

by consultant radiologists as 'unsuitable for EVAR'. 66 had OSR and 52 had Pararenal EVAR (PEVAR).

Results: PEVAR patients were older (74.3yrs vs. 70.8 yrs, $p=0.014$) with higher mean SVS co-morbidity severity scores ($p=0.0001$). All procedures were within 14 days of diagnosis. Mean aneurysm diameter was larger in OSR (OSR 6.6cm vs. PEVAR 5.9cm, $p=0.010$). For PEVAR 83% of endografts were 34mm/36mm. 3-year aneurysm-related survival was significantly higher with PEVAR (100% vs. OSR (92.4%+/-4.37%), $p=0.045$). PEVAR provided an incremental cost-effectiveness ratio of €129,586 saved per QALY gained. 3-year freedom from secondary intervention (PEVAR 83.4% vs OSR 95.5%, $P=0.301$) and all-cause survival (PEVAR 57.1% vs. OSR 84.8%, $p=0.195$) were similar. 30-day morbidity halved with PEVAR (15% vs. 30%, $p=0.059$). Length of hospital stay ($p=0.0007$) was lower and number of patients fit for discharge to their home ($p=0.006$) higher with PEVAR.

Conclusions: PEVAR granted our patients longer Q-TWiSt and Superior Freedom from MACE up to three years. Despite 3-year survival rate of 57%, PEVAR is cost-effective and offered as Endo-bailing for patients living on borrowed time, abolishes the socio-economic catastrophe of managing a rupture PAAA.

TCT-131

Cerebral Ischemia After Thoracic Endovascular Aortic Repair: A Diffusion-Weighted Magnetic Resonance Imaging Study

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Background: The risk of clinical apparent, periprocedural stroke after thoracic endovascular aortic repair (TEVAR) ranges between 2 and 6% and has been associated with increased postoperative mortality. Stroke after TEVAR is thought to be related to multiple emboli, which are dislodged during manipulation of guidewires, catheters and large-bore delivery devices in the diseased aortic arch. Such emboli may also account for clinically silent cerebral ischemia. However, the rate of silent cerebral ischemia in the setting of TEVAR is yet unknown, but may be even higher than the rate of clinical apparent neurological events.

Methods: Twenty patients (12 male, 8 female) who underwent TEVAR were included into this descriptive study; exclusion criteria were a history of stroke, carotid artery disease, renal failure and contraindications for magnetic resonance imaging (MRI). Periprocedural apparent and silent cerebral ischemia was assessed by neurological testing and serial cerebral diffusion-weighted MRI (DW-MRI) at baseline and within the first 10 days (mean: 4.9 days) post procedure.

Results: TEVAR was successful in all patients without immediate clinically apparent neurological deficits. Post-interventional cerebral DW-MRI detected a total of 33 new foci of restricted diffusion in 13 of the 20 patients (65%). Lesions were usually multiple (1-6 lesions per patient) and ranged in size between 15 mm³ and 585 mm³. 17 lesions were found in the left middle cerebral artery and PICA territory, 10 lesions in the right middle cerebral artery and PICA territory. Overstenting of the left-subclavian artery was performed in 9 cases, but was not associated with lateralization of lesions. There were no additional apparent neurological events during the in-hospital period.

Conclusions: TEVAR resulted in a high incidence of new foci of restricted diffusion on cerebral DW-MRI in a pattern suggestive for periprocedural embolization. Although even multiple lesions per patients were found, these lesions were not associated with apparent neurological deficits during the in-hospital period. Further developments in TEVAR should be directed towards reducing the risk of periprocedural cerebral embolization.

TCT-132

Abstract Withdrawn

Chronic Kidney Disease and Acute Renal Insufficiency

Hall D

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TCT-133

Role of arterial stiffness and impaired renal function in the progression of non-culprit coronary lesions after percutaneous coronary intervention

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Background: In the era of drug eluting stent, progression of non-culprit coronary lesion emerged as a new therapeutic target of coronary artery disease. We aimed to clarify the

prognostic factors for the progression of non-culprit coronary lesion after percutaneous coronary intervention (PCI).

