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The Effects of Uremic Toxin on Heart Functions in HD Patients

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The incidence of cardiovascular disease in hemodialysis (HD) patients significantly exceeds that of the general population, and uremic cardiomyopathy is one of the serious cardiovascular complications in HD patients. The accumulation of uremic toxins is a pivotal factor in myocardial injury in HD patients. Studies have demonstrated that indoxyl sulfate (IS), a uremic toxin that is difficult to be cleared by dialysis, can induce vascular inflammation, and cause damage to the heart. Furthermore, in clinical practice, we have found that HD patients are often accompanied by immune dysfunction; Recent studies have indicated that complement C1q and T lymphocytes may contribute to adverse cardiovascular events in HD patients. Consequently, immunity may be implicated in uremia toxin-induced cardiac injury in HD patients. Peripheral blood mononuclear cells (PBMCs) constitute a critical constituent of the immune system, albeit their role in myocardial injury in HD patients remains poorly understood. Extracellular vesicles (EVs) are nanoscale vesicles encapsulated by phospholipid bilayer membranes capable of carrying proteins and/or microRNAs for intercellular information transfer. We advance the hypothesis that the uremic toxin-induced production of "damaging" EVs in PBMCs is a crucial mechanism of myocardial cell immune injury resulting from uremic toxins. Our group's research has revealed that IS activates the NF-κB and apoptosis pathway of cardiomyocytes through PBMCs-EVs-miR-744-5p, resulting in cardiomyocyte hypertrophy. This provides evidence for potential immune mechanisms of myocardial injury in uremia. Subsequently, through an in-depth study of salvianolic acid B (SalB), the main active ingredient in Danshen (Salvia miltiorrhiza), a commonly used drug for the clinical treatment of cardiovascular diseases, it was found that PBMCs-EVs-miR-744-5p was likely to be the underlying molecular mechanism of SalB's function. Our research group has also identified the drug target SY of SalB.

Keywords: uremic cardiomyopathy , indoxyl sulfate, peripheral blood mononuclear cells, extracellular vesicles, miR-744-5p