A Case Report of Statin-Induced Fluctuations in Carcinoembryonic Antigen (CEA) Levels in a Breast Cancer Survivor

Azin Alizadehasl
Iran University of Medical Sciences
Azam Yalameh Aliabadi (Azamyalameh67@gmail.com)
Iran University of Medical Sciences
Seyedeh Fatemeh Hosseini Jebelli
Iran University of Medical Sciences
Sarah Forati
Iran University of Medical Sciences
Yasamin Afsari Zonooz
Iran University of Medical Sciences

Case Report

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Abstract

Introduction: This case report explores the unique fluctuations of carcinoembryonic antigen (CEA) levels in a breast cancer survivor following statin therapy, contributing to the body of knowledge by documenting a potential drug interaction not commonly recognized in medical literature.

Patient's Main Concerns and Clinical Findings: A 63-year-old female breast cancer survivor presented with significant CEA level elevations post-initiation of atorvastatin for cardiovascular screening, which was not explained by conventional diagnostic assessments.

Primary Diagnoses, Interventions, and Outcomes: The primary intervention was the administration of atorvastatin, which coincided with the rise in CEA levels, followed by a marked decrease upon drug cessation, suggesting a statin-induced effect.

Conclusion: The case highlights the necessity for clinicians to be aware of the potential for statins to affect tumor markers like CEA, advocating for personalized monitoring and consideration of such interactions in cancer survivorship care. Further investigation into statin-induced CEA fluctuations is recommended to understand the implications for cancer surveillance and management.

Introduction

Carcinoembryonic antigen (CEA) serves as a crucial serum tumor marker, playing a significant role in both the diagnosis and monitoring of various solid tumors, such as breast cancer. In advanced and metastatic breast cancer, elevated serum CEA levels are frequently observed, ranging from 36–70% according to studies (1, 2). CEA is recognized as a valuable marker in the clinical management of breast cancer patients, particularly for monitoring treatment response and disease progression (3).

The use of carcinoembryonic antigen (CEA) as a breast cancer marker has certain limitations and its utility as a screening tool is limited. recent reports have discouraged the routine use of CEA for monitoring breast cancer due to its low sensitivity, especially in the early stages of the disease (4). CEA may not effectively detect treatable recurrences at an early stage, and its clinically relevant effect on patient mortality remains to be proven (5). Nonmalignant Conditions can also affect the accuracy of carcinoembryonic antigen (CEA) test results, CEA levels can be influenced by age, body mass index (BMI), white blood cell (WBC) count, hemoglobin (HB) levels, aspartate aminotransferase (AST), and glycosylated hemoglobin (HbA1c) (6). Several drugs have been shown to increase the expression of CEA in cancer cells. These medications include antineoplastic drugs like 5-fluorouracil, cytokines such as interferons or interleukin-6, differentiating agents like sodium butyrate, lithium, and protein kinase inhibitors such as staurosporine. (7, 8)(9). It's important to consider the potential influence of these drugs when interpreting CEA test results, especially in patients undergoing treatment with these agents. In the case presented herein, a possible causality between statin medication and possible elevation of CEA is reported.

Patient information

A 63-year-old female, previously diagnosed with breast cancer (two years ago), underwent standard treatment modalities, including chemotherapy, radiation therapy, and hormone therapy, resulting in a full recovery. In the routine cardiovascular screening, coronary CT angiography, revealed coronary artery involvement, leading to the initiation of atorvastatin 20 mg.

Clinical Findings

At the onset of statin therapy, the CEA level was measured at 1.21 ng/mL (Fig. 1).

Surprisingly, over the subsequent nine months of regular atorvastatin intake, the CEA level increased significantly, reaching 26.79 ng/mL.

Diagnostic Assessment

The patient underwent extensive diagnostic evaluations, including endoscopy, colonoscopy, and a PET scan, all of which showed normal results and failed to explain the observed rise in CEA (Fig. 1). Despite the lack of apparent malignancy in imaging studies, the persistent elevation in CEA raised concerns and prompted further investigation.

