TCT-143

Abstract Withdrawn

TCT-144

Impact of chronic kidney disease on platelet reactivity and clinical outcomes of patients undergoing percutaneous coronary intervention

Fabio Mangiacapra[1], Elisabetta Ricottini[1], Emanuele Barbato[2], Giuseppe Patti[1], Vincenzo Vizzi[1], Andrea D'Ambrosio[1], Bernard De Bruyne[2], William Wijns[2], Germano Di Sciascio[1]
[1]Campus Bio-Medico University of Rome, Rome, Italy, [2]Cardiovascular Center Aalst, Aalst, Belgium

Background: Platelet reactivity is a well-established determinant of clinical outcomes after percutaneous coronary intervention (PCI). Conflicting data have been reported concerning the impact of chronic kidney disease (CKD) on residual platelet reactivity (PR) on clopidogrel in patients with coronary artery disease (CAD) undergoing PCI. Aim of the present study was to compare PR and its association with clinical outcomes after PCI in CAD patients with and without CKD.
Methods: In 800 patients treated with clopidogrel we measured PR with the VerifyNow P2Y12 Assay immediately before PCI (results given as P2Y12 reaction units [PRU]). According to previous studies, we defined HPR as a PRU value ≥240 and LPR and a PRU value ≤178. CKD was defined as a glomerular filtration rate <60 ml/min/1.73 m^2. Clinical follow-up at 30 days was obtained in all patients. Net adverse clinical events (NACE) were considered as ischemic (death, myocardial infarction and target vessel revascularization) and bleeding events (according to TIMI criteria).
Results: Patients with (n=173, 21.6%) and without CKD showed similar PRU values (208±67 vs. 207±75; p=0.819). The incidence of 30-day ischemic (12.1% vs. 7.2%; p=0.036) and bleeding events (8.7% vs. 2.1%; p<0.001) was higher in the CKD group. The presence of HPR was associated with higher rates of ischemic events in both patients with (21.1% vs. 7.6%; p=0.012) and without CKD (12.6% vs. 4.7%; p<0.001). Likewise, LPR was associated with higher rates of bleeding in both patients with (19.3% vs. 3.4%; p<0.001) and without CKD (5.1% vs. 0.5%; p<0.001). NACE were significantly higher in CKD patients with HPR or LPR (25.4%) and lowest in those without CKD, HPR or LPR (6.6%; p for trend <0.001). At multivariate analysis, the combination of CKD with LPR or HPR was the strongest predictor of NACE (odds ratio 3.4, 95% confidence interval 2.0-5.6; p<0.001).
Conclusions: We did not find an association between CKD and higher levels of residual PR on clopidogrel. However, the combination of CKD with either high or low platelet reactivity is a strong determinant of adverse events after PCI.

TCT-145

The significance of VARC-defined acute kidney injury after transcatheter aortic valve implantation using the balloon-expandable Edwards bioprosthesis

Muhammed Khawaja[1], Martyn Thomas[2], Abhishek Joshi[3], Kaleab Asrress[4], Karen Wilson[3], Kirsty Bolter[5], Jane Hancock[5], Christopher Young[5], Vinayak Bapat[2], Simon Redwood[6]
[1]King's College London, London, United Kingdom, [2]St Thomas' Hospital, London, United Kingdom, [3]St Thomas' Hospital, LONDON, United Kingdom, [4]King's College London, LONDON, United Kingdom, [5]St Thomas' Hospital, LONDON, United Kingdom, [6]King's College London/ St Thomas' Hospital, London, United Kingdom

