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Anatomic variations of the renal arteries from a local study population using 3D computed tomography angiography reconstruction images from a reference hospital in Cali, Colombia

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Introduction: With the advances in the new image techniques and 3D modeling, angiography computed tomography (A-CT) has become a very useful image for studying vessels. Renal artery (RA) variations are common and have a clinical relevance in pre-operative planning. There are several descriptive studies made in high income countries, but there are not many in middle and low income countries.

Aim: Our objective was to describe prevalence of RA variations in a study population in Cali, Colombia.

Methods: A database was made from a selection of A-CT 3D images from January 1, 2012 to September 30, 2014, from which the RA could be visualized. Patients' under-18 was excluded, also with no 3D A-CT, or not of Colombian nationality. Frequencies, percentages were calculated using Excel.

Results: A total of 560 patients were selected, from which 296 fulfilled all criteria. The most common causes of performing the A-CT were pathologies of the aorta. Variations of the RA were present in 52% of the patients, 54% were men, 77% had unilateral variation and 33% had bilateral variations, 58% in the right side. The two most common variations were extra renal arteries (hiliar and polar) seen in 70% of the patients.

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Human GRK4γ142V variant promotes AT1R-mediated hypertension via renal HDAC1 inhibition and predicts the blood pressure response to angiotensin receptor blockade

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The influence of a single gene on the etiology of essential hypertension may be difficult to ascertain, unless the gene interacts with other genes that are germane to blood pressure (BP) regulation. G protein-coupled receptor kinase type 4 (GRK4) is one such gene. Non-synonymous GRK4 variants, R65L, A142V and A486V are associated with essential hypertension. The dopamine D1 receptor (D1R) and angiotensin II type-I receptor (AT1R) reciprocally regulate renal sodium excretion and BP. hGRK4γ^{142V} transgenic mice have high blood pressure (BP) due to the impairment of the natriuretic function of D1R and increased expression and activity of the AT1R, that counteract the renal physiological effects of the D1R. hGRK4γ^{142V} phosphorylates histone deacetylase type-1 (HDAC1) but not HDAC2 and promotes HDAC1 export from the nucleus to the cytoplasm, resulting in increased AT1R expression. There is specificity of these effects because GRK4WT, but not GRK2WT, decreases AT1R expression. Moreover, AT1R blockade and the deletion of the Agtr1a gene normalize the hypertension in hGRK4γ^{142V} mice. The studies in mice can be translated to humans. In 829 patients with essential hypertension we found that carriers of hGRK4γ^{142V} had a greater decrease in systolic BP in response to angiotensin receptor blockers (ARBs) than non-carrier hypertensive patients. By contrast, those with variants only at hGRK4γ^{486V} were less likely to achieve the BP goal in response to an ARB than those with no variants. These findings illustrate the unique role of GRK4 by targeting receptors with opposite physiological activity for the same goal of maintaining BP homeostasis and thus making the GRK4 a relevant therapeutic target to control BP.

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