Correlation analysis showed that there is not any correlation between the values of AVC and BAC (p=0.877).

Table-1: Baseline Characteristics of Patients (n=320)	
Age, years, mean ± sd	$57.53 \pm 9.34$
Comorbidities;	
- Coronary artery disease, n (%)	29 (9.1 %)
- Diabetes mellitus, n(%)	77 (24.1 %)
- Hypertension, n (%)	195 (60.9 %)
- Atrial fibrillation, n (%)	40 (12.5 %)
Smoking, n (%)	35 (10.9 %)
Medications, n (%)	
RAAS blocker	119 (37.2 %)
Beta blocker	35 (10.9 %)
Calcium channel blocker	50 (15.6 %)
Statin	89 (27.8 %)
Breast artery calcification;	
- n (%)	47 (14.6 %)
<ul> <li>Grade, n, median (min-max)</li> </ul>	0 (-3)
Aortic valvular calcification;	
- n (%)	191 (59.7 %)
<ul> <li>Agatston score, median, (25th-75th percentile)</li> </ul>	0.9 (0.00-6.2)
Aortic peak gradient, median, (25th-75th percentile)	7.29 (5.76-9.00)

Conclusions: Vascular calcifications including breast artery calcification and aortic calcification are related with each other and poor cardiovascular outcomes. Aortic valvular calcification is more frequent in patients with BAC but the severity of AVC is not correlated with BAC severity.

## 305 / #419, POSTER, SAAG: HYPERTENSION, ARTERIAL CALCIFICATION, AND CARDIOVASCULAR DISEASE, 10-07-2020 12:00 PM - 1:00 PM. DIFFERENCES IN POTASSIUM CHANNEL-INDEPENDENT EFFECTS OF PINACIDIL ON THE ISOLATED HUMAN SAPHENOUS VEINS OBTAINED FROM DIABETIC AND NON-DIABETIC PATIENTS

J. Rajković<sup>1</sup>, M. Perić<sup>2</sup>, R. Novaković<sup>3</sup>, V. Djokić<sup>1</sup>, <u>M.Z. Gostimirović<sup>3</sup></u>, H. Heinle<sup>4</sup>, L. Gojković - Bukarica<sup>3</sup>. <sup>1</sup>Institute of Pharmacology, Clinical Pharmacology and Toxicology, Medical Faculty, University of Belgrade, Cardiovascular Pharmacology, Belgrade, Serbia; <sup>2</sup>Dedinje Cardiovascular Institute, Clinic For Cardiac Surgery, Belgrade, Serbia; <sup>3</sup>Institute of Pharmacology, Clinical Pharmacology and Toxicology, Medical Faculty, University of Belgrade, Cardiovascular Pharmacology, Belgrade, Serbia; <sup>4</sup>Institute of Physiology, Gi Physiology, Tubingen, Germany

Background and Aims: Pinacidil is well known potassium (K<sup>+</sup>) channel opener which relaxes blood vessels trough opening ATP-dependent K<sup>+</sup> (K<sub>ATP</sub>) channels located in the plasma membrane of smooth muscle cells. However, numerous in vitro studies were reported K<sup>+</sup> channels-independent effects of pinacidil. Therefore, objective of this study was to investigate K<sup>+</sup> channels-independent component of pinacidil-induced vasorelaxation on isolated human saphenous vein (HSV) obtained from patients with and without type-2 diabetes mellitus.

Methods: Rings of HSV, without endothelium, obtained from patients undergoing coronary bypass surgery, were mounted in organ bath system and isometric tension was being recorded. The experiments followed multiple curve design. Pinacidil (0.01 – 100  $\mu$ M) was used for vaso-relaxation of HSV precontracted with phenylephrine (100  $\mu$ M in the presence of normal Krebs-Ringer solution) or solution with 80 mM K<sup>+</sup>. Results: In the presence of normal Krebs-Ringer solution, pretreatment with nickel (300  $\mu$ M), Na<sup>+</sup>-Ca<sup>2+</sup> exchanger inhibitor, or with nifedipine (1  $\mu$ M), inhibitor of voltage-gated Ca<sup>2+</sup> channels, did not inhibited pinacidil effect on HSV obtained from diabetic patients. The same treatment on HSV from non-diabetic patients indicated that nifedipin caused inhibition of pinacidil effects (P < 0.05). However, in the presence of 80 mM K<sup>+</sup>, pre-treatment with nickel or nifedipine antagonized significantly the effect of pinacidil (P < 0.01, both).

