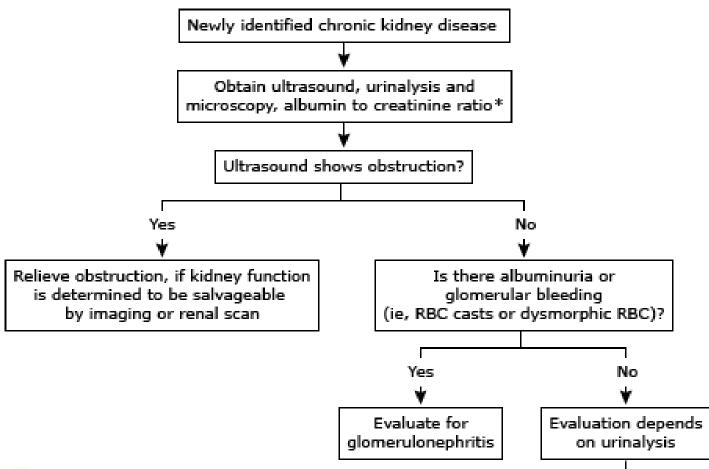
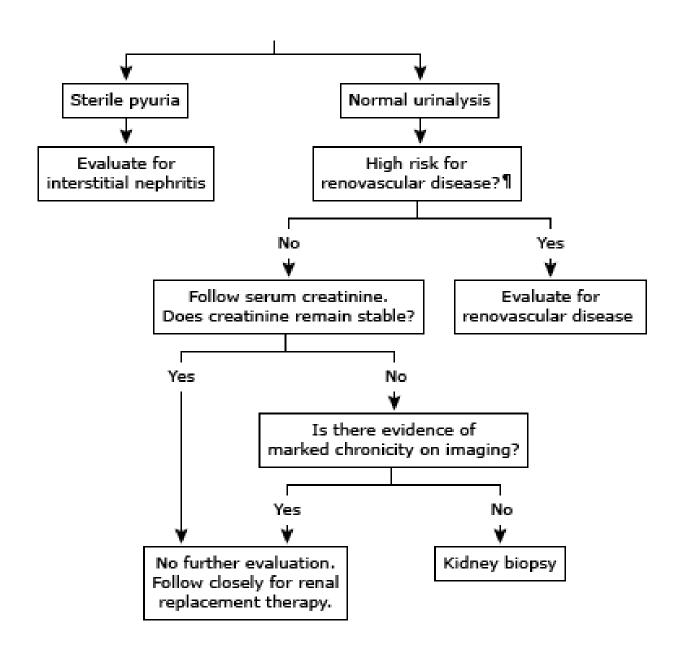
Injuria Renal Aguda vs Insuficiencia Renal Crónica

