P70: FINGER-TOE PULSE WAVE VELOCITY (FTPWV) MEASURED BY POPMèTRE® DEVICE IN PATIENTS WITH ANKYLOSING SPONDYLITIS


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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 ± 15.1 years, males 50.4%, blood pressure 117.8 ± 13.6/69.0 ± 8.8 mmHg). PWV estimated by Mobil-O-Graph (which uses an algorithm of 0.001) and PWV 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 (±SD) of Marfan patients were 6.1 ± 1.3 m/s by Mobil-O-Graph and 8.8 ± 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 ± 5.7 m/s. The values provided by Mobil-O-Graph may be derived from this population from the age factor and the brachial systolic pressure (r = 0.98) according to the formula: PWV = age × 1000 + 0.038 × systolic blood pressure.

Conclusions: The Mobil-O-Graph provides PWV values of an ideal subject for evaluating 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.

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 ± 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 ± 119.2 mmol/L, whereas body mass index (BMI) was 27.8 ± 4.4 kg/m². In-hospital mortality (IH) 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.

References
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 (ft-PWV) in patients with AS.

Methods: Finger-toe pulse wave velocity (ft-PWV) was measured by pOmetrè\textsuperscript{a} 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 < 0.001) compared to controls. Moreover, in the AS group we observed a higher ft-PWV with a mean difference of 1.63 (p < 0.006, 95% CI of 0.50 to 2.7). No significant difference was observed in pPP.

Conclusions: Individuals with ankylosing spondylitis showed increased ft-PWV, central and peripheral blood pressure, this contributes to explain the higher risk of cardiovascular disease in this pathology. pOmetrè\textsuperscript{a} 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.

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<tr>
<th>Table 1. Demographic and clinical characteristics of patients with ankylosing spondylitis and controls.</th>
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<td><strong>Age (years)</strong></td>
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AORTIC PULSE WAVE VELOCITY IN PATIENTS WITH COPD: 5-YEAR DATA FROM THE ARCADE STUDY

Nichola Gale 1, Mahfoudha Al Shezawi 1, Maggie Munnery 2, Barry McDonnell 1, John Cockcroft 1
1Cardiff University, UK
2Cardiff Metropolitan University, UK

Background: Cardiovascular (CV) disease is a major cause of morbidity and mortality in COPD. Aortic pulse wave velocity (AoPWV), an independent predictor of CV risk, is elevated in COPD, 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.

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TROTONIN INCREASE AND SUBENDOCARDIAL OXYGEN SUPPLY AND DEMAND IMBALANCE IN CARDIAC AMYLOIDOSIS

Lucia Salvi 1, Paolo Salvi 2, Andrea Grillo 2, Gianfranco Parati 2, Francesco Banfi 3, Stefano Perlini 3
1Department of Internal Medicine, IRCCS Policlinico San Matteo Foundation University of Pavia, Pavia, Italy
2Istituto Auxologico Italiano, Milano, Italy
3Department of Internal Medicine and Therapeutics, Policlinico San Matteo Foundation, University of Pavia, Italy

Background: The increase in troponin is a cardiac amyloidosis (CA) pecularity. The most acclaimed hypothesis is direct toxicity of amyloid fibrils on cardiac myocytes, 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 sensibility 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. These two phenomena may seriously affect myocardial perfusion. Moreover, amyloid alters the macrostructural organization of myocardium, thus heart may need an increased energy-metabolic supply. SEVR assessment may improve the identification of subclinical myocardial damage in cardiac amyloidosis.