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Innate immunity and susceptibility to infections and nec in crtically ill neonates: Is mbl in infections a pretty woman or an old witch?

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Genetic and/or developmental variations in the innate immune response could play a role in modulating the predisposition to severe infections and to ischemia reperfusion related pathologies, as NEC, in critically ill neonates with the same risk factors.

We prospectively investigated the association of mannose-binding lectin (MBL) serum levels with nosocomial sepsis (NS), their changes overtime during infection, the MBL2 genotype and their relationship with mortality in 365 critically ill neonates, 42 of them underwent major surgery. The median MBL serum concentration on admission, was significantly lower in infected than in uninfected neonates ($p<0.001$), without significant differences between not surgical and surgical neonates. Low MBL serum levels on admission independently increased the risk of infection. The median peak MBL level during infection was higher than the median level on admission ($p < 0.001$) and was correlated with it ($r^2 = 0.83$, $p<0.001$), also dividing the neonates into surgical and not surgical neonates. MBL levels on admission and peak levels during infections were not associated with death ($p=0.57$). We did not find significant difference in the frequencies of MBL2 genotypes between infected and uninfected neonates. Moreover, no association was found between MBL2 genotypes and death.

The pathogenesis of NEC is still unclear and the local pro-inflammatory response of the host could play an important role. To identify subsets of neonates at high risk of NEC is a challenge.

To evaluate the association between Mannose Binding Lectin (MBL) genotype, MBL serum level on admission to the Hospital and NEC in preterm infants we performed a retrospective study on 107 neonates (41 with NEC and 66 controls). MBL-2 genotyping was assessed by PCR and RFLP. The main outcome of the analysis was severe NEC (II-III Bell's stages).

MBL2 genotypes, related to high MBL expression, were more frequent in neonates with severe NEC than in controls. Neonates with NEC showed MBL level on admission > 400 ng/ml more frequently than controls ($p=0.043$) and among neonates with severe NEC, all deceased neonates were carriers of high or intermediate producing *MBL*-2 haplotypes ($p=0.035$).

MBL-2 genotypes associated to high MBL serum levels could represent a risk factor for NEC.

In conclusion Mannose Binding Lectin plays a crucial role in innate immunity against pathogens but we still don't fully understand the clinical value of the MBL response during infections and NEC.