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ORIGINAL ARTICLE

Comparison of echocardiographic findings in patients with nonfunctioning adrenal incidentalomas

Narin Nasıroglu Imga [a,*], Ozgul Ucar Elalmıs [b], Mazhar Muslum Tuna [a], Bercem Aycıcek Dogan [a], Deniz Sahın [b], Tugba Gursoy [b], Yavuz Yalcın [a], Dilek Berker [a], Serdar Guler [a]

[a] *Department of Endocrinology, Ankara Numune Education and Research Hospital, Ankara, Turkey*
[b] *Department of Cardiology, Ankara Numune Education and Research Hospital, Ankara, Turkey*

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Abstract Adrenal incidentalomas (AIs) are usually discovered incidentally after imaging unrelated to adrenal glands. We aimed to evaluate standard risk factors for systemic atherosclerosis and echocardiographic changes in patients with nonfunctioning AIs and compare them with normal subjects. We evaluated 70 patients diagnosed with AIs and 51 healthy controls. Mean levels were determined for HbA1c, LDL, uric acid, fasting plasma insulin, HOMA, and neutrophil-to-lymphocyte ratio (NLR), and these values were found to be significantly higher in the patients than the controls. The mean left atrial diameter, interventricular septum thickness, posterior wall thickness, left ventricular mass, E-wave deceleration time, isovolumetric relaxation time, and the median ratio of the early transmittal flow velocity to the early diastolic tissue velocity (E/Em) were higher in patients with AIs compared to controls. The mitral annular early diastolic velocity was lower in patients with AIs. The mean aortic diastolic diameter, stiffness index (SI), and aortic strain were higher, and aortic distensibility was lower in the patients. The mean right ventricular diameter, right atrial major-axis diameter, and right atrial minor-axis diameter were statistically higher in the patient group than the controls. A negative correlation was found between the NLR and aortic strain and aortic distensibility, while a positive correction was found between the NLR and SI. We found altered left ventricular (LV) and right ventricular (RV) echocardiographic findings in patients with AIs without

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* Corresponding author. Department of Endocrinology, Ankara Numune Education and Research Hospital, Ankara, 06100, Turkey.
E-mail address: xnarinx@yahoo.com (N.N. Imga).

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known cardiovascular disease. Aortic stiffness was also increased. These changes may be related to an increase in cardiovascular risk factors in AI patients.
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Introduction

Adrenal incidentalomas (AIs) are adrenal masses, which are usually discovered incidentally after imaging unrelated to the adrenal glands. The prevalence of AIs has been shown to be 2.3% in autopsies and 0.5—2% in computed tomography (CT) scans [1]. While 85% of AIs are nonfunctional, 15% secrete excess amounts of hormones [2]. Patients with nonfunctioning AIs may have mild hypercortisolism, reduced insulin sensitivity, and increased blood pressure levels when compared with controls [3]. Patients with subclinical Cushing's syndrome (SCS) and AIs had augmented cardiovascular risk similar to overt Cushing's syndrome (CS) [4]. Patients who had AIs with mild hypercortisolism had an increased risk of cardiovascular events and mortality [3—5]. Excessive plasma cortisol and secondary arterial hypertension may be etiologic factors for asymmetric septal hypertrophy, left ventricular (LV) hypertrophy, and diastolic dysfunction [6]. In patients with SCS, mid-wall systolic performance was reduced with diastolic dysfunction, and these structural changes may contribute to increased cardiovascular events in these patients [7]. Previous studies demonstrated that insulin resistance, hypertension, dyslipidemia, fatty liver disease, and metabolic syndrome were observed in patients with nonfunctioning AIs [8]. It is believed that nonfunctioning AIs may secrete small or undetectable cortisol levels that may cause mild systematic changes in these patients. There are many effects of hypercortisolism in the cardiovascular system. Many studies in the literature assessed cardiac changes in CS and SCS, but investigation of nonfunctional AIs remains lacking. Transthoracic echocardiography is an easily available, non-invasive tool that can be used to screen subclinical changes in patients with AIs. In this study, we aimed to evaluate the novel risk factors for systemic atherosclerosis and echocardiographic changes in patients with nonfunctioning AIs and compare these risk factors with normal subjects.

Materials and methods

Patients and methods

We evaluated 70 patients (49 female, 21 male) diagnosed with AIs and 51 healthy controls (43 female, 8 male) between 2012 and 2014 in the Ankara Numune Education and Research Hospital (Endocrinology Department). Healthy subjects who had no adrenal lesions on abdominal imaging were included in the control group. The control group ($n = 51$) consisted of normal subjects who were matched to patients by age, gender, and body mass index (BMI). All controls and patients were measured to determine height (m), weight (kg), waist circumference, and blood pressure. The BMI was calculated as weight/height^2 (kg/m^2). Patients with AIs were randomly included in the study. The AI masses were incidentally detected on CT, magnetic resonance imaging (MRI), or fluorodeoxyglucose (FDG)-positron emission tomography (PET) (FDG-PET)/CT imaging (imaging was not performed for suspected adrenal disease). Imaging suggested a benign adrenal mass if there were the following characteristics: a homogeneous structure with a diameter smaller than 4 cm and Hounsfield Units (HU) $\leq$ 10 in non-contrast CT or in CT with delayed contrast absolute washout $>$ 60%. In MRI scans, the loss of signal intensity on out-phase imaging or on 18F-FDG-PET/CT scans, indicates a reduction or absence of FDG uptake that is consistent with benign mass [9]. Informed consent was obtained from all participants. The local ethics committee of the Ankara Numune Education and Research Hospital approved the study protocol. The functional status of incidentally discovered adrenal masses was determined according to AACE 2009 AI guidelines [10]. Initially, all participants were clinically examined and carefully assessed for symptoms and signs of adrenal hormone excess. All patients underwent an overnight dexamethasone (1 mg) suppression test. Post-dexamethasone morning serum cortisol levels $\leq$1.8 μg/dL were considered a diagnostic criterion for the exclusion of cortisol excess [11]. Pheochromocytoma was excluded if measurements of urinary fractionated metanephrine and normetanephrine excretion were within normal values. In patients with concomitant hypertension or unexplained hypokalemia, the aldosterone/renin ratio was measured to exclude primary aldosteronism. Participants were excluded from the study if they had coronary artery disease, valvular heart disease, severe or uncontrolled hypertension, diabetes mellitus, hyperlipidemia, known malignancy, cortisol overproduction, pheochromocytoma, aldosteronoma, chronic kidney disease, connective tissue disease, chronic obstructive lung disease, pulmonary hypertension, or obesity. Participants who were currently smoking or who had a history of smoking were also excluded. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements were performed during echocardiographic scanning according to the European Society of Hypertension and European Society of Cardiology (ESH/ESC) 2013 Arterial Hypertension Guideline [12]. Pulse pressure was computed as SBP minus DBP.

Echocardiographic examination

All participants (patients and controls) underwent transthoracic 2D-echocardiography (GE Vingmed Ultrasound, Vivid 7Pro, Horten, Norway) using a 2.5 MHz transducer.

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