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Renal Artery Stenosis and Acute Pulmonary Edema-A Possible Correlation beyond Pickering Syndrome

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Abstract

Objectives: The association of renal artery stenosis (RAS) with acute pulmonary edema (APE) is considered specific for bilateral or solitary functioning kidney (SFK) RAS. We aimed to check if APE is also associated with unilateral RAS when both kidneys are functional.

Method: A series of 189 patients with uncontrolled hypertension were investigated for RAS suspicion by duplex ultrasonography. Clinical criteria considered in current guidelines as predictors of RAS were recorded and analysed.

Results: Potentially hemodynamically significant RAS (≥ 50%) was identified in 29% of cases (55/189): unilateral in 35 cases (group A), bilateral or on SFK in 20 cases (group B). The remaining 134 were designated controls (group C). Age, blood pressure and gender did not differ between groups. The presence of acute pulmonary edema was higher in both groups of patients with RAS (23%-group A ($p<0.01$) and 20%-group B vs 8% in control group). The prevalence of azotemia and of azotemia under angiotensin converting enzyme inhibitors or angiotensin II receptor blockers were significantly higher in group B ($p<0.01$ *vs.* group A, $p<0.00001$ *vs.* control). Linear discriminant analysis based on: age, gender, abdominal bruit, vascular disease, renal dysfunction and azotemia under angiotensin converting inhibitors or angiotensin II receptor blockers, had an accuracy of 0.70 for unilateral RAS, 0.85 for bilateral/SFK RAS, and 0.77 for both. This accuracy was not improved when adding APE as a predictive variable.

Conclusions: In a series of hypertensive patients evaluated for renovascular disease the prevalence of APE is higher in patients with RAS. We have found a significant association of RAS with APE for unilateral RAS. This association, little emphasized until present, might contribute to the clarification of the flash pulmonary edema mechanisms beyond those related to bilateral/SFK RAS.

Keywords: Renal artery stenosis; Renovascular disease; Renovascular hypertension; Acute pulmonary edema; Flash pulmonary edema; Acute heart failure; Renal dysfunction

Abbreviations

ACEI: Angiotensin Converting Enzyme Inhibitors; APE: Acute Pulmonary Edema; ARB: Angiotensin Receptors Blockers; RAAS: Renin Angiotensin Aldosterone System; RAS: Renal Artery Stenosis; SFK: Solitary Functioning Kidney

Introduction

Renal artery stenosis (RAS) is defined as narrowing of the main or branch renal arteries. The most important etiologic conditions leading to RAS are the fibromuscular dysplasia-responsible for almost 10% of cases, and the atherosclerotic disease-prevailing in 90% of them. Other causes, like arteritis or extrinsic compressions, are rare [1]. The pathophysiologic consequences of RAS are variable, but a narrowing of more than 50% in lumen diameter could become hemodynamically significant [2]. In such cases, RAS is associated with a critical decrease in the perfusion pressure of the kidneys that triggers the activation of the renin-angiotensin-aldosterone system (RAAS) with two possible consequences: the occurrence of renovascular hypertension and the development of ischemic nephropathy-defined as a reduction in glomerular filtration rate (GFR) in patients with hemodynamically significant renovascular disease affecting the entire functional renal parenchyma. Initially, this condition has been defined as plasma creatinine concentrations higher than 1.5 mg/dl [3-7]. Sometimes, RAS remains asymptomatic even if higher than 50%.

Atherosclerotic RAS is located mostly in the proximal portion of the artery, affects older people [8-11], and is very often associated with other atherosclerotic determinations, especially with aortic, peripheral and coronary artery disease [12-21]. The decrease of systemic blood pressure or renal function improvement after revascularization through percutaneous transluminal renal artery angioplasty is still controversial [22-26]. The CORAL (cardiovascular outcomes in renal atherosclerotic lesions) study did not find any difference associated with revascularization in the incidence of cardiovascular or renal events, nor in blood pressure control [27]. An exception, according to some authors, would be acute pulmonary edema (APE) in the setting of bilateral/solitary functioning kidney (SFK) RAS [28]. Both American College of Cardiology/American Heart Association, and the

European Society of Cardiology guidelines recommend screening for RAS in unexplained APE relying only on series of patients and a limited number of randomized studies (Class I indication and level of evidence B) [29,30].

