INTRODUCTION
Examination of urine is an indispensable part of evaluation of patients with impaired kidney function, particularly proteinuria, hematuria, urinary tract infection, nephrolithiasis and other renal or nonrenal diseases. The relatively simple chemical test performed in the routine urinalysis rapidly provides important information about primary kidney disorder and systemic diseases. Examination of urine sediment provides valuable information about renal parenchyma. Dipstick test can be automated and there is no substitute for careful examination of urine under the microscope. Experience in examining the urine is valuable; studies show that a urinalysis performed by an experienced nephrologist or trained physician is more likely to be better than a urinalysis reported by a clinical chemistry laboratory.

SPECIMEN COLLECTION AND HANDLING
Urine should be collected with minimum contamination.
• Clean catch midstream collection is performed
• If not feasible, bladder catheterization is appropriate for adults—risk of contracting a urinary tract infection is negligible for a single catheterization
• Suprapubic aspiration is used in infants
• High urine osmolality and low pH favor cellular preservation, hence first voided morning urine is preferred
• Chemical composition of urine changes with standing and formed elements degrade over time. Hence, urine is best examined when fresh but a brief period of refrigeration is acceptable
• Bacteria in urine multiply at room temperature, hence bacterial counts from unrefrigerated urine are unreliable (Table 1).

ROUTINE URINALYSIS
Appearance
Selected substance that may alter the physical appearance or odor of the urine has been shown in Table 2.

Specific Gravity
Specific gravity = \frac{\text{Weight of urine of measured volume}}{\text{Weight of distilled water of some volume}}\]

Urine specific gravity is inaccurate surrogate for osmolality.
Urinary specific gravity is usually between 1.001 and 1.035 and urine osmolality is usually between 50–1,000 mOsm/kg.

Methods for Measurement of Specific Gravity
• By hydrometer: Sufficient volume of urine required.
• By refractometer: Calibrated in specific gravity units, a drop of urine is required.
• By dipstick: Small amount of urine is required.

Clinical Importance of Measuring Specific Gravity
• In the absence of proteinuria, glycosuria or iodinated contrast administration, a specific gravity more than 1.018 implies preserved concentrating ability of kidneys.
• Measurement is useful to differentiate prerenal azotemia and acute tubulointerstitial nephritis (ATIN).
Chapter 127  
Urinalysis in Clinical Practice

Section 17

Chemical Composition of Urine

Routine Dipstick Methodology

Proper tabs impregnated with chemical reagents are fixed to a plastic strip. Reagents are chromogenic altered with a chest, which is highly specific. Some interfering substance in urine or extremes of pH may alter the results.

Physiologic urinary pH lies between 4.5 and 8.

• Growth of urea splitting bacteria and loss of carbondioxide (CO₂) raise the pH
• Bacterial metabolism of glucose may produce organic acids and lowers the pH
• These strips are not sufficiently accurate to be used for the diagnosis of renal tubular acidosis (RTA).

Protein Dipstick Method

Protein indicator strips are buffered at an acid pH near their color change point; wetting them with a protein containing specimen indicates a color change.

Protein reaction may be scored as follows:

- Trace = 5–20 mg/dL
- 1+ = 30 mg/dL
- 2+ = 100 mg/dL
- 3+ = 300 mg/dL
- 4+ = > 2,000 mg/dL

Protein strips are highly sensitive to albumin but less so to globulins, hemoglobin or light chains.

Acid Precipitation Test: Turbidimetric Method

- Urine that is negative by dipstick but positive with sulphosalicylic acid is highly suspicious for light chains.
- Tolbutamide, high-dose penicillin, sulfonic acid and radiographic-contrast agents may yield false-positive turbidimetric reactions.
- Protein indicator used for routine dipstick analysis is not sensitive enough to detect microalbuminuria.

Protein Composition of Urine

Protein composition of normal urine is represented in Table 3. Causes of proteinuria according to pathophysiology are shown in Table 4.