Therapeutic Intervention

At a CEA level of 26.79 ng/mL, the patient requested discontinuation of atorvastatin 20 mg, and we adhered to their preference. Remarkably, three weeks post-discontinuation, the CEA level decreased to 19.8 ng/mL and continued to decline, reaching 0.6 ng/mL four months after the cessation of atorvastatin (Fig. 1). The temporal correlation between the discontinuation of atorvastatin and the decline in CEA levels suggested a potential association between statin use and CEA fluctuations in this breast cancer survivor. Subsequently, rosuvastatin 20 mg was initiated as an alternative statin therapy.

Follow-up and Outcomes

After three months of daily use, the CEA marker exhibited an increase to 2.2 ng/mL, raising concerns about a possible class effect of statins on CEA levels in this specific patient (Fig. 1).

Discussion

The observed fluctuations in CEA levels following statin therapy in this breast cancer survivor present a unique and intriguing scenario. The strengths of our approach include the diligent tracking of CEA levels in response to statin therapy and the prompt adaptation of treatment in response to patient concerns,

which is a testament to personalized healthcare. Moreover, the use of comprehensive diagnostic tools to exclude recurrent malignancy exemplifies a meticulous and thorough approach.

However, the limitations are evident in the lack of a control group and the inability to completely rule out other potential causes for CEA elevation, such as minor undetected inflammatory processes or concurrent medications. The case also brings to light the complexity of interpreting tumor markers in the presence of pharmacologic interventions and the need for caution when assessing such markers for disease monitoring.

While statins are widely known for their cardiovascular benefits, emerging evidence suggests potential pleiotropic effects, including anti-inflammatory and anti-cancer properties. The association between statins and cancer outcomes remains a topic of ongoing research, and individual patient responses to these medications may vary. Our review of the medical literature suggests that while statins are not commonly known to affect CEA levels, there is a potential for drugs to influence tumor markers. This aligns with emerging evidence indicating that medications can have unexpected effects on biomarkers used in oncology. The subsequent increase in CEA levels upon initiating Statin medication raises questions about whether this phenomenon is specific to certain statins or represents a broader class effect. Further investigation is warranted to explore the mechanisms underlying the observed changes in CEA levels and their relevance to cancer surveillance in breast cancer survivors.

The rationale for our conclusions is based on the the initial rise in CEA during atorvastatin therapy, followed by a significant decline upon discontinuation, suggests a potential causal relationship. This temporal association, coupled with the lack of other identifiable causes, suggests a drug-related effect.

This case underscores the importance of vigilant monitoring and individualized care in cancer survivors receiving statin therapy. Healthcare providers should be aware of potential interactions between cancer markers and commonly prescribed medications, tailoring treatment plans based on the patient's unique response. Additionally, collaboration between oncologists and cardiologists is crucial to optimize the overall care of cancer survivors with cardiovascular comorbidities. In conclusion, this case report highlights a novel and potentially significant interaction between Atorvastatin and CEA levels in a breast cancer survivor. Further research is essential to elucidate the underlying mechanisms and establish the clinical implications of this observation, ultimately guiding personalized treatment approaches for cancer survivors with cardiovascular risk factors.

Declarations

Availability of data and materials:

The data of this case report are not publicly available due to privacy and ethical restrictions but are available from the corresponding author on reasonable request.

Ethical Approval:

Informed consent was obtained from the patient for the publication of this case report. The research was conducted in adherence to the principles outlined in the Declaration of Helsinki, ensuring ethical standards and patient rights in medical research.

Competing interests:

The authors declare that they have no competing interests.

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Authors' contributions:

A.Y.A. (Azam Yalameh Aliabadi) served as the corresponding author and was primarily responsible for conceptualizing the study, overseeing the research process, and manuscript preparation. A.A. (Azin Alizadehasl), as the supervisor, provided expert guidance, reviewed the study design, and contributed to the final manuscript. S.F.H.J. (Seyedeh Fatemeh Hosseini Jebelli), S.F. (Sarah Forati), and Y.A.Z. (Yasamin Afsari Zonooz) were actively involved in the patient's management and follow-up, data collection, and analysis. All authors critically reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity.

