7TH CARDIAC MARKERS DIALOGUES MEETING, 24–25 SEPTEMBER 2026, GLASGOW · POSTER MP03

Characterization of cardiac troponin fragments present in the blood of patients in early and late stages of acute myocardial infarction

Natalia S. Riabkova[1,2], Ivan A. Katrukha[1,2,*], Alexander E. Kogan[1,2], Amina K. Alieva[3], Dmitry V. Pevzner[3], Anastasia V. Bereznikova[1,2] and Alexey G. Katrukha[1,2]

1 - Hytest Ltd., Turku, Finland; 2 - Department of Biochemistry, Faculty of Biology, Moscow State University, Moscow, Russia; 3 - National Medical Research Centre of Cardiology named after academician E.I. Chazov, Moscow, Russia.

INTRODUCTION

Cardiac troponins I (cTnI) and T (cTnT) are the key biomarkers for the diagnosis of acute myocardial infarction (AMI). It was demonstrated that the kinetics of cTnI and cTnT in blood after AMI differs [1]. cTnI is released predominantly as a single peak, reaching its maximum 24 hours after the onset of symptoms. In contrast, the kinetics of cTnT are biphasic: the first cTnT peak coincides with that of cTnI, whereas the second increase in cTnT concentrations is observed approximately 80 hours after the onset of chest pain. We previously showed that the composition of blood troponin forms changes during the first 30 hours after AMI onset [2]. However, the troponin forms present in the blood 80 hours after AMI remained uninvestigated. In the current study, we compared the composition of fragments of cTnI and cTnT present in the blood of patients at early (~14 h) and late (~80 h) stages of AMI.

MATERIALS AND METHODS

Heparin plasma samples were collected at 6-16 h and 71-89 h following the onset of AMI from 43 AMI patients who had blood flow restored by stenting; cTnT, cTnI, and their fragments were studied by Western blotting (WB), gel filtration (GF), and fluoroimmunoassays (FIA) using monoclonal antibodies specific to various portions of cTnT or cTnI.

