

Urinalysis in the diagnosis of kidney disease

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INTRODUCTION

The urinalysis is an informative and noninvasive diagnostic tool that is readily accessible to the clinician in both the ambulatory and hospital settings. In conjunction with the history, physical examination, and serum chemistries, the urinalysis plays a central role in evaluating acute and chronic kidney disease. In addition, abnormal findings on a routine urinalysis, even in an otherwise asymptomatic patient, may be the first evidence of underlying kidney disease. The urinalysis can also be used in some patients to monitor the course of established kidney disease.

Interpretation of the urinalysis in patients with established or suspected kidney disease will be presented in this topic. Assessment of kidney function, a general approach to the patient with kidney disease, an overview of the indications for kidney biopsy, and the differential diagnosis and evaluation of glomerular disease are discussed separately.

- (See ["Assessment of kidney function"](#).)
 - (See ["Diagnostic approach to adult patients with subacute kidney injury in an outpatient setting"](#).)
 - (See ["The kidney biopsy"](#).)
 - (See ["Glomerular disease: Evaluation and differential diagnosis in adults"](#).)
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WHEN TO PERFORM A COMPLETE URINALYSIS

A complete urinalysis consists of three components: gross evaluation, dipstick analysis, and microscopic examination of the urine sediment. These components are discussed in detail below. (See ['Gross assessment'](#) below and ['Urine dipstick'](#) below and ['Urine sediment'](#) below.)

A complete urinalysis should be performed in the following settings:

- In a patient with evidence of kidney disease, such as someone with albuminuria or an acute or chronic reduction in the glomerular filtration rate.
 - In a patient with suspected kidney disease. Kidney disease may be suspected on the basis of clinical findings (eg, edema) or because of a concurrent illness or condition that is commonly associated with kidney disease (eg, systemic lupus erythematosus, small-vessel vasculitis, newly identified hypertension).
 - In an otherwise asymptomatic patient in order to clarify the significance of incidental findings noted on urine dipstick analyses (eg, microscopic hematuria) when the dipstick was part of a workup for another condition (eg, hypertension, diabetes mellitus).
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OBTAINING THE SPECIMEN FOR ANALYSIS

The urine specimen must be properly collected in order to reliably interpret the findings and thus maximize diagnostic utility. The following technique should be followed whenever feasible [1]:

- The specimen should be collected into a clean dry container.
 - Patients should be asked to clean the external genitalia and provide a midstream specimen for analysis.
 - In patients with indwelling urinary catheters, a recently produced urine sample should be obtained (ie, directly from the catheter tubing), if feasible, rather than a sample from the urometer or drainage bag. This will ensure that the sample represents recently produced urine and to avoid contamination of the sample by debris in the collection bag.
 - The specimen should be examined at room temperature within two hours of retrieval. If this is not feasible, the sample should be refrigerated at 2 to 8 degrees Celsius and then re-warmed to room temperature prior to assessment.
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GROSS ASSESSMENT

Normal urine is clear and light yellow in color. Urine turbidity and color may be altered in a number of settings.

Turbidity — Turbid urine may be seen in the setting of infection, or as a result of precipitated crystals or chyluria [2].

Urine color — The yellow color of urine is lighter when urine is dilute and darker when concentrated, such as after an overnight water restriction. The urine may also have a variety of other colors.

Red to brown urine — The excretion of red to brown urine is observed in a variety of clinical settings [3]. The initial step in the evaluation of this abnormality is centrifugation of the urine to see whether the

red color is in the urine sediment or the supernatant ([algorithm 1](#)).

- If the red color is seen only in the sediment (and the supernatant is not red), the patient has hematuria. (See ['Detection of heme'](#) below.)
- If, on the other hand, the supernatant is red, then the supernatant should be tested for heme with a urine dipstick:
 - If a urine dipstick of the red supernatant is positive for heme, the patient has either hemoglobinuria or myoglobinuria. (See ['Hemoglobinuria and myoglobinuria'](#) below.)
 - If a urine dipstick of the red supernatant is negative for heme, the patient may have one of a variety of unusual conditions. (See ['Other causes of red urine'](#) below.)

Hemoglobinuria and myoglobinuria — Hemoglobinuria is the presence of free hemoglobin (which is normally only present inside intact red blood cells [RBCs]) in the urine. This may occur during episodes of intravascular hemolysis (eg, during an acute hemolytic transfusion reaction or severe malaria treated with certain medications [so-called "blackwater fever" refers to black urine in this condition]). Myoglobinuria is the presence of free myoglobin (which is normally only present in intact muscle cells) in the urine. This may occur during myonecrosis (eg, due to crush injury of muscle). Both hemoglobinuria and myoglobinuria can produce a red or red to brown urine:

- Hemoglobin is relatively poorly filtered, due both to its large size (molecular weight 69,000 of the tetramer and 34,000 of the dimer) and protein binding to haptoglobin. Only the unbound dimer is filtered. Hemoglobinuria will not occur until haptoglobin is fully saturated and the filtered load of free hemoglobin exceeds proximal reabsorptive capacity. Although this does not require a high plasma concentration of the free dimer, the total hemoglobin concentration (protein-bound + tetramer + dimer) at this time generally exceeds 100 to 150 mg/dL, resulting in a red to brown color of the plasma [\[4\]](#). Haptoglobin levels typically fall due to hepatic removal of the haptoglobin-hemoglobin complex.

Hemoglobinuria is often associated with red urine. However, the combination of prolonged transit time through the nephron with glomerular bleeding and an acid urine pH may result in the formation of methemoglobin, which has a smoky brown or "Coca cola" color [\[4\]](#). (See ["Etiology and evaluation of hematuria in adults", section on 'Glomerular versus nonglomerular bleeding'](#).)

- Myoglobin, by comparison, is a monomer (molecular weight 17,000) and is not protein bound. As a result, it is rapidly filtered and excreted, thereby allowing the plasma to retain its normal color unless renal failure limits myoglobin excretion. In addition, although myoglobin can bind to haptoglobin, the binding affinity is low, and thus excess plasma myoglobin does not lower the plasma haptoglobin concentration [\[5,6\]](#). The source of the excess myoglobin is skeletal muscle breakdown (rhabdomyolysis), which is also associated with a marked elevation in the serum creatine kinase concentration.

- (See "[Clinical manifestations and diagnosis of rhabdomyolysis](#)", section on '[Urine findings and myoglobinuria](#)!).
- (See "[Clinical features and diagnosis of heme pigment-induced acute kidney injury](#)", section on '[Urinalysis](#)!).

