Acute Kidney Injury, Analgesic Nephropathy and Toxin-mediated Kidney Injury in an Australian Chronic Kidney Disease (CKD) Cohort

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Aim
To describe CKD attributed to acute kidney injury (AKI), analgesic nephropathy (AN) and toxin-mediated kidney disease (injury/TMI) as seen in public nephrology practice.

Background
AKI is etiologically heterogeneous, with an incidence of 2,000-3,000/million population/yr [1]. The incidence is increasing and has been reported in North America to have risen from 3,227 to 5,224/million person-yrs in the 7yrs to 2003 [2]. Further, AKI is a risk factor for incident CKD [3, 4], progressive CKD [5, 6] and end stage kidney disease [7-9].

Identification of etiologies of AKI has improved with appreciation of the Australian epidemiology of CKD due to AKI, AN and TMKI.

Ongoing registry-based surveillance may reveal changing etiologies.

AKI, AN and TMKI were significant causes for CKD. They had similar CKD stage distribution despite the AKI - Infectious NOS category accounting for a smaller proportion of incident cases.

Methods
CKD.QLD is a registry and research platform involving all consenting patients in public renal practices in Queensland (~10,000). The CKD.QLD registry comprising the first 3,167 patients from four hospital sites was searched for all cases with a primary renal diagnosis related to AKI or etiologic toxins. Identified patients were compared to all patients of the four hospital sites for age, gender and CKD stage.

Results
80 (2.7%) had AKI, 58 (2.7%) had AN and 103 (4.2%) had TMKI, constituting a total of 241 (11.1%) in this analysis (1). TMKI subgroups were NSAID (23.3%), lithium (18.4%), chemotherapy (11.7%), calcineurin inhibitors (10.7%), remi-angiotensin system inhibitors (7.8%), antibiotics (6%), and other miscellaneous (3.8%). The most common AKI etiologies were prerenal (29.2%), supplements (29.2%) and biologic agents (1.9%) (Figure 2). AKI subgroups were prerenal (18.8%), AKI NOS (16.3%), cardiac (12.5%), sepsis (12.5%), vascular (12.5%), interstitial nephritis (10.5%), infective (non-sepsis) (5%), post-contrast glomerulonephritis (5%), obstructive (13.8%), hemolytic-uremic syndrome (2.5%), non-renal trauma (2.5%) and enervation (1.3%) (Figure 2).

In the AKI/AN/TMKI cohort the female:male ratio was 5:3:4 (Figure 3). There were markedly more women in the AN cohort (67:33).

The most commonly affected age group was 65-74 yrs (32.4% vs 38.7%) with mean ages 66.6 yrs (Figure 4). Those with AN were older (74.5 yrs) and TMKI younger (53.9 yrs).

AKI, AN, TMKI and total CKD.QLD populations were most commonly CKD Stage 3B (26.7%-39.7%) (Figure 5).

Conclusions
AKI and AN were significant causes for CKD. They had similar CKD stage distribution despite heterogeneous etiology. Women more commonly had AN. Those with AN were older and with TMKI.

References
4. A Clinical Trials platform
5. A biomarker research platform, and
6. A collaborative, multidisciplinary research and practice improvement website, developed as an initiative of the University of Queensland, Queensland University of Technology, and Queensland Health. CKD.QLD has four major research platforms:
7. A CKD Surveillance Registry, encompassing CKD patients referred to all major public renal units within the State of Queensland
8. A data management platform
9. A Clinical Trials platform
10. A biomarker research platform, and
11. A collaborative, multidisciplinary research and practice improvement website, developed as an initiative of the University of Queensland, Queensland University of Technology, and Queensland Health.