ADQI 11 Figures

Original citation: Acute Dialysis Quality Initiative 11

Figure 1. Overview on mechanisms, histological correlates biomarkers and outcomes in cardiorenal syndrome type 1 in the setting of acute decompensated heart failure. ADHF, acute decompensated heart failure; AKI, acute kidney injury (the term AKI also covers the term "WRF", "worsening renal function which is usually defined as doubling in serum creatinine); AMI, acute myocardial infarction; PE, pulmonary embolism; SVR, systemic vascular resistance; HF, heart failure; CKD, chronic kidney disease; RAS, renal artery stenosis; GFR, glomerular filtration rate; NGAL; neutrophil gelatinase-associated lipocalin; IL-18, interleukin 18; KIM-1, kidney injury molecule 1; L-FABP, liver-type fatty acid binding protein; NAG, N-acetylglucosamine

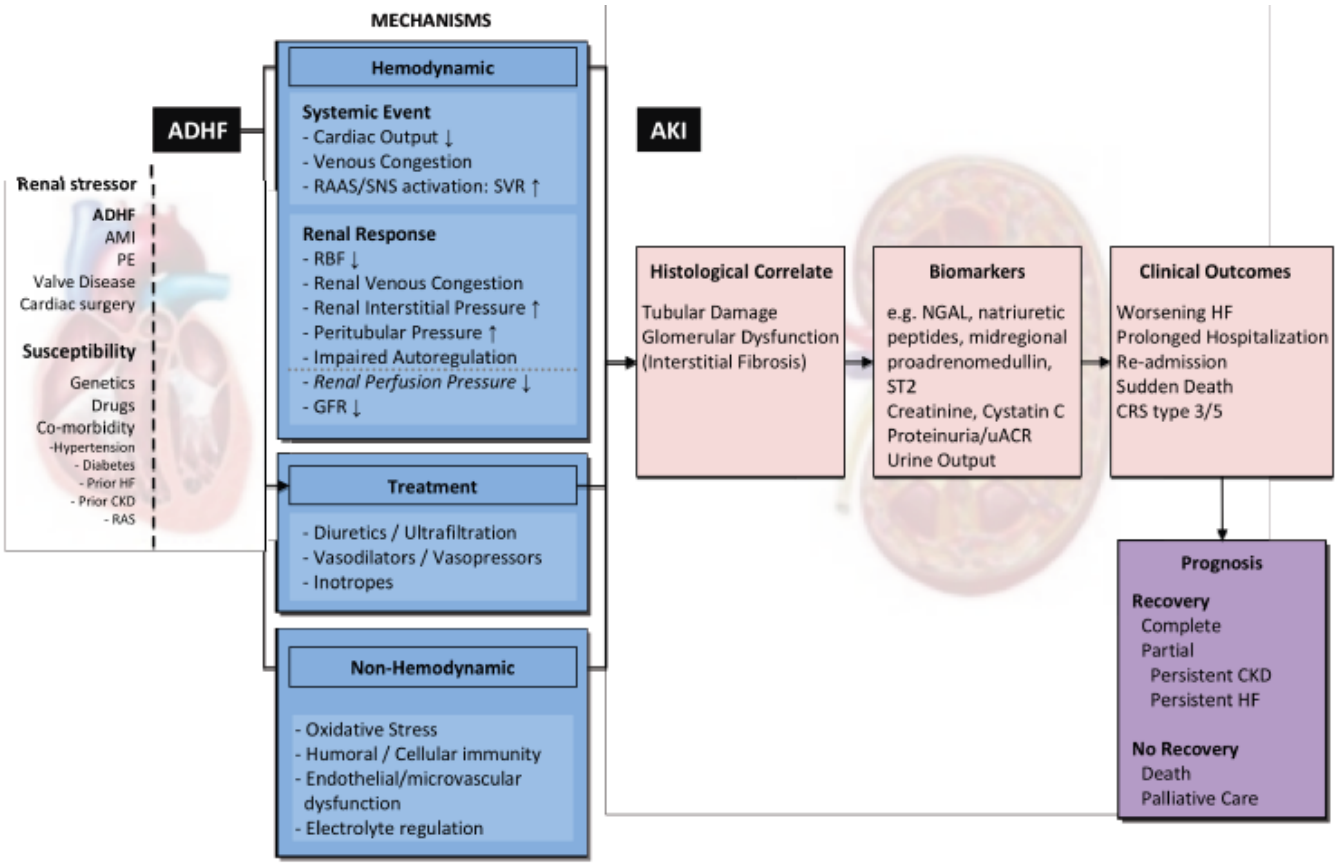

Figure 2. Non-hemodynamic network of pathophysiological interactions in CRS type 1. Note the emerging potential role of macrophages/monocytes as mediator of sodium and fluid retention.

