Elevated Carbohydrate Antigen 125 Level in a Patient with Right Heart Failure

Abstract

Carbohydrate antigen 125 is a known marker for ovarian cancer. A unique case of elevated carbohydrate antigen 125 in a patient with rheumatic heart disease, preserved left ventricular function and isolated right heart failure is reported.

Keywords: Heart failure; Rheumatic heart disease; Right ventricle; CA 125

Case Report

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Introduction

Serum CA125, a high-molecular weight glycoprotein, is a tumor marker widely used for the diagnosis and follow-up of patients with ovarian cancer. Recent studies have shown that CA125 is elevated in heart failure (HF) patients demonstrating a strong correlation with the clinical and echocardiographic findings. We report a case of elevated carbohydrate antigen 125 in a patient with right heart failure due to rheumatic heart disease.

Case Report

A 55-year-old woman with history of rheumatic heart disease, mitral valve and aortic valve replacement four months ago was admitted with dyspnea, abdominal distention and swelling in her lower extremities. Her physical examination was remarkable for ascites and bilateral pedal edema. A chest roentogram showed a small right pleural effusion. Her electrocardiogram was consistent with sinus bradycardia. Her echocardiogram showed a normal left ventricular function with an ejection fraction of 55-60%, moderate pulmonary hypertension, moderately dilated right ventricle with moderately decreased function, moderate tricuspid regurgitation, moderate to severe pulmonic regurgitation, moderate right atrial enlargement and mild left atrial enlargement. Her right heart catheterization showed a right atrial pressure of 28 mmHg, a right ventricular end diastolic pressure of 31 mmHg, a pulmonary artery pressure of 92/36 mmHg, a mean pulmonary artery pressure of 68 mmHg, pulmonary capillary wedge pressure of 22 mmHg, a pulmonary artery saturation of 33% and a cardiac index of 1.7 L/min/m^2. During initial investigation, it was found that her carbohydrate antigen (CA) 125 was markedly elevated to 871 U/ml (upper normal level is 35 U/ml). Investigation for gynecological malignancy by ultrasonography and computer tomography of the abdomen and pelvis was negative. Her ascites was tapped and the analysis did not show any malignant cells. She was started on diuretics and ionotropes with clinical improvement. Her symptoms were resolved and there was no evidence of abdominal distention on exam after few days of hospitalization. Since no obvious malignancy could be identified, her CA 125 was repeated and it was 35 U/ml.

Discussion

The CA 125 is a high molecular weight (approximately 2–5 million daltons) glycoprotein synthesized by epithelial serosal cells, such as the pleura, pericardium and peritoneum [1]. Inflammation or stretch of the serosal surface may be responsible for elevation of CA 125. The biological role of CA 125 in humans is not clearly understood. CA 125 is a tumor marker that is used in ovarian cancer. It has been shown to be useful for diagnosis of suspected cases, to monitor the efficacy of treatment and for early detection of recurrence. Increased CA 125 has also been noted in other neoplastic diseases like lung, breast, uterine and gastrointestinal tract cancer and non-neoplastic diseases like liver cirrhosis, serosal effusion and renal impairment [1].

Nagele et al. [2], while measuring various biomarkers in HF patients, were the first to identify this strong correlation between elevated CA 125 level and the severity of the clinical picture of patients [2]. Other studies investigated the evidence of this correlation in acute failure cases and right-sided failure cases [3,4]. D'Aloia et al. [4] showed a significant correlation of CA 125 levels with the right atrial and systolic pulmonary artery pressure. In a different study by Yilmaz et al. [5], elevated levels of CA 125 reflected right ventricle dysfunction in patients with COPD even before corpulmonale to be evident [5]. Our report adds to the literature a unique case that describe CA 125 elevation in a patient with isolated right heart failure in the setting of rheumatic heart disease [6,7]. Though the exact mechanism remains unclear, the production of CA 125 is hypothesized to be increased secretion by the peritoneal mesothelium, either via a direct stretch activation due to the as cites from the right heart failure or via an indirect activation through inflammatory cytokines such as interleukin-6 [8]. Furthermore, the increased pro-inflammatory state in heart failure likely increases the production of CA 125 from the pericardium as well. In addition, stretching of the pericardium from the dilated heart chambers will likely lead to increased secretion of CA 125 from the visceral pericardial epithelial cells. This is a similar process to the stretching of the myocytes that leads to secretion of

brain natriuretic peptide from the ventricles of the heart. It is important to recognize that benign conditions such as isolated right heart failure can result in markedly elevated CA 125 level.

Conclusion

Our patient presented with symptoms suggestive of volume overload. During initial investigation, it was found that her CA 125 was elevated and after her CHF was appropriately treated and her symptoms were resolved, CA 125 returned back to baseline. Various mechanisms have been hypothesized to explain the association between volume overload/heart failure and CA-125 elevation. In this sense, it is important to recognize that benign conditions such as isolated right heart failure can result in markedly elevated CA 125 level. Further studies are necessary to confirm the sensitivity and specificity and to see if it could be a predictor of mortality and morbidity or used in risk stratification of patients with CHF.

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