201. THE ROLE OF THE INFLAMMATION IN PRETERM BIRTH

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Introduction. Preterm birth is the leading cause of neonatal morbidity and mortality. Although the underlying causes of pregnancy-associated complication are numerous, it is well established that infection and inflammation represent a highly significant risk factor in preterm birth. However, despite the clinical and public health significance, infectious agents, molecular trigger(s), and immune pathways underlying the pathogenesis of preterm birth remain underdefined and represent a major gap in knowledge.
Aim of the study. To carry out a systematic analysis of the data available in the current literature on the impact of inflammation on preterm birth
Materials and methods. Three electronic databases (PubMed, EMBASE and Web of Science) were searched for studies in any language reporting the use of multiplex assays for inflammatior associated with PTB published from January 2015 to March 2020.
Results. Inflammation and complex immunologic abnormalities, occurring in the absence of well-defined infectious triggers, have similarly been correlated with PTB. In addition to chronic inflammation, breakdown of the maternal/fetal tolerance, similar to an allograft rejection, can lead to adverse pregnancy outcome and PTB-evidence observed in chronic chorioamnionitis. Studies support the association between elevated levels of circulating proinflammatory cytokines and PTB, specifically, have implicated IL-1, TNF, and IL-6 as major players in the onset of PTB. Recently, IL-6 was identified as a critical marker of i.a. inflammation and a predictor of PTB; increased amniotic fluid IL-6 levels from the second trimester were associated with the timing and initiation of PTB. More than that infusion of IL-1 or TNF into the amniotic fluid leads to marked increases in i.a. proinflammatory cytokines levels or chorioamnionitis and as results ii lead to PTB.
Conclusions. Inflammation are major risk factors for PTB. However, the molecular triggers and mechanisms underlying the activation of immune pathways associated with induction of PTB remain poorly understood. Of note, adverse pregnancy outcomes have been well correlated with bacterial infections, including chorioamnionitis. However, the sequelae of asimptomatic infections remain poorly understood and warrant further investigations. However, the mechanisms underlying such processes remain underdefined. Additional medical screening of pregnant women for signs of infections and infection-associated immune mediators thus may lead to prevent PTB.
Key words: Inflammation, preterm birth, cytokines.

202. POST-CAESAREAN SEPTIC COMPLICATIONS

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