### Table 3: Protein composition of normal urine

<table>
<thead>
<tr>
<th>Plasma proteins</th>
<th>Excretion (mg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin</td>
<td>12</td>
</tr>
<tr>
<td>Immunoglobulin G</td>
<td>3</td>
</tr>
<tr>
<td>Immunoglobulin A</td>
<td>1</td>
</tr>
<tr>
<td>Immunoglobulin M</td>
<td>0.3</td>
</tr>
<tr>
<td>Light chains</td>
<td></td>
</tr>
<tr>
<td>κ</td>
<td>2.3</td>
</tr>
<tr>
<td>λ</td>
<td>1.4</td>
</tr>
<tr>
<td>β-microglobulins</td>
<td>0.12</td>
</tr>
<tr>
<td>Other plasma proteins</td>
<td>20</td>
</tr>
<tr>
<td>All plasma proteins</td>
<td>40</td>
</tr>
<tr>
<td>Non-plasma proteins</td>
<td>40</td>
</tr>
<tr>
<td>Tamm-Horsfall protein</td>
<td>40</td>
</tr>
<tr>
<td>Other nonrenal derived proteins</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>All non-plasma proteins</td>
<td>40</td>
</tr>
<tr>
<td>Total proteins</td>
<td>80 ± 24 (SD)</td>
</tr>
</tbody>
</table>

Abbreviation: SD, Standard deviation


Proteinuria is evaluated on the basis of their origin and types, which is further investigated in Table 5 and represented in Flow chart 1.

### CHEMICAL COMPOSITION OF THE URINE

Routine Dipstick Methodology

The urine dipstick is a plastic strip to which paper tabs impregnated with chemical reagents have been affixed. The reagents in each are chromogenic. After timed development, the color on the paper segment is compared with a chart. Some reactions are highly specific. Others are sensitive to the presence of interfering substance or extremes of pH. Discoloration of the urine bilirubin or blood may obscure the color.

**pH**

The pH test pads use indicator days that change color with pH. The physiologic urine pH ranges from 4.5 to 8. The determination is most
Nephrology

**TABLE 4 | Causes of proteinuria according to pathophysiology**

**Glomerular proteinuria**
- Primary glomerular disease
  - Minimal change glomerulopathy
  - Immunoglobulin A nephropathy
  - Focal segmental glomerulosclerosis (FSGS)
  - Membranous glomerulonephritis
  - Membranoproliferative glomerulonephritis (MPGN)
  - Fibrillary and immunotactoid glomerulopathy
  - Crescentic glomerulonephritis
- Secondary glomerular disease
  - Multisystem disease: Systemic lupus erythematosus (SLE), vasculitis, amyloid, scleroderma
  - Metabolic disease: Diabetes mellitus, Fabry’s disease
  - Neoplasia: Myeloma, leukemia, solid tumors
  - Infections: Bacterial, fungal, viral, parasitic
  - Drugs, toxins and allergens: Gold, penicillamine, lithium, nonsteroidal anti-inflammatory drug (NSAID), penicillin
  - Familial: Congenital nephrotic syndrome, Alport’s syndrome, nephropathy
- Others: Toxemia of pregnancy, transplant nephropathy, reflux nephropathy
  - Glomerular proteinuria without renal disease
  - Exercise induced, orthostatic, febrile proteinuria

**Tubular proteinuria**
- Drugs and toxins
  - Luminal injury: Light-chain nephropathy, lysozyme (myelogenous leukemia)
  - Exogenous: Heavy metals (lead, mercury, cadmium), tetracycline, aristolochic acid (Balkan nephropathy)
- Tubulointerstitial nephritis
  - Hypersensitivity (drug, toxin)
- Multisystem: SLE, Sjögren’s syndrome, tubulointerstitial nephritis with uveitis
- Others
  - Fanconi’s syndrome

