Urinary indices: DDx

FeNa is the typical go to test for distinguishing between a prerenal cause or acute tubular necrosis as the cause of acute kidney injury. The formula for FeNa is:

$$\text{Fractional Excretion of Sodium} = \frac{\text{Urinary Sodium} \times \text{Serum Creatinine}}{\text{Urinary Creatinine} \times \text{Serum Sodium}} \times 100$$

A FeNa of <1% is typical of prerenal kidney injury, although there are exceptions. A poorly perfused kidney will do it's best to retain water and improve its perfusion and does so by excreting very little sodium, which increases H_2O absorption. A FeNa of >2% is typical of intrinsic kidney injury (although is often seen in post-obstructive AKI) and results from either directly damaged nephrons, which do poor job of sodium reabsorption, or necessarily high FeNa from the few remaining functional nephrons to adequately excrete dietary sodium after intrinsic kidney injury has reduced GFR.

FeNa, however, must be used with caution:

- Its use in patients with underlying chronic kidney disease is unclear
- FeNa should be used in severe AKI when GFR is adequately low. If GFR is still remotely normal, the FeNa will be low, even in cases of intrinsic renal disease due to the very large amount of sodium filtered every day.
- In patients with acute AKI, creatinine may not accurately represent GFR (which is what we are trying to measure with the serum creatinine level). For example, imagine clamping both renal arteries: the GFR is now zero but creatinine will only rise as quickly as it is produced and therefore may lag behind renal injury in the acute phase.
- Other causes of AKI, other than prerenal, can present with a FeNa of <1% including: radiocontrast and heme pigment injury, some non oliguric ATN and AIN cases, and early ATN following ischemic injury.
- Diuretics, by causing natriuresis, disrupt FeNa and artificially elevated the FeNa.

FeUrea, the fractional excretion of Urea, is calculated in a similar way (substitute serum and urinary urea) to the FeNa, however can be used in a situation involving a patient who has received a diuretic.

Lastly, there are a number of under-investigation biomarkers being looked at to help determine the cause of AKI, and predict it earlier. Creatinine is not ideal as it lags behind renal injury and gives no information as to the cause of the AKI. These renal biomarkers are promising and under investigation, but currently not in widespread use.

Further Reading:

Wasung ME, Chawla LS, Madero M. Biomarkers of renal function, which and when? Clin Chim Acta. 2015 Jan 1;438:350-7. doi: 10.1016/j.cca.2014.08.039. Epub 2014 Sep 3. PMID: 25195004.