[C-18] Assessment of Aspirin Induced Platelet Inhibition by Arachadonic Acid Induced Thromboelastography Platelet Mapping

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Introduction

The use of aspirin has been extensively studied in adults but remains relatively untested in the pediatric population. With the increasing use of prosthetic materials in children undergoing repair of congenital heart defects, more children are at risk of a potentially devastating thromboembolic event. In an effort to prevent these events, more children are being treated with aspirin following cardiac surgery. To date there have been a handful of studies in children describing varying degrees of platelet inhibition utilizing different testing modalities such as PFA-100, urinary 11dhTxB2, platelet aggregometry and others[1] [2]. Another test that may provide some insight to platelet inhibition is thromboelastography (TEG). One study[3] has demonstrated the reliability of Arachadonic Acid (AA) induced TEG platelet mapping in an adult population but no one, to our knowledge, has studied it in a pediatric population.

Methods

A retrospective chart review is being performed on approximately 150 pediatric patients presenting for cardiac surgery between May 2007 and November 2013. Patient height, weight, BSA, percentage of platelet inhibition and aspirin dose are being collected. The primary outcome will be percentage of platelet inhibition versus weight based dose of aspirin.

Results

Preliminary data analysis was performed on the 97 complete sets of patient data available at this time. The aspirin dose was plotted two different ways (mg/kg and mg/m^2) versus percentage of platelet inhibition. The Pearson Correlation Coefficient (PCC) was calculated for both of these plots to quantify the relationship between aspirin dose and platelet inhibition. The correlation coefficients were 0.00454 and -0.00142 for these plots respectively. The PCCs of 0.00454 and -0.00142 indicate that there was no relationship between the aspirin dose (mg/kg or mg/m^2) and the amount of observed platelet inhibition as measured by TEG platelet mapping.

Discussion

There are many clinically important reasons why the results of this investigation might show no relationship between aspirin dose and platelet inhibition. First, there may be rampant noncompliance among outpatients presenting for cardiac surgery. This should be recognized as it would present a large barrier to quality long-term care. Secondly, although TEG platelet mapping was validated in one adult study, it may not be an accurate test in a pediatric population. It should be noted that there is no baseline TEG for each patient before they were started on aspirin. It is also possible that there may be some unrecognized confounding factor that skewed the data. Regardless, this retrospective study points out that a single measurement with TEG platelet mapping may not be useful in verifying effective aspirin therapy in children at risk for thromboembolic events following cardiac surgery.

References

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