Editorial

Cardiovascular diseases are the primary cause of death in patients suffering from chronic renal failure requiring hemodialysis. Uremia as a complex syndrome influences all cardiac structures – pericardium, myocardium, endocardium as well as the coronary system. The most important manifestations of this complex symptom, which is also called ‘uremic heart disease’, are hypertensive and anemic heart disease, coronary artery disease and heart valve diseases as a result – among others – of disturbances of calcium and phosphate metabolism. In uremic patients, heart surgery and the entire perioperative management is demanding and has to be done in cooperation with the nephrologist.

Oxidative stress has been postulated to be one of the important risk factors for cardiovascular disorders in uremic patients. In general, uremic patients show a reduced number of red blood cells (RBCs). This results in a reduced antioxidant protection since RBCs are equipped with highly effective antioxidant defense. Most of the antioxidant capacity of the whole blood is localized within the RBCs. Circulating RBCs are mobile radical scavengers and provide antioxidant protection to other tissues and organs. In uremic patients with anemia, a drastic oxidative stress is given inducing cardiovascular injury. The best clinical demonstration to restore antioxidative protection is complete anemia correction by means of erythropoietin.

Particularly noteworthy is the continuous process of lipid peroxidation whose products were elevated in anemic uremic serum. Lipid peroxidation processes result in increased oxidized lipoprotein species (oxLDL). They are key factors of arteriosclerosis. During complete anemia correction, lipid peroxidation products are significantly reduced.

In cardiac failure, an increased sympathetic tone and an activated renin-angiotensin-aldosterone-system (RAAS) initiate the structural and functional transformation of the myocardium (remodeling). Renal anemia is considered to be most important for the development of eccentric left-ventricular hypertrophy (LVH) and arterial hypertension for the concentric LVH. During LVH maximal coronary reserve is already critically reduced, the so-called ‘cardiomyocyte-capillary mismatch’. Thus, severe LVH predisposes ventricular arrhythmia triggered by the critically reduced coronary reserve. This often leads to sudden death. Therefore, an attempt to cause regression of LVH is mandatory.

Cardiac mortality in uremic patients depends on multifactorial causes. To correct only one cause would not be efficient. A multi-interventional therapeutic approach is necessary to achieve improvement in cardiac morbidity/mortality. However, the majority of patients are not receiving optimized cardioprotective therapy and complete anemia correction. Optimized oxygen availability and correction of hormonal abnormality are needed for the diseased heart to heal. Complete anemia correction is essential, safe and economical in uremic patients.

Nephrologists need to be responsible for the cardiac health. Renal physicians can do a lot of good and bad for the cardiac health of their patients.

Profiting from interdisciplinary cooperation with cardiologists and cardiac surgeons and by using up-to-date, sufficient therapy on pathophysiological baselines against cardiac insufficiency, it should be possible, as shown by the following articles, to significantly reduce cardiac morbidity/mortality in uremic patients.

*Hannelore Hampl
Anatole Besarab*