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to explain the rise in Cr and \geq 10% rise in serum cystatin levels from baseline at 24 h. Cr will be assessed at baseline and at 24, 48 and 72 h and CyC will be measured at baseline and at 24 h.

Results: After 48 hrs of CM exposure, Cr increase $\geq 0.5 \text{ mg/dL}$ occurred in 29 patients (11.3%) whereas increase in CyC \geq 10% at 24 hrs after CM exposure occurred in 66 patients (26.08%). The difference in mean Cr and baseline, 24 hrs and 48 hrs after CM exposure was not statistically significant whereas that between CyC at baseline and 24 h after CM exposure was significant. CyC detected CIN 24 hrs earlier as compared to Cr. Similarly the difference between GFR calculated by Cr equation was not statistically significant between pre and post CM exposure but the difference was significant when GFR was calculated using the combined equation. The risk of CIN was higher in patients with pre-existing CKD, diabetes, advanced age, haemodynamic instability, heart failure (LVEF < 40%), patients with ACS and higher contrast volumes.

Conclusions: The assessment of CyC at 24 hours after CM exposure allows an early diagnosis of CIN. Also CyC equation and the combined equation (Cr + CyC) for measurement of GFR accurately identified the patients with CIN even at 24 hrs after CM exposure, in contrast to Creatinine which took 48-72 hrs. Hence the management strategies for CIN can be started earlier by 24-48 hrs when CyC is used as a marker for CIN which would improve long term outcomes.

Pulmonary embolism – Single centre registry data

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Background: Pulmonary embolism (PE) is one of the major cause of cardiovascular mortality. High index of suspicion is necessary for diagnosis. PE likelihood scoring is a useful clinical tool for risk stratification and management. This was a prospective registry to study the clinical features, clinical likelihood scoring, risk stratification and management of PE.

Methods: Consecutive cases hospitalized over a period of 3 years were studied. Revised Geneva score was applied to all cases of suspected pulmonary embolism. Most cases had PE confirmed by CT pulmonary angiography. Risk stratification (Pulmonary Embolism Severity Index from 2015) was used to guide the treatment strategy.

Results: There were 44 patients of pulmonary embolism, 26 being male. The mean age for male patients was 43.8 years and for females 55.4 years. Among 44 patients, 11 (25.0%) patients had surgery or immobilisation in the past 30 days, 15 (34.09%) patients had DVT. 4 (9.09%) patients had diabetes, 3 (6.81%) had hypertension. 6 (13.6%) patients were smokers. 37 (84.0%) patients had breathlessness. ECG revealed sinus tachycardia in 34 (77.2%). Echocardiogram showed dilated right atrium (RA) & right ventricle (RV) in 32 (72.7%) patients and RV dysfunction in 10 (22.7%) patients. The mean modified Geneva score was 8.18 for males and 9.7 for female. CT pulmonary angiography was done in 33 patients which confirmed PE in 32 cases. Risk stratification according to expected pulmonary embolism-related early mortality rate stratified 11 (25.0%) patients in high risk group, 27 (61.3%) patients in intermediate risk group & 6 (13.6%) patients in low risk group. 27 (61.3%) patients were thrombolysed with tenecteplase & 3 (6.8%) with reteplase. 1 patient had emergency surgical thrombo-endartectomy, recovering fully after an eventful post-operative period. Posttreatment most patients showed improvement.

Conclusion: Modified Geneva score along with imaging investigations helped in establishing diagnosis. Thrombolytic therapy in high risk cases and selected intermediate risk cases was safe and effective.

Assessment of short term effects of sildenafil therapy in patients with secondary pulmonary hypertension



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Background: It is well proven that sildenafil improves pulmonary hemodynamics and exercise capacity in patients with primary pulmonary hypertension. However, the drug armamentarium for secondary pulmonary hypertension is limited. Sildenafil may also be helpful in this subgroup. Certain studies have shown promising results but none of the magnitude to promulgate new recommendations.

Methods: In this double-blind, placebo-controlled study, we randomly assigned 106 patients with symptomatic secondary PAH (idiopathic DCMP, heart failure with preserved EF, COPD, and other lung parenchymal disease, valvular heart disease) to placebo or sildenafil (53 in each group). Sildenafil was given orally 25 mg TID for 6 weeks. The primary end point was the change from baseline to week 6 in the distance walked in 6 minutes. We also assessed clinical improvement (improvement in 6 minute walk test, and NYHA functional class, change in Borg dyspnoea index) and change in hemodynamic parameters (PASP, LVEF).

Results: Of the 106 patients, included secondary PAH was due to COPD in 21 (19.8%), valvular heart disease in 53 (50%), heart failure with preserved EF in 16 (15%), idiopathic DCMP in 11 (10.2%) and other lung parenchymal diseases in 5 (5%). The mean increase in the distance walked after 6 weeks of therapy was 54 min sildenafil group and 13 m in placebo group p = 0.04. In the sildenafil group significantly greater number of patients improved by at least one functional class (23% vs 11%, p = 0.003). The mean NYHA class at 6 weeks was 2.0 ± 0.2 in the sildenafil group versus 2.8 ± 0.4 in the placebo group, p = 0.02. The mean PASP significantly decreased in the sildenafil group at 6 weeks (48 ± 6 mmHg), compared to placebo (58 ± 6 mmHg), p = 0.02. LVEF was higher in the sildenafil group, $60 \pm 10\%$ versus $55 \pm 10\%$ in the placebo group, but did not reach statistically significant difference.

Conclusion: Sildenafil improves exercise capacity, functional class, and hemodynamics in patients with PAH. PDE-5 inhibition may represent an important therapy for patients with secondary PAHif, the benefits observed in our study are confirmed in larger clinical trials.

Use of dabigatran versus warfarin in patients of atrial fibrillation at Asian heart hospital – An institutional based study



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Background: Atrial fibrillation increases the risks of stroke and death. Although warfarin reduces the risks of stroke and death, it increases the risk of hemorrhage along with its other drawbacks.