facilitate formation of cancer stem cells<sup>7</sup>. Colonic inflammations due to chronic infections are known to play a role in the development of colon cancer<sup>9-12</sup>. Phenolic polysaccharides, a main component of the blackstrap molasses used in the MSQ 15D dietary supplement are potent anti-inflammatory and anti-carcinogenic compounds<sup>8</sup> and likely play an important role in suppressing the underlying causes of colonic tumorigenesis. As the gut is a main point of entry of pathogens into the body, maintaining the health of the digestive system should be a major concern for both prevention and therapy.

This study demonstrates the result of this treatment strategy in a case of stage IV colon adenocarcinoma. This patient went through standard surgery and chemotherapy regimens, but his condition kept deteriorating. When conventional options failed, he chose the MSQ 15D dietary composition as his sole therapeutic modality. This initially led to the arrest of disease progression. Gastrointestinal complications from a nearly fatal *Salmonella* infection led to duodeno-jejunectomy followed by a long period of limited

food intake along with considerable weight loss. This resulted in a two-month cessation in the MSQ 15D therapy. From November 2006, that patient was on a path of recovery, regaining much of his lost weight. A computed tomography scan in February 2007 found no tumor in his body. He regained energy and returned to work. Although advised to watch his diet and avoid heavy meals in general and particularly late at night, he found this advice difficult to follow. His lack of self-discipline along with the absence of basic medical services over a weekend time period caused his death. Autopsy demonstrated no tumors in his body. His case was still a success in light of the treatment's ability to achieve tumor regression in a multidrug-resistant, progressing, metastatic disease.

This case study again demonstrates that a novel nutritional therapy may provide a tool for the therapy of metastatic colon carcinoma, and gives further proof to the hypothesis that a common therapeutic approach can be designed for cancer. Further studies are warranted to investigate the utility of this nutritional strategy in a larger population of stage IV colon cancer patients.

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