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Angiography related increases of high sensitivity cardiac troponin T among patients undergoing an elective coronary angiography without coronary intervention

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The high sensitivity assays of cardiac troponin are regarded as the biochemical reference standard for the diagnosis of myocardial infarction. The higher analytical sensitivity of those novel tests allows detection of hitherto undetectable troponin levels in the absence of myocardial infarction in apparently healthy population. Hence, it is currently feasible to measure minor cTn rising as in intermittent ischemia without apparent myocardial infarction. However, the diagnostic specificity decreases as the analytical sensitivity increases. Therefore, it is essential to differentiate between acute and chronic myocardial damage conditions, thus, cardiac troponin results should not be interpreted in isolation but within the extent clinical context and on the basis of kinetic changes observed in serial measurements.

Coronary intervention related myocardial injury is designated as myocardial infarction type 4a, but there are no data addressing troponin releases after an uneventful coronary angiography.

The present study investigated whether diagnostic coronary angiography is followed by a release of cardiac troponin T as measured by a high sensitivity troponin assay (hs-cTnT) and to study potential pathomechanisms for myocardial injury following coronary angiography in the absence of a coronary intervention. This approach reveals significant increases of hs-cTnT within the study population after undergoing an elective diagnostic coronary angiography (CAG) for suspected stable CAD, while absolute and relative troponin kinetic changes (δ) are fairly comparable within study settings. This could be explained through a micro-myocardial injury due to limited ischemia induced by the examination, whereas possible peripheral embolization of platelet microaggregates or intracoronary thrombus cannot be excluded. Further, rheological, hemodynamic and chemical properties or thrombogenic potential of the injected contrast medium may induce myocardial microcirculatory disturbance or even limited myocardial toxicity.

Moreover, this data suggest hs-cTnT increases are associated with the severity of the examination rather than the complexity of the coronary artery disease. Several independent potential determinants of a postprocedural hs-cTnT increase were identified including age, left ventricular impairment, consumption of contrast agent and fluoroscopy time.

This approach underlines the extreme sensitivity of the hs-cTnT assay as an indicator of myocardial injury rather than myocardial infarction due to myocardial ischemia. The use of this assay may allow identification of myocardial injury during diagnostic coronary angiography reflecting the severity of the procedure.