Background: Acute kidney injury (AKI) is a potentially serious complication of transcatheter aortic valve implantation (TAVI) that has recently been re-defined by the Valve Academic Research Consortium (VARC). The aim of this study was to identify the incidence and risk factors for AKI after TAVI.
Methods: We performed a retrospective analysis of data from 248 consecutive patients undergoing TAVI using the Edwards bioprosthesis at St Thomas' Hospital, London, UK. AKI was defined as a VARC-modified Risk, Injury, Failure, Loss, and End-stage (RiFLE) kidney disease score ≥2.
Results: Of 248 patients who underwent TAVI using the Edwards bioprosthesis 89 (35.9%) of patients suffered an acute kidney injury as defined by the a score of ≥2 on the VARC-modified RiFLE score. The overall mean pre-procedural creatinine was 116.6±69.3 μmol/L with an overall peak creatinine of 148.8±94.4 mmol/L (p<0.001). Patients with AKI had greater mean pre-procedural (134.7±74.5 v. 106.5±64.2 mmol/L (p<0.001), 48h (206.8±89.1 v. 98.0±40.0 mmol/L (p<0.001)) and 72h creatinine concentrations (205.9±88.3 v. 99.3±39.6 mmol/L (p<0.001)). A higher VARC-modified RiFLE score was associated with increased mortality (p<0.001). Kaplan Meier analysis according to incidence of RiFLE score ≥2 (i.e. AKI) demonstrated significantly increased mortality at 30 days (13.5% v. 3.8%), 1 year (31.5% v. 15.0%) and overall (40.4% v. 19.5%; logrank p<0.001) at a median follow up of 379 days (interquartile range 113-729 days). Multivariate logistic regression analysis revealed that the variable with the strongest independent association with risk of AKI was DM (OR 3.17, 95%CI 1.67-6.05, p<0.001), followed by peripheral vascular disease (OR 2.54, 95%CI 1.34-6.44, p=0.007) and the pre-procedural stage of chronic kidney disease (OR 1.57, 95%CI 1.11-2.21, p=0.010).
Conclusions: Greater than 1/3 of patients sustain AKI after TAVI using the Edwards bioprosthesis, as defined by the VARC-modified RiFLE score. AKI was associated with increased mortality at both 30-days and at 1-year. A history of diabetes mellitus, peripheral vascular disease and higher chronic kidney disease stage had the strongest independent associations with post-TAVI AKI.

TCT-146

The Effect of Drug-Eluting Stents on Clinical and Angiographic Outcomes in Renal Failure Patients with Dialysis: Multicenter Registry in Asia

Sunao Nakamura[1], Hisao Ogawa[2], Jang-Ho Bae[3], Yeo Cahyadi[4], Wasan Udayachalerm[5], Damras Tresukosol[6], Sudaratana Tansuphaswadikul[7]
[1]New Tokyo Hospital, Chiba, Japan, [2]Kumamoto University Hospital, Kumamoto, Japan, [3]Konyang University Hospital, Daejeon, Korea, Republic of, [4]Husada Hospital, Jakarta, Indonesia, [5]King Chulalongkorn Memorial Hospital, Bangkok, Thailand, [6]Faculty of Medicine Siriraj Hospital, Bangkok, Thailand, [7]Chest Disease Institute, Nonthaburi, Thailand

Background: Patients treated with renal failure have been reported having high incidence of mortality and other complications rate after PCI. Optimal treatment of PCI for renal failure patients with dialysis is still unknown. Aim: The aim of this study is to compare the safety and efficacy of Sirolimus (SES), Paclitaxel (PES), EPC capture (ECS), Zotarolimus (ZES-R/ Endeavor Resolute), BiolimusA9 (BES) and Everolimus-eluting stent (EES) on the outcome of percutaneous coronary intervention in renal failure patients with dialysis (CRF-HD).
Methods: A prospective analysis of 1013 patients with CRF-HD (258 SES, 244 PES, 77 ECS, 118 ZES-R, 128 BES, 188 EES) in six high volume Asian centers after successful stenting was performed. The study endpoints were 30 days major adverse cardiac events (MACE) and 12, 24 and 36 months target lesion revascularization (TLR) and MACE.
Results: The baseline clinical characteristics between 5 groups were similar. See table for clinical results.
Conclusions: The use of drug-eluting stents in patient with CRF-HD was safe with low acute complication. Patients treated with PES and EES showed lesser incidence of restenosis rate and TLR compared with other drug-eluting stents.

		SES	PES	ECS	ZES-R	BES	EES
Number of patients		258	244	77	118	128	188
Multivessel disease (%)		89.1	87.7	85.7	83.4	79.6	89.3
MACE at 30 days (%)		0.8	1.2	1.3	0.8	0.8	0.5
Reference diameter (mean: mm)		2.80	2.79	2.88	2.85	2.86	2.91
Lesion type: % of B_2, C (%)		64.0	67.7	56.6	63.7	67.1	61.9
Stent length (mean: mm)		28.9	29.5	26.2	26.9	29.9	27.8
MLD at baseline (mean: mm)		2.65	2.63	2.68	2.59	2.60	2.66
12 months	TLR (%)	15.1	9.8*	20.8	16.9	18.8	9.5*
	MACE (%)	19.4	12.7*	23.4	19.5	20.3	12.2*
24 months	TLR (%)	17.8	11.9*	23.4	19.5	20.3	13.8*
	MACE (%)	22.4	14.8*	26.0	22.9	22.6	17.0*
36 months	TLR (%)	19.4	12.7*	26.0	21.2	21.9	17.0*
	MACE (%)	25.2	16.8*	29.9	25.4	25.0	20.2*

*p<0.05 vs. SES, ECS, ZES-R and BES.

