# NCCU JOURNAL CLUB: EARLY TROPONIN I IN CRITICAL ILLNESS

## Early troponin I in critical illness and its association with hospital mortality: a cohort study.

## Or…. is routine troponin measurement a good idea?

## Presented by Dr Odysseas Papazachariadis

Elevated troponin is a biomarker of myocardial distress, frequently seen in ICU patients and has been shown to be associated with poor outcome.

This study hypothesised that routine troponin measurement would help classify ICU patients and better assess risk, or put another way, is there is an independent relationship between early Troponin I (TnI) and hospital mortality and is routine early blood TnI measurement of any prognostic use?

They tried to answer this question by analysing retrospectively collected data obtained from two ICUs, Glasgow Royal Infirmary (GRI) and St. Thomas' Hospital (STH), London. It is important to note that they do not claim to add to the understanding of the pathophysiology of TnI increase in ICU patients.

The authors followed the study population from the first day of admission to 6 months later. TnI, APACHE II, and demographic data was collected in the first 24h, and mortality over the subsequent 6 months. Data were then fitted in a regression model with TnI and APACHE II score as input variables, and mortality as outcome. Finally, they correlated TnI to APACHE II and demographic subsets – age, chronic disease, presenting pathology, acute physiology score, and additional diagnoses.

Results were encouraging in the sense that reliable predictions can be made based on TnI levels at admission. However, no benefit of adding TnI data to the preexisting APACHE II data was found in any of the groups they examined. TnI levels correlated to individual APACHE II subsets, mainly the APS.

Perhaps inevitably, the study has some methodological flaws, although this should not take away from an interesting hypothesis generating study, it is almost impossible to undertake research of this nature without compromises. In the main, this study is confounded by selection bias, the authors

were not in position to actively impose a protocol. One of the most interesting areas of discussion will be the study populations, particularly within the GRI subset, of 3073 patients admitted, 43.9% had troponin levels measured and 56.1% did not. There were significant differences between these populations (patients who did not have TnI taken were younger, were more likely to have an elective admission and had lower APACHE II scores). Within the TnI group, 55.3% had TnI measured routinely while 44.7% at clinical discretion, again creating two potentially different subpopulations. One tempting criticism is that the study focused on selected patients that had a greater probability of a TnI increase and poorer prognosis, but independent of their TnI level. There are other differences between the groups and these will be discussed at journal club.

The key issues to discuss at journal club are:

> Should this study influence what we do, why?
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> Does troponin leak matter?
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> Where does the balance lie in neurocritical care between treatments which exacerbate troponin leak but also improve cerebral blood flow?

**Open Access Resources**

Here is a good, recent review by our friends at Papworth.

BioMedCentral Critical Care – The role of cardiac troponin I as a prognosticator in critically ill medical patients.

St Emlyn's – Why hyper-troponin-aemia does not always equal acute myocardial infarction.

The Role of Cardiac Troponin I in Prognostication of Patients with Isolated Severe Traumatic Brain Injury

Nice Deranged Physiology blog post.