

Case Report

“Resistant” Hypertension: The Wrong Approach (And a Very Wrong One...or Two?)

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Keywords

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Abstract

A Patient is described where an incorrect BP measurement (under-cuffing) led to an over treatment with a wrong ACE/I – ARB association. Both mistakes induced an acute renal failure that partially recovered after saline-hydration and drug discontinuation. These mistakes appear at an amazing rate. The correct measurement of BP is the first step in a successful hypertension treatment. The use of a wrong cuff is surprisingly widespread. The incorrect measurement of BP can induce over treatment with a consequent increased risk of renal, myocardial and cerebral damage. The use of drug associations not recommended such as ACE/I + ARB may lead to adverse events.

ABBREVIATIONS

ARB: Angiotensin Receptor Blockers; ACE/I: Angiotensin Converting Enzyme Inhibitors; BP: Blood Pressure; RAAS: Renin-Angiotensin-Aldosterone System; RAS: Renin-Angiotensin System.

INTRODUCTION

“Numquam aliquid ad mentulam canis fecisti?” Gaius Valerius Catullus, 1st Century BC

Hypertension is a primary risk factor for cardiovascular disease and kidney damage. The correct measurement of blood pressure is the first step of an accurate diagnosis and treatment: there is evidence that a cuff too narrow or too short (under-cuffing) can induce an over estimation of blood pressure, and there is also growing evidence that a cuff too wide or too long (over-cuffing) may under estimate blood pressure [1-3]. A careful treatment can prevent the appearance or the worsening of target organ damage, as highlighted by the latest guidelines [4]. On the contrary, arbitrary associations among anti hypertensive drugs can induce detrimental effects [5].

CASE PRESENTATION

An 81 year old Caucasian male came to our observation from the emergency unit of our Hospital for acute renal failure (CKD stage 4) with serum Creatinine 3.0 mg/dL, serum K⁺ 7.0 mEq/L, and serum Uric Acid 8.07 mg/dL (all determinations had been carried out in the Clinical Laboratory of our Hospital by Roche/Hitachi AutoAnalyzer cobas c701/702/ISE8000). Heart objectivity: systolic aortic bruit and diastolic mitral bruit; no abdominal or respiratory signs, no peripheral edema, arm circumference 36 cm, BMI 26 kg/m². At the first visit in our

division, supine BP was 100/60 mm Hg (after repeated measures this value showed to be stable) after drug withdrawal (48 h). The patient had a history of resistant hypertension treated in last year with a double inhibition of the renin-angiotensin system (captopril 50 mg/day and losartan 100 mg/day), thiazide and loop diuretics (butizide 5 mg/day and torasemide 20 mg/day) and an aldosterone-blocker (potassium canreonate 50 mg/day). There were no other evident cardiovascular risk factors in his history and a previous (1.5 years old) creatinine value was 0.8 mg/dL. The abdominal echography (performed with an Esaote Techos MPX with a 3.5 MHz probe) resulted negative for adrenal lesions. Renal size was in the normal range for age, with a normal corticomedullary ratio, without haemodynamically significant stenosis of the renal arteries. A previous screening for secondary hypertension (urinary free cortisol, urinary metanephrines, plasma electrolytes) had been negative. During hospitalization, after a careful adequate intravenous hydration, the renal function gradually recovered (Table 1) to sub-optimal values (creatinine at discharge: 1.3 mg/dL, calculated CKD-EPI GFR was 51 mL/min). The BP, measured daily with an appropriate cuff (arm circumference 32-42 cm), by means of an Omron 907 oscillometric automated device, progressively rose during the hospitalization and was maintained to normal values (130/80 to 120/70 mmHg) by introducing valsartan 40 mg oad and furosemide 12.5 mg bid (the BP remained in the above range after three months from

Table 1: Potassium and Creatinine serum levels during the hospitalization period.

	Admission	Day 03	Day 05	Discharge - Day 07
Creatinine (mg/dL)	3.0	2.5	1.9	1.3
Potassium (mEq/L)	7.0	5.5	4.5	4.0

the discharge). An echocardiography, performed for screening, showed both severe aortic stenosis and mitral regurgitation with a prevailing component of severe stenosis (likely rheumatic in origin), while the left ventricle had a normal systolic function. Neither dilation nor hypertrophy were evident.

DISCUSSION

The incorrect measurement of BP with a cuff too small for the patient's arm can induce over treatment. The first goal in hypertension evaluation and treatment should be a correct measurement and the selection of a suitable cuff may be viewed as a first step. The quality of BP assessment in Italian public hospitals [6] has been published. The authors administered more than 1300 questionnaires and showed a consistent level of inaccuracy in BP measurement: in the majority of the analyzed settings the circumference of patient's arm was never recorded, the BP was always measured at a single arm, and never in different periods of the day. Our patient had been labeled as "resistant" hypertensive on the basis of repeated incorrect (but rather usual) office measurement (i.e. under-cuffing), leading to overestimation of BP levels. Mistake over mistake, he was administered with a rather inconsistent cocktail of drugs including a double blockade of the RAS (ARB and ACE/I), the association of an aldosterone receptor inhibitor (i.e. a triple RAAS blockade), plus some diuretics (it is to be noted that months after the discharge, the BP continued to be maintained at appropriate levels with just two drugs at low dose). Not only this patient had been incorrectly diagnosed and treated, but received an even worse follow up (if any), leading to a silent development of renal failure. The recovery of the renal function after drug withdrawal seems in keeping with a pre-renal mechanism, as well as with a functional interference by the double RAS blockade.

The association of an ARB and an ACE/I may be unsafe in terms of renal function [5], the kidney being even a major target organ of the hypertensive damage.

This is why recent guidelines advise against the combination of ACE/I and ARB to avoid renal damage and hyperkalaemia with the further serious risk of myocardial damage [4]. Not only there is no benefit in terms of CV event reduction in combining an ARB with an ACE inhibitor, but this association does worsen the renal function [5].

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