**Overflow proteinuria**
- Myeloma, light-chain disease, amyloidosis, hemoglobinuria, myoglobinuria

**Tissue proteinuria**
- Acute inflammation of urinary tract
- Uroepithelial tumors

**TABLE 5 | Evaluation of proteinuria based on type**

<table>
<thead>
<tr>
<th>Origin of proteinuria</th>
<th>Investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glomerular</strong></td>
<td>Quantify by protein-to-creatinine ratio or 24-hour protein excretion</td>
</tr>
<tr>
<td></td>
<td>Measure serum albumin and cholesterol serology work-up</td>
</tr>
<tr>
<td></td>
<td>Plasma and urine protein electrophoresis (UPEP)</td>
</tr>
<tr>
<td><strong>Tubular</strong></td>
<td>Alpha-microglobulin, retinol-binding protein, β2-microglobulin-to-albumin ratio</td>
</tr>
<tr>
<td></td>
<td>Heavy metal screen</td>
</tr>
<tr>
<td></td>
<td>Plasma and urine electrophoresis</td>
</tr>
<tr>
<td><strong>Abnormal plasma proteins</strong></td>
<td>Serum protein electrophoresis</td>
</tr>
<tr>
<td></td>
<td>UPEP and Bence-Jones protein</td>
</tr>
<tr>
<td></td>
<td>Erythrocyte hemolysis</td>
</tr>
<tr>
<td></td>
<td>Reticulocyte count, blood film</td>
</tr>
<tr>
<td></td>
<td>Lactate dehydrogenase</td>
</tr>
<tr>
<td></td>
<td>Haptoglobin</td>
</tr>
</tbody>
</table>

Protein

Protein measurement uses the protein error of indicators principle. The pH at which some indicators change color varies with the protein concentration of the bathing solution. Protein indicator strips are buffered at an acid pH near their color change point. Wetting them with a protein containing specimen induces a color change. The protein reaction may be scored from trace to +4 or by concentration. Highly alkaline urine, especially after contamination with quaternary ammonium skin cleansers, may produce false-positive reactions.

Protein strips are highly sensitive to albumin but less so to globulins, hemoglobin or light chain. If light-chain proteinuria is suspected, more sensitive assays should be used. With acid precipitation test, an acid that denatures protein is added to the urine specimen and the density of the precipitate is related to the protein concentration. Urine that is negative by dipstick but positive with sulfosalicylic acid is highly suspicious for light chains. Tolbutamide, high-dose penicillin, sulfonamides, and radiographic-contrast agents may yield false-positive turbidimetric reactions. More sensitive and specific tests for light chains are preferred.

If the urine is very concentrated, the presence of a modest protein reaction is less likely to correspond to significant proteinuria in a 24-hour collection. Even so, it is unlikely that a 3+ or 4+ reaction would be seen solely because of a high urine concentration or conversely that the urine would be dilute enough to give a negative reaction despite significant proteinuria. The protein indicator used for routine dipstick analysis is not sensitive enough to detect microalbuminuria.

Blood

Reagent strips for blood rely on the peroxidase activity of hemoglobin to catalyze organic peroxide with subsequent oxidation of an indicator dye. Free hemoglobin produces a homogeneous color. Intact red cells cause punctate staining. False-positive reactions occur if the urine is contaminated with other oxidants such as povidone-iodine, hypochlorite or bacterial peroxidase. Ascorbate yields false-negative results. Myoglobin is also detected, because it has intrinsic peroxidase activity. A urine sample that is positive for blood by dipstick analysis but shows no red cells on microscopic examination is suspected for myoglobinuria or hemoglobinuria. Pink discoloration of serum may occur with hemolysis, but free myoglobin is seldom present in a concentration sufficient to change the color of plasma.

Specific Gravity

Specific gravity reagent strips actually measure ionic strength using indicator dyes with ionic strength-dependent dissociation constants (pKa). They do not detect glucose or nonionic radiographic-contrast agents.

Glucose

Modern dipstick reagent strips are specific for glucose. They rely on glucose oxidase to catalyze the formation of hydrogen peroxide, which reacts with peroxidase and a chromogen to produce a color change. High concentrations of ascorbate or ketoadic acids reduce test sensitivity. However, the degree of glycosuria occurring in diabetic ketoadidosis is sufficient to prevent false-negative results despite ketonuria.

