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Prophylactic Dexamethasone in Paediatric Open Heart Surgery Ptx3 Blood Pentraxin Elevations: Potential Clinical Significance

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Abstract

Administration of glucocorticoids before Cardio Pulmonary Bypass (PCB) may reduce the systemic inflammatory response and improve clinical outcomes. Pentraxin long PTX3 is a novel inflammatory parameter that may play a cardioprotective role by modulating inflammation. 29 children undergoing open heart surgery were enrolled in the study. Fourteen children received dexamethasone (first dose 1.5 mg/kg IV or IM one day before surgery; second dose 1.5 mg/kg IV before initiating bypass surgery) and fifteen children were used as controls. Blood PTX3, short C Reactive Protein (CRP), Interleukin-1 Receptor II (IL-1RII), fibrinogen, and Partial Thromboplastin Time (PTT) were measured at different time points. PTX3 levels were significantly increased during CPB in both Dexamethasone (+D) and Dexamethasone (-D)-treated and Non-Dexamethasone (-D)-treated subjects, but were significantly higher in +D than in -D patients. . CRP levels were significantly increased in both +D and -D patients in the postoperative days, with values significantly higher in -D patients than in +D patients. Fibrinogen and PTT values were significantly higher in -D patients than in +D patients on the first postoperative day.

Keywords: Gynaecological; Surgery; COVID-19; Global Screening

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Introduction

Plasma IL-1RII concentrations increased in the postoperative period in both groups. Prophylaxis with dexamethasone in pediatric patients undergoing CSCC for cardiac surgery is associated with a significant increase in blood PTX3, which may help reduce inflammatory parameters and improve patient clinical outcomes. Cardio Pulmonary Bypass (PCP) procedures during cardiac surgery produce systemic effects, especially in young children. Exposure of cellular and humoral blood components to biocompatible composites of extracorporeal vessels induces a Systemic Inflammatory Response (SIR) associated with leukocyte and endothelial cell activation and release of cytokines, leading to myocardial, kidney and lung dysfunction and has negative effects on the body clinical course after surgery [1, 2].

Discussion

Various anti-inflammatory strategies have been used to minimize CPP-associated organ dysfunction, including steroid prophylaxis with different usage histories. Although administration of Gluco Corticoids (GC) before CSCC reduces inflammatory responses, the benefit of glucocorticoids on clinical outcomes in adult and pediatric patients is controversial. Pentraxins are a superfamily of proteins that belong to the humoral arm of innate immunity and include the classic short pentraxins (C- Reactive Protein (CRP) and human and murine serum amyloid P components, respectively) and other pentraxins long. CRP, the prototype of the short family of pentraxins, is a human acute phase protein. It is produced in the liver in response to inflammatory signals, mainly IL-6, it interacts with various ligands, and it is involved in innate resistance to various pathogens [2, 3].

Long pentraxin PTX3 is a novel inflammatory marker, archetype of

the long pentraxin family, produced by innate immune cells and vascular cells in response to proinflammatory signals. PTX3 is a multifunctional protein and plays a complex and non-redundant role in vivo, recognizing a variety of pathogens, regulating complement activity, and facilitating pathogen recognition by macrophages and Dendritic Cells (DCs). Some evidence linking PTX3 and cardiovascular diseases: PTX3 production by smooth muscle cells is stimulated by atherogenic LDL, localization in atherosclerotic lesions, and high expression levels are observed in the heart during inflammatory responses. PTX3 levels increase rapidly in patients with Acute Myocardial Infarction (AMI), becoming the sole independent predictor of death. Furthermore, plasma PTX3 levels are elevated in patients with unstable angina and in stented patients, suggesting that PTX3 is a candidate to be a novel prognostic marker in ischemic heart disease. However, in addition to the role of PTX3 as a cardiovascular biomarker involved in the inflammatory response, recent in vivo and in vitro data suggest a cardioprotective role for PTX3 through a regulatory role in inflammation. In this regard, increased levels of PTX3 may reflect a host defense response [4].

