of the aortic valve was the most common valvular abnormality. Phosphocalcic and lipid parameters, levels of hemoglobin, CRP and uric acid did not predispose to cardiac calcifications in our patients.

Discussion: In hemodialysis patients, the pathogenesis of cardiovascular calcification is complex and cannot be attributed to a passive process. This process involves several factors that can promote or inhibit calcification. The new multi-slice ultrafast scanner is a very sensitive method for topographic and quantitative assessment of coronary calcification and is a better alternative to invasive techniques.

Conclusion: Our study confirms the high prevalence of cardiac calcification in hemodialysis, and highlights the importance of early screening, and treatment of predisposing factors.

0141
Topography of the coronary tree calcification in hemodialysis
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Introduction: Cardiac disease is the first leading cause of death in hemodialysis. On these patients, cardiovascular calcifications occur at an earlier age and are developing faster than in the general population.

Materials and methods: Forty-nine patients on chronic hemodialysis, 26 men and 23 women, mean age 56.4 years, with a mean duration of 85 months on hemodialysis underwent screening for coronary calcification (CC) by a 64 slice cardio-sonar with ECG synchronization and without contrast injection. CC were studied at the anterior inter ventricular artery (AIV), the right coronary artery (RCA) , the left coronary artery (LCA), the circumflex artery (Cx), the diagonal artery (Diag) and the posterior inter ventricular artery (PIA). Agatston coronary calcification score (ACCS) was calculated by a pre supplied software.

Results: Coronary calcification concerned 69.4 % of cases and were distributed as follows: 69.4% AIV, RCA 36.7%, 32.7% Cx, Diag 29.6% 20.4% LCA, PIA 8.2%. CC sat in one artery in 22.4 % of cases, in 2, 3 or 5 arteries in 10.2% of cases, respectively, in 4 arteries in 14.3 % of cases and at 6 divisions in one patient. The mean ACCS was 331.1, and 522.2 in the 51 patients treated for ischemic heart disease (p = 0.09). The mean ACCS by coronary division was: AIV/AV 88.5, 69.8 CX, RCA 46.6, 15.8 Diag, LCA 6, PIA 2.8. Coronary calcification were significantly associated with conventional cardiovascular risk factors (age, male sex, systolic blood pressure, diabetes, history of ischemic heart disease).

Discussion: In this study, the topography of CC is superimposable to coronary atherosclerosis with which CC share several risk factors. Autopsy studies confirm that CC in patients with renal failure are more intense and are associated with more complex histological alterations in comparison with general population. Other studies confirm that total and individual coronary artery calcium scores are independent predictors of mortality in hemodialysis patients

Conclusion: Our results confirm the high prevalence of CC in hemodialysis and encourage early and regular screening.

0195
Early predictive factors of LV remodeling after STEMI: assessment by coronary angiogram and cardiovascular magnetic resonance
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Background/aim of the study: Several months after acute ST elevation myocardial infarction (STEMI), many patients will develop left ventricular (LV) remodeling and heart failure. The aim of this study was to identify early predictive factors for LV remodeling (LVR) assessed by coronary angiogram and cardiovascular magnetic resonance (CMR) after STEMI.

Methods: We prospectively included 52 patients with a first STEMI. All patients were successfully revascularized within 12 hours of chest pain onset using percutaneous coronary intervention (PCI). Angiographic parameters such as TIMI flow and blush grade were recorded. Index of microvascular resistance (IMR) was measured immediately after successful reperfusion. CMR was performed at days 4+/-2 days and at 6 months after STEMI. Comprehensive CMR included cine, T2-weighted, and late gadolinium enhancement (LGE) imaging allowing assessment of ventricular function, infarct size (IS), microvascular obstruction (MVO) and myocardial haemorrhage. LVR was defined as a >20% increase of LV volume at 6 months.

