Topic	**Sub-clinical AKI and CKDu – One Disease or Two?**
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While chronic kidney disease (CKD) may follow recognized systemic or renal pathology such as diabetes, vasculitis or glomerulonephritis, increasing experimental and clinical evidence suggests that CKD may result from multiple episodes of acute kidney injury (AKI). Such AKI may be “subclinical” causing no increase in creatinine. Alternatively, clinically overt AKI may simply have been missed because serum creatinine was not measured at an appropriate time point before the creatinine decreased (recovered) to a near “normal” reference range. In the absence of measurement of renal reserve or kidney biopsy our understanding of apparent recovery is limited. Serum creatinine is the most commonly used surrogate marker of kidney function. Episodes of AKI revealed by kidney damage biomarkers highlight that using serum creatinine as a surrogate measure of kidney fails to detect subclinical AKI that nevertheless leads to loss of more than 50% of the kidney parenchyma. As serum creatinine increases minimally as the GFR decreases from 100 to 50% of normal, many patients and their doctors are unaware that there has been major loss of nephrons before these patients present with overt CKD. CKDu is frequent in low income countries where subjects are prone to diseases causing AKI, but such subjects are frequently not investigated unless severe AKI is present or overt CKD has developed. This lecture will highlight that AKI can induce subclinical CKD and that this can progress from subclinical to overt CKD. This concept explains the frequent observation of extensive fibrosis and damage on kidney biopsy in subjects where there has been little or no history of kidney disease before a reduced estimated GFR or the presence of proteinuria or hypertension alerts the treating doctor to the possibility of kidney disease.