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### **P70: FINGER-TOE PULSE WAVE VELOCITY (FTPWV) MEASURED BY POPMÈTRE<sup>®</sup> DEVICE IN PATIENTS WITH ANKYLOSING SPONDYLITIS**

Guillermo Alanis-Sánchez, Carlos Ramos-Becerra, Ernesto Cardona-Muñoz, Diego Castañeda-Zaragoza, David Cardona-Muller, Hakim Khettab, Stephen Laurent, Pierre Boutouyrie, Hasan Obeid, Magid Hallab, Valeria Diaz-Rizo

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## Poster Session I – Special Populations I

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## UNRELIABLE PULSE WAVE VELOCITY VALUES PROVIDED BY ALGORITHM-BASED DEVICE: A STUDY IN MARFAN SYNDROME

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**Objective:** To evaluate the reliability of algorithm-based aortic pulse wave velocity (PWV) estimated by the Mobil-O-Graph (IEM, Germany) compared to a standard non-invasive measurement of aortic PWV (carotid-femoral PWV), in a population of patients with a genetic disorder causing premature stiffening of the arterial wall: Marfan syndrome. **Methods:** In this study, 107 patients with confirmed Marfan syndrome were enrolled (mean age  $37.7 \pm 15.1$  years, males 50.4%, blood pressure  $117.8 \pm 13.6/69.0 \pm 8.8$  mmHg). PWV estimated by Mobil-O-Graph (which uses an algorithm based mainly on age and pressure acquired by oscillometric method) was compared with carotid-femoral PWV measured by PulsePen tonometer (DiaTecne, Italy). For each method, two measurements were performed simultaneously, in a single session.

**Results:** Mean values of PWV ( $\pm$ SD) of Marfan patients were  $6.1 \pm 1.3$  m/s by Mobil-O-Graph and  $8.8 \pm 3.1$  m/s by carotid-femoral PWV, with a weak correlation between the two ( $r = 0.34$ ). The average underestimation by the Mobil-O-Graph was  $-2.7 \pm 5.7$  m/s. The values provided by Mobil-O-Graph may be derived in this population from the age square and the brachial systolic pressure ( $r^2 = 0.98$ ) according to the formula:  $PWV = \text{age}^2/1000 + 0.038 * \text{systolic blood pressure}$ .

**Conclusions:** The Mobil-O-Graph provides PWV values of an ideal subject for a given age and pressure, but may not be able to evaluate the cardiovascular risk expressed by aortic PWV in patients with specific alterations of aortic wall properties, as demonstrated in this population with Marfan syndrome. The use of algorithms for the evaluation of PWV should therefore be discouraged in special populations at high cardiovascular risk.

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## THE HIDDEN PREDICTOR OF CARDIOVASCULAR OUTCOME

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**Background:** Hyperuricemia is common in patients with hypertension, diabetes and obesity. Whether it is an independent cardiovascular risk factor (CVRF) or not remains controversial.

**Purpose:** To determine the prognostic value of uricemia in the setting of acute coronary syndrome (ACS).

**Methods:** Retrospective single-center study comprising 1187 patients consecutively admitted into a cardiac intensive care unit for ACS, in whom uricemia was measured during hospitalization. Follow-up targeted all-cause mortality (FUM), reinfarction, percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG) and acute heart failure (AHF). Statistical analysis was performed using SPSS, version 25.

**Results:** Mean age was  $68.0 \pm 13.3$  years and 30.4% were female. Prevalence of CVRF was as follows: hypertension, 76.9%; diabetes, 33.4%; dyslipidemia, 65.6%; smoking, 35.5%; chronic kidney disease (CKD), 20.5%. Uricemia was  $377 \pm 119.2$   $\mu\text{mol/L}$ , whereas body mass index (BMI) was  $27.8 \pm 4.4$  kg/m<sup>2</sup>. In-hospital mortality (IHM) was 6%, while median follow-up time was 6 years, encompassing the following event rates: FUM, 36.9%; reinfarction, 19.4%; PCI, 21.1%; CABG, 2.3%; AHF, 16.6%. Uricemia was higher in males ( $p = 0.001$ ) and in patients with hypertension ( $p < 0.001$ ), diabetes mellitus ( $p = 0.009$ ) and CKD ( $p < 0.001$ ) and lower in patients with dyslipidemia ( $p = 0.031$ ) and smokers ( $p = 0.03$ ). Age and BMI displayed weak correlation

with uricemia. Hyperuricemia had no effect on the burden of reinfarction, PCI and CABG. In a model of logistic regression including the above-mentioned CVRF, hyperuricemia was an independent predictor of IHM ( $p = 0.009$ , Hosmer-Lemeshow  $p = 0.685$ ), FUM ( $p < 0.001$ , Hosmer-Lemeshow  $p = 0.056$ ) and AHF ( $p = 0.001$ , Hosmer-Lemeshow  $p = 0.367$ ).

