



## **The concentration of troponin I is increased in patients with acute-onset atrial fibrillation.**

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Some preliminary evidence shows that atrial fibrillation (AF) in patients admitted to the emergency department (ED) with clinical symptoms suggestive of myocardial ischemia is frequently associated with elevations of cardiospecific troponin I (TnI) [1].

Since definitive information about the frequency of troponin release during acute-onset AF (AAF) is lacking to the best of our knowledge, we carried out a retrospective observational study by retrieving data from the electronic database of our hospital about all ED visits for AAF (defined as onset of symptoms within 48 hours) recorded in the year 2013. According to currently available guidelines, the definition of AAF included both first-diagnosed AF and paroxysmal AF (PAF) [2]. The ED of the Academic Hospital of Parma is a large urban facility, with approximately 90.000 visits per year. The concentration of TnI was measured in all patients with the contemporary sensitive immunoassay Beckman Coulter AccuTnI, on Unicel DxI (Beckman Coulter, Brea, CA, USA) [3]. The concentration of cTnI was also assessed, during the same period, in a population of 125 ostensibly healthy blood donors (77 males and 48 females, age range 21-54 years), who had this biomarker measured for definition of the local 99<sup>th</sup> percentile of the upper reference limit (URL), as specified by the recommendations of the Clinical and Laboratory Standards Institute (CLSI) [4]. Results of TnI were finally expressed as median, interquartile range (IQR) and range (minimum and maximum values). The quality of TnI data was systematically validated throughout the study period by regular internal quality control (IQC) procedures and participation in an External Quality Assessment Scheme (EQAS). Differences between groups were assessed by Wilcoxon-Mann-Whitney test (for continuous variables) and  $\chi^2$  test with Yates' correction (for categorical variables), using Analyse-it (Analyse-it Software Ltd, Leeds, UK). The investigation was performed in accord with the Declaration of Helsinki and under the terms of all relevant local legislation.

The 99th percentile URL calculated from our local population of blood donors was 0.03  $\mu\text{g/L}$ . Overall, 474 cases of AAF were recorded in the ED in the year 2013. The median concentration of TnI was found to be significantly higher in patients with AAF (0.01  $\mu\text{g/L}$ ; IQR, <0.01 to 0.02  $\mu\text{g/L}$ ; range, <0.01 to 0.54  $\mu\text{g/L}$ ) than in controls (<0.01  $\mu\text{g/L}$ ; IQR, <0.01 to <0.01  $\mu\text{g/L}$ ; range, <0.01 to 0.03  $\mu\text{g/L}$ ;  $p < 0.001$ ) (Fig. 1). The frequency of values exceeding the 99<sup>th</sup> URL was also significantly greater in the group of patients with AAF (86/474, 18%) than in the control population (3/125, 2%;  $p < 0.001$ ).

The results of this study confirm the existence of an association between AAF and increased values of TnI, even when this biomarker is measured with a contemporary-sensitive immunoassay in a patient population with no symptoms of myocardial ischemia.

Due to a substantially increased risk of cardiovascular mortality and morbidity, AF requires an early and appropriate management. AAF represents a frequent reason for ED evaluation and the appropriate management strategy (e.g., rate-control versus rhythm control, antithrombotic therapy) should hence be based on timely risk stratification of stroke, bleeding and cardiovascular morbidity.

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Along with conventional risk assessment tools such as CHA<sub>2</sub>DS<sub>2</sub>-VASc and HAS-BLED scores, cardiospecific troponins may hence provide an important complementary information about the thromboembolic risk and the prognosis of patients with AF [6]. Interestingly, an increased troponin value in these patients does not invariably mirrors the presence of relevant coronary artery stenosis, but it is most commonly associated with tachycardia per se, thus reflecting a cardiac injury (e.g., myocyte necrosis and/or fibrosis) which may be directly attributed to AF [7].

## References

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