TCT-147

High dose Atorvastatin Pretreatment for Preventing Contrast-Induced Nephropathy in Patients Receiving Primary Percutaneous Coronary Intervention: Prespecified Substudy of a Prospective Randomized Clinical trial

Sang-Ho Jo[1], Hyun-Sook Kim[2], Jin-Ho Choi[3], Seung-Hyuk Choi[3], Joo-Yong Hahn[3], Sung Yun Lee[4], Young Bin Song[3]
[1]Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do , Korea, Republic of, [2]Hallym University Sacred Heart Hospital, Anyang, Kyeonggi-do, [3]Samsung Medical Center, Seoul, Korea, Republic of, [4]Ilsan Paik Hospital, Goyang , Korea, Republic of

Background: Controversies persist whether statin pre-treatment can prevent contrast-induced nephropathy(CIN). We evaluate the efficacy of high dose atorvastatin on CIN occurrence.
Methods: We studied whether atorvastatin 80mg loading and subsequent use for 5days (high dose group[HD]) could prevent CIN as compared to those received atorvastatin 10mg (routine dose group[RD]) with same schedule in patients with ST-elevation myocardial infarction undergoing primary angioplasty. Primary endpoint was incidence of CIN, defined as a ≥ 25% or ≥ 0.5 mg/dL increase in baseline serum creatinine within 5 days after contrast administration. Secondary endpoint was 1- and 6-month renal function change and composite of all cause mortality, renal failure, heart failure and target vessel revascularization.
Results: One hundred and ten patients were allocated to HD and 108 to RD from August 2007 to February 2009. CIN incidence was 5.5% (6/110) in HD and 10.2% (11/108) in RD, a nonsignificant difference (p=0.193). CIN occurred significantly less in HD than RD, 0% vs. 16.7% (p=0.024) in subgroups of renal insufficiency (creatinine clearance [CrCl]≤60mL/min) and 4% (1/25) and 23.1% (6/26) respectively, (p=0.048) in old patients≥70. Composite of clinical outcomes at 6-month was comparable in HD and RD (7.9% and 13.1%, p=0.26). CrCl rat 1-month tended to be higher, in HD than in RD, 81

POSTERS

mL/min and 72.6 mL/min (p=0.059) but similar at-6month, 80.2 mL/min and 72.2 mL/min (p=0.167).
Conclusions: High dose atorvastatin treatment does not prevent CIN in patients receiving primary angioplasty. However it demonstrated potential of lowering CIN in patients with renal insufficiency and elderly.

TCT-148

Blood Transfusion And The Risk Of Acute Kidney Injury Following Transcatheter Aortic Valve Implantation.

Rutger-Jan Nuis[1], Josep Rodes-Cabau[2], Jan Malte Sinning[3], Leen van Garsse[4], Joelle Kefer[5], Johan Bosmans[6], Antonio Dager[7], Nicolas Van Mieghem[8], Marina Urena[9], Nikos Werner[10], Georg Nickenig[3], Jos Maessen[11], Parla Astarci[5], Luis Benitez[12], Eric Dumont[13], Ron Van Domburg[14], Peter De Jaegere[15]
[1]Thoraxcenter, Erasmus Medical Center, Rotterdam, Rotterdam, Zuid Holland, [2]Quebec Heart and Lung Institute, Quebec, Canada, [3]University Hospital Bonn, Bonn, Germany, [4]University Hospital Maastricht, Maastricht, Netherlands, [5]University Hospital Saint-Luc, Brussels, [6]University Hospital Antwerp, Antwert, Belgium, [7]Angiografía de Occidente, SA, Cali, colombia, [8]Erasmus MC, Rotterdam, Netherlands, [9]Quebec Lung and Heart Institute, Laval University, Québec, Quebec, [10]University Hospital Bonn, Bonn, NRW, [11]University Hospital Maastricht, Maastricht, [12]Angiografia de Occidente S.A, Cali, Cali, [13]Quebec Heart and Lung Institute, Laval University, Quebec, QC, [14]Thoraxcenter, Erasmus MC, Rotterdam, Netherlands, [15]Thoraxcenter, Erasmus Medical Center, Rotterdam, Rotterdam, Netherlands

