Chronic renal allograft damage after transplantation: what are the reasons, what can we do?

Abstract:
Chronic renal allograft damage is one of the main problems after kidney transplantation. This review enumerates causes, describes available therapeutic options, and discusses options of the future. Alloantigen-dependent and alloantigen-independent factors are responsible for allograft damage. Prevention of renal allograft damage starts with interventions that occur surrounding the explantation in cadaveric organs. These include the use of dopamine or machine perfusion systems. Followed by the critical phase of ischemia/reperfusion injury, the LCN2/lipocalin-2, HAVCR1, and p38 MAPK pathway are new players involved in that process. Innate immunity plays a part, too. Cold ischemia time is associated with genes of apoptosis. Nondonor-specific antibodies like antihuman leukocyte antibodies-Ia or angiotensin type 1 receptor may also play a role. Recent research indicates that genetic polymorphism like the Ficolin-2 Ala258Ser polymorphism and the mannose-binding lectin-2 polymorphism are involved in that process. New therapeutic options are rare and in the future. However, there is some evidence that drugs interfering with metalloproteinases, sexual hormones like dihydroandrosterone, and mesenchymal stem cell therapy may be of importance. Taken together, although the understanding of chronic rejection has improved, the available therapeutic options remain scarce.