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Figures

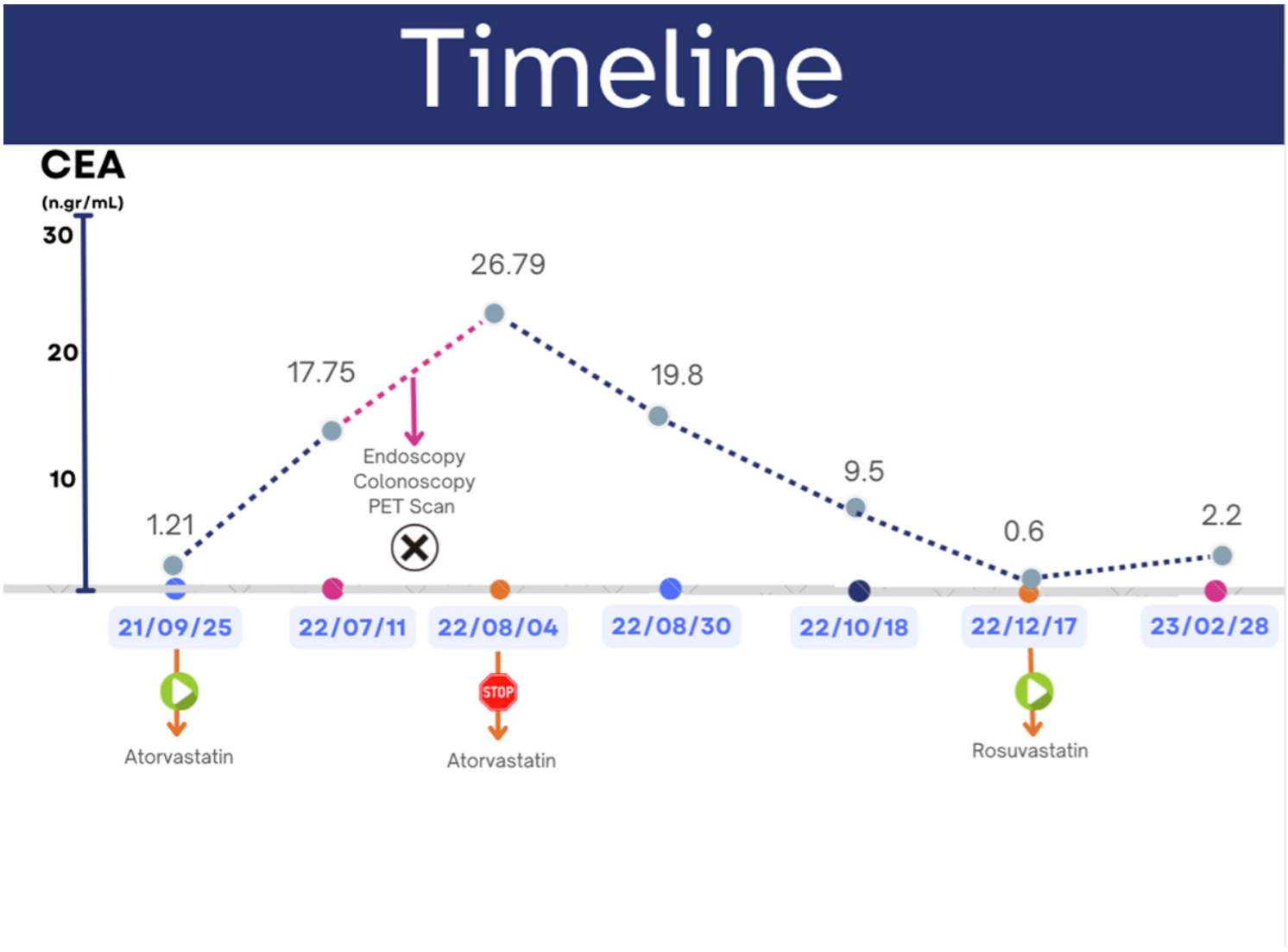

Figure 1

Patients Time line.

