ELDERLY WOMAN WITH MASSIVE PERICARDIAL EFFUSION, CARDIAC TAMPOANADE, AND HYPOTHYROIDISM

To the Editor: A 74-year-old woman, was brought for geriatric consultation after having a fall while walking in the street. She reported feeling dizzy; she had no cranial trauma or loss of conscience. On past history, family reported having high blood pressure diagnosed 3 months before, treated with enalapril and spironolactone. She was reported to be independent for basic activities of daily living but had difficulty managing finances 3 months before consultation and had stopped preparing meals 1 month before. She reported fatigue, hyperoxia, and moderate exertional dyspnea. Physical examination revealed an elderly woman with moderate alopecia, no neck vein distention, a pericardial rub, a systolic murmur most audible on the mitral area, and generalized nonpitting edema. On cognitive and affective screening there were no abnormalities. Routine laboratory data showed hemoglobin of 11 g/dl; mean corpuscular volume of 91 fl; white blood cell count of 3,900/μL, platelets of 288,000/μL, glucose of 92 mg/dL and creatinine of 0.6 mg/dL. Because of cardiac abnormalities detected on clinical grounds, a transthoracic echocardiogram was performed. It showed pericardial effusion with hemodynamic compromise because of diastolic collapse of the right chambers, approximate volume of 300 mL, and concentric parietal hypertrophy, diastolic dysfunction, and ejection fraction of 72%. Thyroid function tests were ordered and found a thyroid-stimulating hormone (TSH) of 36.9 mIU/mL. Hospitalization for pericardiocentesis was suggested, but the patient and family refused treatment. Levothyroxine 12.5 μg/d was initiated. One month later, the patient presented for consultation again, so a new echocardiogram was performed that indicated a small pericardial effusion with postural hypotension. J Hum Hypertens 1994;8:711–716.

References


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REFERENCES

DO ADIPONECTIN ISOFORMS IN OLDER ADULTS WITH OR WITHOUT CORONARY ARTERY DISEASE DIFFER ACCORDING TO GLUCOSE TOLERANCE STATES AND LIFESTYLE FACTORS?

To the Editor: Adiponectin, an adipose tissue–derived protein, is an important regulator of insulin sensitivity and inflammation. The hormone plays a role in the suppression of the metabolic derangements that may result in type 2 diabetes mellitus (DM), obesity, atherosclerosis, and non-alcoholic fatty liver disease, and is an independent risk factor for metabolic syndrome. Adiponectin circulates in three isoforms: a trimer (low molecular weight (LMW)), a hexamer (trimer-dimer) of medium molecular weight (MMW), and a larger multimeric high-molecular-weight (HMW) isoform. Various studies have demonstrated that the HMW adiponectin isoform is primarily responsible for direct associations between adiponectin and several metabolic parameters, including insulin sensitivity and lower abdominal fat accumulation. We read with great interest the recently published study by Rizza and colleagues on adiponectin isoforms in elderly patients with or without coronary artery disease. The data from this study confirmed that adiponectin isoform levels are higher in older adults with coronary heart disease. The special importance of this article is that LMW adiponectin may have a protective role in this special group, but there are several points that merit discussion.

As shown in the study design section, subjects without known type 2 DM underwent an oral glucose tolerance test, but the participants with pre-diabetes were evaluated as patients without DM, not as a separate group. This is important because circulating blood adiponectin levels are altered in subjects with disordered glucose metabolism.

Smoking has been known to affect adipocytokines and to cause decreases in adiponectin levels. Moderate alcohol consumption is associated with higher adiponectin concentrations in healthy individuals. Furthermore, physically active older women have higher adiponectin concentrations, but Rizza and colleagues, in their research, did not check lifestyle-related factors, which may affect adipocytokine status.

For these reasons, we would like to ask the authors whether they can present some new results by categorizing the patients according to confounders such as glucose tolerance status and lifestyle factors. This may provide readers with clearer information about adiponectin isoforms in older populations. We hope that this letter might prompt them to provide additional perspective on these questions.

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