An immune system "mistake" could be the cause of organ failure in intensive care patients

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An immune system "mistake" resulting from the evolutionary process could be the cause of organ failure in intensive care patients

At least one in every two intensive care patient displays Systematic Inflammatory Response Syndrome (SIRS). This clinical picture resembles sepsis but without any identifiable bacterial or fungal infection being present. Until now the exact cause of this syndrome was unknown. Researchers at MedUni Vienna Department of Internal Medicine II have now discovered a possible cause of this "pseudo-sepsis": the immune system mistakes the mitochondria that find their way into the blood following circulatory collapse or cardiac arrest for bacteria and so attacks them. The explanation for this goes back 1.5 billion years and is based on the so-called endosymbiotic theory.

The endosymbiotic theory states that, over the course of evolution, single-celled organisms have joined forces symbiotically to form a higher organism (endosymbionts). In this case, single-celled organisms absorbed bacteria, which then became their mitochondria, also known as the powerhouses of the cells, because it is here that the energy-providing molecule adenosine triphosphate (ATP) is produced.

In the case of Systemic Inflammatory Response Syndrome (SIRS), the results obtained by the MedUni Vienna researchers suggest that the immune system "mistakes" the mitochondria for the bacteria from which they originally arose (since the DNA structure of the mitochondria is the same as that of the bacteria) and so responds to them with an inflammatory reaction. "When patients suffering from shock and organ damage release higher levels of mitochondrial DNA and this bonds to special signal-transmitting molecules, TLR 9 receptors, the risk of organ failure increases and the chances of surviving the disease decrease," explains Walter Speidl of the University Department of Internal Medicine II (cardiology department). Researchers now want to find out whether this "pseudo-sepsis" can be prevented by inhibiting TLR 9 receptors.

The recent study, which has now been published in the leading magazine *Critical Care Medicine*, involved 233 patients from the intensive cardiology unit in Vienna General Hospital. The study demonstrated that patients with high plasma levels of mitochondrial DNA and also augmented TLR 9 receptor expression, had a significantly higher mortality risk. Nearly every second intensive care patient is affected by some form of SIRS, which is associated with high mortality. The symptoms include high fever or hypothermia, rapid heart rate and hyperventilation as well as an elevated white blood cell count.

More information: Konstantin A. Krychtiuk et al.

"Mitochondrial DNA and Toll-Like Receptor-9 Are Associated With Mortality in Critically Ill Patients," *Critical Care Medicine* (2015). DOI: 10.1097/CCM.0000000000001311

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