G79 Can Renal Acute Tubular Necrosis Be Differentiated From Autolysis at Autopsy?

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After attending this presentation, attendees will have an understanding of the morphological characteristics used to define acute tubular necrosis (ATN) and how certain characteristics may be used to differentiate between ischemic ATN and autolysis in postmortem samples.

This presentation will impact on the forensic community by providing morphological characteristics to be used in the microscopic examination of postmortem renal tissue to determine and/or confirm analyses of ATN as contributing to cause of death. The use of diagnostic criteria will augment the identification of ischemic ATN as distinct from the process of autolysis.

Acute tubular necrosis is the most common cause of acute renal failure and accounts for 50% of all cases of acute renal failure in hospitalised patients and greater than 75% of critical care/intensive care unit cases. Acute renal failure affects about 5% of hospitalized patients and has a high mortality rate of 50%. It is a commonly held view amongst autopsy pathologists that it is generally not possible to diagnose ATN at autopsy because of the presence of autolysis, and that the only way the condition can be diagnosed is by identifying mitotic figures in the tubular epithelium. This belief may lead to the under-reporting of the condition as a finding upon microscopic analysis of renal tissue.

The Department of Forensic Medicine, Glebe, Sydney autopsy database was queried for cases where an antemortem diagnosis of ATN was made. Antemortem hospital medical charts for each case were searched for a diagnosis of ATN based on clinical and biochemical parameters. A total of 57 cases over a 5 year period were found. These cases were compared to a similar number of age and sex matched controls, who died suddenly as a result of self-inflicted hanging but were otherwise healthy.

A total of 114 deidentified and randomized kidney sections were examined. Serial tissue sections from each case were stained with H&E, Martius Scarlet Blue (MSB), Masson's Trichrome and anti-human Ki-67 immunoperoxidase. Morphological characteristics compared were proliferating epithelial cells (as visualized by Ki-67 positivity); fibrin thrombi in glomeruli; tubular epithelial whorls; mitoses in tubular epithelium; presence of tubular casts; degree of autolysis; tubulorrhexis; tubular epithelial flattening; interstitial inflammation, and interstitial edema.

All results were expressed as mean ± standard deviation. Differences between groups were determined by two sample t-test. A *p value* < 0.05 was considered to be statistically significant.

Statistically significant differences were between the cases exhibiting ATN and the controls in the following morphological characteristics: number of tubular epithelial whorls, proliferating cells, tubulorrhexis, and interstitial edema. The mean number of tubular epithelial whorls in ATN cases was 1.93 ± 5.15; no whorls were found in any control cases ($p < 0.001$). The mean number of proliferating cells in ATN cases was 19.5 ± 29 and in control cases was 5 ± 9.2 ($p = 0.0001$). The mean number of tubules exhibiting tubulorrhexis in ATN cases was 0.0309 ± 0.0826 and in control cases was 0.007 ± 0.0258 ($p = 0.041$). The mean degree of interstitial edema (as determined by proportion of fields exhibiting the condition) in ATN cases was 0.533 ± 0.412 and in control cases was 0.195 ± 0.312 ($p < 0.001$).

The remaining morphological characteristics (fibrin thrombi, tubular casts, degree of autolysis, mitotic figures, tubular epithelial flattening and interstitial inflammation) were analysed and showed no statistically significant differences between the two groups.

Acute tubular necrosis can be reliably differentiated from autolysis at autopsy. The presence of characteristic tubular epithelial whorls is highly diagnostic of ATN. When taken together with tubulorrhexis, interstitial edema and epithelial proliferation, a diagnosis of ATN can be reliably made at autopsy.

Autopsy, Acute Tubular Necrosis, Renal Pathology

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