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MOG1, a novel therapeutic agent for cardiac traffickopathies

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Despite tremendous improvements in its prevention and treatment, cardiac-arrhythmias are the major cause of morbidity and mortality worldwide accounting for >1.2 million hospitalizations and 400,000 sudden-deaths each year in the United States. The cardiac voltage gated sodium channel, Nav1.5, is crucial for maintaining normal cardiac rhythm. Loss-of-function mutations in the Nav1.5-encoding gene SCN5A cause a vast array of cardiac disorders including Brugada syndrome (BrS), sick sinus syndrome (SSS), dilated cardiomyopathy (DCM), cardiac conduction disease (CCD) and atrial fibrillation (AF). However, no effective treatment is available for these syndromes, except for invasive implantation of defibrillators or pacemakers in some cases. Defects in cell-surface-trafficking of ion-channels have been demonstrated to be a unique molecular mechanism underlying a variety of arrhythmic disorders. As *SCN5A*-mutations causing BrS, DCM and SSS act by loss-of-function; agents increasing cardiac-sodium-current (I_{Na}) may aid towards safe, effective treatment. In 2008, we reported that a 20 kD protein MOG1 is a novel cofactor modulating Nav1.5-function. In 2013, we demonstrated the therapeutic potential of MOG1 *in vitro* to rescue the Nav1.5-trafficking-defects and reduced INa associated with BrS, DCM and SSS. We are currently evaluating the therapeutic potential of MOG1 *in vivo*. Reduced I_{Na} has also been associated with inherited cardiovascular disorders like myocardial infarctions/ ischemia and heart-failure. Thus, the MOG1-therapy to facilitate membrane-trafficking of Nav1.5 may be utilized for the trafficking-defective subset of cardiac channelopathies.

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A comparison of rescue and primary percutaneous coronary interventions for acute ST elevation myocardial infarction

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Background: To perform a comparative analysis of in-hospital results obtained from patients with acute ST elevation myocardial infarction (STEMI), who underwent rescue or primary percutaneous coronary intervention (PCI). The aim is to determine rescue PCI as a practical option for patients with no immediate access to primary PCI.

Methods: From Cardiology PCI Clinic of the National Hospital of Sri Lanka (NHSL), we selected all consecutive patients who underwent early percutaneous coronary intervention for acute STEMI presenting with ≤ 24 h door-to-balloon delay for primary PCI and ≤ 72 h door-to-balloon delay, (90 minutes after failed thrombolysis) for rescue PCI from March 2013 to April 2015 and their in-hospital results were analyzed, comparing rescue and primary PCI patients.

Results: We evaluated 159 patients, of which 78 underwent rescue PCI and 81 underwent primary PCI. The culprit left anterior descending (LAD) vessel (76.9% vs. 58.8%; P=0.015) was more prevalent in rescue than in primary patients. Thrombus aspiration was less frequent in rescue group (19.2% vs. 40.7%; P=0.003). The degree of moderate-to-severe left ventricular dysfunction reflected by the ejection fraction <40% (24.3% vs. 23.7%; P=0.927) and prevalence of multivessel disease (41.0% vs. 43.8%; P=0.729) revealed no significant difference. Coronary stents were implanted at similar rates in both strategies (96.2% vs. 92.6%; P=0.331). Procedural success (97.4% vs. 97.5%; P=0.980) and mortality rates (5.1% vs. 3.8%; P=0.674), were similar in the rescue and primary groups.

Conclusion: In-hospital major adverse cardiac events (MACE) are similar in both rescue and primary coronary intervention groups, supporting the former as a practical option for patients with no immediate access to PCI facilities

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