Results: LVR was observed in 34.8% of the patients (18/52). TIMI flow and blush grade after PCI was not different between patients with and without LVR (3 and 2 for TIMI flow and 2 and 2 for Blush grade respectively, p=0.952). However the IMR level markedly differed between patients with and without LVR (73.95 vs. 27.23, p=0.0293). After multivariate analysis IMR>40 was the strongest angiographic factor to predict LVR (OR 15 (1,030-218.4), p=0.03). Regarding CMR, patients with LVR had lower LVEF (43% vs. 48%; p=0.01), larger IS (51 mg vs 32 mg; p=0.002) and greater MVO extent (4.5seg vs. 2seg; p=0.03) when compared to patients with no LVR.

Conclusion: IMR assessed by coronary angiogram, as well as IS and MVO extent assessed by CMR, are strong predictive factors of LV remodeling 6 months after STEMI.

0031
Predictive factors of left ventricular remodeling after myocardial infarction. Angiographic and MRI point of view
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Background/aim: several months after myocardial infarction, many patients will develop left ventricular remodeling and heart failure. The aim of this study was to bring out predictive factors of left ventricular remodeling especially angiographic and MRI ones.

Methods: prospective cohort study.

Results: 16 patients of the 46 included had left ventricular remodeling 6 months after myocardial infarction. Angiographic data: there was statistical association between index of microcirculatory resistance (IMR) >40 and left ventricular remodeling (OR 15 (1,030-218.4), p=0.0308). IMR level was statistically significant higher in left ventricular remodeling group (73.95 versus 27.23, p=0.0293). MRI data: there was a statistically significant relationship between transmurality (late gadolinium enhancement) and remodeling (OR 25.46 (1.397-463.8), p=0.0019), the number of akinetic segments was statistically significant higher in left ventricular remodeling group (6,26 versus 4, p=0.0012), and the number of segments with microvascular obstruction too (4,43 versus 2,26, p=0.0392).

Conclusion: High level of IMR, number of segments with microvascular obstruction and transmural late gadolinium enhancement can be considered as predictive factors of left ventricular remodeling 6 months after a myocardial infarction.

Keywords: angiography; MRI; IMR; microvascular obstruction; left ventricular remodeling

0276
Dynamics of the perfusion reserve during adenosine-induced stress in rats
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Introduction: In clinical routine, myocardial perfusion MRI is generally performed with a stress/rest protocol using adenosine as a short living (10s half-life)
stressor. Adenosine allows visualizing ischemic risk zones in the myocardium and is typically injected 3 to 4 minutes prior to stress imaging. To gain insight into perfusion dynamics under stress, we used a non-invasive and fast imaging procedure (cine-ASL) to study the longitudinal effect of a continuous injection of adenosine on myocardial blood flow (MBF) and MBF reserve in rats.

**Methods:** MBF and MBF reserve were monitored every 5min following adenosine injection (140μg/kg/min) on 17 healthy Wistar rats at 4.7T. For the Stress group (N=6), adenosine was continuously injected for 40min. For the Stress/Recovery group (N=6), adenosine was injected for 25min and then stopped for the last 15min. Finally, for the Control group (N=7), a saline solution was injected for 40min.

**Results:** Mean group MBF at rest was 5.4±0.5mL/g/min in a ROI covering the entire myocardium. Mean group MBF data are reported as a function of time after onset of adenosine (Fig). In every studied animal of the 2 first groups subjected to stress, an initial MBF increase was found within the first 5min following adenosine infusion. But, the maximum MBF response to adenosine was always obtained later, between 5-10min, followed by a continuous MBF decrease until 25min. Maximum stress perfusion was 10.8±1.4mL/g/min leading to a reserve of 2.1±0.3, consistent with previously reported study.

**Conclusion:** This study focuses on MBF response dynamics to adenosine. In rats, maximum MBF response to adenosine was found between 5 to 10min. Such dynamic measurements give more detailed insight into rodent coronary reserve and coronary responses to infused vasodilators and may give complementary information on microvascular functional defects in non-ischemic heart disease models.