J Clin Cell Immunol 2014, 5:5
http://dx.doi.org/10.4172/2155-9899.S1.019

OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference and Exhibition on
Clinical & Cellular Immunology
September 29-October 01, 2014 DoubleTree by Hilton Baltimore-BWI Airport, USA

Molecular mechanisms in the pathogenesis of sepsis

Valentina Pop-Began, D Pop-Began, V Grigorean and **C Popescu**
Clinical Emergency Bagdasar Arseni Hospital, Romania

Innate immune system is an universal form of host defense against infections. The recognition of the innate immunity is based on a limited number of encoded receptors that have evolved to recognize microbial metabolism products. Recognition of these molecular structures allows the immune system to distinguish own infectious components from non-communicable structures. Immune suppression is a hallmark of sepsis. The complement system is activated in the early stages of sepsis, generating large amounts of anaphylatoxin C5a. Complement and TLR family are two major upstream sensors and effectors systems of innate immunity. It was found that TLR4 and complement system are involved in initiating the inflammatory response in sepsis. Clinical studies in which TLR4 was blocked have not shown beneficial effects (41). Toll-like receptors (TLRs) that are a subfamily of PRRS have emerged as the crucial receptors for the recognition of DAMPs. Recently, in the complex cascade of sepsis was highlighted a special form of non-coding genetic material called microRNA. The individual role of every microRNA and the exact role of microRNA network is under investigation. Currently, studies are performed in order to find micro RNA to be used as biomarkers of sepsis. Researches are performed to determine microRNA, small fragments of non-coding RNA, in order to distinguish patients with sepsis and healthy patients, and if the plasma levels of microRNA correlate with the severity of the disease. Recent researches reports that the regulation of gene expression through microRNA plays a very important role in the following cellular processes, for example: Apoptosis, the differentiation process, and the cell cycle.

validor2004@yahoo.com

Presence of partial RH1 by fetal RHD genotyping in maternal plasma: Clinical and biological expression at birth. About a case

Guinchard Emmanuelle
EFS Rhône-Alpes, France

Introduction: Mrs. B. Marie, caucasian, 5th pregnancy, presents a poly- alloimmunization already known during a previous pregnancy: A non evolutive anti-RH1 + an anti-RH2 + an anti-JK1. RHD genotyping in maternal plasma fetal performed at 23 weeks and 33 weeks shows an amplification of exon 10, but no amplification of exons 4 and 5. The study of the maternal buffy coat showed no amplification. It may be a partial D inherited from his African father whose RHD gene could not be analyzed. The birth took place at 37 weeks: at birth, the child had no anemia, moderate hyperbilirubinemia quickly contained by phototherapy, no transfusion was necessary.

Results and hypothesis: At birth, the direct antiglobulin test was positive. The elution test found the presence of anti-RH2, anti-JK1 and anti-RH1 while the newborn was Rh: -1.2 and JK: 1. The sequencing of the RHD gene showed an aspect of partial D associated with a deletion of some exons: How is the expression profile? It may be a non-functional gene and anti-RH1 found in the eluate was present in the free state in the close environment of the erythrocyte (Matuhasi-Ogata phenomenon). Conversely, in case of a gene with partial antigen expression, the result of RH1 phenotype at birth may be due to saturation of antigenic sites by corresponding antibodies.

Conclusion: A new sample a few months after birth would confirm its RH1 phenotype.

Biography

I am an M.D/PhD in Biology. I work currently in a laboratory of a Health Clinical Center, CHU of Lyon. I specialize in immuno-haematology as well as in NIPT: fetal RHD genotyping on maternal plasma. I was invited to present this at several international conferences (ESHG 2012) and I am the author of a recently published article (Non-invasive fetal RHD genotyping: Validation of the method with 200 patients, TCB, 2014).

emmanuelle.guinchard@efs.sante.fr