Other causes of red urine — A red urine supernatant that is negative for heme can be seen in several conditions. These include:

- Use of certain medications such as [rifampin](#) or [phenytoin](#)
- Consumption of food dyes
- Ingestion of beets (beeturia), rhubarb, or [senna](#)
- Acute intermittent porphyria ([picture 1](#))

Other urine colors — Rarely, the urine has other colors. These include:

- White urine, which may be due to polyuria, phosphate crystals, chyluria [7,8], or [propofol](#) [9].
- Pink urine, presumably due to uric acid crystals, which may occur following [propofol](#) administration [10-12].
- Green urine, which may be due to the administration of [methylene blue](#) [13], [propofol](#) [14-18], or [amitriptyline](#).
- Black urine, which may be due to hemoglobinuria [19,20], myoglobinuria, melanuria [21], or ochronosis. The black urine in ochronosis, which usually results from alkaptonuria (also called "black urine disease"), is caused by the urinary excretion of homogentisic acid. The black color may only be apparent after the urine stands for some time, permitting the oxidation of homogentisic acid. (See "[Disorders of tyrosine metabolism](#)", section on '[Alkaptonuria](#)!).
- Purple urine, which may be due to bacteriuria in patients with urinary catheters [22]. (See "[Catheter-associated urinary tract infection in adults](#)", section on '[Pathogenesis](#)!).

These and other urine colors can occur in children with inborn errors of metabolism ([table 1](#)).

URINE DIPSTICK

The urine dipstick provides a rapid semiquantitative assessment of urinary characteristics on a series of test pads embedded on a reagent strip. Most dipsticks permit the analysis of the following core urine parameters: heme, leukocyte esterase, nitrite, albumin, hydrogen ions, specific gravity, and glucose. Some dipsticks include test pads for additional parameters including urobilinogen and ketones. Users should be familiar with the specific characteristics of the reagent strips they are using and adhere to manufacturer instructions regarding the amount of urine required and the time that needs to elapse before interpreting the color on any given test pad.

Detection of heme — Heme acts as a pseudoperoxidase, and when heme-containing urine is exposed to peroxide and a chromogen on the test pad, a color change takes place [23]. However, a positive dipstick for heme may result not only from urinary red blood cells (RBCs), but also from free hemoglobin or free myoglobin. In addition, the dipstick may be falsely positive if there is semen present in the urine [24]. Thus, a positive dipstick does not establish the presence of RBCs in the urine, and the diagnosis of hematuria requires confirmation with microscopy [25]. (See '[Hemoglobinuria and myoglobinuria](#)' above and "[Etiology and evaluation of hematuria in adults](#)".)

The detection of heme by urine dipstick is thought of as a highly sensitive test for the presence of RBCs (eg, one to two RBCs per high-powered field) [26]. False-negative results are said to be unusual and, as a result, a dipstick that is negative for heme theoretically excludes the presence of RBCs [27,28]. However, urinary ascorbic acid can interfere with the peroxidase reaction, thereby yielding false-negative results [29]. As an example, one study showed that, in the presence of urinary ascorbic acid, the urine dipstick was negative for heme in 70 percent of patients with microscopically documented RBCs. Given the frequency of vitamin C ingestion, this may limit the value of the urine dipstick as a screening test for hematuria. Manufacturers have attempted to allay such concerns through the production of dipsticks that oxidize ascorbic acid, thereby minimizing the risk of false negatives [30].

Detection of leukocyte esterase — Leukocyte esterase released by lysed neutrophils and macrophages is a marker for the presence of white blood cells (WBCs). However, a concentrated urine may impede cell lysis and therefore produce a false-negative result. Proteinuria and glucosuria may also lead to a false-negative test for leukocyte esterase [23].

Nitrite — Many *Enterobacteriaceae* species, the most common microorganisms causing urinary tract infections, elaborate the enzyme nitrate reductase, which confers the ability to convert urinary nitrate to nitrite. Thus, nitrite-positive urine may indicate underlying bacteriuria. However, bacteriuria or frank infection may still be present in the absence of nitrite positivity. This would occur with organisms expressing low levels of nitrate reductase (eg, enterococcus), or when urine dwell time in the bladder is short [2].

Protein — The urine dipstick test for protein is most sensitive to albumin and provides a semiquantitative means of assessing albuminuria. There are several important limitations of dipstick testing for the urine concentration of albumin:

- In most cases, moderately increased albuminuria in the range of 30 to 300 mg/day (formerly called "microalbuminuria") cannot be detected with dipstick testing. This is important in some patients at high risk for kidney disease, such as those with diabetes, since therapy with an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) would be considered in such patients.
 - (See "[Moderately increased albuminuria \(microalbuminuria\) in type 2 diabetes mellitus](#)", [section on 'Summary and recommendations'](#)).

- (See "[Moderately increased albuminuria \(microalbuminuria\) and cardiovascular disease](#)", [section on 'Summary and recommendations'](#).)
- A patient with severely increased albuminuria that is normally detectable by the dipstick (more than 300 mg/day, formerly called "macroalbuminuria") may still have a negative dipstick if the urine is very dilute.
- Even if the urine dipstick is positive, the semiquantitative categories of albuminuria that are reported (trace, 1+, 2+, and 3+) are **not** necessarily reliable. A dilute urine, for example, will underestimate the degree of albuminuria. By contrast, a concentrated urine may register as 3+ but may **not** indicate high-grade albuminuria.
- Recent exposure to iodinated radiocontrast agents can induce transient albuminuria [\[31\]](#). However, this may not be observed with newer non-ionic contrast agents [\[32\]](#).

A patient with a persistently positive dipstick test for protein should have albuminuria quantified with assessment of the albumin-to-creatinine ratio on a random (spot) urine sample or with a 24-hour urine collection. (See "[Patient education: Collection of a 24-hour urine specimen \(Beyond the Basics\)](#)" and "[Assessment of urinary protein excretion and evaluation of isolated non-nephrotic proteinuria in adults](#)".)

The clinical significance of dipstick proteinuria has been demonstrated in multiple settings [\[33,34\]](#), and the presence and degree of proteinuria have become a fundamental part of chronic kidney disease staging [\[35\]](#) and the prognostication of progression [\[36,37\]](#). This issue is discussed in detail elsewhere.

- (See "[Assessment of urinary protein excretion and evaluation of isolated non-nephrotic proteinuria in adults](#)".)
- (See "[Definition and staging of chronic kidney disease in adults](#)".)
- (See "[Chronic kidney disease and coronary heart disease](#)".)

Detection of non-albumin proteinuria — The dipstick is insensitive to non-albumin proteins, most notably potentially nephrotoxic immunoglobulin light chains. A screen for the presence of such proteins may be performed with the sulfosalicylic acid test.

Sulfosalicylic acid (SSA) detects all proteins in urine and may be useful in patients with acute kidney injury (AKI) of unclear etiology and a urine dipstick that is negative for protein. A positive SSA test in conjunction with a negative dipstick usually indicates the presence of non-albumin proteins in the urine, most often immunoglobulin light chains. (See "[Kidney disease in multiple myeloma and other monoclonal gammopathies: Etiology and evaluation](#)".)