The association of bilateral/SFK RAS with APE was first reported by Pickering et al., in 1988, in hypertensive patients with azotemia in which revascularization prevents the APE reoccurrence [31]. Subsequent data confirmed this observation [32-43]. In 2011, Messerli et al., advanced the designation of ``Pickering syndrome'' for flash APE in patients with bilateral/SFK RAS [44].

Higher rates of flash APE were reported in bilateral/SFK RAS compared with unilateral RAS [34,44]. There are no conclusive data about an increased risk of APE in patients with unilateral RAS compared to those without any RAS, or with hemodynamically non-significant RAS. In this study, we aimed to verify if APE is associated with unilateral RAS as well. We also attempt to quantify the predictive value of APE events on a later RAS diagnostic.

Material and Methods

A series of 189 patients with uncontrolled hypertension have been investigated for RAS, with duplex ultrasonography, in a hospital clinic. Screening for RAS was requested by physicians accordingly to criteria listed in current guidelines. We considered such indicators to be: hypertension started before age of 30, or after the age of 55, accelerated/malignant/difficult to control hypertension, unexplained renal dysfunction, unexplained atrophic kidney, size discrepancy between kidneys of greater than 1.5 cm, development of new azotemia or worsening renal function after administration of an Angiotensin converting enzyme inhibitor (ACEI) or an Angiotensin II receptor blocker (ARB), systemic atherosclerotic disease, unexplained congestive heart failure, and sudden and unexplained pulmonary edema [29,30]. Demographic and medical data were recorded from patient documents on the occasion of vascular ultrasound evaluation.

Ultrasound imaging was performed according to AIUM (American Institute of Ultrasound in Medicine) practice guideline for the performance of renal artery duplex sonography [45] and carried out with HP Agilent Sonos 5500 equipment and a transducer of 2-4 MHz. Duplex ultrasound is recommended as the first-line imaging test in RAS detection. From a technical point of view, it is more easily available and affordable than other non-invasive imagistic methods, like CT or magnetic resonance angiography, avoiding the adverse outcomes caused by contrast medium. Still, the accuracy of this method is highly dependent on sonographer. In our laboratory we have accredited this method with a sensitivity of 92%, a specificity of 83% and an accuracy of 87% compared to digital subtraction angiography that is considered the gold standard for RAS diagnosis. A peak systolic velocity ≥ 180 cm/sec in the main renal arteries was taken as discriminant for RAS ≥ 50%.

Azotemia was defined according to the criteria applied in the series described by Pickering et al. and in the majority of communications that followed, meaning a level of serum creatinine>1.5 mg/dl [31-35]. Renal function was evaluated through estimated GFR (eGFR) based on the MDRD (modification of diet in renal diseases) study equation and on the CKD-EPI (chronic kidney disease-epidemiology collaboration) study equation, considering that the last one is more accurate for values higher than 60 ml/min/1.73m^2 [46-47]. Azotemia under IECA/ARB was affirmed when a high level of creatinine (at least 0.25 mg/dl above the normal range) normalized after these drugs were stopped. Coronary artery disease included history of stable pectoral angina and any form of acute coronary syndrome. Peripheral artery disease was defined as a history of revascularization or any clinical or investigational evidence of hypoperfusion in this territory.

Patients were divided in three groups: group A with unilateral RAS, group B with bilateral/SFK RAS and a control group without evidence of RAS ≥ 50%. We used the Kruskal-Wallis test for quantitative variables, and the χ^2-square test for qualitative variables. We also used the linear discriminant analysis for variables that usually considered as predictors for RAS. We used the R2.15 package from the R Foundation in Ubuntu 14.04.

Results

The main results of the study are presented in Table 1, containing the characteristics of patients in group A, group B and the control group. Patients included in the study were known to have high, uncontrolled blood pressure under at least two antihypertensive drugs.

The total prevalence of RAS ≥ 50% was 29%. Of this case: 64% had unilateral RAS and 36% had bilateral/SFK RAS. There were no differences between group A, group B and the control group for mean age, gender distribution and mean blood pressure values. For an average age of 55 years, atherosclerotic disease was the most probable etiology of RAS.