Ketones

Ketone reagent strips depend on the development of a purple color after acetoacetate reacts with nitroprusside. Some strips can also detect acetone, but none react with β-hydroxybutyrate. False-positive results may occur in patients who are taking levodopa or drugs such as captopril or mesna that contain free-sulhydril groups.
Urobilinogen

Urobilinogen is a colorless pigment that is produced in the gut from metabolism of bilirubin. Some is excreted in feces and the rest is reabsorbed and excreted in the urine. In obstructive jaundice, bilirubin does not reach the bowel, and urinary excretion of urobilinogen is diminished. In other forms of jaundice, urobilinogen is increased. The urobilinogen test is based on the Ehrlich reaction, in which diethylaminobenzaldehyde reacts with urobilinogen in acid medium to produce a pink color. Sulfonamides may produce false-positive results and degradation of urobilinogen to urobilin may yield false-negative results. Better tests are available to diagnose obstructive jaundice.

Bilirubin

Bilirubin reagent strips rely on the chromogenic reaction of bilirubin with diazonium salts. Conjugated bilirubin is not normally present in the urine. False-positive results may be observed in patients receiving chlorpromazine or phenazopyridine. False-negative results occur in the presence of ascorbate.

Nitrite

The nitrite screening test for bacteriuria relies on the ability of Gram-negative bacteria to convert urinary nitrate to nitrite, which activates chromogen. False-negative results occur when there is infection with Enterococcus or other organisms that do not produce nitrite, when ascorbate is present or when urine has not been retained in the bladder long enough (approximately 4 hours) to permit sufficient production of nitrite from nitrate.

Leukocytes

Granulocyte esterases can cleave pyrrole amino acid esters, producing free pyrrole that subsequently reacts with a chromogen. The test threshold is five to fifteen white blood cells (WBCs) per high-power field (HPF). False-negative results occur with glycosuria, high-specific gravity, cephalixin or tetracycline therapy or excessive
oxalate excretion. Contamination with vaginal debris may yield a
positive test result without true urinary tract infection.

**Microalbumin Dipstick**

Albumin-selective dipsticks are available for screening for
microalbuminuria in patients with incipient diabetic nephropathy.
The most accurate screening occurs when first morning specimens
are examined, because exercise can increase albumin excretion.
One type of dipstick uses colorimetric detection of albumin
bound to gold-conjugated antibody. Normally, the urine albumin
concentration is less than the 20 µg/L detection threshold for these
strips. Unless the urine is very dilute, a patient with no detectable
albumin by this method is unlikely to have microalbuminuria.

Because urine concentration varies widely, however, this assay has
the same limitations as any test that only measures concentration.
This strip is useful only as a screening test and more formal testing is
required if albuminuria is found.

A second type of dipstick has tabs for measurement of both
albumin and creatinine concentration and permits calculation of
the albumin-to-creatinine ratio. In contrast to the other dipstick tests
described in the chapter, these strips cannot be read by simple visual
comparison with a color chart. An instrument is required, but this
system is suitable for point-of-care testing. When present on more
than one determination, an albumin-to-creatinine ratio of 30–300
µg/mg signifies microalbuminuria.

**Microscopic Examination of the Spun Urinary Sediment**

**Specimen Preparation and Viewing**

The contents of the urine are reported as the number of cells or casts
per HPF (400X) after resuspension of the centrifuged pellet in a
small volume of urine. The accuracy and reproducibility of this semi-
quantitative method depends on using the correct volume of urine.
Twelve milliliters of urine should be spun in a conical centrifuge tube
for 5 minutes at 1,500–2,000 rpm (450 g). After centrifugation, the tube
is inverted and drained. The pellet is resuspended in the few drops of
urine that remain in the tube after inversion by flicking the base of the
tube gently with a finger or with the use of a pipette. Care should be
taken to fully suspend the pellet without excessive agitation.

A drop of urine is poured onto a microscope slide or transferred
with the pipette. The drop should be sufficient in size that a standard
22 mm × 22 mm coverslip just floats on the urine. If too little is used, the
specimen rapidly dries out. If an excess of urine is applied, it will
spill onto the microscope objective or stream distractingly under
the coverslip. Rapid commercial urine stains or the Papanicolaou
stain may be used to enhance details. Most nephrologists prefer the
convenience of viewing unstained urine. Subdued light is necessary.
The condenser and diaphragm are adjusted to maximize contrast
and definition. When the urine is dilute and few formed elements
are present, detection of motion of objects suspended in the urine
ensures that the focal plane is correct. One should scan the urine
at low power (100X) to get a general impression of its contents
before moving to high power (400X) to look at individual fields.
It is useful to scan large areas at low power and then move to high
power when a structure of interest is identified. Cellular elements
should be quantitated by counting or estimating the number in at
least 10 representative HPFs. Casts may be quantitated by counting
the number per low-power field, although most observers use less-
specific terms such as rare, occasional, few, frequent and numerous.