The SSA test is performed by mixing one-part urine supernatant (eg, 2.5 mL) with three-parts 3 percent SSA and assessing whether the urine becomes turbid, which suggests the presence of proteinuria. The

SSA test is described in detail elsewhere. (See ["Assessment of urinary protein excretion and evaluation of isolated non-nephrotic proteinuria in adults", section on 'Sulfosalicylic acid test'.](#))

Hydrogen ion concentration — The urine hydrogen ion concentration, expressed as the pH, reflects the degree of acidification of the urine. The urine pH ranges from 4.5 to 8, depending upon the systemic acid-base balance. The urine pH is most often used clinically in patients with metabolic acidosis. The appropriate renal response to acidemia is to increase urinary acid excretion, with the urine pH falling below 5. A higher value suggests the presence of renal tubular acidosis. A discussion of the urine pH in the diagnosis of renal tubular acidosis is presented elsewhere. (See ["Overview and pathophysiology of renal tubular acidosis and the effect on potassium balance".](#))

In some settings, the urine pH is not indicative of acid excretion by the kidneys. As an example, infection with any pathogen that produces urease, such as *Proteus mirabilis*, can result in a urine pH above 7 to 7.5, even if urinary acidification by the kidney is normal.

Specific gravity — The osmolality of the urine can be inferred by measuring the urine specific gravity, which is defined as the weight of the solution compared with the weight of an equal volume of distilled water. The urine specific gravity generally varies with the osmolality, rising by approximately 0.001 for every 35 to 40 mosmol/kg increase in urine osmolality ([figure 1](#)). Thus, a urine osmolality of 280 mosmol/kg (which is isosmotic to normal plasma) is usually associated with a urine specific gravity of 1.008 or 1.009.

However, there is an important difference between these measures: the urine osmolality is determined by the number of particles in the urine (eg, urea, sodium, potassium), while the specific gravity is determined by both the number and size of the particles in the urine. This becomes important clinically when there are large molecules in the urine, such as glucose or radiocontrast media. In these settings, the specific gravity can exceed 1.030 (suggesting a highly concentrated urine) despite a urine osmolality that may be dilute to plasma.

By contrast, there are no causes of a falsely low urine specific gravity. As an example, a specific gravity ≤ 1.003 is indicative of a maximally dilute urine (≤ 100 mosmol/kg).

In most clinical settings, the urine osmolality can be measured directly, and estimation using the urine specific gravity is unnecessary. Where available, direct measurement of the urine osmolality is most useful in the evaluation of patients with the following disorders (hyponatremia, hypernatremia, polyuria, and AKI):

- Hyponatremia (see ["Osmotic demyelination syndrome \(ODS\) and overly rapid correction of hyponatremia", section on 'Overly rapid rate of correction'](#) and ["Diagnostic evaluation of adults with hyponatremia"](#))
- Hypernatremia (see ["Etiology and evaluation of hypernatremia in adults"](#))
- Polyuria (see ["Evaluation of patients with polyuria"](#))

- AKI (see "[Etiology and diagnosis of prerenal disease and acute tubular necrosis in acute kidney injury in adults](#)")

Glucose — When present in the urine, glucose triggers the production of peroxide, which in turn leads to the oxidation of a chromogen in a reaction catalyzed by peroxidase [2]. As is the case with the dipstick test for heme, ascorbic acid can produce a false-negative test for glycosuria [29]. Glycosuria may be due to either the inability of the kidney to reabsorb filtered glucose in the proximal tubule despite normal plasma glucose concentration, or to an overflow scenario related to high plasma glucose concentrations overwhelming the capacity of the renal tubules to reabsorb glucose. In patients with normal kidney function, significant glycosuria does not generally occur until the plasma glucose concentration exceeds 180 mg/dL (10 mmol/L).

When glycosuria occurs with a normal plasma glucose, a primary defect of proximal tubule reabsorption needs to be considered. In this setting, glycosuria may coexist with additional manifestations of proximal tubular dysfunction, including phosphaturia (leading to hypophosphatemia), uricosuria, renal tubular acidosis, and aminoaciduria. This constellation is called the Fanconi syndrome and may result from a variety of disorders, including multiple myeloma, heavy metal exposure, and treatment with certain medications including tenofovir, [lamivudine](#), [cisplatin](#), valproic acid, and aminoglycosides [38]. Glycosuria with normal plasma glucose will be evident in patients receiving sodium-glucose cotransporter 2 inhibitors, [39-42]. Glycosuria may also be an isolated defect (isolated renal glycosuria) associated with genetic mutations affecting renal glucose transport.

- (See "[Kidney disease in multiple myeloma and other monoclonal gammopathies: Etiology and evaluation](#)".)
- (See "[Etiology and diagnosis of distal \(type 1\) and proximal \(type 2\) renal tubular acidosis](#)".)

URINE SEDIMENT

Microscopic examination of the urine sediment is an essential part of the urinalysis, as it enables confirmation and clarification of urine dipstick findings and also the identification of structures that are not evaluated by the urine dipstick (eg, epithelial cells, casts, crystals).

To perform the urine sediment examination, 10 mL of urine is centrifuged at 3000 rpm for five minutes. Most of the supernatant is then poured out, and the pellet is resuspended with gentle shaking of the tube. A pipette can then be used to place approximately 50 microL (or a small drop) of resuspended sediment on a glass slide, followed by application of a coverslip [23]. An alternative approach is to tilt the test tube and insert the corner of the coverslip into the tube to extrude a single drop of resuspended sediment; the coverslip is then laid onto the slide, thereby allowing a thin film of sediment to become interposed between the coverslip and the slide.

A brightfield (or if available, phase-contrast) microscope with a binocular head, built-in light source, and polarizing filters should be used [1]. The light intensity should be subdued because some structures may be missed if the light is excessively bright. As the specimen is reviewed, the fine adjustment on the microscope should be manipulated to appreciate structures in different levels of depth within the sample [2]. The entire specimen is initially scanned at low power (100x) with particular attention to the edges of the coverslip where casts tend to migrate. High power (400x) should then be used to better characterize structures that were identified at lower power. Polarized light may be used to search for lipid-laden elements or crystals as warranted by the clinical context.

The urine sediment examination should be performed by a clinician trained in urine microscopy because the diagnostic yield may be substantially greater compared with a urinalysis performed by laboratory staff [43].

Cells — Cellular elements that may be found in the urinary sediment include red blood cells (RBCs), white blood cells (WBCs), and epithelial cells from all levels of the urinary tract.