**Conclusion:** Hyperuricemia is an independent predictor of mortality and AHF in the setting of ACS.

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## RELATIONSHIP BETWEEN CAROTID INTIMA-MEDIA THICKNESS, ENDOTHELIAL FUNCTION, AORTIC STIFFNESS AND CARDIOVASCULAR EVENTS AMONG METABOLIC SYNDROME SUBJECTS

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**Objective:** The aim of this study was to evaluate predictive value of main arterial markers for cardiovascular (CV) events in subjects with metabolic syndrome (MetS).

**Methods:** A prospective study enrolled 2728 middle-aged ( $53.9 \pm 6.2$  years old, 63% women) MetS patients of the Lithuanian High Cardiovascular Risk primary prevention program without overt CV disease. Subjects were followed-up for  $3.9 \pm 1.7$  years for fatal or non-fatal myocardial infarction (MI) or stroke after their initial assessment including evaluation of carotid intima-media thickness (cIMT), aortic augmentation index (Aix), aortic pulse wave velocity (aPWV), brachial flow-mediated dilatation (FMD), carotid stiffness index, and cardio-ankle vascular index (CAVI).

**Results:** 83 (3%) patients had at least one cardiovascular event during the follow-up period. Univariate analysis showed association of CV events with higher mean blood pressure, aPWV, Aix, cIMT, and lower FMD (all  $p < 0.05$ ). Cox proportional hazard regression analysis revealed association between CV events, increase in cIMT (HR 1.31, 95% CI 1.14–1.50,  $p < 0.001$ ), aPWV (HR 1.29, 95% CI 1.04–1.60,  $p = 0.019$ ), Aix (HR 1.53, 95% CI 1.16–2.02,  $p = 0.003$ ) and decrease in FMD (HR 0.83, 95% CI 0.71–0.97,  $p = 0.016$ ) even after the adjustment for age, gender, and common CV risk factors.

Using two-level survival trees analysis, we discovered relation between cIMT > 794 and higher CV risk ( $p < 0.001$ ) and even higher risk with aPWV > 11.1 m/s ( $p = 0.023$ ). Whereas cIMT  $\geq$  794  $\mu\text{m}$  together with the FMD cut-off point of 6.5% also resulted in higher risk ( $p = 0.003$ ).

**Conclusions:** Our follow-up study reveals association between CV risk, increased aortic PWV, cIMT and decreased brachial FMD among middle-aged MetS patients.

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## FINGER-TOE PULSE WAVE VELOCITY (FTPWW) MEASURED BY POPMÈTRE® DEVICE IN PATIENTS WITH ANKYLOSING SPONDYLITIS

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**Background:** Ankylosing spondylitis (AS) is an inflammatory autoimmune disease. AS is a prototype form of spondyloarthropathies (SpA). The precise ethology of AS has not been fully understood. But Inflammation has a critical role in the pathogenesis of the disease. Extra skeletal organs may also be affected by this disease and is also associated with an increase of cardiovascular risk. The effect of large arteries appears by a stiffness that can be an element of disease monitoring.

**Objective:** The objective of this study was to evaluate the finger-toe Pulse Wave Velocity (ftPWV) in patients with AS.

**Methods:** Finger-toe pulse wave velocity (ft-PWV) was measured by pOpmetre<sup>®</sup> allowed to explore arterial stiffness.

**Results:** Demographic and clinical characteristics are presented in Table 1. Twenty-two patients with AS and 24 controls were included in our study, subjects with AS exhibited greater pSBP ( $p < 0.001$ ), pDBP ( $p < 0.001$ ), pPP ( $p < 0.001$ ) and MBP ( $p < .001$ ) compared to controls. Moreover, in the AS group we observed a higher ftPWV with a mean difference of 1.63 ( $p < 0.006$ , 95% CI of .50 to 2.7). No significant difference was observed in pPP.

**Conclusions:** Individuals with ankylosing spondylitis showed increased ftPWV, central and peripheral blood pressure, this contributes to explain the higher risk of cardiovascular disease in this pathology. pOpmetre<sup>®</sup> is a no operator depended, simple and practical device, highlighted an increase in arterial stiffness in patients with AS by measuring the ft-PWV. It could play a role in this disease monitoring and in prediction of cardiovascular complications.

	AE	Controls	p
Age (years)	42.4±12.1	40±9.9	.45
Males/Females	3/15	6/17	.36
BMI	25.5±3.9	27.1±3.5	.16
sBP (mmHg)	116.4±14	102.5±7	< .01
pSBP (mmHg)	127.6±13	113.8±8	<.01
pDBP (mmHg)	78.3±7	67.7±8	<.01
MBP (mmHg)	98.0±8	85.1±4	<.01
pPP (mmHg)	47±7	46.8±9.3	.94
ftPWV	7.8±2.3	6.1±1	<0.006

Table 1. Demographic and clinical characteristics of patients with ankylosing spondylitis and controls.

