

nel through the Na channel's supposed calcium sensitivity. While there are proponents for both sides, there has been relatively little research that provides strong evidence for either case. In this experiment, the effect of calmodulin on Nav 1.5 is tested by preparing a set of cardiac cells (of the human specie) with the Nav 1.5 C-Termini and CaM protein, which were then to be placed in solutions with varying concentrations of calcium. We took special care to test multiple concentrations of calcium, as previous studies have tested very low concentrations, with Manu Ben-Johny's team from the John Hopkins laboratory in particular testing up to a meager 50 micromolar, despite producing a well-respected paper (By comparison, the average Na channel can naturally sustain a concentration of almost 1-2 millimolar and on some occasions, reaching even higher concentrations). After using light scattering and observing the signals given off by the calcium interacting with these Nav1.5/CaM complexes across the varying calcium concentrations, the overall pattern indicated that there was a one to one stoichiometry between calmodulin and Nav 1.5. More importantly, it indicated calcium sensitivity of the Na channel. With this research, a definitive answer has been drawn regarding the importance of calmodulin in calcium modulation in Na channels. Not only does this have the effect of creating a foundation for further research into the structure and function of Na channels, but it also gives deep insight into fundamental functions of the channel that can play a major role into the creation of drugs to treat the many cardiac diseases associated with dysfunction of the channel.

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## Chronic and acute ischemic heart disease

### BIOMARKERS

#### 44. Copeptin as early marker of acute non-ST elevation myocardial infarction in patients suspected with acute coronary syndrome

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Rapid diagnosis and management of AMI have great impact on morbidity and mortality. Diagnosis which is based on elevation of cardiac biomarkers has its limitations. Copeptin is the C-terminal part of the vasopressin prohormone. The pathophysiology mode of release should theoretically add diagnostic information of cardiac cell necrosis. One of the major limitations of cardiac biomarkers is the delayed release in circulation. So looking for a new marker with a short diagnostic time window is needed. Aim is to determine the role of copeptin as an early marker for acute non-ST elevation MI (NSTEMI).

This study included 88 patients with chest pain. They were divided into 2 groups. Group (1); included 30 patients with diagnosis of NSTEMI. Diagnosis of AMI was established according to the universal definition of MI. Group (2); included 58 patients with diagnosis of unstable angina (UA). Full medical history, physical examination, 12 lead ECG, random blood glucose level, renal function, total cholesterol, triglyceride, cardiac troponin I and Copeptin were obtained on admission. Follow up cardiac troponin I was done. Inclusion criteria: Defined as chest pain of  $\leq 6$  h duration since onset, suggestive of myocardial ischemia, and lasting  $>20$  min. at rest. Exclusion criteria: Patients with positive First cardiac troponin were rolled out, patients with ST segment elevation were rolled out. Other exclusion criteria: Patients presenting after a cardiac arrest, Trauma or major surgery within the last 4 week; pregnancy; IV drug abuse; age less than 18 years; shock and sepsis. Patients who were included had second troponin I re- done and copeptin analysis done. In group 1 (NSTEMI) 28 patients had ECG changes and only 2 had NSTEMI without ECG changes. In group 2 (UA) 23 patients had ECG changes and 35 patients had normal ECG. Males and females were 49 and 39. Age in G1 and G2 was  $60 \pm 4$  and  $53 \pm 5$ . Copeptin analysis was done 6 h after Infarction or chest pain. All the patients with NSTEMI (30) had positive copeptin and positive troponin except one only who had + troponin only and another one who had + copeptin only. Of the 58 patients without MI none had the two tests positive, only one had + troponin and one had + copeptin. Using ROC curve: copeptin had sensitivity 100% and specificity 82.8% with using cut off point 13.2 pmol/l. So copeptin can be used for early detection of myocardial infarction. Copeptin seems to be an ideal confirmatory marker for rapid rule out of AMI. If the two tests (with troponin) are positive, this is evident MI; if the two are negative it rules out MI.

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## Chronic ischemic heart disease

#### 45. Ezetimibe and statins yields on silent holter ambulatory myocardial ischemia

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Further cholesterol lowering may affect silent ischemia detected on holter monitoring. Cholesterol lowering is associated with a reduction in cardiovascular morbidity and mortality. Statins are the main drugs for cholesterol lowering. Ezetimibe when added to statins gives further reduction in cholesterol but its long-term effect on cardiovascular morbidity and mortality and ischemic events is not known. This study sought to determine whether further cholesterol lowering with ezetimibe will also results in a reduction of myocardial ischemia during daily