**Cellular Elements**

Cellular elements in the urine have been shown in [Figures 1A to F](#).

**Erythrocytes**

Red blood cells (RBCs) may find their way into the urine from
any source between the glomerulus and the urethral meatus.
The presence of more than two to three erythrocytes per HPF is
considered as pathologic. Erythrocytes are biconcave disks 7 µm in
diameter. They become crenated in hypertonic urine. In hypotonic
urine, they swell or burst, leaving ghosts. Erythrocytes originating
in the renal parenchyma are dysmorphic, with spicules, blebs,
submembrane cytoplasmic precipitation, membrane folding and
vesicles. Those originating in the collecting system retain their
uniform shape. Some experienced observers reported success
differentiating renal parenchymal from collecting system bleeding
by systematic examination of erythrocytes using phase contrast
microscopy (Flow chart 2).

**Leukocytes**

Polymorphonuclear leukocytes (PMNs) are approximately 12 µm in
diameter, and are most readily recognized in a fresh urine sample
before their multilobed nuclei or granules have degenerated.
Swollen PMNs with prominent granules displaying Brownian motion
are termed “glitter” cells. Polymorphonuclear leukocytes indicate
urinary tract inflammation. They may occur with intraparenchymal
diseases such as glomerulonephritis or interstitial nephritis. They are
a prominent feature of upper or lower urinary tract infection. They
also may appear with periureteral inflammation, as in regional ileitis
or acute appendicitis.

**Renal Tubular Epithelial Cells**

Tubular cells are larger than PMNs, ranging from 12 µm to 20 µm in
diameter. Proximal tubular cells are oval or egg-shaped, and tend to
be larger than the cuboidal distal tubular cells, but because their size
varies with urine osmolality, they cannot be reliably differentiated.
In hypotonic urine, it may be difficult to distinguish tubular cells
from swollen PMNs. A few tubular cells may be seen in a normal
urine sample. More commonly, they indicate tubular damage or
inflammation from acute tubular necrosis (ATN) or interstitial
nephritis.

**Other Cells**

Squamous cells of urethral, vaginal or cutaneous origin are large,
flat cells with small nuclei. Transitional epithelial cells line the
renal pelvis, ureter, bladder and proximal urethra. They are rounded
cells several times the size of leukocytes and often occur in clumps.
In hypotonic urine, they may be confused with swollen tubular
epithelial cells.

**Casts and Other Formed Elements**

Based on their shape and origin, casts are appropriately named.
Immunofluorescence studies demonstrate that they consist of a
matrix of Tamm-Horsfall urinary glycoprotein (uromodulin)
in the shape of the distal tubular or collecting duct segment in which
they were formed. The matrix has a straight margin that is helpful in
differentiating casts from clumps of cells or debris (Figures 2A to E).

**Hyaline Casts**

Hyaline casts consist of the protein alone. Because their refractive
index is close to that of urine, they may be difficult to see, requiring
subdued light and careful manipulation of the iris diaphragm.
Hyaline casts are nonspecific. They occur in concentrated normal
urine as well as in numerous pathologic conditions.

**Granular Casts**

Granular casts consist of finely or coarsely granular material.
Immunofluorescence studies show that fine granules are derived
from altered serum proteins. Coarse granules may result from degeneration of embedded cells. Granular casts are nonspecific but are usually pathologic. They may be seen after exercise or with simple volume depletion and as a finding in ATN, glomerulonephritis or tubulointerstitial disease.

**Waxy Casts**

Waxy casts or broad casts are made of hyaline material with a much greater refractive index than hyaline casts—hence, their waxy appearance. They behave as if they were more brittle than hyaline casts and frequently have fissures along their edge. Broad casts form in tubules that have become dilated and atrophic due to chronic parenchymal disease.

