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Abstract

Neonatal infection remains the primary cause of death in extremely-low-birth-weight infants (ELBWIs). Alpha 1 acid glycoprotein (α1AG), an acute-phase protein, has been shown to be elevated in sporadic cases of septic ELBWIs prior to abnormal clinical signs. To delineate the roles of inflammation, delivery, and feeding in postnatal α1AG changes in ELBWIs, 75 ELBWIs of 26.5 ± 2.2 weeks of gestation born between May 2011 and August 2017 were retrospectively studied. The dependence of α1AG levels obtained on days 0–5 on the clinical variables was examined by incorporating interactions with age, followed by estimations of regression coefficients between clinical variables and α1AG levels at the early and late postnatal ages, defined by their standard deviation. Chorioamnionitis ($p < 0.001$), funisitis ($p = 0.045$), vaginal delivery ($p = 0.025$), enteral feeding ($p = 0.022$), and probiotics ($p = 0.005$) were associated with early α1AG elevations. Hypertensive disorder of pregnancy ($p < 0.001$) and gestational age ($p = 0.001$) were associated with late α1AG elevation; premature rupture of membranes ($p < 0.001$), funisitis ($p = 0.021$), body weight z-scores ($p < 0.001$), and enteral feeding ($p = 0.045$) were associated with late α1AG reduction. Postnatal α1AG changes in ELBWIs were associated with variables representative of age, growth, delivery, inflammation, and enteral feeding, potentially reflecting the process of sensitization to extrinsic microbes in utero, at birth, and thereafter.