Red blood cells — Hematuria can be benign or reflect serious underlying disease ([figure 2](#)). The evaluation of patients with hematuria is discussed in detail separately, but some of the major issues will be briefly reviewed here. (See ["Etiology and evaluation of hematuria in adults"](#).)

Hematuria may be grossly visible or microscopic. Microscopic hematuria is commonly defined as the presence of two or more RBCs per high-powered field in a spun urine sediment [26] ([picture 2](#)). The urine color change in gross hematuria does not necessarily reflect a large degree of blood loss, since as little as 1 mL of blood per liter of urine can induce a visible color change. As previously mentioned, red to brown urine can be observed in patients without actual hematuria [25]. (See ['Red to brown urine'](#) above.)

Hematuria may be transient or persistent. Transient hematuria is relatively common in young patients and may occur following exercise or sexual intercourse [26]. Menstruation may confound the evaluation of hematuria, and the urinalysis should be repeated when the patient is not menstruating. However, even transient hematuria can represent underlying malignancy, especially in patients over the age of 50 years. Transient hematuria can also occur with urinary tract infection (eg, cystitis or prostatitis). This is typically accompanied by pyuria and bacteriuria, and patients may often complain of dysuria. (See ["Etiology and evaluation of hematuria in adults"](#).)

Persistent hematuria should always be evaluated. Among the more common pathologic causes are kidney stones, malignancy, and glomerular disease. A study of Israeli army recruits showed that, even in asymptomatic individuals, those with isolated persistent hematuria were 18 times more likely to develop end-stage renal disease (ESRD) over a follow-up period that exceeded 20 years [44].

Distinguishing between glomerular and nonglomerular causes is the first key step in the evaluation of unexplained hematuria. Isomorphic RBCs have an appearance similar to erythrocytes in the circulation (small, anucleated cells shaped as biconcave discs) and can be seen with any cause of hematuria. By

contrast, dysmorphic RBCs (which have an altered morphology) are suggestive of glomerular disease [45]. There are no uniform criteria for defining dysmorphic RBCs, and defining a clinically relevant proportion of dysmorphic cells is debatable; therefore, the practical utility of describing dysmorphic RBCs in the diagnosis of glomerular disease has been questioned [46]. However, RBCs that have membrane protrusions (ie, acanthocytes) are a readily definable subset of dysmorphic RBCs (

[picture 3A-B](#)) that have a sensitivity of 52 percent and specificity of 98 percent for the diagnosis of glomerulonephritis [47]. The concomitant presence of RBC casts and/or albuminuria in a patient with hematuria increases the likelihood that the observed hematuria is of a glomerular origin [48]. (See ["Etiology and evaluation of hematuria in adults"](#), section on ['Glomerular versus nonglomerular bleeding'](#) and ['Red blood cell casts'](#) below.)

Less commonly, urinary RBCs with unique morphologies may suggest underlying systemic illness. This includes sickled RBCs in patients with underlying sickle cell trait/anemia and elliptocytes in patients with hemolysis [49]. (See ["Overview of the clinical manifestations of sickle cell disease"](#) and ["Hereditary elliptocytosis and related disorders"](#).)

White blood cells — Although the entire spectrum of WBCs may be seen in the urine, neutrophils and eosinophils are the cell types of greatest practical interest to the clinician-microscopist. Neutrophils are intermediate in size compared to RBCs and renal tubular epithelial cells and can be identified by their characteristic granular cytoplasm and multilobed nuclei ([picture 4](#)). Urinary neutrophils are commonly associated with bacteriuria. However, if the corresponding urine culture is negative (ie, sterile pyuria), interstitial nephritis, renal tuberculosis, and nephrolithiasis should be considered [50]. (See ["Sampling and evaluation of voided urine in the diagnosis of urinary tract infection in adults"](#) and ["Clinical manifestations and diagnosis of acute interstitial nephritis"](#), section on ['Clinical features'](#).)

Urine eosinophils can be detected by applying Wright's or Hansel's stain to the urine sediment [51]. The presence of eosinophiluria has classically been considered a marker of acute interstitial nephritis. However, in a case series of adults with biopsy-proven acute interstitial nephritis, only 34 percent of patients had eosinophiluria [52]. Thus, testing for eosinophiluria should **not** be used to establish or exclude a diagnosis of acute interstitial nephritis [53,54].

Epithelial cells — Epithelial cells may appear in the urine after being shed from anywhere within the genitourinary tract. Renal tubular cells are 1.5 to 3 times larger than white cells and are further distinguished by a round, large, centrally-located nucleus ([picture 5](#)). Transitional epithelial cells originate anywhere from the renal pelvis to the proximal urethra and are slightly larger than renal tubular epithelial cells. They may have a pear-like or oval appearance ([picture 6](#)). Squamous epithelial cells are derived from the distal urethra or external genitalia. They are large and irregular in shape with a small central nucleus, and their presence represents contamination by genital secretions ([picture 7](#)).

Casts — Casts are cylindrical structures that are formed in the tubular lumen; several factors favor cast formation: urine stasis, low pH, and greater urinary concentration [2]. Casts will assume the shape and

size of the renal tubule in which they are formed. All casts have an organic matrix composed primarily of Tamm-Horsfall mucoprotein, which comprises the basic architecture for any cast. Casts are defined by the nature of the cells or other elements that are embedded in the cast matrix.

Some casts can be found in healthy individuals, while others are diagnostic of significant renal disease. The observation of cells within a cast is highly significant since their presence is diagnostic of an **intrarenal** origin.

Red blood cell casts — The finding of RBC casts suggests an underlying proliferative glomerulonephritis, for which numerous etiologies exist ([picture 3C](#)). However, due to their limited sensitivity, the absence of RBC casts, particularly in a patient with hematuria and a high pre-test probability, does not rule out a proliferative glomerulonephritis [47]. RBC casts are not exclusive to the setting of proliferative glomerulonephritis. In one study, 6 of 21 patients (nearly 30 percent) with biopsy-proven acute interstitial nephritis in one study had RBC casts in the urine [55]. This implies that RBCs which extrude into the renal tubules from an inflamed interstitium can also lead to cast formation.

- (See ["Etiology and evaluation of hematuria in adults"](#), section on 'Glomerular versus nonglomerular bleeding').
- (See ["Clinical manifestations and diagnosis of acute interstitial nephritis"](#), section on 'Clinical features').

White blood cell casts — White blood cell (WBC) casts are indicative of interstitial or, less classically, glomerular inflammation ([picture 8A-B](#)). In a biopsy series of patients with confirmed acute interstitial nephritis, only 3 percent of patients had WBC casts in their urine sediment [52]. This highlights that, in the presence of a reasonable clinical suspicion for acute interstitial nephritis, the absence of WBC casts should not diminish consideration of this important diagnosis.

