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The role of N-acetylcysteine in the prevention of peri-procedural myocardial infarction after percutaneous coronary revascularization

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Background: The use of N-acetylcysteine (NAC) has been associated with lower acute renal failure after primary percutaneous coronary intervention (PCI) and lower in-hospital mortality. The utility of NAC in the prevention of contrast-induced nephropathy (CIN) has been questioned by recent studies. Since, NAC also has known benefits on microvasculatureand protective agent against reperfusion injury in patients having myocardial infarction (MI), we sought to determine whether NAC would reduce incidence of peri-procedural MI in patients undergoing PCI or reduce infarct size in patients undergoing PCI for MI.

Methods: We retrospectively analyzed 5,034 patients who underwent PCI at St. Mary's Hospital; Rochester, MN Patients were grouped into those who receiving NAC pre-PCI vs. no NAC pre-PCI and then further stratified by presence of MI or no MI at time of procedure. Patients with cardiogenic shock or ST-Elevation myocardial infarction who immediately went to PCI were excluded. The primary endpoint was the rise in cardiac biomarkers after PCI, stratified by presence or absence of MI.

Results: Of the patients analyzed 3,162 patients were without MI and 1,872 patients with MI. Across both groups, patients receiving NAC were older, more likely to have diabetes, and have hypertension. In those patients without MI patients who received NAC had a slightly higher baseline troponin (0.1 vs 0.0, P<0.001). Post-PCI, the maximum troponin remained higher in the NAC group (0.2 vs 0.1, P<0.001) without and the difference in the CK-MB (7.7 vs 7.2, P=0.51). There was no difference in in-hospital myocardial infarction (4.4 vs 4.1%) In the MI patients there was no difference in the baseline troponin and CK-MB. Post-PCI, the max troponin was lower in those who received NAC, 2.1 vs 3.2 (P<0.001), and max CK-MB was lower 31.1 vs 103.2 (P<0.001). In those with MI, NAC use was not associated with a difference in in-hospital death (4.4 vs 4.3%).

Conclusion: In patients without an MI NAC does not reduce post-PCI procedural myocardial infarction. However, NAC reduced infarct size in MI patients who undergo PCI. Further studies are needed to clarify the role of NAC in the MI patient focusing on myocardial preservation.

Biography

Sara Negrotto is a first year Internal Medicine resident at Mayo Clinic in Rochester, MN. She grew up in East Tennessee and completed her medical degree at East Tennessee State University in 2013. She is interested in a career in Interventional Cardiology.

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