

## Case Report: Carcinoembryonic Antigen (CEA) in Postoperative Follow-up and Treatment Monitoring of Colorectal Adenocarcinoma

[K.S. Price](#)<sup>\*</sup>, M.D., and [D.M.P. Thomson](#)<sup>†</sup>, M.D., Ph.D., F.R.C.P.(C)

\* To whom correspondence should be .

<sup>†</sup> Department of Medicine, Montreal General Hospital, McGill University, 1650 Cedar Ave., Montreal, QC, Canada H3G 1A4

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### THE CASE

A 53 year-old married secretary and mother of three was referred to the Montreal General Hospital in April 1991 by her family physician for investigation of a one month history of bright red blood per rectum associated with the passage of stool. The patient was otherwise well and reported a good appetite. She denied weight loss, fatigue, fevers, night sweats, and any lumps or itch. The patient described scant streaks of red blood on the toilet tissue and on the surface of formed stool. She denied any change in frequency, color, or consistency of stool. In addition, she reported no indigestion, nausea, vomiting, abdominal pain, tenesmus, or urgency of defecation.

Her past medical history was significant for 30 pack-years of smoking and an abdominal hysterectomy sparing the ovaries in 1981. There was no history of ethanol abuse, nor was the patient taking any drugs, either illicit, over-the-counter, or by prescription. There was no personal or family history of cancer.

Physical examination was unremarkable, with normal vital signs, no lymphadenopathy, and a benign abdomen with no hepatosplenomegaly or appreciable masses on palpation. Digital rectal exam was remarkable only for OB+ stool. Blood tests, including leukocyte count, hemoglobin, hematocrit, electrolytes and liver enzymes, were all normal. Colonoscopy and barium enema were performed, revealing a 3.5 cm mass at the rectosigmoid junction. Chest radiograph and abdominal CT were normal. An anterior rectosigmoid resection and oophorectomy were performed in April 1991. Pathology revealed a moderately differentiated adenocarcinoma with invasion of pericolonic fat and 15 of 15 lymph nodes negative for malignant cells. The diagnosis of Dukes Stage B2 colorectal cancer was made. Unfortunately, no CEA levels had been taken before surgery; however, CEA levels were monitored postoperatively on approximately a three-month basis ([Table 1](#)). The patient remained well and declined further treatment until March 1992, when, asymptotically, the CEA level rose and abdominal ultrasound revealed three liver lesions. Biopsy confirmed metastasis, and treatment with somatostatin was started. Colonoscopy was normal, demonstrating disease-free anastomosis. The patient remained asymptomatic until July 1993, when she complained of right upper quadrant fullness. Serial abdominal CT scans showed increased size of the liver metastases which was correlated with a rise in CEA level. Intrahepatic administration of 5-fluorouracil (5-FU) with leucovorin was begun in view of the disease progression. The patient responded to monthly 5 day regimens of 5-FU with

reduction of CEA levels and shrinkage of liver lesions by CT. Treatment was stopped in February 1994.

At the time of this report, the patient remains well, with follow-up every four months.

## DISCUSSION

The present case illustrates the utility of the CEA assay in the clinical context. CEA was originally discovered as a marker for colorectal carcinoma in 1965 (1), and a radioimmunoassay for detecting this marker in the blood was developed shortly thereafter (2). The CEA assay has potential clinical application in detection, diagnosis, prognosis, treatment monitoring and therapy for cancer. As demonstrated here, CEA testing is clinically indicated for the monitoring of both tumor recurrence and treatment response in colorectal cancer (3). In addition, preoperative CEA levels are believed to be of prognostic value in colorectal (4,5) and breast (6) cancer. However, current CEA assays are not of sufficient sensitivity or specificity to be used for screening, and CEA measurements by themselves are not diagnostic of cancer.

As is often the case, the rise in CEA postoperatively in this patient heralded the recurrence of disease before the development of symptoms, thus enabling earlier treatment (7). Furthermore, serial CEA testing in conjunction with imaging showed the lack of response to somatostatin and prompted a successful change in treatment that was appreciated by a decline in CEA in conjunction with tumor bulk. Unfortunately, preoperative CEA levels were not obtained in this case and therefore were not available to aid in the formulation of the prognosis. Yet, the fact that postoperative levels taken were within the normal range was encouraging, suggesting that all of the tumor mass was removed. It must be emphasized, however, that truly complete surgical resection of cancer, on both macro- and microscopic levels, is impossible. In this context, CEA holds additional great promise in the form of radioimmunolocalization and immunotherapy of CEA-producing tumors. In particular, in a case such as the one presented here (i.e., Dukes Stage B2), where the risk for recurrence is high, the development of improved adjuvant therapies utilizing, for example, anti-CEA antibodies as carriers of anti-tumor agents, or recombinant CEA gene products as stimulators of an anti-tumor immune response, could dramatically change prognosis. The potential for relapse raises the important issue of continued CEA testing in patients with high risk of tumor recurrence once all treatment options have been used. In the event of future recurrence, what can be offered? Although, at the time of this report, the patient presented here is well and does not wish to be burdened with an advanced warning and regular reminder of the potential for relapse, patient attitudes vary, and the complexity of long-term management in individuals at high risk for recurrence underscores the need for continued research toward new treatments for colorectal cancer.

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## BIOGRAPHY

**Kursteen S. Price, M.D.** received B.Sc. and M.Sc. degrees in Physiology from McGill University (Montreal, QC, Canada) in 1990 and 1992 for work on the role of endotoxin in graft-versus-host disease. She received her M.D. degree from McGill in 1996, is presently a second-year resident in Internal Medicine at McGill, and continues her work on graft-versus-host disease.

**David M.P. Thomson, M.D., Ph.D., F.R.C.P.(C)** earned his M.D. *cum laude* in 1964 from the University of Western Ontario (London, ON, Canada) and completed his postgraduate medical training at the Montreal General Hospital (Montreal, QC, Canada). As a resident in Clinical Immunology and Allergy, through the influence of Dr. Phil Gold, he became interested in cancer immunology, and it was in Dr. Gold's laboratory that they developed the CEA radioimmunoassay. From 1970-72, he performed Ph.D. work in tumor immunology at the University of London (London, England), under the supervision of Dr. Peter Alexander, and received his doctorate in 1973. Upon his return to Canada, Dr. Thomson pursued his research interest in tumor immunology. Presently, he is a Professor of Medicine at McGill University and a Senior Physician at the Montreal General Hospital.

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