Renal tubular epithelial cell casts — These may be observed in any setting where there is desquamation of the tubular epithelium, including acute tubular necrosis (ATN), acute interstitial nephritis, and proliferative glomerulonephritis ([picture 18B](#)).

Granular casts — Granular casts represent degenerated cellular casts or the aggregation of proteins within a cast matrix [2] ([picture 9](#)). Granular casts may be coarse or fine in nature, although the clinical significance of this distinction is unclear. Coarse, deeply-pigmented granular casts (ie, "muddy brown" or heme-granular casts) are considered characteristic of ATN, the leading cause of acute kidney injury (AKI) in hospitalized patients [56].

In patients with ischemic or toxic injury to the tubular epithelial cells, cell sloughing into the tubular lumen, due either to cell death or to defective cell-to-cell or cell-to-basement membrane adhesion, may lead to the formation of granular and/or epithelial cell casts. (See ["Pathogenesis and etiology of ischemic acute tubular necrosis"](#), section on 'Epithelial cell injury and dysfunction').

Hyaline casts — Hyaline casts are only slightly more refractile than water and have a transparent, empty appearance ([picture 10](#)). Hyaline casts may be observed with small volumes of concentrated urine or with diuretic therapy and are generally nonspecific.

Waxy casts — Waxy casts are thought to be the last stage in the degeneration of a granular cast. They are homogeneous in appearance and are characterized by sharp indentations and darker edges that are more distinct ([picture 11](#)). Waxy casts are nonspecific and may be observed in a variety of acute and chronic kidney diseases.

Broad casts — Broad casts are wider than other casts, a characteristic believed to be due to their formation in large dilated tubules with little flow ([picture 12](#)). The presence of broad casts is typically associated with advanced chronic kidney disease.

Crystals — Whether crystals form in the urine depends upon a variety of factors, including the degree of concentration of constituent molecules, the urine pH, and the presence of inhibitors of crystallization. Many different forms may be observed in normal patients and in those with defined disorders:

- Uric acid crystals – Uric acid crystals as well as amorphous urates are observed in acid urine, a milieu that favors the conversion of the relatively soluble urate salt into the insoluble uric acid ([picture 13A-B](#)). (See ["Uric acid renal diseases"](#).)
- Calcium oxalate or calcium phosphate crystals – Calcium oxalate crystals, which are not dependent upon the urine pH, may appear in the monohydrate form with a characteristic "dumbbell" appearance or in the dihydrate form as an envelope-like structure. Calcium phosphate crystals only form in a relatively alkaline urine and have a characteristic coffin-like structure ([picture 14A-B](#)). (See ["Kidney stones in adults: Epidemiology and risk factors"](#).)
- Cystine crystals – Cystine crystals, with their characteristic hexagonal shape, are diagnostic of cystinuria ([picture 15](#)). (See ["Cystine stones"](#).)
- Magnesium ammonium phosphate crystals – Magnesium ammonium phosphate (struvite) and calcium carbonate-apatite are the constituents of struvite stones ([picture 16](#)). Normal urine is undersaturated with ammonium phosphate. Struvite stone formation occurs only when ammonia production is increased and the urine pH is elevated, which decreases the solubility of phosphate. Both increased ammonia production and increased urine pH occur only in the setting of a urinary tract infection with a urease-producing organism, such as *Proteus* or *Klebsiella*. (See ["Pathogenesis and clinical manifestations of struvite stones"](#).)

The observation of crystals in the urine is useful in patients with known or suspected kidney stones and is a risk factor for recurrent calcium oxalate [57] or cystine [58] stone formation. In addition, crystalluria may have diagnostic utility in other settings:

- The presence of magnesium ammonium phosphate crystals, which is consistent with infection (see "[Pathogenesis and clinical manifestations of struvite stones](#)")
- The combination of AKI and calcium oxalate crystals, which may suggest ethylene glycol ingestion (see "[Methanol and ethylene glycol poisoning: Pharmacology, clinical manifestations, and diagnosis](#)", section on 'Clinical features of overdose')
- The presence of a larger number of uric acid crystals occurring in association with AKI, which may suggest tumor lysis syndrome (see "[Tumor lysis syndrome: Definition, pathogenesis, clinical manifestations, etiology and risk factors](#)" and "[Tumor lysis syndrome: Definition, pathogenesis, clinical manifestations, etiology and risk factors](#)", section on 'Clinical manifestations')

Microorganisms — Bacteria are often seen in the urine, although the clinical significance of bacteriuria is generally guided by patient symptoms ([picture 17](#)). Fungi are also frequently present ([picture 17](#)). (See ["Asymptomatic bacteriuria in adults"](#).)

THE ASSESSMENT OF LIPIDURIA

Lipid droplets, composed primarily of cholesterol esters and, to a lesser degree, cholesterol, are commonly seen on urinalysis in patients with conditions that are associated with the nephrotic syndrome [59,60]. These fat droplets may be free within sloughed tubular cells (called oval fat bodies) or within casts (called fatty casts) ([picture 3D](#)). Fat droplets have a characteristic "Maltese cross" appearance under polarized light ([picture 3E](#)).

Fat droplets are round and may be confused with red cells. They can be differentiated from red cells under routine light microscopy by their variable size (ranging from larger to much smaller than red cells), their dark outline, and the "Maltese cross" appearance under polarized light.

The origin of urinary lipid is not well understood [60]. The initial step is the filtration of lipoprotein-bound cholesterol, particularly HDL-cholesterol. Filtration of lipoproteins is minimal in healthy individuals but is markedly enhanced when glomerular permeability to macromolecules is increased in the nephrotic syndrome. Some of the filtered lipoprotein is taken up by the proximal tubular cells. The cholesterol will be seen in the urine sediment as an oval fat body when the cell is desquamated and/or as free droplets or in fatty casts if the lipid is extruded from the cells.

Because of the apparent requirement for increased glomerular permeability, lipiduria is almost always diagnostic of some form of glomerular disease (see "[Glomerular disease: Evaluation and differential diagnosis in adults](#)"). One exception is autosomal dominant polycystic kidney disease [61,62]. In one report, urinary oval fat bodies were observed in 21 of 35 patients with autosomal dominant polycystic kidney disease whose average proteinuria on dipstick was only 1+ [61]. Oval fat bodies were also seen in fluid directly aspirated from renal cysts, and lipid droplets were observed in the epithelial cells lining the cyst wall. Lipiduria may occasionally be seen in other nonglomerular diseases, such as acute or chronic interstitial nephritis or even prerenal azotemia [62].