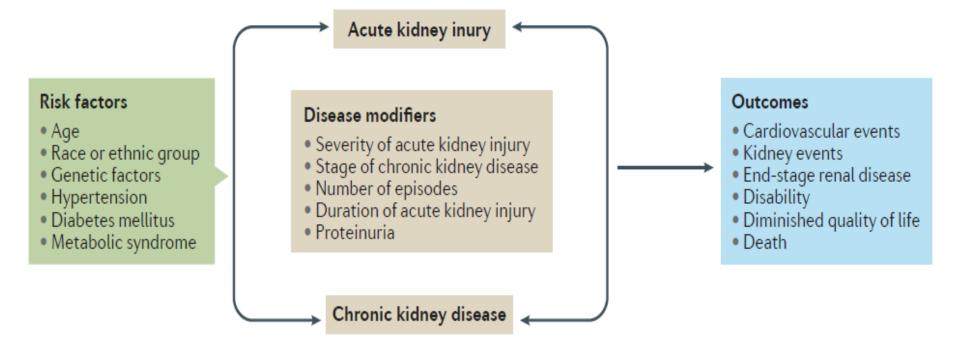
Ivan Goic Boroevic

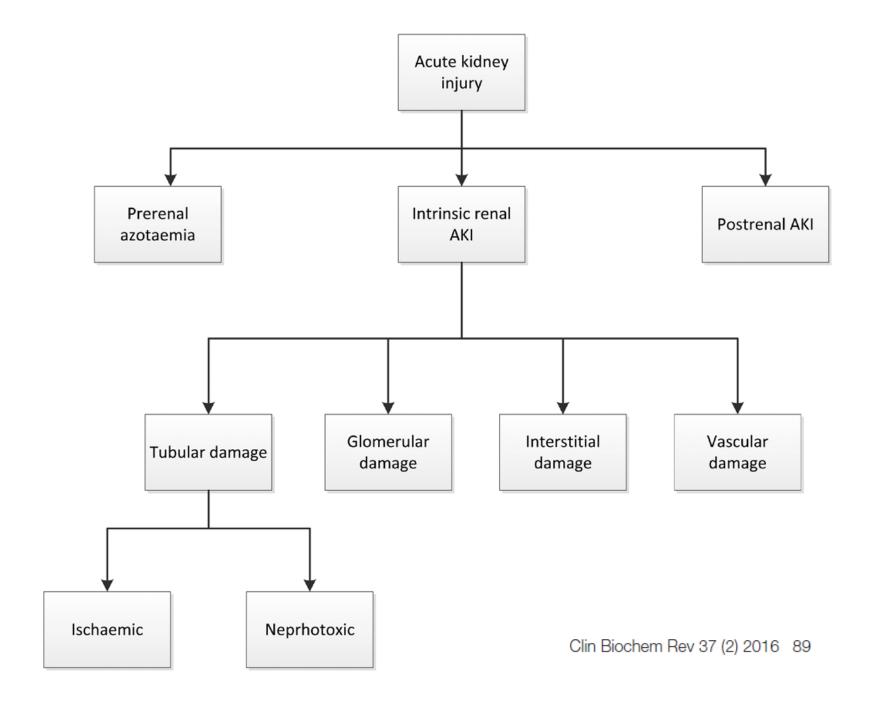


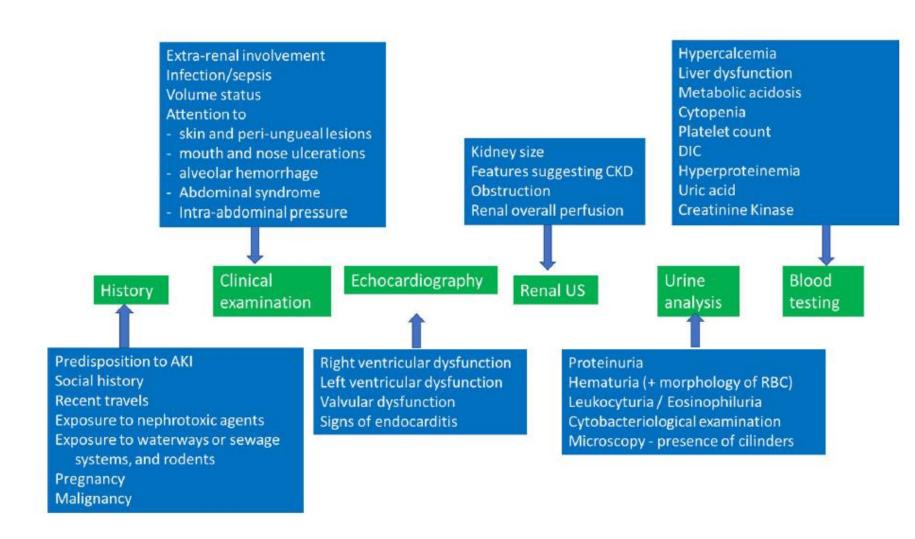




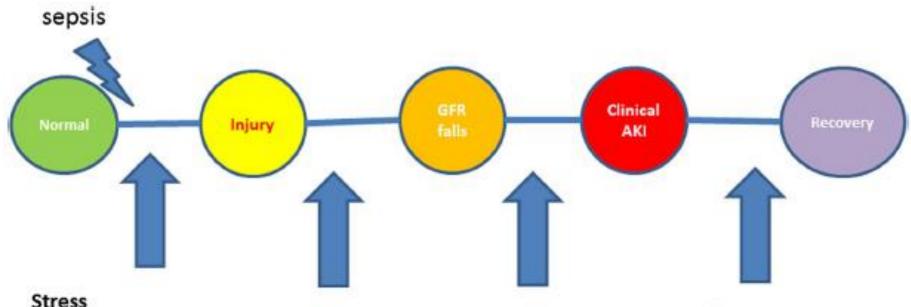








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Biomakers

(events: iron removal, cell cycle arrest) (Molecules: NGAL, TIMP-2, ILGFBP-7)

Injury Biomarkers

(events: enzyme release, cell exfoliation, protein loss) (Markers: tubular cells in urine, glutathione transferase in urine, KIM-1, albuminuria)

Functional loss biomarkers

(event: loss of excretion) (Markers: cystatin C, rise in creatinine, fall in UO)

Recovery Biomarkers

(fall in creatinine, increased UO, decrease in NGAL, decreased cystatin C, decreased Injury biomarkers

Fig. 1 Potential contribution of novel renal injury biomarkers to the detection, prevention, and treatment of septic AKI