Methods: We retrospectively examined 401 patients who underwent PCI during February 2010 to January 2011 in our institute. Among them, 275 patients were performed follow-up coronary angiography (CAG) 6-12 months after PCI. Patients with target lesion revascularization (n=39) were excluded. Finally, total of 236 patients were included in this study. Progression of non-culprit lesion was defined as clinically driven PCI because of the development of coronary lesion which was not significant at initial PCI but significant at follow-up CAG, and was associated with ischemic symptom and/or abnormal results of functional study.

Results: Thirty three patients (14%) underwent additional clinically driven PCI to treat non-culprit coronary lesions. There was no difference in background clinical characteristics between patient with or without progression of non-culprit lesion PCI. Prevalence of chronic kidney disease (CKD) (61% vs. 31%, $p=0.001$) and multi-vessel disease (MVD) (55% vs. 35%, $p=0.027$) were significantly higher and statin use (61% vs. 72%, $p=0.187$) was tended to be lower in patients with non-culprit lesion PCI than those without. Brachial-ankle pulse wave velocity (baPWV) was significantly higher in patients with non-culprit lesion PCI than those without (1838±371 vs. 1589±313cm/s, $p<0.001$). High density lipoprotein cholesterol level at follow-up CAG was tended to be lower in patients with non-culprit lesion PCI than those without (54±15 vs. 58±16mg/dL, $p=0.147$). Multivariate analysis showed that higher baPWV, CKD, MVD, and lower HDL at follow-up CAG were independent determinants for progression of non-culprit coronary lesion.

Conclusions: In conclusion, higher baPWV, CKD, MVD, and lower HDL at follow-up CAG were independent determinants of non-culprit coronary lesion PCI, suggesting important prognostic role of arterial stiffness and impaired renal function in the progression of non-culprit coronary artery lesion.

TCT-134

Abstract Withdrawn

TCT-135

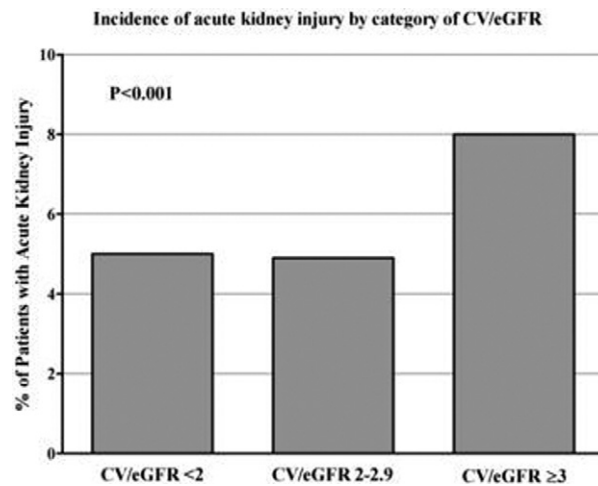
Contrast Use And Acute Kidney Injury In Contemporary Clinical Practice

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Background: Recent insurance-based observational data indicate that the ratio of contrast volume (CV) use to calculated creatinine clearance predicts acute kidney injury (AKI). Whether these findings will be observed in other populations, is uncertain.

Methods: 11,986 CATH and PCI procedures were performed at a single center (Wake Forest Baptist Medical Center) between January 2007 and December 2011 and were evaluated for AKI post-procedure. A contrast minimization strategy including automated contrast injection was used in all patients. Procedures with missing pre- or post-creatinine or renal failure with dialysis were excluded (n=4,117). Glomerular filtration rate was estimated (eGFR).

Results: High risk baseline characteristics were significantly more prevalent with AKI (n=430) compared to no AKI (n=7,439), including CHF, diabetes, prior renal failure, acute MI, and elevated baseline creatinine. Per institutional standard of practice CV use was 20 mL lower per procedure for those with AKI ($p<0.001$), but CV/eGFR was 30% higher for those with AKI ($p<0.001$). After multivariable adjustment, CV/eGFR remained a significant independent predictor of AKI, with CV/eGFR ≥ 3.0 having 2.18 (1.59-3.00) increased odds of AKI vs. CV/eGFR < 3.0 (see Figure).



Conclusions: In this prospective observational registry CV/eGFR was independently predictive of AKI, with a ratio ≥ 3.0 associated with two-fold greater risk of AKI. These data provide strong support for the use of this metric as a risk-reduction method for AKI