CORRELATION OF URINARY FINDINGS WITH KIDNEY DISEASES

Various patterns of urinary findings may suggest specific categories of kidney disease. As with all diagnostic tests, these urinary findings must be interpreted in the context of the history, physical exam, and available laboratory data ([table 2](#)). Patterns of urinary findings and the diagnoses to which they point include:

Hematuria with dysmorphic RBCs, RBC casts, and proteinuria — This constellation of findings (eg, dysmorphic red blood cells [RBCs], RBC casts, and proteinuria) is suggestive of a proliferative glomerular disease, which, in the setting of rapidly declining kidney function, constitutes a nephrologic emergency ([picture 3A-E](#)). (See "[Glomerular disease: Evaluation and differential diagnosis in adults](#)".)

Heavy proteinuria with absent or minimal hematuria — Heavy proteinuria with oval fat bodies, lipid-laden casts, and absent or minimal hematuria is indicative of nonproliferative glomerular diseases including severe diabetic nephropathy. In addition, this pattern may be seen with membranous nephropathy, focal segmental glomerulosclerosis, minimal change disease, and amyloidosis, each of which has both primary and secondary forms. (See "[Overview of heavy proteinuria and the nephrotic syndrome](#)".)

Granular or epithelial cell casts and renal tubular epithelial cells — In a patient with acute kidney injury (AKI), the presence of granular and/or epithelial cell casts with or without free renal tubular epithelial cells is strongly suggestive of acute tubular necrosis (ATN) ([picture 18A-B](#)). The number of observed granular or renal tubular casts may also have prognostic significance; in a study of patients with ATN who were diagnosed based upon clinical criteria, a semiquantitative assessment of the burden of granular or renal tubular epithelial cell casts on low-powered fields was associated with an increased risk of death or the subsequent need for renal replacement therapy [\[63\]](#).

The presence and quantity of granular casts or renal tubular epithelial cells in the urine was evaluated as a diagnostic tool in 267 patients with hospitalized AKI [\[64\]](#). Prior to performing a urinalysis, trained clinicians were asked to make a clinical diagnosis of ATN, prerenal AKI, or other. Since kidney biopsies were not routinely performed, the final diagnosis of ATN versus prerenal disease was determined by whether the renal function improved within 48 hours after fluid resuscitation and/or hemodynamic manipulation. The following observations were made:

- A pre-urinalysis diagnosis (based upon the history, physical examination, and other available data) of ATN had a positive predictive value of 86 percent. In such patients who also had at least one granular cast or tubular epithelial cell in the urinalysis, the positive predictive value increased to 100 percent. Of patients with a pre-urinalysis diagnosis of ATN and no granular casts or tubular epithelial cells, 44 percent had a final diagnosis of ATN. Thus, a diagnosis of ATN is still possible even if the urinalysis has no granular casts or tubular epithelial cells.

- A pre-urinalysis diagnosis of prerenal AKI had a positive predictive value of 77 percent. In such patients, the absence of granular casts or tubular epithelial cells increased the positive predictive value to 91 percent for a final diagnosis of prerenal AKI.

The presence of granular casts and tubular epithelial cells in the urine was also associated with progression of AKI in the hospital [65]. Patients whose urinalyses revealed six or more granular casts per low-powered field (without tubular epithelial cells), six or more tubular epithelial cells per high-powered field (without granular casts), or any number of granular casts and tubular epithelial cells present in the same specimen had a significantly greater likelihood of developing worse kidney function, initiation of dialysis, or death compared with patients who had no granular casts or tubular epithelial cells (54-67 percent versus 9 percent).

Isolated pyuria — Isolated pyuria is usually indicative of bacterial urinary tract infection. The differential diagnosis is broad if a concurrent urine culture is negative and includes a partially or recently treated urinary tract infection, non-bacterial infections (including tuberculosis), prostatitis, interstitial nephritis, and nephrolithiasis [50].

Normal or near-normal urinalysis — In patients with AKI, a relatively normal urinalysis (few cells with little or no proteinuria and no casts other than hyaline casts) can be associated with ATN but may also indicate one of the following diseases:

- Prerenal AKI due to either an actual or effective decrease in circulating volume
- Urinary tract obstruction
- Hypercalcemia
- Cast nephropathy in multiple myeloma
- Vascular disease that produces glomerular ischemia but not infarction (eg, hypertensive emergency, scleroderma, thrombotic microangiopathies) or that affects extra-glomerular vessels (eg, cholesterol atheroemboli, polyarteritis nodosa)
- Tumor lysis syndrome
- Acute phosphate nephropathy

In patients with chronic kidney disease, a normal urinalysis most commonly indicates states of persistently decreased effective circulating volume, such as in patients with heart failure, urinary tract obstruction, chronic tubulointerstitial diseases, light chain cast nephropathy, and ischemic or hypertensive nephrosclerosis.

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see ["Patient education: Blood in the urine \(hematuria\) in adults \(The Basics\)"](#))
 - Beyond the Basics topics (see ["Patient education: Blood in the urine \(hematuria\) in adults \(Beyond the Basics\)"](#) and ["Patient education: Glomerular disease \(Beyond the Basics\)"](#))
-

SUMMARY

- A complete urinalysis consists of three components: gross evaluation, dipstick analysis, and microscopic examination of the urine sediment. A complete urinalysis should be performed in the following settings (see ['When to perform a complete urinalysis'](#) above):
 - In a patient with evidence of kidney disease, such as someone with albuminuria or an acute or chronic reduction in the glomerular filtration rate.
 - In a patient with suspected kidney disease. Kidney disease may be suspected on the basis of clinical findings (eg, edema) or because of a concurrent illness that is commonly associated with kidney disease (eg, active systemic lupus erythematosus, secondary hypertension).
 - In a patient with known or suspected kidney stones. A complete urinalysis may identify crystals that clarify the etiology of the kidney stone.
 - A complete urinalysis is also needed to clarify the significance of findings noted on urine dipstick analyses (eg, microscopic hematuria) from otherwise asymptomatic individuals who may have had the urine dipstick as part of a workup for another condition (eg, hypertension, diabetes mellitus).
- The urine specimen must be properly collected in order to reliably interpret the findings and thus maximize diagnostic utility. The following technique should be followed whenever feasible (see ['Obtaining the specimen for analysis'](#) above):
 - The specimen should be collected into a clean dry container.

