

Theranostic Strategies for Drug-Induced Acute Kidney Cancer

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Brief Report

Drug-induced predisposition to acute renal failure (ARF) is a hitherto uncharacterized, underappreciated, and difficult to detect aspect of nephrotoxicity that has the potential to have a significant human and societal impact. Urinary *GM2AP* was identified as the first of a new class of biomarkers for the increased risk of acute renal failure following subnephrotoxic gentamicin treatment in our study. Gentamicin-predisposed animals with no signs of renal cancer develop ARF when exposed to a second potentially nephrotoxic drug, which is also given at subnephrotoxic doses that are safe for non-predisposed individuals. Subnephrotoxic gentamicin had no effect on renal *GM2AP* gene expression or protein levels, as measured by RT-PCR, Western blot, and immunostaining, and its serum level was unaffected. Further research indicates that the higher quantity of *GM2AP* in the urine is most likely due to gentamicin-induced tubular protein mishandling.

Risk markers have the potential to improve ARF prevention by improving our ability to monitor acquired predisposition to ARF

in a proactive manner. In terms of medication nephrotoxicity aetiology, we identified regenerating islet derived protein III beta (Reg IIIb) and gelsolin as potentially distinct urine indicators of gentamicin nephrotoxicity. Indeed, urinary reg IIIb and gelsolin levels distinguish gentamicin-induced nephrotoxicity from cisplatin-induced nephrotoxicity. Reg IIIb is overexpressed in the kidneys of gentamicin-treated rats and is excreted in the urine, whereas gelsolin is excreted in the glomerular ultrafiltrate. Our findings provide proof-of-concept for the aetiological diagnosis of AKI using urine biochemical analysis, with potential applications for improved drug theranostic and more customised care of polymedicated and critically sick patients at multifactorial risk of AKI. Furthermore, our research has discovered new urine indicators that distinguish ischemia from toxic acute kidney cancer.

Kidney cancer, commonly referred to as renal cancer, is a type of cancer that begins in the kidney. Symptoms may include blood in the urine, an abdominal mass, or back ache. Fever, weight loss, and fatigue are all possible side effects. Spread to the lungs or brain can result in complications.

Renal cell cancer (RCC), transitional cell cancer (TCC), and Wilms tumour are the three most common kinds of kidney cancer. RCC accounts for over 80% of kidney malignancies, with TCC accounting for the majority of the remainder. Smoking, certain pain drugs, previous bladder cancer, being overweight, high blood pressure, certain chemicals, and a family history are all risk factors for RCC and TCC. A family history of Wilms tumour, as well as specific genetic abnormalities such as WAGR syndrome, are risk factors. Symptoms, urine testing, and medical imaging may all point to a diagnosis. A tissue sample confirms it.

Kidney masses are categorised based on the type of the cells in the tumour or how they show on radiography. Cancer is a malignant tumour, which is an uncontrolled proliferation of aberrant cells. However, kidney masses can be caused by normal tissue growth (benign), inflammatory (immune system reaction), or vascular growth (cells of the blood vessels).