中文題目： 腎臟組織病理上病變對於急性腎小管壞死後續恢復之影響

英文題目： The impact of renal histopathological lesions on recovery from biopsy-proven acute tubular necrosis

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**Background:**

Acute tubular necrosis (ATN) is a common cause of acute kidney injury (AKI), especially in hospitalized patients. Despite the improvement of modern medicine, ATN remains the important cause for developing chronic kidney disease and subsequent morbidity. Patients with acute on chronic kidney disease may have an increased risk of long-term dialysis and mortality. However, the impact of renal histopathological lesions on the prognosis of ATN is not well understood.

**Methods:**

Between January 1, 2000 and December 30, 2004, we retrospectively reviewed all patients with AKI in Changhua Christian hospital renal pathology database. 70 patients underwent renal biopsy had a diagnosis of ATN with or without other renal histopathological lesions during the study period. 14 patients were excluded because of no baseline data or loss of follow-up. Therefore, a total of 56 patients were selected for analyzing clinical and histopathological characteristics and followed up for renal function.

**Results:**

In this retrospective, single-center, observational cohort study, 56 patients who had AKI with biopsy-proven ATN were analyzed. AKIs were stratified according to the RIFLE classification. There were 15 ATN alone patients, 25 ATN with primary nephropathy patients, and 16 ATN with secondary nephropathy patients. Compared with ATN alone or ATN with primary nephropathy patients, an impaired recovery of renal function (serum creatinine and eGFR) was observed in ATN with secondary nephropathy patients during the 1-year follow-up period. The Kruskal-Wallis test showed that ATN with secondary nephropathy patients had more diabetes, angiotensin-converting-enzyme (ACE) inhibitor or angiotensin II receptor blocker (ARB) use, urinary protein, global obsolescence of glomeruli, and interstitial fibrosis.

**Conclusion:**

In contrast to primary nephropathy, secondary nephropathy may associate with more advanced glomerular sclerosis or interstitial fibrosis and contribute to impaired recovery from acute tubular necrosis.