RESULTS

Fluoroimmunoassay analysis

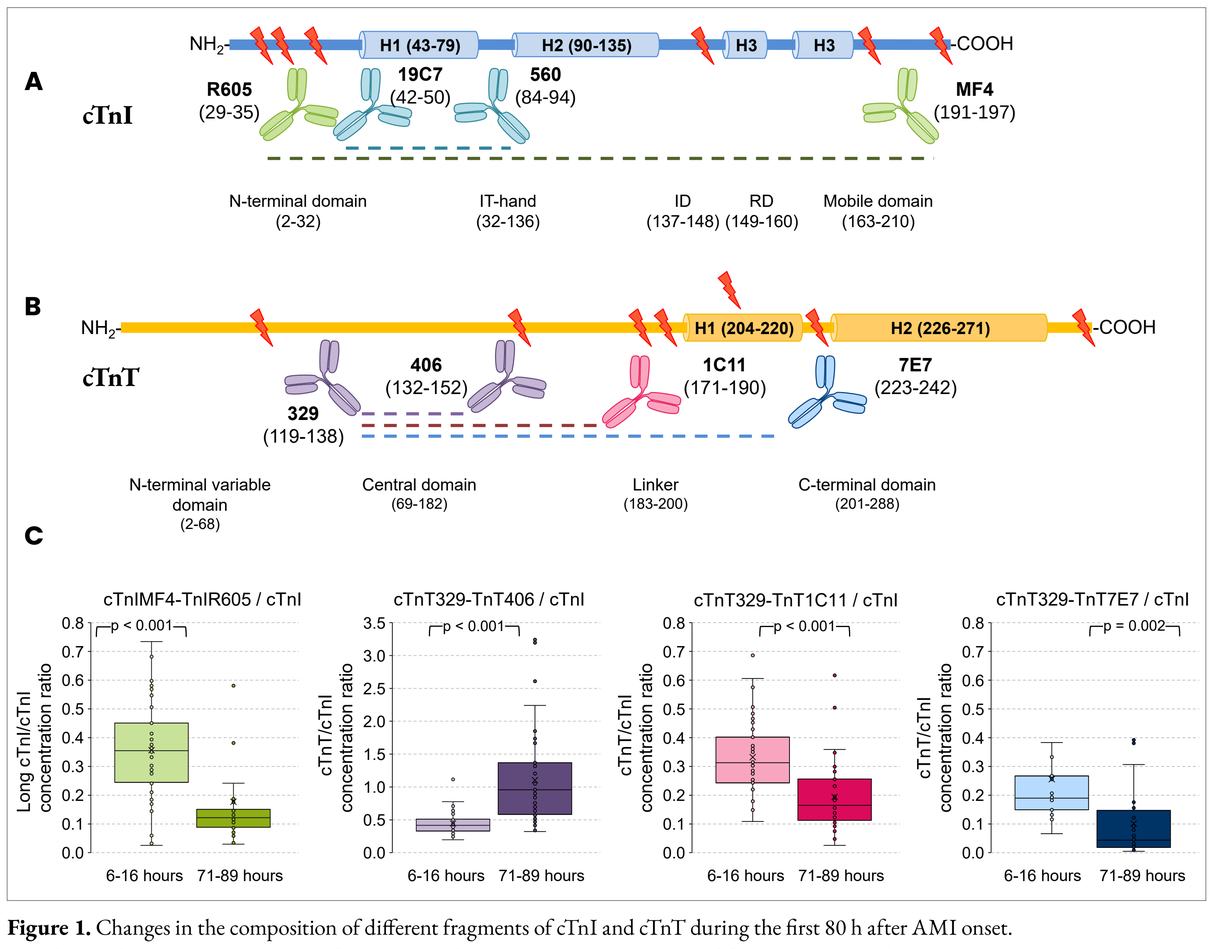

Figure 1. Changes in the composition of different fragments of cTnI and cTnT during the first 80 h after AMI onset.

A and B – Schematic representation of anti-cTnI and anti-cTnT mAb epitopes used for FIA, respectively.

C – Comparison of the ratios of cTnI and cTnT concentrations measured by assays specific to different regions of the cTnI or cTnT with the cTnI concentration measured by the TnI19C7-TnI560 assay. Only 20 out of 43 patients were studied by the TnT329-TnT7E7 assay.

Western blotting

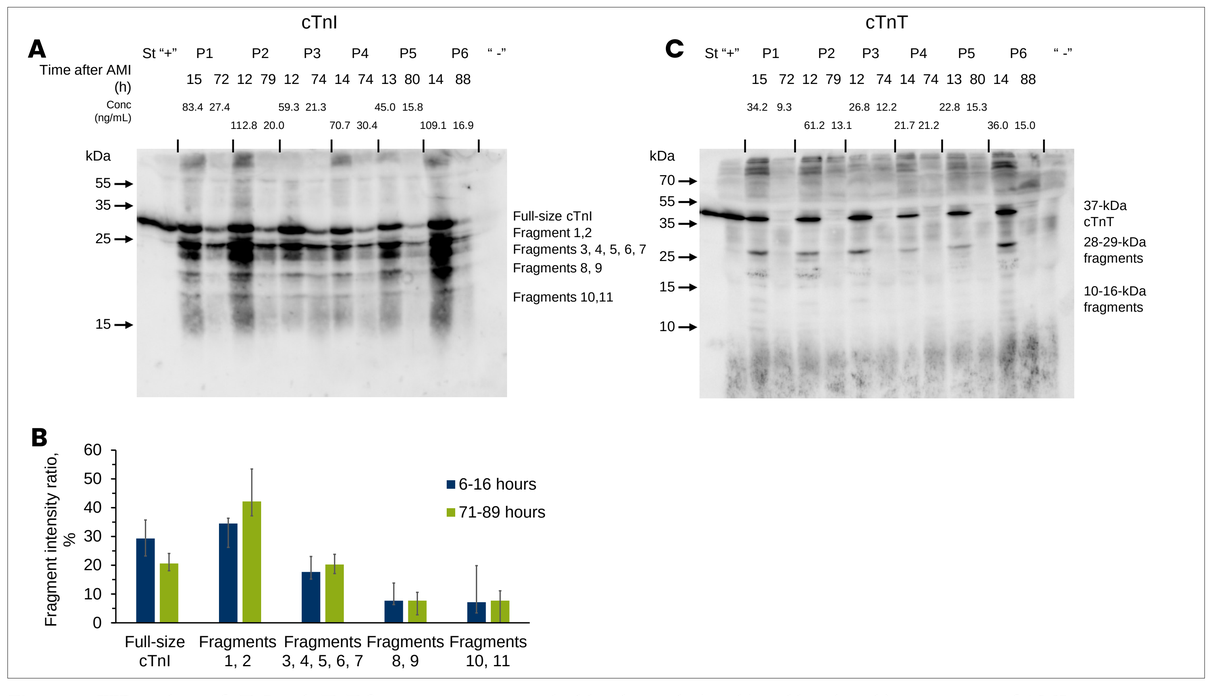

Figure 2. WB analysis of cTnI and cTnT fragments present in the blood at early (~10 h) and late (~80 h) time points of AMI.