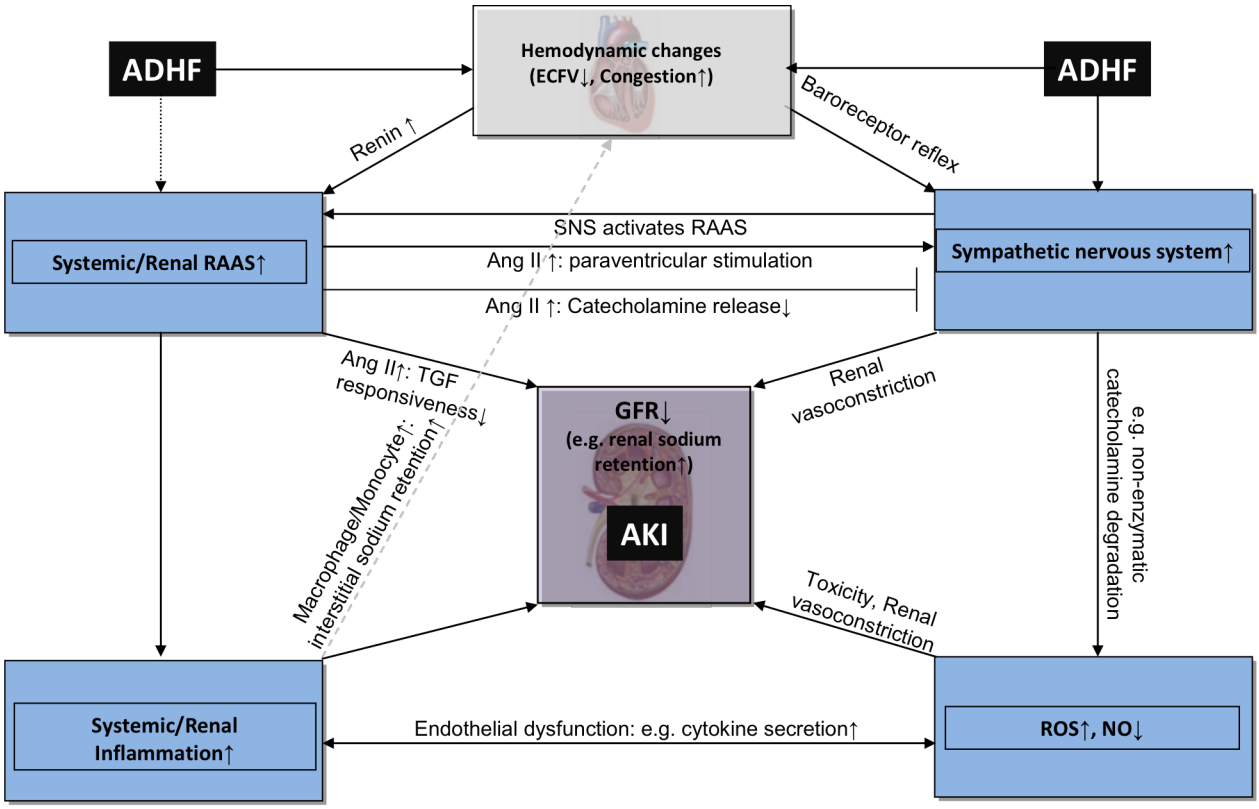

Figure 3. Proposed definition of CRS Type 2 in stable chronic heart failure.

