Title: Hyperglycemia is not a risk factor for post-spine fusion infection in cerebral palsy patients

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Introduction: Despite improvements in surgical technique and antibiotic therapy, reported rates of infection following surgery vary from 3.4-9.7% [1] and as high as 14% in patients with cerebral palsy [2]. In a previous study, we identified GERD and histamine blockers as risk factors for post-spine fusion infection [3]. Several observational studies have also found correlations between hyperglycemia in children and poorer outcomes such as increased mortality, length of stay, as well as infection. However, these studies have been in previously healthy children with head trauma [4], in heterogeneous PICU populations where underlying diagnoses are not specified [5], or in a group of cardiac surgery patients less than 1 year of age with increased morbidity associated with congenital heart disease [6]. To our knowledge, no prior studies have looked at hyperglycemia and infections following spine fusion in patients with cerebral palsy. We sought to determine if hyperglycemia was an added risk factor in this pediatric surgical population with similar pre-operative co-morbidities.

Methods: Following institutional IRB approval, patients that developed infection following spinal fusion from January 1998-July 2008 (N=30) were identified by our Infection Control Officer. From our spine database of 665 pediatric patients we selected 37 patients who did not develop infection, but had similar co-morbidities. We compared age, weight, sex, spine curve, intraoperative blood loss, and administration of packed red blood cells between groups. We then determined glucose level at 4 time points selected based on prior published studies: preoperative, first intraoperative, admission to PICU or floor, and maximum glucose level within 7 days of surgery. Statistical analysis was performed using Mann – Whitney U at the 4 time points while glucose levels were compared within groups using one-way ANOVA (GraphPad Prism Version 4).

Results: There were no statistical differences in age, weight, sex, spine curvature, intraoperative blood loss, and administration of red blood cells between groups. There was no difference in preop glucose (p=0.91), intraop glucose (p=0.11), admission glucose (p=0.72), or 7day max glucose (p=0.92) between groups (fig. 1). There was a significant glucose difference within groups at the 4 time points for both non-infected (p<0.0001) and infected patients (p<0.0001) (fig. 2).

Conclusion: We demonstrate no difference in glucose levels between high risk cerebral palsy patients with similar comorbidities with and without infection following spinal fusion. However, both groups develop relative hyperglycemia following surgery. The role of glucose homeostasis in pediatric patients is not well understood. Our results suggest that the mechanism of hyperglycemia in certain high risk pediatric patients may be different from that of adults and may be a normal response.

References:

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