

CASE H—EVALUATION OF DECREASED URINE OUTPUT

A 35 year old man with a history of alcohol abuse was admitted with loss of appetite and diarrhea; he also had a diagnosis of cirrhosis. He was begun on treatment with vancomycin because of a positive blood culture. By the fourth day on antibiotics, he was noted to have decrease in his urine output to less than 300 mL per day; urine electrolytes and osmolality were ordered. What is the differential diagnosis for a decrease in urine output? How do laboratory tests help in this case in deciding between the various causes?

TIME	Na	K	Cl	CO ₂	BUN	Creatinine	Glucose	Osmolality
Admission	136	3.3	103	28		0.6	125	n/a
4 d later	133	4.1	112	18	10	4.5	72	287
Urine (day 4)	10	22.5	19					187

In a patient with decreased urine output, the two major diagnoses considered are (1) decreased blood flow to the kidneys, often termed prerenal azotemia, and (2) acute damage to the tubules, usually due to acute damage to the tubules (usually “acute tubular necrosis”). In this case, the history would have been compatible with either diagnosis.

Cirrhosis often is associated with decreased blood flow to the outer part of the kidney where most glomeruli are found; this causes a decrease in kidney function called “hepatorenal syndrome”. In any case of prerenal azotemia, the kidneys attempt to regulate their blood flow by producing renin and, ultimately, aldosterone, which causes maximal sodium retention. The decreased blood volume causes production of ADH, producing a concentrated urine.

In acute tubular necrosis (ATN), however, the intrinsic damage to the tubules renders them incapable of responding to either hormone. Common causes of ATN are shock and exposure to drugs which damage the renal tubules, including aminoglycosides and drugs such as vancomycin. Typically, response to ADH is lost first, reducing free water clearance to zero and resulting in production of urine with an osmolality near that of serum. Sodium excretion typically rises within about 24 hours, due to the lack of proximal tubular sodium reabsorption. If aldosterone had been increased before this event takes place, the increased distal tubular sodium reabsorption may cause urine sodium excretion to be low for as long as 48-72 hours.

At the time of initial investigation, the low urine sodium excretion suggested the diagnosis of prerenal azotemia to the physicians. However, the low free water clearance in this case implied the presence of acute tubular necrosis. By the next day, urine output remained low but urine sodium excretion increased. The patient continued to have low urine output, and he ultimately required hemodialysis for a period of approximately 1 month before his renal function improved enough that he no longer needed to be dialyzed. His renal function never returned to normal. This case illustrates the importance of free water clearance as an early indicator of ATN.

cases of acute
renal failure
and dialysis