Conclusions: Pinacidil endothelium-independent vasorelaxation of HSV from diabetic and non-diabetic patients includes  $K_{ATP}$  channels-independent effects. In K<sup>+</sup> channel-independent component of the pinacidil-induced vasorelaxation, Na<sup>+</sup>-Ca<sup>2+</sup> exchanger and voltage-gated Ca<sup>2+</sup> channels are partly involved. However, we have to investigate further additional K<sup>+</sup> channel-independent mechanism(s) of pinacidil.

## 306 / #751, POSTER, SAAG: HYPERTENSION, ARTERIAL CALCIFICATION, AND CARDIOVASCULAR DISEASE, 10-07-2020 12:00 PM - 1:00 PM. ASSOCIATION BETWEEN NON-FASTING ATHEROGENIC INDEX AND ANATOMICAL COMPLEXITY OF CORONARY ARTERY DISEASE IN PATIENTS WITH CHRONIC CORONARY SYNDROME

E.S. Farag<sup>1</sup>, A. Bendary<sup>2</sup>, M. Bendary<sup>3</sup>, I. Ibrahim<sup>1</sup>, H.G. Abomandour<sup>1</sup>. <sup>1</sup>Zagazig University, Cardiology Department, Zagazig, Egypt; <sup>2</sup>Benha University, Cardiology, Banha, Egypt; <sup>3</sup>National Cancer Institute, Cairo University, Biostatistics, Cairo, Egypt

Background and Aims: Fasting atherogenic index (AI), triglycerides/HDL-C ratio, has been found to be associated with the presence and severity of coronary artery disease(CAD). Recent guidelines have recommended the use of non-fasting lipid assay as being more reflective of atherogenic burden of plasma lipoproteins.We aimed to study the association between non-fasting AI and severity of CAD as assessed by SYNTAX score (SXscore).

Methods: 400 patients referred to coronary angiography for suspected stable CAD were included and classified according to SXscore into 3 groups; SXscore < 22 (n=199), SXscore 23-33 (n= 174) and SXscore > 33 (n= 27). Fasting and non-fasting, after a standard meal, lipid assays were done on the day of coronary angiography.

Results: The mean age of patients was 56  $\pm$  9 years with males representing 70.3%. Non-fasting AI was significantly higher than fasting AI (P= 0.008)Both fasting and non-fasting AI were significantly higher in patients with severe CAD (P value for each <0.001). On multivariate logistic regression analysis, non-fasting, but not fasting, AI was significantly associated with SXscore > 22 (for non-fasting AI OD 1.15, 95% CI 1.04 – 1.27, P= 0.006).

Conclusions: Non-fasting, in contrast to fasting, AI is significantly associated with the severity of CAD. Therefore, control of non-fasting AI has to be considered as a therapeutic target among those patients.

## 307 / #690, POSTER, SAAG: LIPID-LOWERING THERAPIES, 10-07-2020 12:00 PM - 1:00 PM. IDENTIFICATION OF A NEW CLASS OF SMALL MOLECULE PROPROTEIN

## IDENTIFICATION OF A NEW CLASS OF SMALL MOLECULE PROPROTEIN CONVERTASE SUBTILISIN/KEXIN TYPE 9 (PCSK9) INHIBITORS

<u>N. Ferri</u><sup>1</sup>, M.G. Lupo<sup>1</sup>, M. Radi<sup>2</sup>, J. Lebrun<sup>3</sup>. <sup>1</sup>University of Padua, Pharmaceutical And Pharmacological Sciences, Padua, Italy; <sup>2</sup>University of Parma, Department Of Food And Drug, Parma, Italy; <sup>3</sup>Faculteit Farmaceutische, Biomedische en Diergeneeskundige Wetenschappen, Universiteit Antwerpen, Antwerp, Belgium

Background and Aims: Drawbacks of anti-PCSK9 mAbs, such as, high cost, subcutaneous administration, and potential immunogenicity, emphasize the need for small-molecule orally bioavailable drugs. Objective of this study was the in vitro pharmacological study of new potential small molecule with anti PCSK9 activities.

Methods: By a high-throughput screening approach one small molecule capable to reduce the secretion of PCSK9 was identified. From this first hit we synthesized thirteen structurally related compounds and assessed