	Patients with unilateral RAS (Group A)	Patients with bilateral/SFK-RAS (Group B)	Patients without RAS (Control group)
Number of patients	35/189 (19%)	20/189 (10%)	134/189 (71%)
Mean age (years)	59.6 ± 12^{y}	58.85 ± 15.7^{y}	55.14 ± 14.9^{y}
Gender (females from all)	16/35	8/20	66/134
Mean systolic blood pressure (mmHg)	197 ± 49	194 ± 35	189 ± 41
Mean diastolic blood pressure (mmHg)	103 ± 20	100 ± 21	102 ± 21
Azotemia	4/35 (11%)^{x}	9/20 (45%)^{a,x}	29/134 (22%)
eGFR-MDRD (ml/min/1.73 m^2)	67.8 ± 27	49.9 ± 31.8^{b,y}	67.1 ± 28.5
eGFR-CKD-EPI (ml/min/1.73 m^2)	67.8 ± 21	48.1 ± 28.9^{b,y}	67.5 ± 28

Mean serum creatinine level (mg/dl)	1.21 ± 0.77	1.91 ± 1.19[b,y]	1.31 ± 0.76
Azotemia under ACEI/ARB	4/35 (11%)[y]	11/20 (55%)[c,y]	11/134 (8%)
Abdominal bruit	5/35 (14%)	4/20 (20%)	6/134 (4%)
Peripheral arterial disease	12/35 (34%)[c]	9/20 (45%)[c]	11/134 (8%)
Coronary artery disease	17/35(49%)	12/20 (60%)[a]	42/134 (31%)
History of acute pulmonary edema	8/35 (23%)[a]	4/20 (20%)	11/134 (8%)

Table 1: Characteristics of patients with unilateral renal artery stenosis in comparison with patients with bilateral renal stenosis or on solitary functional kidney and with the control group. RAS: Renal Artery Stenosis; SFK: Solitary Functional Kidney; Azotemia Defined as a Serum Creatinine >1.5 mg/L; ACEI: Angiotensin Converting Enzyme Inhibitors, ARB: Angiotensin II Receptor Blockers; Egfr-MDRD: Estimated Glomerular Filtration Rate according to Modification of Diet in Renal Diseases Study Equation; Egfr-CKD: EPI Estimated Glomerular Filtration Rate according to Chronic Kidney Disease-Epidemiology Collaboration Study Equation; [a]Statistically Significant Compared to Control for $p<0.05$; [b]Statistically Significant Compared to Control for $p<0.01$; [c]Statistically Significant Compared to Control for $p<0.001$; [x]Statistically Significant between Group A and B for $p<0.05$; [y]Statistically Significant between Group A and B for $p<0.01$; [z]Statistically Significant between Group A and B for $p<0.001$.

The prevalence of azotemia, the mean serum creatinine level and the rate of azotemia under angiotensin converting enzyme inhibitors or angiotensin II receptor blockers were significantly higher only in group B compared with group A or the control group ($p=0.01$, and $p<0.00001$, respectively). Renal function based on eGFR was significantly lower in bilateral/SFK RAS by comparison with the other groups. The mean distribution of the serum creatinine level under IECA/ARB was 3.13 ± 3.70 mg/dl. The presence of peripheral or coronary artery disease prevailed in each category of patients with RAS.

The presence of APE was higher in both groups of patients with RAS (23%-group A ($p<0.01$) and 20%-group B vs 8% in control group). No correlation has been found between APE and renal function or history of coronary artery disease in each group of patients. Ejection fraction of the left ventricle was available from echocardiographic evaluation in only 16 of 23 patients with APE and in all these cases had a value ≥ 40%.

Based on linear discriminant analysis we have found the following formula as an optimal predictor of RAS: [0.22 × female gender-0.0044 × age+1.22 × abdominal bruit+1.61 × vascular disease+0.037 × augmented plasma creatinine+1.28 × azotemia while on ACEI/ARB]. The accuracy (area under the curve) in predicting RAS was: 0.77-for all the patients with RAS, 0.70-for unilateral stenosis, 0.85-for bilateral/SFK RAS. However, adding EPA to the previous variables did not improve the discriminative power for RAS prediction: 0.78 - for all the patients with RAS, 0.70-for unilateral stenosis and 0.85-for bilateral/SFK RAS.