**Red Blood Cell Casts**

Red blood cell casts indicate intraparenchymal bleeding. The hallmark of glomerulonephritis, they are seen less frequently with tubulointerstitial disease. Red blood cell casts have been described along with hematuria in normal individuals after exercise.

Fresh RBC casts retain their brown pigment and consist of readily discernible erythrocytes in a tubular cast matrix. Over time, the heme color is lost, along with the distinct cellular outline. With further degeneration, RBC casts are hard to distinguish from coarsely granular casts. Red blood cell casts may be diagnosed by the company they keep. They appear in a background of hematuria with dysmorphic red cells, granular casts and proteinuria. Occasionally, the evidence for intraparenchymal bleeding is a hyaline cast.
Nephrology

with embedded red cells. These have the same pathophysiologic implication as RBC casts.

White Blood Cell Casts
White blood cell casts consist of WBCs in a protein matrix. They are characteristic of pyelonephritis and are useful distinguishing that disorder from lower urinary tract infection. They may also be seen with interstitial nephritis and other tubulointerstitial disorders.

Tubular Cell Casts
Tubular cell casts consist of a dense agglomeration of sloughed tubular cells or just a few tubular cells in a hyaline matrix. They occur in concentrated urine but are more characteristically seen with the sloughing of tubular cells that occurs with ATN.

Bacteria, Yeast and Other Infectious Agents
Bacillary or coccoid forms of bacteria may be discerned even on an unstained urine sample. Examination of a Gram-stain preparation of unspun urine allows estimation of the bacterial count. One organism per HPF of unspun urine corresponds to 20,000 organisms per cubic millimeter. Individual and budding yeasts and hyphal forms occur with Candida infection or colonization. Candida organisms are similar in size to erythrocytes, but they are greenish spheres, not biconcave disks. When budding forms or hyphae are present, yeast cells are obvious. Trichomonas organisms are identified by their teardrop shaped motile flagellum.

Lipiduria
In the nephrotic syndrome with lipiduria, tubular cells reabsorb luminal fat. Sloughed tubular cells containing fat droplets are called over fat bodies. Fatty casts contain lipid-laden tubular cells or free-lipid droplets. By light microscopy, lipid droplets appear round and clear with a green tinge. Cholesterol esters are anisotropic; cholesterol-containing droplets rotate polarized light, producing a “Maltese cross” appearance under polarized light. Triglycerides appear similar by light microscopy, but they are isotropic. Crystals, starch granules, mineral oil and other urinary contaminants are also anisotropic. Before concluding that anisotropic structures are lipid, the observer must compare polarized and bright-field views of the same object (Figures 3A to D).

Crystals
Crystals may be present spontaneously, or they may precipitate with refrigeration of a specimen. They can be difficult to identify because they have similar shapes. The pH is an important clue to identity, because solubility of a number of urinary constituents

Flow chart 2: Evaluation of hematuria

Abbreviations: RBCs, Red blood cells; WBC, White blood cell; UA, Uric acid; GBM, Glioblastoma multiforme; ANCA, Antineutrophil cytoplasmic antibody; VDRL, Venereal Disease Research Laboratory; HIV, Human immunodeficiency virus; ASLO, Antisteptolysin O; IVP, Intravenous pyelography; CT, Computed tomography
is pH dependent. The three most distinctive crystal forms are: (1) cystine, (2) calcium oxalate and (3) magnesium ammonium (triple) phosphate. Cystine crystals are hexagonal plates that resemble benzene rings. Calcium oxalate crystals are classically described as "envelope-shaped" but when viewed as they rotate in the urine under the microscope, they appear bipyramidal. Coffin lid-shaped triple, phosphates are rectangular with beveled ends. Oxalate may also occur in dumbbell-shaped crystals. Urate may have several forms, including rhomboids or needles.

