# POLYCYSTIC KIDNEY DISEASE WITH HIGHLY ELEVATED γ-GLUTAMYL TRANSPEPTIDASE

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Patients with polycystic kidney disease (PKD) may be asymptomatic until the later ages owing to the underlying disorder. A renal proximal tubule associated enzyme, γ-glutamyl transpeptidase (γ-GT), was reported to be normal in PKD. We report an elderly case with highly elevated γ-GT with no other potential source.

***Key words:*** Polycystic kidney disease, gamma glutamyl transpeptidase, elderly

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## INTRODUCTION

Polycystic kidney disease (PKD) is characterized by the growth of multiple cysts in the kidneys. Autosomal dominant, the most common form that develops between the ages of 30 and 40, and autosomal recessive PKD, which begins early in life, are the inherited types (1).

γ-glutamyl transpeptidase (γ-GT) is a membrane-bound enzyme that is an integral part of the γ-glutamyl cycle functioning in the degradation and neo-synthesis of glutathione in the kidney (2,3). It is primarily synthesized in the brush border membrane of proximal tubules (4), but is present in cell membranes in pancreas, liver, spleen, heart, brain, seminal vesicles and endothelium (5-7).

## CASE

A-74 year old man was admitted to our hospital with a clinical picture of fatigue and vertigo for 20 days. In his history, there was only rarely seen dark urine. He had no history of alcohol use, chronic hepatic or renal disease, and drug intake. The physical examination was normal. CBC and CRP were normal, erythrocyte sedimentation rate was 33 mm/hour, and RF was negative. Serial urine analyses were normal. In detailed blood chemistry, the only abnormal finding was γ-GT of 334 U/L (normal range 7-50 U/L), which persisted over the following days. Liver function tests, cholestatic markers and renal function tests were normal (AST: 25 U/L, ALT: 32 U/L, ALP: 225 U/L, Total Blb: 1,0 mg/dL, Urea: 25 mg/dL, Cr: 1,1 mg/dL). Estimated GFR was 65 ml/min. In abdominal ultrasonography the only pathological finding was enlargement and multiple cysts in both kidneys. Thoracoabdominopelvic CT showed only polycystic renal disease with no enlargement in intra or extrahepatic bile ducts. Cranial CT revealed only slight age related changes. The patient was treated for his vertigo and improved satisfactorily before discharge. In outpatient follow-up, there was no decline in serum γ-GT levels in the following months (666, 276 and 336 U/L).

## DISCUSSION

The cysts in polycystic kidney disease originate as expansions of renal tubules. Cysts arise in every tubule segments in autosomal dominant type whereas they are derived from collecting tubules in autosomal recessive type (8). Although γ-GT synthesis occurs mainly in the kidneys, elevated γ-GT is interestingly not common in patients with cystic renal disease. The reason for this is not so clear but it may theoretically be related to the pathophysiological mechanisms that cysts derived from tubules rapidly close off from the original nephron after formation, but retain their functional capacity and show similarity with hepatic cysts (9). No significant difference was found in serum

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and urine γ-GT activity in 32 patients with PKD when compared to patients with renal plus hepatic cysts and healthy controls (10). For our patient, who had no symptom even until he was 74 years old, we have found no pathology in the liver, intestine, pancreas etc. that might possibly be source of elevated γ-GT. Such an increase in γ-GT in an accidentally diagnosed PKD patient may suggests that the cysts not always close off from the nephron and subsequently may drain into venous system, though it is quite hard to get any direct evidence. Finally, renal cystic disease may be a reason of elevated γ-GT levels of unknown origin, and if the liver and the biliary tree are normal, should clinically be suspected more frequently.

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