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SHORT RESEARCH ARTICLE

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Sonographic evaluation of hypertension: Role of atrophic index and renal resistive index

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Abstract

Hypertension can cause structural and functional renal damage. Intrarenal ultrasound parameters have been extensively investigated in hypertensive patients and among the parameters introduced, the renal resistive index (RI) is associated with the progression of chronic kidney disease and hypertension. Atrophic index (AI) is an indirect anatomical ultrasound index that reports the atrophic changes of the renal parenchyma and it is mainly studied in chronic glomerular diseases. The present study aimed to evaluate renal RI and AI in hypertensive patients with normal renal function. AI showed correlations with all parameters associated with renal function reduction (age, creatinine, and intrarenal arterial stiffness). AI, in combination with RI, can represent in hypertensive patients an additional marker for renal damage progression.

KEYWORDS
atherosclerosis, atrophic index, hypertension—vascular disease, renal disease, renal resistive index

1 | INTRODUCTION

Hypertension is an independent risk factor for morbidity and mortality able to promote vascular complications with organ damage. The European Society of Hypertension recommends laboratory analysis such as glomerular filtration rate and albumin excretion for the diagnosis and follow-up of renal complications in hypertensive patients.[1]

Also, Doppler and renal ultrasound could help to evaluate morphological changes associated with hypertension, including alterations of parenchymal echogenicity, reduction of longitudinal diameter, cortical thickness and hemodynamic parameters modifications such as resistive index (RI).[2]

Among renal Doppler parameters, RI is the most used and provides useful information for the expression of arterial impedance and renal vascular resistance highlighting arteriolar damage.

In course of kidney disfunction with changes in microcirculation, an increase in RI values is demonstrated. This process is related to reduction of post-glomerular capillaries. Sclerosis and chronic ischemia lead to a reduction of the intrarenal arterioles, which, in turn, may cause an increase in vascular resistance.[2] An association between RI and the complications-hypertension related such as hypertrophy of the left ventricle and microalbuminuria, have been reported.[3,4]

The relationship between intrarenal hemodynamic parameters and systemic vascular changes is widely investigated showing a direct association between RI and carotid intima-media thickness, marker of atherosclerosis, supporting the role of RI as marker of systemic vascular impairment.[5]

Atrophic index (AI) is an indirect anatomical ultrasound index that evaluates the degree of atrophy in the renal parenchyma and it is considered normal if less than 0.70.[6]

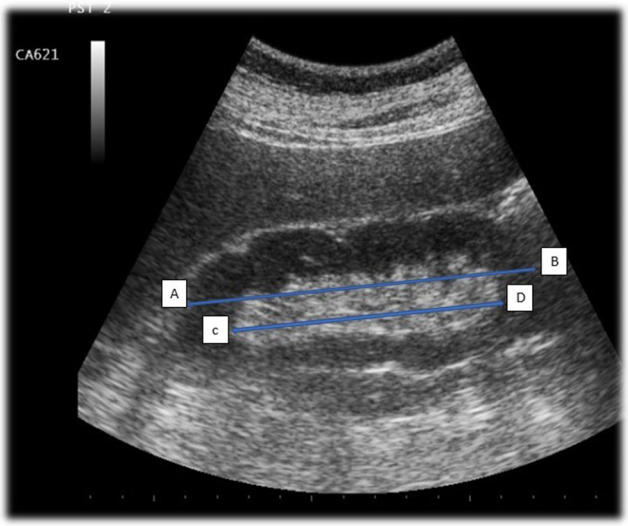

FIGURE 1 The distance between A and B represents renal length; the distance between C and D represents renal sinus length. Atrophic index is calculated as the ratio between renal sinus length/renal length

The aim of the study is to evaluate RI and AI in hypertensive patients with normal renal function.

2 | METHODS

The subjects' written consent was obtained according to the Declaration of Helsinki and the study was conducted in agreement to local ethics committee's directives.

Eighty-one hypertensive patients and 40 matched healthy controls (HC) were enrolled in the present study and eco-color-Doppler and renal ultrasonography were performed. Exclusion criteria were renal artery stenosis, glomerulonephritis, diabetes, acute renal failure, heart and lung disease.

2.1 | Renal and doppler ultrasound

Renal US was performed using standard gray scale B-mode imaging in hypertensive patients and HC using a Toshiba Aplio Ultrasound System SSA-790 equipped with a convex 3.5-MHz probe.

Bilateral renal lengths were measured as the greatest pole-to-pole distance (mm) in the sagittal plane. AI was calculated as the ratio between renal sinus length/renal length to quantify atrophic changes in renal parenchyma (Figure 1). After observation of the interlobar arteries by placing the probe at three different positions (mesorenal, superior, and inferior) by color Doppler ultrasonography, peak systolic velocity (PSV) and diastolic velocity (DV) were evaluated. RI is measured as = (PSV–DV)/PSV (Figure 2). Pulsatility index (PI) was calculated as (peak systolic frequency shift–minimum diastolic frequency shift)/mean frequency shift), and systolic/diastolic (S/D) ratio was also measured.