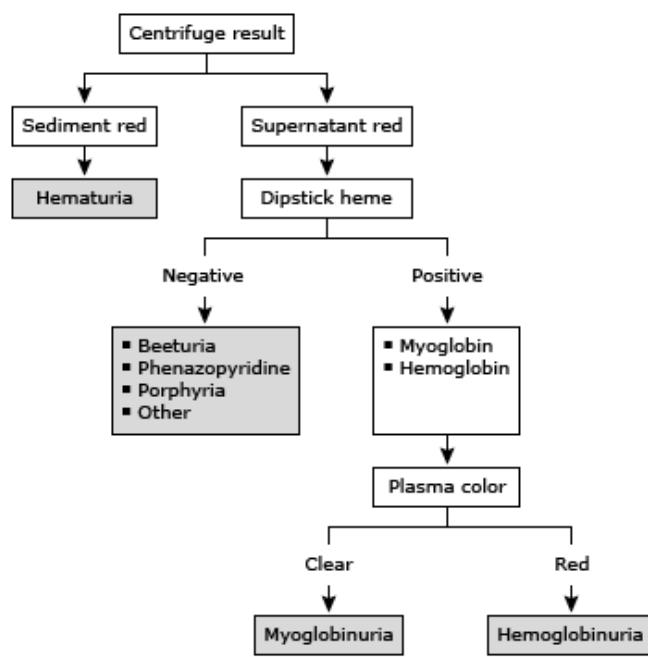
- Patients should be asked to clean the external genitalia and provide a midstream specimen for analysis.
- In patients with indwelling urinary catheters, a recently produced urine sample should be obtained (ie, directly from the catheter tubing), if feasible, rather than a sample from the urometer or drainage bag.
- The specimen should be examined at room temperature within two hours of retrieval. If this is not feasible, the sample should be refrigerated at 2 to 8 degrees Celsius and then re-warmed to room temperature prior to assessment.
- Normal urine is clear and light yellow in color. Urine turbidity and color may be altered in a number of settings. (See '[Gross assessment](#)' above.)
- The urine dipstick provides a rapid semiquantitative assessment of urinary characteristics on a series of test pads embedded on a reagent strip. Most dipsticks permit the analysis of the following core urine parameters: heme, leukocyte esterase, nitrite, albumin, hydrogen ions, specific gravity, and glucose. (See '[Urine dipstick](#)' above.)
- Microscopic examination of the urine sediment is an essential part of the urinalysis, as it enables confirmation and clarification of urine dipstick findings and also the identification of structures that are not evaluated by the urine dipstick (eg, epithelial cells, casts, crystals). To perform the urine sediment examination, 10 mL of urine is centrifuged at 3000 rpm for five minutes. Most of the supernatant is then poured out, and the pellet is resuspended with gentle shaking of the tube. A pipette can then be used to place approximately 50 microL (or a small drop) of resuspended sediment on a glass slide, followed by application of a coverslip. A brightfield (or if available, phase-contrast) microscope with a binocular head, built-in light source, and polarizing filters should be used to examine the urine. (See '[Urine sediment](#)' above.)
- The following diagnostically useful structures may be identified with microscopic examination:
 - Red blood cells (RBCs) ([picture 2](#)), which can be benign or reflect serious underlying disease ([figure 2](#)). (See '[Red blood cells](#)' above.)
 - White blood cells (WBCs) ([picture 4](#)). (See '[White blood cells](#)' above.)
 - Renal tubular epithelial cells ([picture 5](#)), transitional epithelial cells ([picture 6](#)), and squamous epithelial cells ([picture 7](#)). The latter represent contamination by genital secretions. (See '[Epithelial cells](#)' above.)
 - RBC casts, which are usually diagnostic of glomerular hematuria and in the right clinical context, are suggestive of an underlying proliferative glomerulonephritis ([picture 3C](#)). (See '[Red blood cell casts](#)' above.)

- WBC casts, which are indicative of kidney inflammation, the cause of which may be infectious (eg, pyelonephritis) or non-infectious (eg, interstitial nephritis, proliferative glomerulonephritis) ([picture 8A-B](#)). (See '[White blood cell casts](#)' above.)
- Renal tubular epithelial cell casts, which may be observed in any setting where there is desquamation of the tubular epithelium, including acute tubular necrosis (ATN), acute interstitial nephritis, and proliferative glomerulonephritis ([picture 18B](#)). (See '[Renal tubular epithelial cell casts](#)' above.)
- Granular casts, which represent degenerated cellular casts or the aggregation of proteins within a cast matrix ([picture 9](#)). Coarse, deeply-pigmented granular casts (ie, "muddy brown" or heme-granular casts) are considered characteristic of ATN. (See '[Granular casts](#)' above.)
- Hyaline casts, which are only slightly more refractile than water and have a transparent, empty appearance ([picture 10](#)), may be observed with small volumes of concentrated urine or with diuretic therapy and are generally nonspecific. (See '[Hyaline casts](#)' above.)
- Crystals, such as uric acid crystals ([picture 13A-B](#)), calcium phosphate or calcium oxalate crystals ([picture 14A-B](#)), cystine crystals ([picture 15](#)), and magnesium ammonium phosphate crystals ([picture 16](#)). (See '[Crystals](#)' above.)
- Bacteria or fungi ([picture 17](#)). (See '[Microorganisms](#)' above.)
- Lipid droplets ([picture 3D](#) and [picture 3E](#)), composed primarily of cholesterol esters and, to a lesser degree, cholesterol, are commonly seen on urinalysis in patients with conditions that are associated with the nephrotic syndrome. (See '[The assessment of lipiduria](#)' above.)
- Various patterns of urinary findings may suggest specific categories of kidney disease. As with all diagnostic tests, these urinary findings must be interpreted in the context of the history, physical exam, and available laboratory data ([table 2](#)). (See '[Correlation of urinary findings with kidney diseases](#)' above.)

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GRAPHICS

Approach to the patient with red or brown urine



Graphic 55923 Version 5.0

Urine in acute intermittent porphyria



Photograph of urine from a normal subject (left) and a subject with acute intermittent porphyria (middle). The colors are compared with a dilute aqueous solution of red wine (right).

AIP: acute intermittent porphyria

Provided by Shigeru Sassa, MD, PhD.

Graphic 57844 Version 3.0

Urinary clues to inborn errors of metabolism

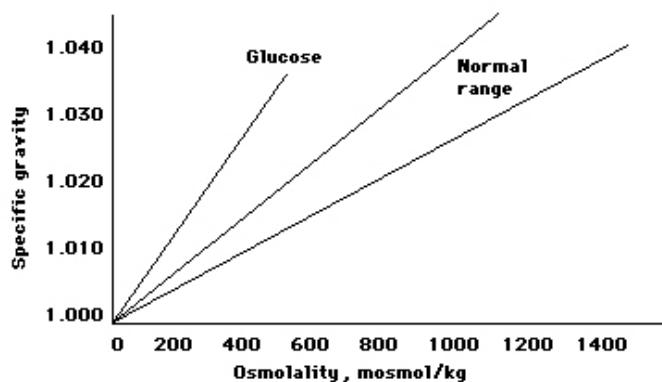
		Potential disorder
Urine color		
Black (upon standing/oxidation)		Homogentisic aciduria (alkaptonuria)
Blue		Tryptophan malabsorption
Pink		Disorders with hematuria, kidney stone formation
Port wine (upon standing/oxidation)		Porphyrias
Yellow-orange		Disorders with increased uric acid
Urine odor*		
Acrid, sweaty feet		Glutaric acidemia II
Cabbage		Tyrosinemia
Fishy		Trimethylaminuria, dimethylglycinuria
Maple syrup, curry		Maple syrup urine disease
Mousy		Phenylketonuria
Sweaty feet		Isovaleric acidemia
Sweet		Beta-ketothiolase deficiency
Swimming pool		Hawkinsinuria

* Only in acute phases or depending on food intake.

