SUMMARY

Iron is a very important biogenous trace element, which is involved in many of cell processes in organism. For its character iron can be also involved in Fenton reaction, where a toxic hydroxyl radical is produced. The iron metabolism is very carefully regulated in order to avoid formation of hydroxyl radical. Iron Responsive Proteins-Iron Responsive Elements (IRPs-IREs) system is involved in the regulation of iron metabolism on cell level, small peptide hormon hepcidin is involved in the regulation on systemic level.

Hepcidin was discovered in 2000 as a peptide with antimicrobial properties. It is a key regulator of iron metabolism, as was discovered later. The target of hepcidin is ferroportin-the only known iron cell exporter. The expression of hepcidin is downregulated by hypoxia and anemia and upregulated by iron overload and inflammation.

Hemodialyzed patients suffer from anemia very often. This anemia is caused by many factors, e.g. inadequate production of erythropoietin, chronic inflammation, chronic oxidative stress, blood loss during hemodialysis process or lower lifetime of red blood cells.

We realized three studies on the patients with end-stage renal disease in our laboratories. Our aim was to find a relationship of hepcidin and other parameters of iron metabolism, inflammation and erythropoiesis. We expected that this relationship should be close.

Our results showed, that the evaluation of iron metabolism status in patients with end-stage renal disease is quite difficult. We expected that hepcidin, which is considered to be one of the inflammatory markers, would copy the levels of other parameters of inflammation as CRP and IL-6. We also expected, that the relation between glomerular filtration or residual kidney function and hepcidin level would exist. The levels of hepcidin and inflammatory markers were higher in hemodialyzed patients compared to the healthy controls, but no strong correlation between these parameters was found.

The hepcidin level in patients with end-stage renal disease seems to be influenced by many factors, which effect complexly. It is inflammation, which is maybe not the most crucial factor, anemia, hypoxia, hepcidin retention, production of hepcidin by the fat tissue in patients with better nutritional status, defence against infection, diurnal variability of hepcidin or its intra-individual variability. There is necessary to solve some preanalytical, analytical and clinical aspects of hepcidin estimation in future. Then we will be able to consider the usefullness of hepcidin determination. The efforts for improvement of this situation will be stronger, because hepcidin is investigated in the other medical fields than only in nephrology or hematology at the moment.