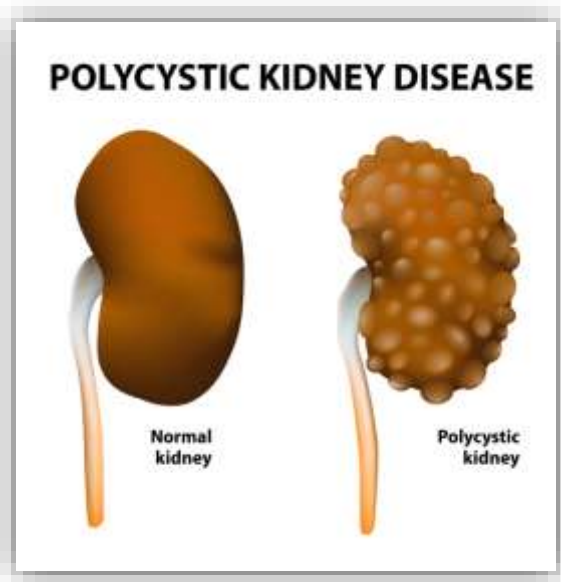


Effects of hydrogen-rich water in Polycystic Kidney Disease



Various factors are considered to be mechanisms of the increase in the sizes of cysts in patients with polycystic kidney disease. Autosomal-Dominant Polycystic Kidney Disease (ADPKD) causes multiple cysts in the bilateral kidneys, which increases and impairs normal kidney tissue. Approximately half of ADPKD patients progress to end-stage renal failure by 60 years of age. ADPKD is the most common hereditary kidney disease. ADPKD is caused by mutations of two genes (PKD1, PKD2) encoding Polycystin 1 (PC 1) and Polycystin 2 (PC 2). The vasopressin (AVP) R2 receptor present in the renal tubule (collecting duct) is activated by AVP and works to increase water permeability through Adnylcyclase (AC) and cAMP to retain moisture. In PKD cells, the increase in cAMP caused by vasopressin increases the sizes of cysts.

Thus, drinking a large amount of water and tolvaptan (a vasopressin V 2 receptor) antagonist is expected to suppress the increase in cyst numbers and suppress the progression of renal disorder.

Hydrogen in aqueous solution shows antioxidant action by reducing hydroxyl radical. Consumption of hydrogen-rich water alleviated renal injury in spontaneous hypertensive. Hydrogen is also thought to increase the ATP production in mitochondria. ATP is converted intracellularly into cyclic AMP(cAMP) by Adnylcyclase (AC). Thus, as cAMP—which is involved in the increase in the cysts—increases, the antioxidant effect may be canceled out.

Vasopressin is one of the causes, and drinking large volumes of water shows an effect of suppressing an increase in cysts. Hydrogen-rich water can be produced with relatively little effort, while taking tolvaptan requires the intake of large amounts of water. If its effectiveness can be improved by changing the water to hydrogen-rich water, it might be a useful treatment with few side effects. It is suggested that hydrogen-rich water is useful not only for kidney injury but also for diseases of various organs.



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