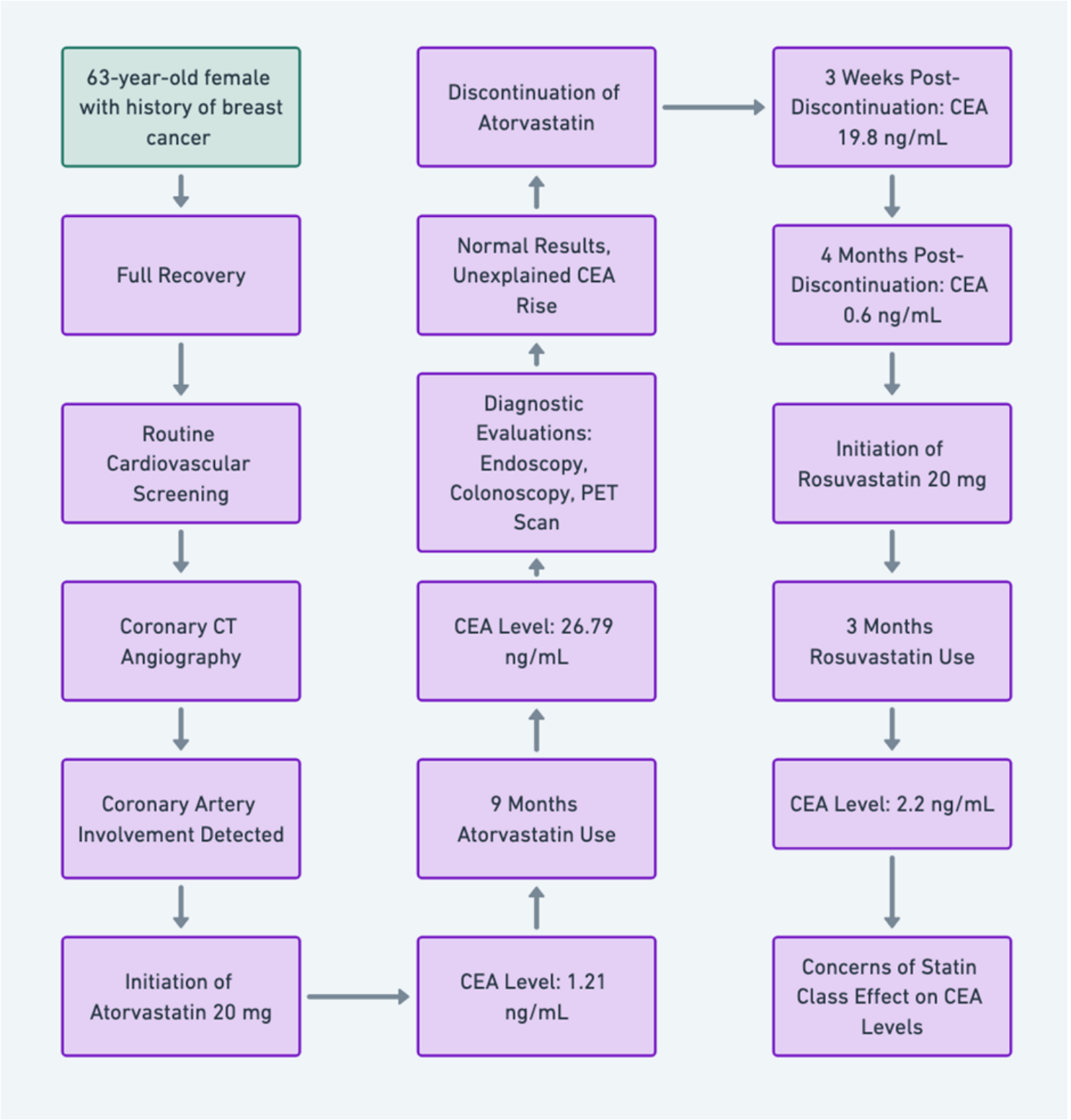

Figure 2

Graphical event sequence.