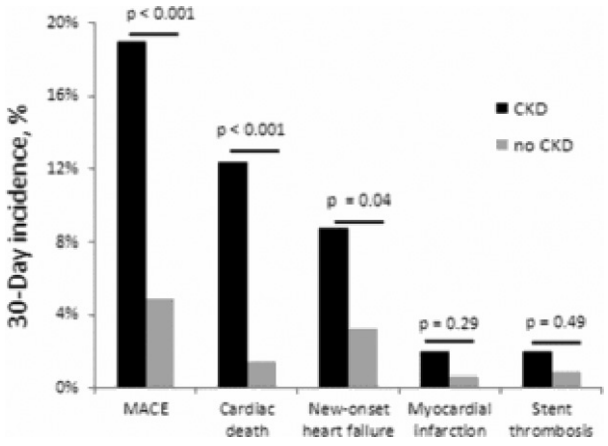
Background: Blood transfusion is associated with acute kidney injury (AKI) after transcatheter aortic valve implantation (TAVI). We sought to elucidate in more detail the relation between blood transfusion and AKI and its effects on short- and long-term mortality.
Methods: 995 patients with aortic stenosis underwent TAVI with the Medtronic-CoreValve or the Edwards Valve in 7 centers. AKI was defined by the Valve Academic Research Consortium (absolute increase in serum creatinine ≥0.3 mg/dl (≥26.4 μmol/l) or ≥50% increase ≤72 hr). Logistic and Cox regression was used for predictor and survival analysis.
Results: AKI occurred in 20.7% (n=206). The number of units of blood transfusion ≤24 hr was the strongest predictor of AKI (≥ 5 units, OR: 4.81 [1.45-15.95], 3-4 units, OR: 3.05 [1.24-7.53], 1-2 units, OR: 1.47 [0.98-2.22]) followed by peripheral vascular disease (OR: 1.48 [1.05-2.10]), history of heart failure (OR: 1.43 [1.01-2.03]), leucocyte count ≤72 hrs after TAVI (OR: 1.05 [1.02-1.09]) and EuroSCORE (OR: 1.02 [1.00-1.03]). Potential triggers of blood transfusion such as baseline anemia, bleeding-vascular complications and peri-operative blood loss were not identified as predictors. Patients with severe baseline anemia had 2.4 times less blood loss but on average received 2.3 fold more units of blood transfusions in comparison to patients without anemia before TAVI (p<0.001). AKI and life-threatening bleeding were independent predictors of 30-day mortality (OR: 3.04 [1.52-6.07], OR: 5.39 [2.14-13.57], respectively) while transfusion (≥3 units), baseline anemia and AKI predicted mortality beyond 30 days.
Conclusions: AKI occurred in 21% of the patients after TAVI. The number of blood transfusions but not the indication of transfusion predicted AKI. AKI was a predictor of both short- and long-term mortality whereas blood transfusion predicted long-term mortality. These findings indicate that outcome of TAVI may be improved by more restrictive use of blood transfusions.

TCT-149

Impact of Chronic Kidney Disease on Myocardial Infarct Size and Adverse Events in ST-Elevation Myocardial Infarction: Results from the INFUSE-AMI Trial

Usman Baber[1], Roxana Mehran[2], Sorin Brener[3], Akiko Maehara[4], Dariusz Dudek[5], Thomas Neunteuff[6], D. Christopher Metzger[7], C. Michael Gibson[8], Gregg Stone[9]
[1]Mount Sinai School of Medicine, New York, NY, [2]Mount SInai School of Medicine, New York, NY, [3]New York Methodist Hospital, Brooklyn, NY, [4]Cardiovascular Reserach Foundation and Columbia University Medical Center, New York, NY, [5]University Hospital, Krakow, Poland, [6]AKH Vienna Austria, Vienna, Vienna, [7]Wellmont CVA Heart Institute, Kingsport, USA, [8]Beth Israel Deaconess Med Ctr - Harvard Medical School, Boston, USA, [9]Columbia University Medical Center and the Cardiovascular Research Foundation, New York, NY

