

# Kidney Attack: Overdiagnosis of Acute Kidney Injury or Comprehensive Definition of Acute Kidney Syndromes

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## Commentary

By Professor Richard Glasscock

When an authority such as Claudio Ronco writes about Acute Kidney Injury (AKI) we should all pay very close attention. He bemoans the complexities of definition of this common 'syndrome' and yearns for something akin (no pun intended) to acute myocardial injury and infarction, long ago called a 'heart attack'. Recently, advocacy for the term 'kidney attack' has surfaced as a new term for AKI. Admittedly, 'heart attacks' are due universally to ischemia of heart tissue, most often resulting from an acute coronary artery thrombosis. Unfortunately, no such uniformity underlies a 'kidney attack', which can be the consequence of not only an ischemic insult but also of toxic, allergic (immunologic), metabolic or infectious disturbances. Like 'heart attacks', high mortality risk attends 'kidney attacks', but our understanding of biomarkers for AKI remains rather rudimentary compared to 'heart attacks', albeit with significant improvement in recent years. Such biomarkers take on special importance because 'kidney attacks' are generally quite silent, compared to the chest pain and other symptoms of 'heart attacks'. While lacking any biochemical reference or staging of severity, the concept of 'kidney attack' allows for separation from elevations in serum creatinine or reductions in urine output so commonly used to define AKI. Indeed, a modern definition of 'kidney attack' as a corollary of 'heart attack'

would include both evidence of a functional disturbance (reduction of GFR) and/or evidence of kidney tissue damage (biomarker). Viewed in this fashion, AKI or 'kidney attack' can be categorized as 'renal angina' (slight decrement in renal function without a necessary biomarker evidence of tissue injury); serum creatinine increase AKI (CRIAKI) (with a decrement in function and biomarker evidence of renal tissue injury); and non-serum creatinine increase AKI (NCRIAKI) (with no demonstrable decrement in renal function but with biomarker evidence of renal tissue injury). Parallels exist for each of these non-overlapping categories in 'heart attacks'. While not yet proven to have meaningful clinical value, this 'kidney attack' approach is novel and has appeal because of its corollaries with coronary artery disease. The main issue, as I see it, is that the diversity of conditions that can lead to 'kidney attacks' compared to the narrow span of lesions responsible for 'heart attacks' jeopardizes its utility at the bedside. Nevertheless, the concept of 'kidney attacks' may stimulate research that leads to even better biomarkers of renal tissue injury and more accurate and timely ways to assess decrements in kidney function in AKI.

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