Release of soluble cytokine receptors may be a mechanism against inflammatory responses. IL-1 is an important cytokine in inflammation and an important target of GC-mediated immunosuppressive activities. GC suppresses IL-1β production but increases cell surface expression of the IL-1(R) II receptor thereby increasing the release of the soluble form of the receptor itself. IL-1 RII has no signal transduction properties, acts as a "decoy" target for IL-1, binds with high affinity to IL-1 and blocks its binding to IL-1RI signalling. Here we analyzed the effect of dexamethasone prophylaxis in paediatric patients undergoing CSCC on blood levels of PTX3, IL-1RII and other inflammatory parameters [5].

29 children admitted to the Cardiovascular Surgery Unit of the G. Gaslini Institute were included in the study. Inclusion criteria were body weight < 10 kg and type of surgery (biventricular repair), excluding neonates, and all residual intra cardiac shunts that could preclude pulmonary oxygen exchange analysis. This study protocol was approved by the ethics committee of the G. Gaslini Institute in Genoa, Italy. Patients were randomized into two groups: (a) +D: 14 children received prophylactic dexamethasone; (b) -D: 15 controls. Patients in group +D received two doses of dexamethasone 1.5 mg/kg intravenously or intramuscularly. The first dose in the evening before the procedure, the second dose 30 minutes before the start of CPB. All patients in both groups were treated with the same regimen of anaesthesia and extracorporeal fluids. Blood samples were obtained preoperatively before steroid administration (T1), 10 min after initiation of CPB (T2), after removal of the aortic clamp (T3), at end of CPB (T4) and on day 1 (T5) and day 2 (T6) postoperative day. The method used to assign interventions to trial participants was a simple random assignment with an assignment sequence generated by an automated process (using SPSS software), with an assignment rate of 1:1. The two groups of patients were homogenous in terms of sex, age and weight [6, 7].

Conclusion

Clinical assessment of the patient included duration of SCC and aortic clamp removal, duration of mechanical ventilation, length of stay in the Intensive Care Unit (ICU), postoperative blood loss, and alveolar oxygen gradients arteries at T5. No associated complications were observed during the operative period. Blood samples were taken at the indicated times (see above) into tubes containing Ethylene Diamine Tetra Acetic Acid. Plasma was obtained by centrifugation of the sample and stored at -80˚C until use. Plasma PTX3 levels were measured by internal ELISA as previously described. The limit of detection was 100 pg/mL and the inter-assay variability was 8 to 10%. IL-1RII was assayed by sandwich ELISA using anti-IL-1RII 8.5 monoclonal antibody and anti-IL-1RII polyclonal antibody, both produced by some of us. The lowest detection limit of this test is 20 pg/mL. CRP was measured with an immune turbidimetric assay (Roche Diagnostic S.p.A Milano, Italy). Fibrinogen and PTT were measured using a photometric and turbidimetric detection system (BCS-XP, Siemens Healthcare Diagnostic, Deerfield, Ill, USA) descriptive analysis was first performed; Qualitative data are reported as frequencies and absolute percentages, and quantitative data are reported as mean values with the first and third quartiles (first through third quarters). The comparison of qualitative data between two groups of patients (treated and untreated) was performed using the chi-squared test. The comparison of quantitative data between the two groups of patients was performed using the Mann-Whitney U test [8].

The comparison of quantitative data at different time points was evaluated by non-parametric analysis of variance (Friedman test) and post hoc analysis was performed using the Wilcoxon test adjusted with Bonferroni correction. To assess the difference between quantitative data at each time point between the two groups of patients (treated versus untreated), the Bonferroni correction adjusted Mann-Whitney U test was applied use. In the data have been presented as medians with first and third quartiles. All statistical tests were two-sided and values less than 0.05 were considered statistically significant. Statistica software (StatSoft Co., Tulsa, OKla, USA) was used for all statistical analyses [9, 10].

Acknowledgement

None

Conflict of Interest

None

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