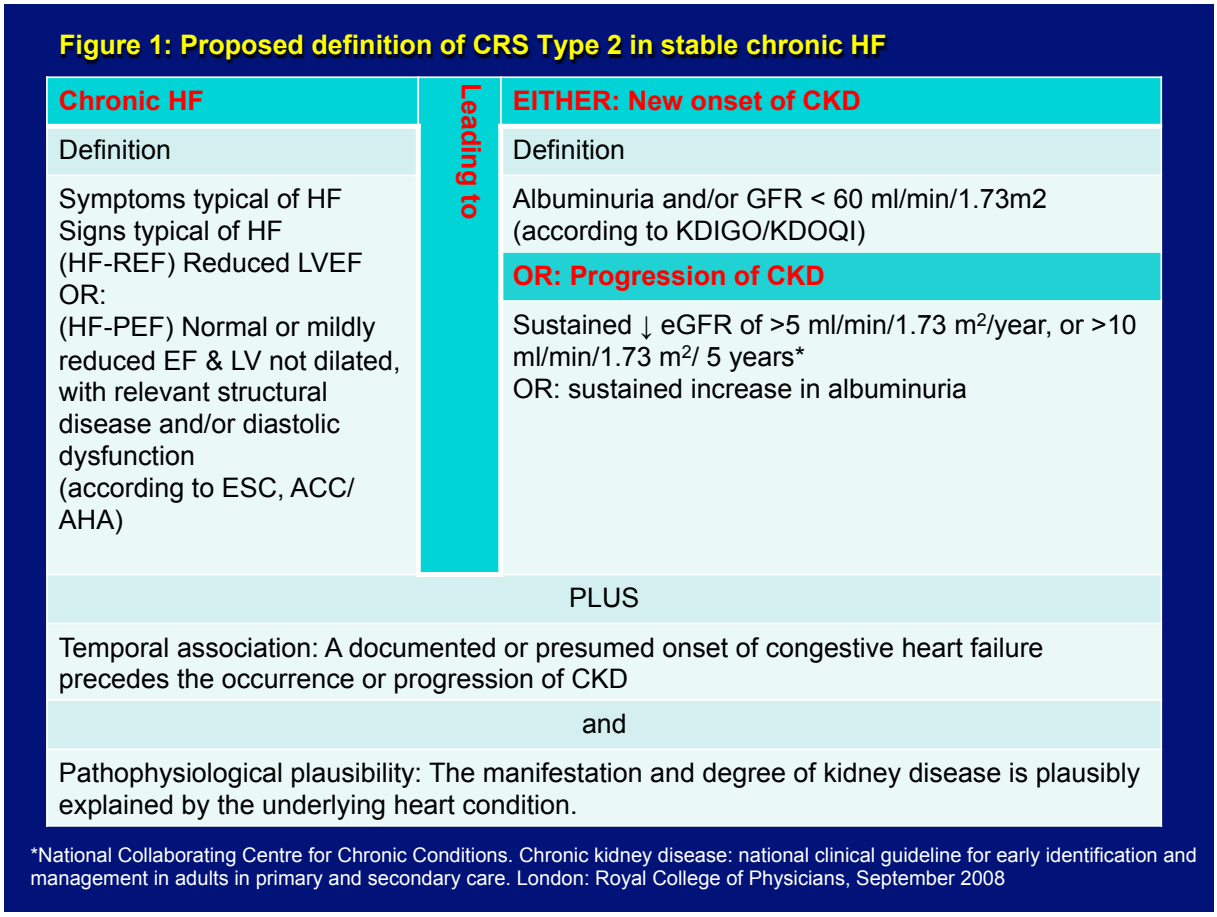

Figure 4. Predominant pathophysiologic mechanisms of cardiorenal syndrome type 2 in stable chronic heart failure.