A – A representative WB showing the composition of cTnI fragments extracted from the blood of patients at early (12-15 h) and late (72-88 h) stages of AMI. Immunostaining with a mixture of the mAbs TnI560 and TnI16A11.

B – Relative proportions of cTnI fragments in the blood of patients (n=16) at early (blue) and late (green) time points after the AMI onset. Median ± 25-75 quartiles.

C – A representative WB showing the composition of cTnT fragments extracted from the blood of patients at early and late time points of AMI. Immunostaining with a mixture of the mAbs TnT329, TnT406, and TnT7E7.

RESULTS

Gel filtration

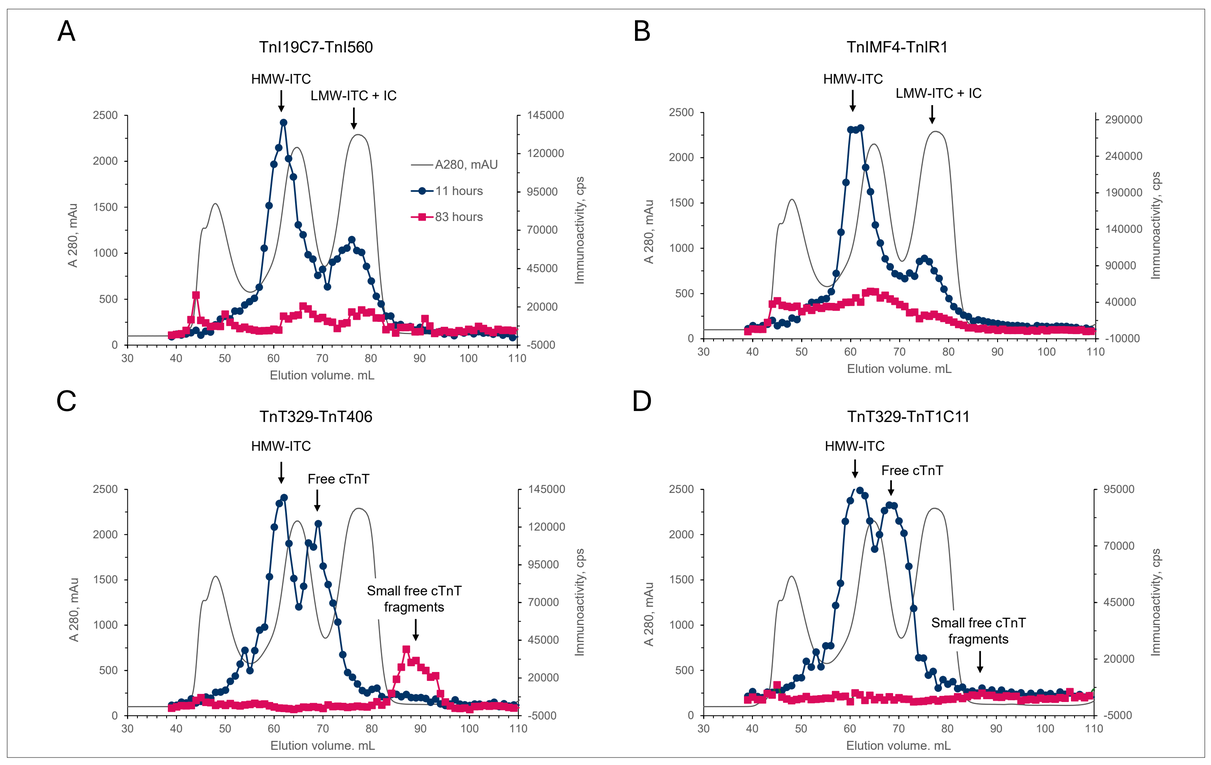

Figure 3. Gel filtration analysis of early and late blood samples of a representative AMI patient. Citrate plasma samples were collected 11 h (dark blue) and 83 h (magenta) after the AMI onset. Immunoactivity in the gel filtration fractions was measured using:

A – TnI19C7-TnI560 assay, initial cTnI concentrations were 85.0 and 15.1 ng/mL, respectively.

