Methods: Using the 2004–2013 National Inpatient Sample, the largest publicly available inpatient care database representing more than 95% of the United States population, we extracted data using the ICD 9 code 410.7 (NSTEMI) as the primary diagnosis and the ICD 9 code 585 (CKD) as the secondary diagnosis. We excluded patients with missing information on age, sex, and mortality for a final sample size of 7,774. We used prevalence logistic and linear regression models with random effect to adjust for confounding.

Results: Among 3,784,774 admissions for NSTEMI, 31% (1,174,006) underwent PCI. Compared to NSTEMI patients undergoing PCI with no kidney dysfunction, NSTEMI patients undergoing PCI with severe kidney dysfunction (stage 5) had a significantly longer length of stay (7.5 days vs 3.5 days, p<.0001), higher cost of hospitalization ($33,620 vs $21,477, p<.0001), greater likelihood to be discharged to a skilled nursing facility (16% vs 5%, p<.0001), greater likelihood to be on Medicare (86% vs 52%, p<.0001), and greater likelihood to be from the lowest income bracket, <$25,000 (33% vs 27%, p<.0001). NSTEMI patients with any degree of CKD who were on Medicare had a 14% greater mortality and were 52% less likely to undergo PCI compared to NSTEMI who were self-pay (p<.0001). Women with NSTEMI with any of CKD were 25% less likely to undergo PCI compared to men with NSTEMI and any degree of CKD.

Conclusion: Among NSTEMI patients undergoing PCI, increasing severity of chronic kidney disease was associated with significantly longer length of stay, higher cost of hospitalization, and a greater likelihood to be discharged to a skilled nursing facility. This subset of patients was also more likely to be below a lower income bracket and to be on Medicare. This knowledge may fuel further studies to identify tools to lessen the socioeconomic differences among NSTEMI patients with varying stages of CKD and address the disparities in utilization of advanced technology in this specific subset of patients.

CANCAN WE TEACH HEART FAILURE DRUGS NEW TRICKS?

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Carvedilol vs. metoprolol tartrate on mortality in patients with heart failure with a reduced ejection fraction and atrial fibrillation or sinus rhythm: a post-hoc analysis of COMET

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 on behalf of COMET study group.

Methods and results: COMET enrolled 3,029 out-patients with chronic stable heart failure; 252 had an implanted pacing device (usually a right ventricular pacemaker or ICD); of those who did not, 2209 patients were in SR at baseline, 552 in AF and 16 in undetermined rhythm.

Of patients in sinus rhythm, 1360 were titrated to full doses of beta-blockers (median resting heart rate 66.00 bpm (IQR: 60.0, 75.0)), of those in AF, 324 (median resting heart rate 70.00 bpm (62.0, 78.8)) and of those with a device, 117 (median resting heart rate of 70.00 bpm (61.0, 72.0)); 471, 111 and 58 in respectively were on study medication but not on target doses; their heart rates were 67.5bpm (61.0, 72.0)), 70.0bpm (61.0, 72.0)) and 68.5bpm (61.0, 72.0) respectively on study medication but not on target doses; their heart rates were 67.5bpm (61.0, 72.0)), 70.0bpm (61.0, 72.0)) and 68.5bpm (61.0, 72.0) respectively at the first maintenance visit.

For the entire population (n=3,029), all-cause mortality was lower in patients assigned to carvedilol (hazard ratio 0.83 [95% CI 0.74–0.93], p=0.0017) and there was no significant interaction (p-value = 0.06) by the original allocated rhythm which did not account for pacing devices. In a multivariable model with predefined variables (age, sex, diabetes, ischaemic heart disease, duration of HF, NYHA, BNP, LVEF, sodium, potassium, creatinine, sodium, use of aldosterone antagonists or statins) and confined to patients taking any dose of study medication at the first maintenance visit, the hazard ratio for mortality on carvedilol compared to metoprolol was 0.86 (p-value = 0.01) for those in sinus rhythm, 0.56 (p-value = 0.001) for those in AF and for those assigned by the attending physician's score to receive a value 1.06 (p-value = 0.81). The interaction between treatment effect and rhythm (sinus or AF) at baseline was significant (p=0.03).

Conclusions: Patients randomly assigned to and taking carvedilol had a lower mortality compared to and taking metoprolol tartrate. The difference in mortality was substantially greater in patients in AF compared to sinus rhythm (after excluding patients with pacing devices). This requires further exploration.

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Effect of ivabradine on mortality in patients with heart failure and a reduced left ventricular ejection fraction not receiving a beta-blocker: an analysis from SHIFT

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Methods and results: A post-hoc analysis of the SHIFT data was conducted investigating the effect of ivabradine on all-cause and cardiovascular mortality amongst the subgroup of patients who were not prescribed a beta-blocker. Of 6505 patients valid for this analysis, 685 (10.5%) were not taking a beta-blocker of whom 426 (62.6%) were in atrial fibrillation or paroxysmal atrial tachycardia. Their mean age was 64 (SD11) years and LVEF 29 (SD5)%.

Patients not taking beta-blockers had a mean baseline heart rate of 84 (SD12)bpm, which had dropped to 68 (SD11) bpm at 28 days in those assigned to ivabradine and to 81 (SD14)bpm in those assigned to placebo. Patients assigned to placebo who were not taking beta-blockers had a higher risk profile and higher mortality (27.3%) compared to those taking beta-blockers (15.7%; p=0.009).

Overall, 552 patients assigned to placebo died compared to 503 assigned to ivabradine (unadjusted HR 0.91; 95% CI 0.80–1.02; p=0.11). Amongst patients taking beta-blockers, there were 459 deaths amongst those assigned to placebo compared to 432 assigned to ivabradine (unadjusted HR 0.94; 95% CI 0.83–1.08; p=0.38). Amongst those not taking beta-blockers there were 93 deaths in those assigned to placebo but only 71 in those assigned to ivabradine (unadjusted HR 0.70; 95% CI 0.52–0.96; p=0.026). A test for interaction was of borderline significance (p=0.089). For those not taking beta-blockers there was a similar trend for cardiovascular mortality (81 versus 63 deaths; unadjusted HR 0.72 (0.52–1.00); p=0.05).

Conclusions: In SHIFT, patients who were not prescribed beta-blockers had higher heart rates and higher mortality than those prescribed beta-blockers and tended to have a greater benefit from ivabradine. This retrospective post-hoc analysis is consistent with the hypothesis that patients with HF in sinus rhythm would receive a beta-blocker whenever possible but, when this is not the case, that ivabradine may reduce mortality in the absence of a beta-blocker. The strikingly similar point-estimate for the effect of beta-blockers and ivabradine “monotherapy” compared to placebo on all-cause mortality for patients with HF with an ejection fraction <30% should be further explored.