question survey to assess their knowledge of heart disease, infective endocarditis, and endocarditis prophylaxis. Out of 133 patients (100%), 102 patients knew the name of their heart disease. Fifty patients correctly defined endocarditis, but only 38 knew hygiene measures that could prevent endocarditis. Thirty patients knew that they needed to take "a medicine" before dental procedures and just 18 of those patients knew that an antibiotic was necessary. Among doctors, all knew what infective endocarditis is, but 30% of family doctors and 67% of dentists hesitated to name the antibiotic of choice and its dosage. The most recent guidelines recommend prophylaxis only in patients with underlying cardiac conditions with the higher risk of adverse outcomes, including patients with a previous history of infective endocarditis, patients with prosthetic heart valve or prosthetic material used for valve repair, patients with a valvulopathy after cardiac transplantation, and patients with a specific congenital heart disease. But it is a particularity of Moldova that the number of patients with rheumatic valve disease is high, that is why we consider forming a group of moderate risk for infective endocarditis and to include them in prophylaxis regimens. Many adults with heart diseases have inadequate knowledge of their cardiac lesion, of endocarditis and of endocarditis prophylaxis. Educational efforts for them need to be updated and reinforced regularly. The use of definite criteria for identifying groups of risk and prescribing antibiotics regiments for IE prophylaxis can decrease its incidence and rate of complications.

Renal Damage and Hypercholesterolemia

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Objectives of this study are to investigate/indicate the potential mechanisms of hypercholesterolemia-induced renal injury. We found out that podocyte activation, such as after renal mass reduction, with hyperlipidemia may contribute to podocyte injury those results in development of segmental sclerosis associated with secondary damage to the tubulointerstitium. Other studies stress the pathogenetic roles of macrophage influx and mesangial cell activation/injury (as evidenced by glomerular hypertrophy and matrix accumulation) in lipid-induced glomerular damage. Another hypothesis for renal effect of hypercholesterolemia suggests that hypercholesterolemia impairs systemic vascular reactivity in response to endothelium-dependent vasodilators, which may be mediated partly through increased formation oflipid peroxides. One of the underlying mechanisms for impaired vascular reactivity is an increased release of oxygen radicals that react with nitric oxide (NO) resulting in decrease of NO's bioavailability and form of peroxynitrite. The impairment also likely is related to increased oxidizability of LDL. Furthermore, oxidized LDL may affect NO bioavailability by modulating the expression of the enzyme endothelial NO synthase. Finally, hypercholesterolemia is associated with pro-inflammatory changes and impaired regulation of tissue perfusion, which may lead to neovascularisation in the renal cortex, which precedes signs of overt renal morphological damage resulting in renal disease progression. Recent experimental studies on hypercholesterolemia-induced renal damage exhibit that hyperlipidemia contributes to the progression of renal disease Further studies are needed to investigate the pathogenetic mechanisms.