Discussion

In most of the literature, APE prevalence in RAS is evaluated without a RAS-free control group, based on series of patients undergoing renal angiography and revascularization through percutaneous transluminal renal artery angioplasty. Initially Pickering et al. reported a 23% (13/55) prevalence of recurrent APE in the presence of RAS and renal dysfunction [31]. Later on Messina et al. found a rate of 65% for APE in 17 patients with bilateral/SFK RAS [32]. Subsequently, Bloch et al. found a significantly higher proportion of APE in patients with bilateral/SFK RAS (41%) compared with unilateral RAS (12%) [34]. In a review of the literature including these observations, the weighted prevalence of APE was estimated at 14.3% for bilateral/SFK RAS and at 3.5% for unilateral RAS [44]. Our data are difficult to be compared with these results due to the selection of our patients: uncontrolled hypertensives evaluated through duplex ultrasonography for a high suspicion of RAS. In this category of patients the prevalence of APE in bilateral/SFK was approximately equal to that in unilateral RAS and higher than in the control group, but did not reach statistical significance probably due to the small number of cases. One study with control group was realized in 732 hypertensive patients undergoing coronary angiography which analysed the prevalence and predictors of RAS in this category of patients [48]. They found a medical history of pulmonary edema in 6.9% (6/87) of patients with RAS ≥ 50% and 1.4% (9/645) of those without or with RAS<50%. These results are indicating a lower prevalence of APE than in our study, but they were patients evaluated for coronary artery disease and not for a high presumption of RAS.

The main finding of our study is the significant association of RAS with APE, not only for bilateral/SFK RAS, but also for unilateral RAS. Starting with the experimental model of Goldblatt it is considered that patients with bilateral/SFK RAS are evolving with intravascular volume expansion due to impaired natriuresis [49]. Unilateral RAS is characterized by another hemodynamic pattern consisting in the activation of neurohormonal pathways, like RAAS or sympathetic nervous system, which are promoting severe forms of arterial hypertension with important remodeling of the cardiovascular system [50-53]. A recent study has shown higher values of systolic blood pressure, E/e' ratio, concentric left ventricular hypertrophy and lower estimated glomerular-filtration-rate in renovascular hypertension compared with essential hypertension [54]. Therefore, in the presence of a hemodynamically significant unilateral RAS two pathophysiological mechanisms implicated in the appearance of flash pulmonary edema can become manifest in certain conditions: the deterioration of diastolic dysfunction and the impairment of the pulmonary capillary blood-gas barrier due to a burden of vasoactive mediators [55-56]. In a review of the literature, 58% of patients with Pickering syndrome had coronary artery disease, but its association with unilateral RAS is not yet clarified [44].

Our best prediction model for RAS had an accuracy of 0.77. This result is comparable with the validation of a known prediction rule, proposed by Krijnen et al. in 1998, and based on the logistic regression analyses in a cohort belonging to DRASTIC (Dutch Renal Artery Stenosis Intervention Cooperative) study [57-58]. But, although sudden, unexplained APE is considered a strong predictor of RAS, adding APE to our model did not improve the accuracy of RAS prediction.

There is little published information about the prevalence of RAS in patients with APE. In one study, with a small number of patients, RAS >50% was identified through gadolinium-enhanced MRI in 9/20 (45%) of patients with APE: 6 with bilateral and 3 with unilateral topography [59]. More data are needed in order to evaluate the clinical impact of RAS for the evaluation of patients with APE.

Our study has some limitations. It was realized in a single clinic, which did not allow inclusion of a larger number of patients. The diagnosis of RAS was based on duplex ultrasound, which is currently considered to be a first line screening test. Through this method we were not able to evaluate the true hemodynamic significance of the stenosis. Finally we did not have detailed data about cardiac structure and function that could contribute to the understanding of APE pathophysiology in patients with RAS.

Conclusions

In a series of patients with uncontrolled hypertension, evaluated for renovascular disease, the prevalence of APE is higher in patients with unilateral RAS than in controls. This association, little emphasized until present, might contribute to the clarification of the flash pulmonary edema mechanisms, beyond those related to bilateral/SFK RAS. Although sudden, unexplained APE is considered a strong predictor of RAS, the prediction accuracy did not improve after addition of APE. Therefore, more data are needed to establish the contribution of RAS to the appearance of flash pulmonary edema.

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