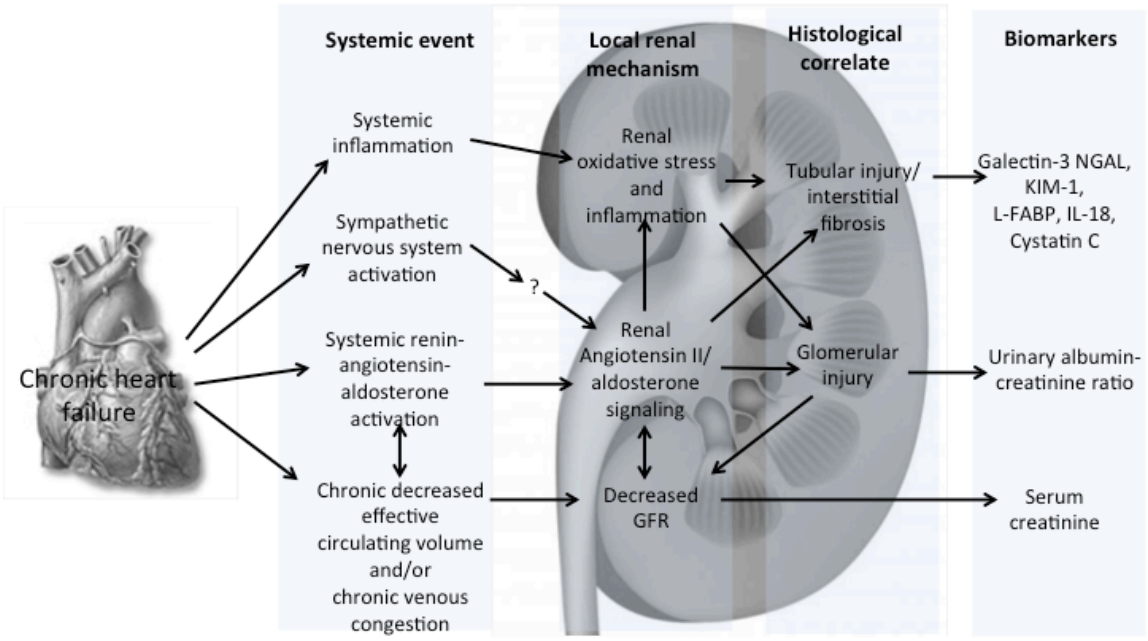

Figure 5. Repeated acute events (heart failure decompensation and/ or acute kidney injury [AKI]) may contribute to the progression of chronic heart and kidney dysfunction.

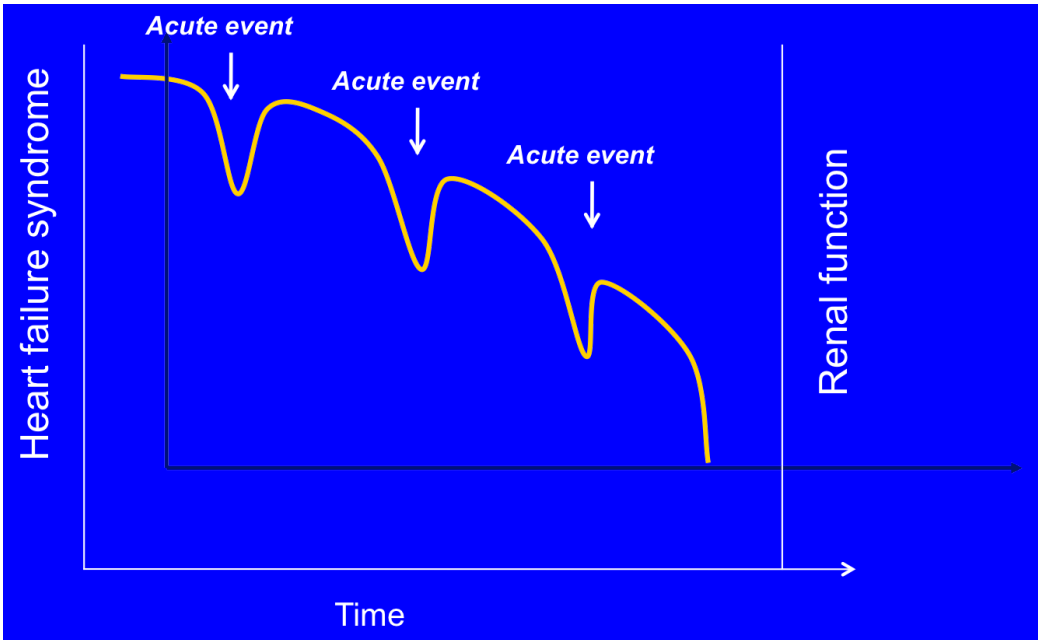

Figure 6. Summary of the demographic contributors, clinical susceptibilities and pathophysiologic mechanisms for development of CRS Type 3.

