SUMMARY

Hyperglycemia in critically ill patients was considered for many years an adaptive response to stress conditions being present in both patients with and without previous history of diabetes.

Hyperglycemia is caused mainly by peripheral insulin resistance induced by the factors acting counteracting insulin signalling at the postreceptor level. Furthermore, hyperglycemia itself can then increase serum levels of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin-6 (Il-6) and interleukin-8 (Il-8) and others. On the contrary, peripheral insulin resistance induced by pro-inflammatory cytokines may further potentiate hyperglycemia.

White adipose tissue represents in addition to its energy storage function also a very active endocrine active organ. In addition to regulation of a number of metabolic processes it also significantly modulates the inflammatory response. In critically ill patients, adipose tissue changes its morphology, i.e. the adipocytes are shrinking and adipose tissue is abundantly infiltrated by macrophages. Paradoxically, overweight and obese critically ill patients have lower mortality than underweight, lean and morbidly obese subjects.

In our studies, we selected population of the patients undergoing elective major cardiac surgery with extracorporeal circulation as a suitable model of stress conditions, and examined tissues which were easily accessible for the sampling.

We measured mRNA expression of two recently discovered factors with an important role in the regulation of metabolic pathways and inflammation - fibroblast growth factor -21 (FGF-21) and adipose fatty acid binding protein (A-FABP) in epicardial and subcutaneous adipose tissue, skeletal and cardiac muscle and their

serum levels in perioperative period to identify their possible role in the development of hyperglycemia and insulin resistance in critically ill patients.

We demonstrated marked increase in FGF-21 serum levels during cardiac surgery with significant increase of its mRNA expression in epicardial adipose tissue. At the same time, we have also seen a relatively strong FGF-21 mRNA expression in skeletal muscle suggesting its possible role as an additional source of FGF-21.

Data from our second study demonstrated that cardiac surgery increased A-FABP mRNA expression in peripheral monocytes but not in epicardial and subcutaneous adipose tissue, cardiac or skeletal muscle. Cardiac surgery significantly increased in A-FABP serum levels.

Both studies suggested possible influence of investigated adipokines on the development of stress response, inflammatory response and insulin resistance after cardiac surgery.

Key words: adipocyte fatty acid binding protein; fibroblast growth factor-21; adipose tissue; skeletal muscle; cardiac surgery; insulin resistance