

	1	2	3	4	5	6	7	8	9	10
Specific gravity	1025	1010	1014	1010	1012	1010	1015	1015	1012	1.010
Dipstick: Protein	---	2+	3+	1+	1+	1+	4+	1+	2+	Trace
Blood	---	---	3+	1+	2+	3+	---	4+	4+	---
Sediment:	Hyaline casts	Waxy casts	10-15 wbc	0-4 wbc	25-30 wbc	TNTC wbc	No cells	TNTC rbc	50-100 rbc	No cells
	No cells	No cells	TNTC rbc	5-10 rbc	20-30 rbc	50-100 rbc	No casts	rbc casts	0-5 wbc	Waxy casts
			Rbc casts	Granular casts	Granular casts	Wbc casts	+ oval		Gran casts	
			Granular casts	Renal tubular	No bacteria	3+ bacteria	Fat bodies		Rbc casts	
				Epithelial cells						

Match the urinalysis:

- A) Asymptomatic chronic renal failure
 - B) CHF
 - C) Membranous Glomerulopathy
 - D) SLE (diffuse proliferative glomerulonephritis)
 - E) Acute tubular necrosis
 - F) Acute pyelonephritis
 - G) Allergic interstitial nephritis
1. decompensated CHF
 2. 48 yo male with creatinine of 2.8 mg/dl and 15 yr history of "protein" on urinalysis
 3. 38 yo BF with arthralgia, rash, thrombocytopenia, + ANA, and lower extremity edema
 4. 50 yo WM with pitting edema, cholesterol 400 mg/dl, creatinine 1.0 mg/dl, and albumin 2.3 mg/dl, normal BP
 5. 62 yo BF with 15 yr history of Type II diabetes mellitus
 6. 40 yo male developed rash, arthralgia, creatinine increase from 1.0 mg/dl to 2.7 mg/dl 4 wk after starting Dilantin
 7. UGI bleed
 8. 26 yo female with fever, dysuria, flank pain, nausea, vomiting, wbc count at 20,000 μm^3
 9. 50 yo male following emergency surgery for ruptured abdominal aortic aneurysm.
 10. 27 yo male IV drug user with fever, wbc count 18,000 μm^3 and new systolic murmur
 11. 48 yo male with palpable purpura of distal lower extremities, ESR 95, creatinine 3.2 mg/dl
 12. 65 yo male with rise in creatinine from 1.2 mg/dl to 2.1 mg/dl after 2 weeks of Gentamycin
 13. decompensated cirrhosis
 14. 80 yo male with chronic bladder outlet obstruction 2^o benign prostatic hypertrophy.

PATTERNS OF URINALYSIS

I. Casts: All types of casts formed within the renal tubules. The matrix of all casts is thought to be Tamm-Horsfall protein which is produced by cells of the thick ascending limb of Henle.

A. Types of Casts:

- (1) Hyaline casts: These are the only normal casts.
- (2) Granular casts: Pathologic casts which can be seen in either glomerular or tubular injury. It usually signifies that there is active injury present. The granules are thought to be fragments of cellular debris such as sloughed brush border membranes.
- (3) Fatty casts: A hyaline cast which contains fatty oil droplets, seen in pure nephrotic syndrome.
- (4) Cellular casts:
 - a. White blood cell casts: White blood cells incorporated in the matrix of the cast most commonly seen in pyelonephritis.
 - b. Red blood cell casts: Red cells in the matrix almost **pathognomonic** of glomerulonephritis, acute glomerular injury.
 - c. Renal tubular epithelial cell casts: Slough renal tubular epithelial cells incorporated in the matrix, seen in acute tubular injuries.
- (5) Waxy casts: This is a sign of chronic renal disease. It is non-specific and can be seen in either glomerular or tubular injury. It does not mean active injury, it simply reflects the presence of chronic renal disease.

II. Patterns of urinalysis:

Any pathologic process that leads to renal injury should lead to an abnormal urinalysis with potential changes in GFR which will be reflected in the serum creatinine.

- a. Nephrotic urine: This urine contains protein and lipid. It is a non-inflammatory, non-proliferative state.
- b. Nephritic urine: Nephritic implies active inflammation with cellular infiltration, i. e. proliferative changes. This will be manifest by the presence of varying levels of protein and almost invariably there will be hematuria, either gross or microscopic. Frequently there will be red blood cell casts.
- c. Mixed nephritic and nephrotic: There are signs of proliferation/inflammation with hematuria and potentially red blood cell casts, but there is also heavy proteinuria and lipiduria which frequently leads to signs of nephrotic syndrome.
- d. Tubular urine: In this situation only the tubules are injured, the glomeruli are not directly involved. This type urine, therefore, will not have heavy proteinuria. There may be microscopic hematuria since damaged tubules are surrounded by capillaries. There may be renal tubular epithelial cells, the hallmark is the presence of granular casts. It is invariably tubular injury that leads to loss of ability to concentrate the urine so specific gravity will typically be 1010. When urine fails to be diluted or concentrated, it ends up being in similar osmolality to plasma which is normally about 300. Thus, a specific gravity of 1010 correlates with a urine osmolality of about 300 (which is similar to plasma).

- (1) Non-inflammatory tubular injury: This would be typical of ischemia (ATN) or direct tubular toxins such as aminoglycosides. In this pattern there is no evidence of cellular infiltration damaging tubules. Thus, there is very little pyuria.
- (2) Inflammatory tubulitis (tubular injury): In this situation there is cellular infiltrate directly injuring tubules such as allergic interstitial nephritis. In this situation there will almost invariably be sterile pyuria present in the urinalysis.
- (3) Pyelonephritis is infected tubules. Thus, there is pyuria and bacteruria.
- (4) Obstructive uropathy will typically present with the tubular pattern of urine. There may be red blood cells if the urinary tract is being injured by tumor or trauma.

E. Mesangial Pattern: Mesangial pattern shows microscopic or gross hematuria and probably red blood cell casts in the absence of major proteinuria.

These patterns of urinalysis can be correlated with renal histology. A renal injury could occur by damaging the microcirculation such as the afferent and efferent arterioles (vasculitis), or by directly injuring the glomerulus proper, or by directly injuring renal tubules. Glomerular injury can be broken down into diseases that primarily affect the glomerular basement membrane or which affect mainly the mesangium. Glomerular injury can also be thought of as those which have active inflammation or without inflammation. Diseases affecting only the glomerular basement membrane in a non-inflammatory manner should lead to a pure nephrotic urine. Diseases which have active proliferative inflammation that involve the mesangium only lead primarily to hematuria. Diseases which involve active inflammation/proliferation involving both the mesangium and the capillary loop should result in a nephritic urine. Disease only involving the tubules should lead to the tubular pattern of urine. Diseases involving the microcirculation such as the afferent arteriole will lead to altered glomerular filtration rate and frequently to signs of glomerular injury with proteinuria and hematuria.