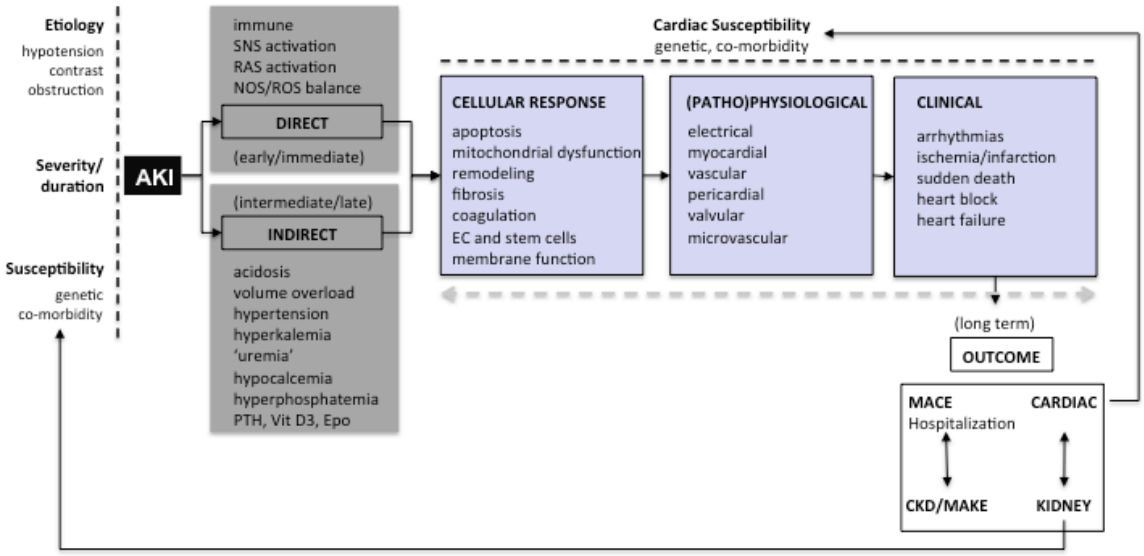

Figure 7. Summary of the interaction of clinical complications of AKI and theoretical negative physiologic effects on myocardial performance.

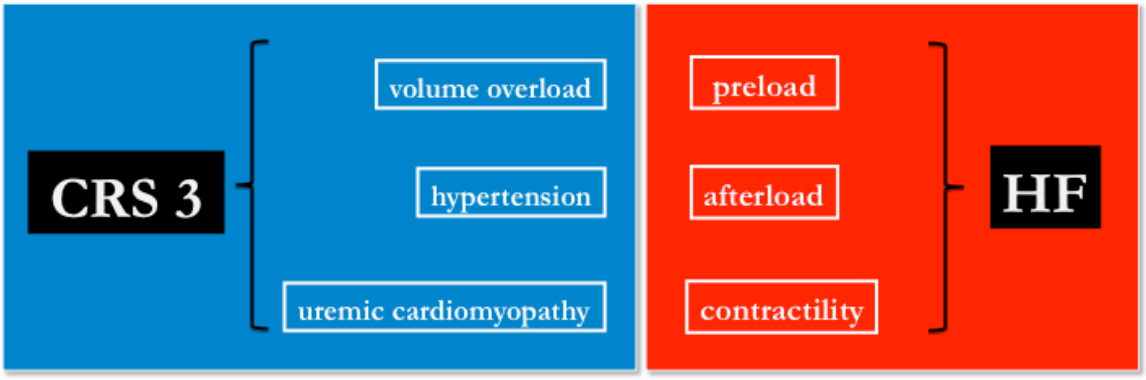

Figure 8. Hyperkalemia. A) A 54-year-old woman with acute renal failure due to glomerulonephritis status, was admitted with acute onset of shortness of breath. Her clinical examination and radiographic findings were consistent with acute pulmonary oedema. ECG shows normal sinus rhythm with tall T waves best seen in leads V3, V4 and V5. The tall T wave is symmetrical, narrow, scooped inwards and 'tented'. K^+ level was 5.8 mEq/L. This classic appearance of the T wave is usually associated with hyperkalaemia. The PR interval, QRS duration and QTc interval are all within normal range. The P wave is wide (> 0.12 sec in duration) and bifid in shape – so called 'P-mitrale' in lead II. There is a negative component in lead V1, which is widened and slurred, consistent with left atrial enlargement. B) ECG shows significant widening of the QRS complex to 158 ms with right bundle branch block morphology associated with coved ST segment elevation in lead V1, resembling 'Brugada ECG pattern', which can be seen in severe hyperkalaemia.