Background: Chronic kidney disease (CKD) patients have less favorable outcomes after ST-elevation myocardial infarction (STEMI) for yet unclear reasons.
Methods: The INFUSE-AMI trial randomized patients with STEMI due to proximal or mid LAD occlusion to intracoronary bolus abciximab (ClearWay RX catheter) vs. no abciximab, and to thrombus aspiration (Export) vs. no aspiration. We compared infarct size as % of LV mass assessed by magnetic resonance imaging at 30-days, myocardial reperfusion and incidence of adverse events between patients with vs. patietns without CKD. CKD was defined as a creatinine clearance < 60 ml/min.
Results: Patients with CKD (n=59, 14.4%) were older, more often female, diabetic and less likely to undergo angiography within 3 hours of symptom onset (50.8% vs. 72.2%, p=0.001) compared to those without CKD (n=349, 85.5%). Following PCI, final thrombolysis in myocardial infarction (TIMI) 3 flow (86.4 vs. 92.8%, p=0.12) and myocardial blush grade (MBG) 3 (64.4% vs. 70.5%, p=0.35) was observed similarly in both groups. Median infarct size was non-significantly larger in CKD patients (19.3% vs. 17.0%, p=0.34). The incidence of 30-day adverse events, were significantly higher in those with CKD (Figure). There were no significant differences in stent thrombosis, reinfarction or revascularization between groups.



Conclusions: Differences in infarct size between patients with and without CKD presenting with STEMI are modest and unlikely to account for the significantly higher short-term cardiac risk in those with CKD.

TCT-150

Prognostic Value of Different Definitions of Contrast Induced Acute Kidney Injury in STEMI: Analysis from the HORIZONS-AMI Trial

Rudolf Jarai[1], George Dangas[2], Kurt Huber[3], Roxana Mehran[4], Gregg Stone[5], Ke Xu[6]
[1]Wilhleminenhospital, Vienna, Austria, [2]Department of Cardiology, Mount Sinai Medical Center, New York, New York City, NY, [3]Dept Cardiology and Emergency Medicine, Wilhelminenhospital, Vienna, Austria, Vienna, Austria, [4]Mount Sinai Hosptial, New York, USA, [5]Columbia University Medical Center and the Cardiovascular Research Foundation, New York, NY, [6]Cardiovascular Research Foundation, New York, NY

Background: Several definitions of acute kidney injury (AKI) are in use, and the optimal absolute and relative increase of serum creatinine increase after contrast administration to define contrast-media induced acute kidney injury (CI-AKI) is still a matter of debate. Moreover, the prognostic relevance of AKI according to the varying definitions in STEMI has not been established.
Methods: Serum creatinine concentration data within 48h after coronary angiography was present in 2975 STEMI pts in the HORIZONS-AMI trial. Patients were analyzed according to different AKI definitions (AKIN-, modified AKIN- (mAKIN), Waikar-Bonventre (W-B), percent-changes of creatinine (<25%, 26-50%; 51-75%; >75%) and to the commonly used "standard-definition" of a relative increase in serum creatinine of ≥25% or an absolute increase of ≥0.5 mg/dL). The primary endpoint was all-cause mortality at 3 years.
Results: Depending on definitions the incidence of CI-AKI ranged from 5.0% to 15.5%. Similarly, 3-year mortality rates differed substantially with respect to the different CI-AKI definitions (Table). Absolute changes of creatinine were strongly associated with all-cause mortality, starting with an increase of 0.3mg/dl absolute increase of creatinine above baseline (HR 3.68 p<0.001; Figure) – the cutoff level used in the AKIN- and modified AKIN-definitions. The increased risk associated with >0.3mg/dl absolute increase of creatinine was independent of the amount of contrast-media used.

	AKIN	mAKIN	W-B	Percent-changes	Standard
no CI-AKI	4.2	4.2	4.8	4.5	4.5
CI-AKI I	24.0	14.5	23.0	8.7	16.0
CI-AKI II	60.0	23.0	58,5	21.4	
CI-AKI III	36.5	55.6	53.5	47.3	
p-value	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001

Conclusions: The modified AKIN criteria might be the optimal definition for contrast-induced AKI after primary PCI in STEMI.

POSTERS