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Non Invasive Imaging

ADENOSINE MONOPHOPHATE DEAMINASE-1 (AMPD1) DEFICIENCY AND RESPONSE TO REGADENOSON

Poster Contributions
Hall C
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Background: Adenosine monophosphate deaminase-1 (AMPD1) C34T (rs17602729) is a common polymorphism (SNP) resulting in AMPD-1 deficiency with reduced clearance of AMP and increased production of adenosine in skeletal muscle. Regadenoson (RGD), an adenosine analogue, exhibits large variability in hemodynamic response and incidence of side effects (SE). We hypothesized that AMPD1 deficiency in carriers of C34T SNP may affect heart rate (HR) and blood pressure (BP) response, as well as incidence of SE from RGD.

Methods: We prospectively enrolled 274 patients undergoing RGD stress testing. AMPD-1 C34T genotyping was done by Taqman assay. Primary outcomes included (1) change in BP and (2) change in HR from baseline, after administration of RGD in AMPD-1 carriers as compared to wild type (WT) subjects. The incidence of self reported adverse SE was analyzed.

Results: Of 274 patients, 229 (84%) were wild type and 45 (16%) were carriers of AMPD-1 C34T SNP. Systolic BP rise with RGD was significantly greater in AMPD-1 C34T carriers as compared to WT subjects (15±25 vs 9±13 mmHg, p=0.02; multivariate analysis: p=0.006). A higher incidence of SE was observed in patients with AMPD1 (C34T) (LR+=4.2; p=0.04) (Fig). There was no significant difference in HR change between groups (31±14 vs 30±14;p=0.6).

Conclusions: Carriers of the AMPD-1 variant had greater rise in systolic BP and a greater incidence of SE with RGD stress testing. Reduced AMPD-1 function may modulate systemic effects of RGD.