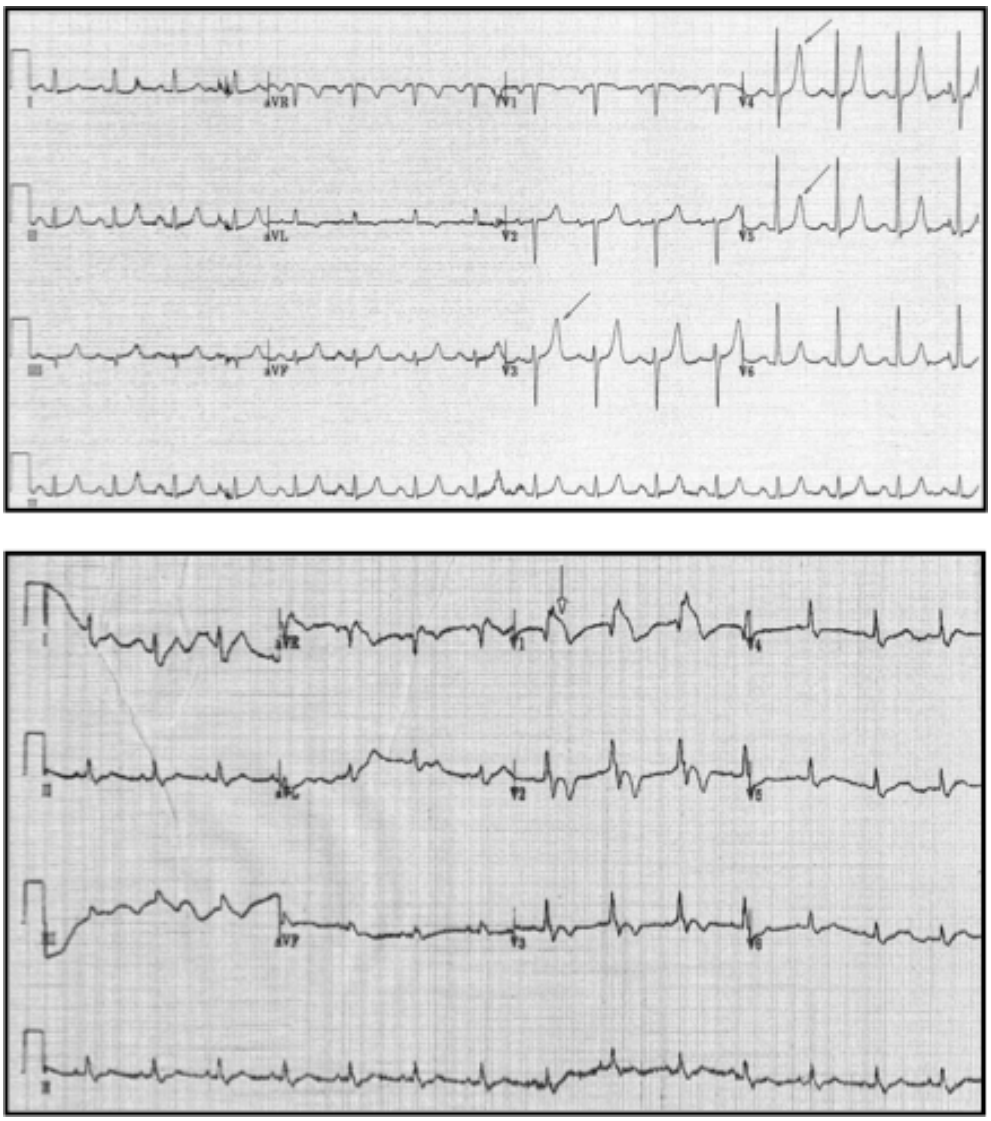

Figure 9. Mechanisms of left ventricular dysfunction in CRS type 3.