**P71**  
**TROPONIN INCREASE AND SUBENDOCARDIAL OXYGEN SUPPLY AND DEMAND IMBALANCE IN CARDIAC AMYLOIDOSIS**

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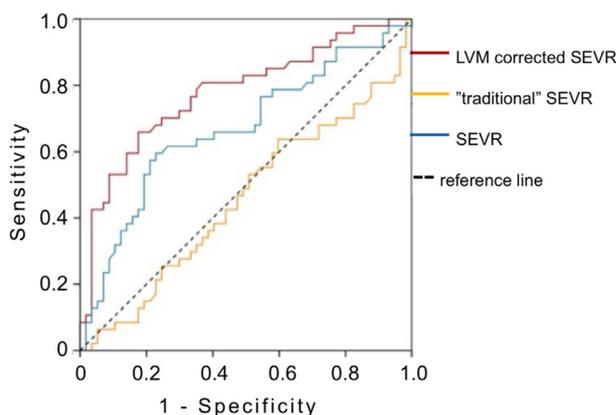
**Background:** The increase in troponin is a cardiac amyloidosis (CA) peculiarity. The most acclaimed hypothesis is direct toxicity of amyloid fibrils on cardiomyocytes, but a subendocardial ischemia due to discrepancy between oxygen supply and demand imbalance has not been investigated yet.

**Methods:** 129 outpatients attending the Pavia Amyloid Center were enrolled, 66 of them were affected by CA. Aortic stiffness was assessed measuring carotid-femoral pulse wave velocity (PWV). The subendocardial viability ratio (SEVR) was used to quantify the relationship between subendocardial oxygen supply and demand. Echocardiogram data were used to quantify left ventricular diastolic pressure and left ventricular mass index (LVMI).

**Results:** Troponin was higher in CA ( $p < 0.0001$ ); there was an inverse correlation between troponin and SEVR ( $p = 0.0002$ ). Troponin was strongly correlated with LVMI ( $p = 0.0003$ ). Both the increase in TnI and the reduction of SEVR were related to low values of ejection fraction. The ROC curves showed that SEVR had a greater sensitivity and specificity (AUC = 0.778) than EF% and PWV in identifying pathological troponin values.

**Conclusions:** There is a close relationship in CA between troponin values and the reduction in the SEVR. Ischemic suffering, with undamaged coronary arteries, may be a cause of cardiac myocytes damage in amyloidosis. LVMI

increases with disease progression. On the other hand, amorphous amyloid mass modifies the microcirculation. These two phenomena may seriously affect myocardial perfusion. Moreover, amyloid alters the macrostructural organization of myofibrils, thus heart may need an increased energy-metabolic supply. SEVR assessment may improve the identification of subclinical myocardial damage in cardiac amyloidosis.



**P72**  
**AORTIC PULSE WAVE VELOCITY IN PATIENTS WITH COPD: 5-YEAR DATA FROM THE ARCADE STUDY**

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**Background:** Cardiovascular (CV) disease is a major cause of morbidity and mortality in COPD 1. Aortic pulse wave velocity (AoPWV), an independent predictor of CV risk, is elevated in COPD 2, however, there have been no longitudinal studies of AoPWV in COPD. The Assessment of Risk in Chronic Airways Disease Evaluation (ARCADE) aims to study CV risk factors longitudinally, in COPD. We hypothesised that patients with COPD would have increased AoPWV over 5 years compared to controls.

**Methods:** Thus far, 26 patients with COPD and 26 controls subjects have completed the assessments at baseline and after 5 years. Assessments included: AoPWV (SphygmoCor device), blood pressure (BP), heart rate, BMI and lung function (spirometry).

**Results:** At baseline, patients and controls were similar in age, gender, BP, heart rate and BMI but patients had a trend of greater PWV ( $p < 0.055$ ). After 5 years both COPD and comparators had increased AoPWV ( $p < 0.05$ ). There was no change BP in COPD, but BP increased in controls ( $p < 0.05$ ), while lung function declined in patients with COPD ( $p < 0.05$ ) but not controls. The rate of change of AoPWV was similar in patients and controls after adjustment for changes in BP (Figure 1) ( $p > 0.05$ ).

**Conclusions:** Although the increase in AoPWV over 5 years was similar in COPD and controls, AoPWV was greater in patients with COPD than controls at baseline and after 5 years which may suggest earlier stiffening in COPD. Further longitudinal assessments will inform the understanding of the development of arterial stiffness and may indicate possible therapeutic targets.

