

Fractional Excretion of Sodium (FENa) [Updated]

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Abstract

Fractional Excretion of Sodium (FENa) quantifies the proportion of filtered sodium that is excreted in the urine and is widely used to help distinguish between different causes of acute kidney injury (AKI), particularly in oliguric patients. FENa provides a more reliable measure of renal sodium handling than urine sodium concentration alone, as it accounts for urine volume and the influence of antidiuretic hormone. Despite its utility, FENa interpretation requires careful consideration of the clinical context, as low values may also occur in non-volume-depleted states such as glomerulonephritis, hepatorenal syndrome, and cardiorenal syndrome.

FENa is best applied in oliguric AKI patients without confounding factors such as diuretic use, chronic kidney disease, urinary tract obstruction, or acute glomerular diseases. No absolute FENa value can definitively diagnose pre-renal AKI, and serial measurements may provide additional diagnostic insight. The physiologic rationale for using a FENa <1% as a marker of pre-renal azotemia stems from the fact that at a normal glomerular filtration rate, this represents sodium excretion exceeding typical dietary intake. Ultimately, FENa should be interpreted as one piece of a broader clinical puzzle that includes history, physical examination, and other laboratory data.

HOW TO USE

When to Use

Patients with oliguria and/or acute kidney injury of unclear etiology.

Pearls and Pitfalls

FENa is only clinically validated in patients with oliguric acute kidney injury WITHOUT any of the following: diuretic use, chronic kidney disease (CKD), urinary tract obstruction, or acute glomerular disease.

Using urine Na concentration alone is less accurate because it does not account for urine volume and water handling of the kidney by antidiuretic hormone (ADH).

Single measurements of serum creatinine are “snapshots” in time and do not reflect true glomerular filtration rate (GFR). The most accurate measurement of GFR is the average of the sum of 24h creatinine clearance and urea clearance.¹⁻⁴

Why to Use

May give an additional data point in patients whose volume status is difficult to assess. It also provides a more ac-

curate assessment of kidney function than urine sodium alone (for example, a severely hypovolemic patient may have a relatively high urine sodium, as a fraction of total urine volume, despite having little sodium in the urine).^{1, 2}

NEXT STEPS

Advice

No absolute FENa indicates true “pre-renal” disease. Always consider history, clinical context, physical exam, and current medications. Obtaining repeat FENa or urine studies throughout a patient’s hospital course can give more clinical clues.

Non-volume depleted states with low urine sodium (and consequently low FENa) include acute glomerulonephritis, cardiorenal syndrome, hepatorenal syndrome, contrast-related nephropathy, and rarely, acute obstruction and early acute interstitial nephritis (AIN) or acute tubular necrosis (ATN).^{3,4}

EVIDENCE

Evidence Appraisal

A FENa cutoff of <1% for pre-renal azotemia was clinically validated in a prospective study, though there was overlap between patients with true pre-renal azotemia and acute tubular necrosis. Specificity for pre-renal azotemia (with a cutoff of <1%) was decreased in non-oliguric acute renal failure.²

At a normal GFR of 180 L/day and Na concentration of 140 mEq/L, the filtered sodium load is 26,100 mEq/day (=145 × 180). A FENa of 1% in this setting represents the excretion of 261 mEq/day. This is higher than the average sodium intake of 80 to 250 mEq/day. This is the physiologic basis for hypothesizing that patients with normal GFR have a FENa below 1%.

Validity: The formula is strongly supported by renal pathophysiology. The derivation is well described in the literature, and comparisons to clinical diagnoses of acute kidney injury subtypes have been performed. However, the absence of a single gold standard for AKI subtype diagnosis limits definitive validation.

Reliability: The formula performs consistently in specific contexts (e.g., oliguric AKI without diuretics), but external validations show variability in reliability when confounders (diuretics, CKD, sepsis) are present. There are also discrepancies in sensitivity/specificity across different studies and populations.

Applicability: The formula is easy to calculate and interpret in appropriate settings (oliguric AKI without interfering factors), but its usefulness is limited in patients on diuretics, those with CKD, or in non-oliguric AKI.

Limitations and sources of error: The formula assumes stable sodium handling and is prone to misclassification in the presence of diuretics, sepsis, or CKD, and alternative markers (e.g., FE urea, biomarkers of tubular injury) may provide better discrimination in complex cases.

Formula

$$\text{Fractional Excretion of Sodium (FENa)} = (\text{PCr} \times \text{UNa}) / (\text{PNa} \times \text{UCr}) \%$$

How the equation is derived:

FENa is a measure of tubular resorption of Na.

$$\text{FENa} = (\text{Na excreted} / \text{Na filtered}) \times 100$$

$$\text{Na excreted} = \text{UNa} \times \text{urine volume}$$

$$\text{Na filtered} = \text{PNa} \times (\text{UCr} \times \text{urine volume}) / \text{PCr}$$

Table 1. Interpretation of FENa formula

	Pre-Renal*	Intrinsic	Post-Renal
FENa	<1%	>1%	>4%
Urine Na (mmol/L)	<20	>40	>40

*Pre-Renal: Anything causing decreased effective renal perfusion: hypovolemia, heart failure, renal artery stenosis, [sepsis](#), etc.

REFERENCES

1. Espinel CH. The FENa test. Use in the differential diagnosis of acute renal failure. *JAMA*. 1976;236(6):579-581. doi:[10.1001/jama.1976.03270060029022](https://doi.org/10.1001/jama.1976.03270060029022)
2. Miller TR, Anderson RJ, Linas SL, et al. Urinary diagnostic indices in acute renal failure: a prospective study. *Ann Intern Med*. 1978;89(1):47-50. doi:[10.7326/0003-4819-89-1-47](https://doi.org/10.7326/0003-4819-89-1-47)
3. Pahwa AK, Sperati J. Urinary fractional excretion indices in the evaluation of acute kidney injury. *J Hosp Med*. 2016;11(1):77-80. doi:[10.1002/jhm.2501](https://doi.org/10.1002/jhm.2501)
4. Steiner RW. Interpreting the fractional excretion of sodium. *Am J Med*. 1984;77(4):699-702. doi:[10.1016/0002-9343\(84\)90368-1](https://doi.org/10.1016/0002-9343(84)90368-1)