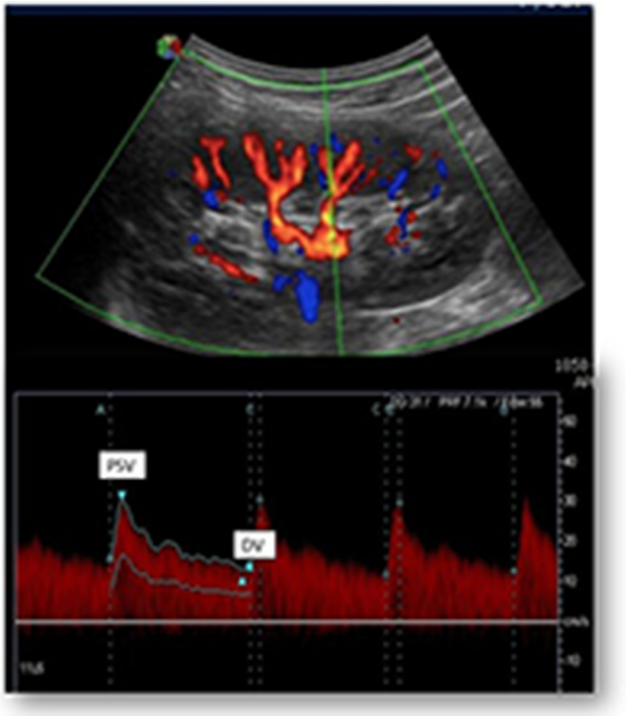

FIGURE 2 Renal resistive index is calculated as peak systolic velocity (PSV) - diastolic velocity (DV)/PSV

TABLE 1 Clinical and Doppler characterization of the study population

Clinical characterization of the study population	
Age in years, mean ± SD	58 ± 8
Male sex, *n* (%)	45 (55.5)
Disease duration in years, mean ± SD	5.5 ± 3.5
BMI (kg/m^2), median (IQR)	25.3 ± 2.7
Systolic blood pressure (mmHg), mean ± SD	133.6 ± 7
Diastolic blood pressure (mmHg), mean ± SD	83.6 ± 3.5
Serum creatinine (mg/dl), mean ± SD	1.3 ± 0.5
eGFR, (ml/min), mean ± SD	68.8 ± 16
Renal length, (mm) mean ± SD	105.8 ± 5.7
Atrophic index, mean ± SD	0.71 ± .05
	1.36 ± 0.04
Resistive index, mean ± SD	0.67 ± 0.09
Systolic/Diastolic ratio, mean ± SD	3.23 ± 0.08

Abbreviations: BMI, body mass index; eGFR, estimated glomerular filtrate rate.

3 | RESULTS

A total of 81 (45 males) hypertensive patients with median age of 58 ± 8 were enrolled. The degree and duration of hypertension were respectively stage 1 and 5.5 ± 3.5 years. Anthropometric, biochemical and ultrasound parameters are resumed in Table 1.

The parenchymal indices measured by renal Doppler ultrasound showed the following mean values: AI 0.71 ± 0.05; PI 1.36 ± 0.04; RI 0.67 ± 0.09; S/D ratio 3.23 ± 0.08. AI showed significant differences between HC normotensive subjects and hypertensive patients (0.63 ± 0.05 vs. 0.71 ± 0.05; $p < .001$). The AI showed a significant correlation with all the indices evaluated by Doppler ultrasound (PI [rho = 0.41; $p < .0001$], RI [rho = 0.45; $p < .0001$], and S/D ratio [rho =0 .39; p < .001]). Furthermore, a significant correlation was demonstrated between AI and creatinine values (rho = 0.36; $p = .002$), eGFR (CKD-Epi [rho = -0.47; $p < .0001$]) and age (rho =0 .49, P < .0001). Except for age, our study shows that AI correlates with all those parameters that suggest a reduction in renal function due to hypertension (creatinine, eGFR, and intraparenchymal indices).

4 | DISCUSSION

It has already been proved that RI of interlobar arteries seems to be a reliable marker of intrarenal arteriosclerosis[7] while AI in combination with RI could predict tubular interstitial involvement in glomerulonephritis.[6] Glomerulosclerosis and tubulointerstitial damage represent the main mechanisms leading to end stage renal disease.[8]

Blood pressure elevation over time can lead to renal arterioarteriolosclerosis, glumerulosclerosis, tubulointerstitial fibrosis promoting one of a major causes of chronic and end-stage renal disease.[7]

Performance of Doppler and renal ultrasonography with hemodynamic parameters and AI could represent an early marker of fibrosis and sclerosis in hypertensive patients with serum creatinine still in normal range.

Bipolar renal diameter is an important parameter to diagnose chronic kidney disease able to give information about renal atrophy.[9] However, in the renal longitudinal length is also included sinus fat which does not represent functioning kidney tissue. For this reason, we suggest measuring AI to better evaluate atrophic renal index because it represents a ratio between maximum renal sinus diameter and longitudinal diameter and can be considered a more reliable index to detect renal functional tissue. Large studies are needed to evaluate the exact role of AI in hypertensive patients.

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CONFLICT OF INTEREST DISCLOSURE

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Antonietta Gigante and Rosario Cianci made substantial contributions to conception and design and interpretation of data; Adolfo Maria Perrotta, Oriana De Marco made substantial contributions to acquisition of data; and Silvia Lai, Edoardo Rosato and Antonietta Gigante made substantial contributions to analysis of data. All authors participated in drafting the article or revising it critically for important intellectual content. All authors gave final approval of the version to be submitted.

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