**Characteristic Urine Sediments**

The urine sediment is a rich source of diagnostic information. Occasionally, a single finding (e.g. cystine crystals) is pathognomonic. More often, the sediment must be considered as a whole and interpreted in conjunction with clinical and other laboratory findings. Several patterns bear emphasis. In the acute nephritic syndrome, the urine may be pink or pale brown and turbid. Blood and moderate proteinuria are detected by dipstick analysis. The microscopic examination shows RBCs and RBC casts as well as granular and hyaline casts; WBC casts are rare. In the nephrotic syndrome, the urine is clear or yellow. Increased foaming may be noted because of the elevated protein content. In comparison with the sediment of nephritic patients, the nephrotic sediment is bland. Hyaline casts and lipiduria with oval fat bodies or lipid-laden casts predominate. Granular casts and a few tubular cells may also be present, along with few RBCs. With some forms of chronic glomerulonephritis, a "telescoped" sediment is observed. This term refers to the presence of the elements of nephritic sediment together

---

**Figures 2A to E:**

- **A:** Hyaline cast; **B:** Muddy-brown granular casts and amorphous debris from a patient with acute tubular necrosis (ATN) (original magnification 100X); **C:** Waxy cast (open arrows) and granular cast (solid arrow) from a patient with lupus nephritis and a telescoped sediment. Note background hematuria; **D:** Red blood cell (RBC) cast. Background hematuria is also present; **E:** Tubular cell cast. Note the hyaline cast matrix.
Figures 3A to D: Lipid: (A) Oval fat bodies, as seen by bright-field illumination; (B) Same field as in A viewed under polarized light; (C) Lipid-laden cast, bright-field illumination; (D) Same field as in C viewed under polarized light.

### TABLE 6 | Common urinary crystals

<table>
<thead>
<tr>
<th>Description</th>
<th>Composition</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crystals found in acid urine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amorphous</td>
<td>Uric acid (UA)</td>
<td>Cannot be distinguished from amorphous phosphates except by urine pH; may be orange tinted by urochromes</td>
</tr>
<tr>
<td>Rhomboid</td>
<td>Sodium urate</td>
<td></td>
</tr>
<tr>
<td>Rosettes</td>
<td>UA</td>
<td></td>
</tr>
<tr>
<td>Bipyramidal</td>
<td>Calcium oxalate</td>
<td>Also termed “envelope-shaped”</td>
</tr>
<tr>
<td>Dumbbell-shaped</td>
<td>Calcium oxalate</td>
<td></td>
</tr>
<tr>
<td>Needles</td>
<td>UA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sulfur drugs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Radiographic-contrast material</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cystine</td>
<td></td>
</tr>
<tr>
<td>Hexagonal plates</td>
<td>Cystine</td>
<td>Presence may be confirmed with nitroprusside test</td>
</tr>
<tr>
<td><strong>Crystals found in alkaline urine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amorphous</td>
<td>Phosphates</td>
<td>Indistinguishable from urates except by pH</td>
</tr>
<tr>
<td>“Coffin lid” (beveled rectangular prisms)</td>
<td>Triple (magnesium ammonium phosphate)</td>
<td>Seen with urea-splitting infection and bacteriuria</td>
</tr>
<tr>
<td>Granular masses or dumbbells</td>
<td>Calcium carbonate</td>
<td>Larger than amorphous phosphates</td>
</tr>
<tr>
<td>Yellow-brown masses with or without spicules</td>
<td>Ammonium biurate</td>
<td>—</td>
</tr>
<tr>
<td>Plate-like rectangles, fan-shaped, starburst</td>
<td>Indinavir</td>
<td>Causes nephrolithiasis or renal colic. <em>In vitro</em> solubility increased at very low pH. The lowest urine pH achievable <em>in vivo</em> may not actually be acid enough to lessen crystalluria.</td>
</tr>
</tbody>
</table>
with broad or waxy casts, indicative of tubular atrophy and dipstick findings of heavy proteinuria. In pyelonephritis, WBC casts and innumerable WBCs are present, along with bacteria. In lower tract infections, WBC casts are absent. The sediment in ATN shows tubular cells, tubular cell casts and muddy-brown granular casts. The typical urinary findings in individual kidney disorders are discussed in their respective chapters (Table 6 and Figures 4A to E).

BIBLIOGRAPHY