B – TnIMF4-TnIR1 assay, cTnI concentrations were 48.3 and 1.8 ng/mL.

C – TnT329-TnT406 assay, cTnT concentrations were 56.5 and 25.2 ng/mL.

D – TnT329-TnT1C11 assay, cTnT concentrations were 51.5 and 4.3 ng/mL.

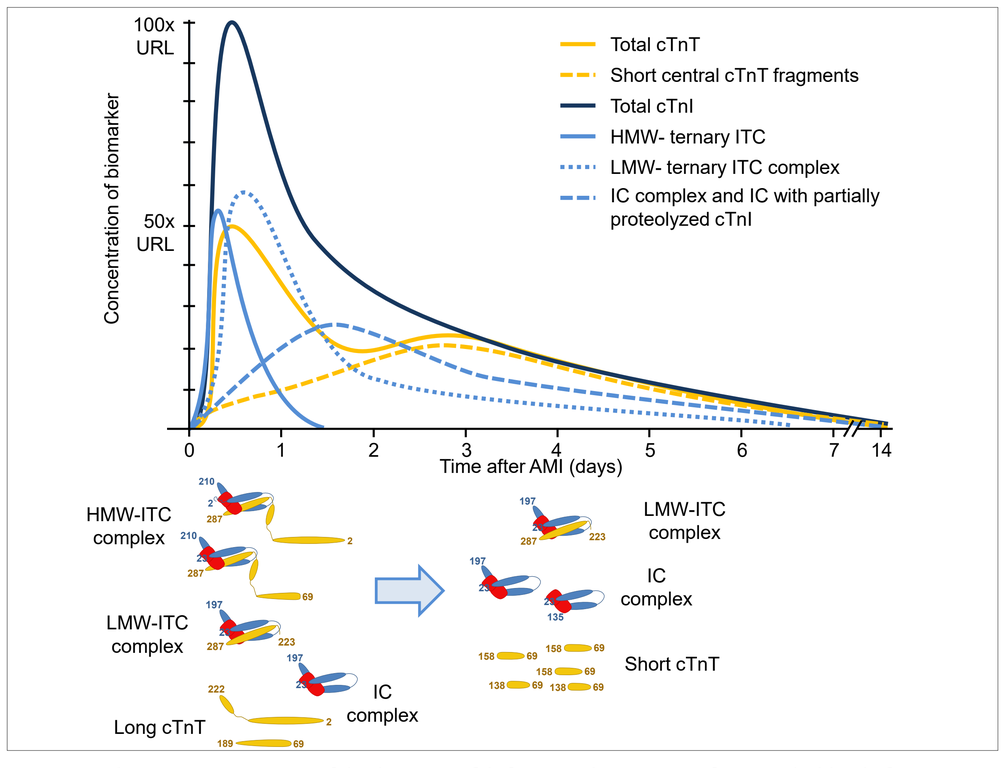

Figure 4. Schematic representation of the dynamics of different cardiac troponin forms in the blood of AMI patients after reperfusion.

CONCLUSIONS

The composition of cardiac troponin forms in blood changes over time after AMI: if at the early phase troponins were present as a mixture of full-size and truncated ternary ITC complexes, binary IC complex, and long free cTnT molecules, 80 hours after the AMI onset mostly binary IC complex and small free cTnT fragments were observed.

The dynamics of cTnI and cTnT proteolytic fragments in the blood of AMI patients differ. While at the late time point after AMI onset cTnI was present as a set of high-molecular weight and low-molecular weight fragments, cTnT was detected only as small peptides.

An increase in the cTnT/cTnI ratio observed 80 hours after the AMI onset indicates an excessive release of free small central fragments of cTnT into the blood flow which suggests the presence of various mechanisms of cardiac troponin release from the damaged myocardium.

REFERENCES

1. Laugaudin G, Kuster N, *et al.* Kinetics of high-sensitivity cardiac troponin T and I differ in patients with ST-segment elevation myocardial infarction treated by primary coronary intervention. Eur Heart J Acute Cardiovasc Care. 2016 Aug;5(4):354-63. doi: 10.1177/2048872615585518.

2. Katrukha IA, Riabkova NS, *et al.* Fragmentation of human cardiac troponin T after acute myocardial infarction. Clin Chim Acta. 2023;542:117281. doi: 10.1016/j.cca.2023.117281.