^{*}NGAL: neutrophil gelatinase associated lipocalin; TIMP: tissue inhibitor of metallo-proteinase; ILGF8P: insulin like growth factor binding protein KIM: kidney injury molecule; UO: urinary output

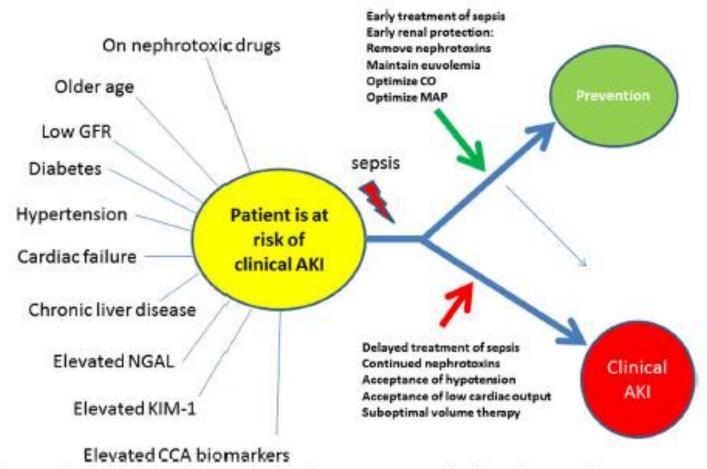
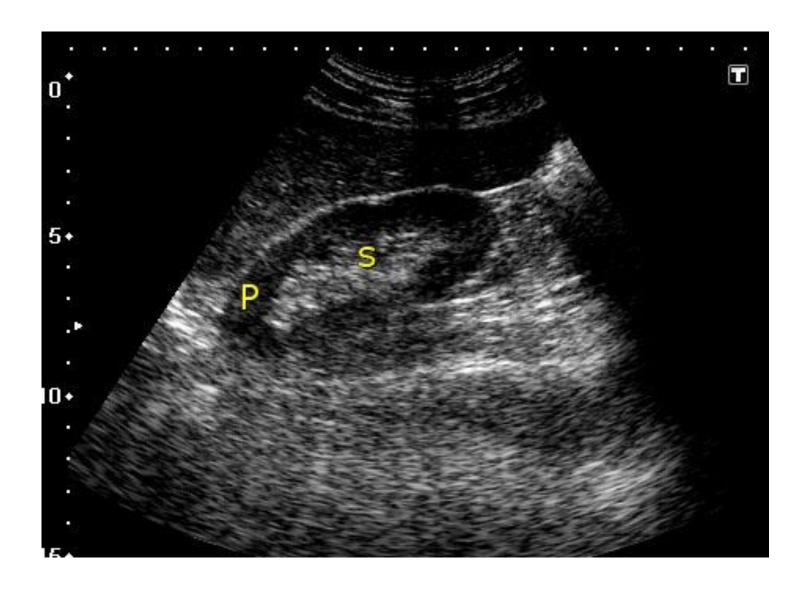
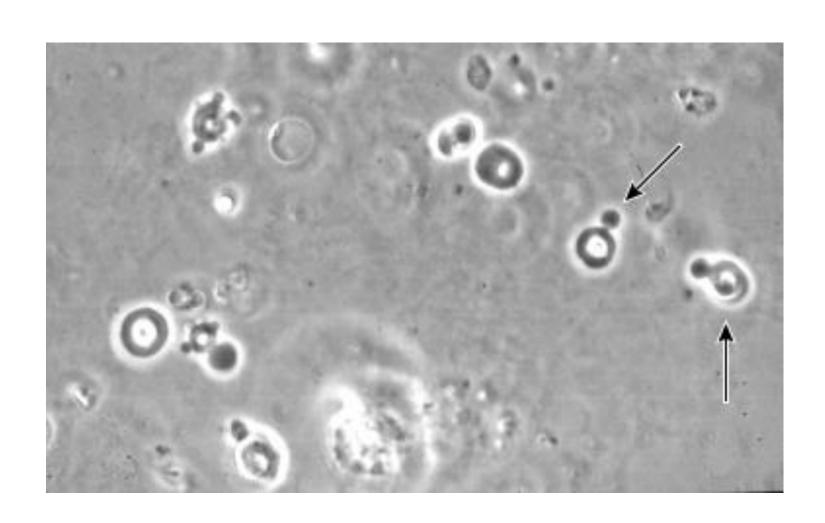


Fig. 2 Diagram illustrating how identification of a patient at risk of septic AKI can correctly inform all aspects of care





En resumen

Toda insuficiencia renal es aguda hasta que se demuestre lo contrario