



DAVID L. LONGWORTH, MD, AND JAMES K. STOLLER, MD, EDITORS

## The clinical usefulness of urinalysis

**Q1** A 52-YEAR-OLD MAN WITH CHRONIC hypertension, atherosclerotic peripheral vascular disease, and a baseline serum creatinine concentration of 1.5 mg/dL is admitted for coronary artery bypass surgery. The postoperative course is complicated by two episodes of hypotension, pneumonia (treated with penicillin), and painful bluish lesions on the tips of three toes. Six days after surgery the serum creatinine concentration is 5.4 mg/dL.

The acute renal failure could be due to:

- ☐ Acute tubular necrosis
- ☐ Acute interstitial nephritis
- ☐ Bladder outlet obstruction
- ☐ Atheroembolic renal disease
- ☐ All of the above

**A1** Based on this patient's history, his acute postoperative renal failure could be due to any of the causes listed. In any older man who has undergone surgery, is confined to bed, and is receiving narcotics, bladder outlet obstruction should always be considered; it is easily detected and corrected. Acute tubular necrosis is likely because patients with abnormal kidney function and a serum creatinine concentration of 1.5 mg/dL are vulnerable to the adverse renal effects of hypotension. Acute interstitial nephritis is most commonly seen after treatment with beta-lactam antibiotics, which this patient received. Atheroembolic renal disease causing renal failure should be suspected in any patient who has extensive vascular disease, has had recent vascular or cardiovascular surgery, has had recent intra-arterial catheters, or has peripheral cutaneous indicators (livedo reticularis or microinfarcts of the

skin) of peripheral atheroembolization. In addition to the history, the urinalysis will frequently help to discriminate among causes of acute renal failure in cases such as this one. A urinalysis that shows no protein, blood, or abnormalities in the urine sediment points toward obstruction or prerenal causes of acute renal failure. What diagnosis would be suggested in this case by each of the following urinalyses?

**Q2** If the dipstick urinalysis showed 1+ protein and 2+ blood, microscopic examination revealed many white blood cells and red blood cells, and Hansel's stain was positive for eosinophils, the acute renal failure would most likely be due to:

- ☐ Acute tubular necrosis
- ☐ Acute interstitial nephritis
- ☐ Bladder outlet obstruction
- ☐ Atheroembolic renal disease
- ☐ All of the above

**A2** These findings could be produced by either acute interstitial nephritis or atheroembolic renal disease. Acute interstitial nephritis is an inflammatory renal interstitial injury caused by a reaction to a drug, most commonly the beta-lactam antibiotics (especially methicillin) and nonsteroidal anti-inflammatory drugs. The urinalysis most consistent with acute interstitial nephritis will show hematuria, leukocyturia, and eosinophiluria, though there are a considerable number of cases without all of these urinary features. In addition to the findings in the urine sediment and the temporal relationship between drug exposure and renal disease, other signs that suggest interstitial nephritis are fever, skin rash,

and eosinophilia. Discontinuation of the offending drug results in recovery of renal function in most cases.

Atheroembolic renal disease must be suspected in any patient with atherosclerotic vascular disease who has recently undergone cardiac or vascular surgery or insertion of intra-arterial lines or catheters. The urinalysis can produce the same results as in acute interstitial nephritis. Additional features that suggest atheroembolic renal disease include skin changes (livedo reticularis or small cutaneous infarctions on the feet or toes), eosinophilia (present in 70% to 80% of cases), and hypocomplementemia (present in 50% to 70% of cases). Definite diagnosis is made by biopsy showing vascular cholesterol crystals in the kidney, skin, or muscle. Most patients with atheroembolic renal disease do not recover renal function and many slowly worsen, presumably due to the continuation of the same microembolic process.

**Q3** If the urinalysis showed trace protein and no blood, and the sediment had many dirty brown casts, five to 10 renal tubular cell casts, and occasional white blood cell casts, the acute renal failure would most likely be due to:

- ☐ Acute tubular necrosis
- ☐ Acute interstitial nephritis
- ☐ Bladder outlet obstruction
- ☐ Atheroembolic renal disease
- ☐ All of the above

**A3** Acute tubular necrosis causes acute renal failure most commonly in hospitalized patients who have mildly to moderately impaired renal function (serum creatinine concentration > 1.5 mg/dL) and who suffer a superimposed insult such as hypotension, sepsis, or exposure to a nephrotoxin such as

an aminoglycoside or a radiocontrast agent. The urine sediment that shows many brown pigmented casts and renal tubular cells strongly supports this diagnosis. Because routine hospital laboratory urinalyses rarely detect these urine sediment features, the physician must examine the urine personally.

**Q4** A 60-year-old man comes to your office with a 3-month history of fatigue and gradually worsening edema of the feet and legs. The serum creatinine concentration is 1.4 mg/dL. The dipstick urinalysis shows 4+ protein and no blood. The sediment contains granular and hyaline casts and oval fat bodies. This presentation is consistent with:

- ☐ Glomerulonephritis with nephrotic proteinuria
- ☐ Acute interstitial nephritis
- ☐ Acute tubular necrosis

**A4** Glomerulonephritis with nephrotic proteinuria is the likely diagnosis. Acute interstitial nephritis and acute tubular necrosis do not cause heavy proteinuria or fat droplets in the urine. A urinalysis with 4+ protein and fat droplets always indicates glomerular disease (as contrasted with tubulointerstitial disease). The edema is likely secondary to the hypoalbuminemia, which often accompanies nephrotic syndrome.

PHILLIP M. HALL, MD  
Department of Nephrology  
and Hypertension  
The Cleveland Clinic Foundation

#### SUGGESTED READING

Rose BD, Black RM. Manual of clinical problems in nephrology. Boston: Little, Brown, and Co., 1988.