Adapted from: Wappner RS, Hainline BE. Inborn errors of metabolism. In: Oski's Pediatrics. Principles and Practice, 3rd ed, McMillan JA, DeAngelis CD, Feigin RD, Warshaw JB (Eds), Lippincott, Williams & Wilkins, Philadelphia, 1999. p.1823 and Saudubray JM, Chappentier C. Clinical phenotypes: Diagnosis/algorithms. In: Metabolic and Molecular Bases of Inherited Disease, 8th ed, Scriver CR, Beaudet AL, Sly WS, Valle D (Eds), McGraw-Hill, New York, 2001. p.1327.

Graphic 74441 Version 3.0

Urine osmolality versus specific gravity

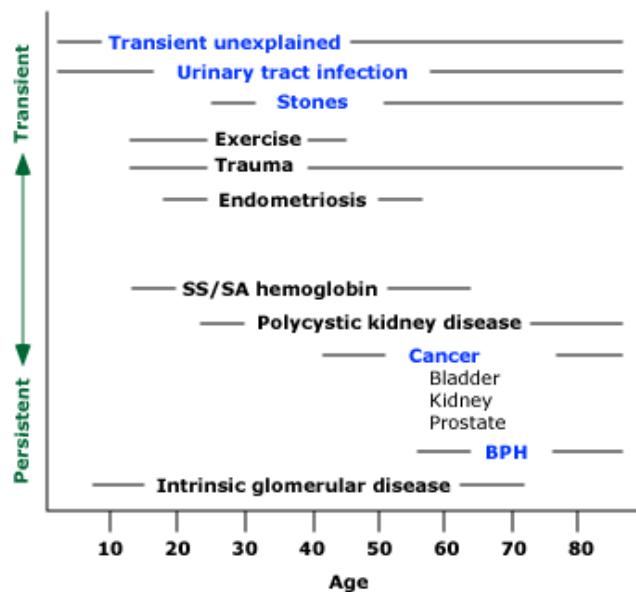


Relationship between the specific gravity and osmolality of the urine from normal subjects who have neither glucose nor protein in the urine. For comparison, the relationship between the specific gravity and osmolality for glucose solutions is included. Glucose is larger than the main solutes in normal urine such as sodium, potassium, ammonium, and urea; as a result, a glucose solution has a higher specific gravity at a given osmolality than normal urine.

Data from Miles B, Paton A, deWardener H, Br Med J 1954; 2:904.

Graphic 75070 Version 1.0

Major causes of hematuria by age and duration



Schematic representation of the major causes of hematuria in relation to the age at which they usually occur (horizontal axis), transience or persistence (vertical axis), and frequency (blue implies more frequent).

BPH: benign prostatic hyperplasia.

Graphic 61296 Version 1.0

Phase-contrast micrograph showing monomorphic red cells in urine sediment

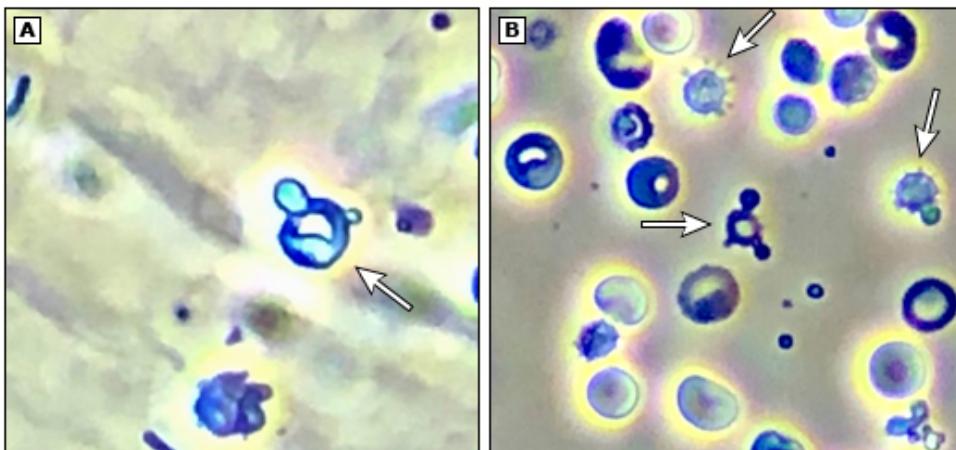


Urine sediment viewed by phase-contrast microscopy showing many red cells and an occasional larger white cell with a granular cytoplasm (arrows). The red cells have a uniform size and shape, suggesting that they are of nonglomerular origin.

Courtesy of Harvard Medical School.

Graphic 80282 Version 4.0

Phase-contrast micrograph showing dysmorphic RBCs in urine sediment

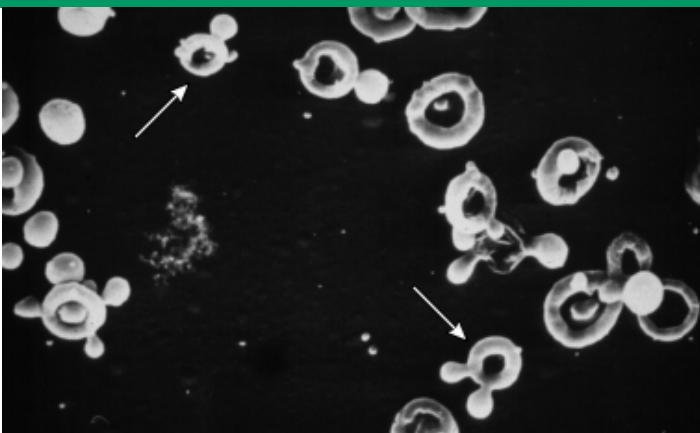


Phase-contrast microscopy showing dysmorphic red blood cells (RBCs) and acanthocytes in the urinary sediment of a patient with glomerular hematuria. Acanthocytes (arrows) can be recognized as ring forms with vesicle-shaped protrusions.

Courtesy of Juan Carlos Q Velez, MD.

Graphic 130438 Version 1.0

Scanning electron micrograph showing dysmorphic red cells in urine sediment



Scanning microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows).

Courtesy of Hans Köhler, MD.

Graphic 62064 Version 3.0

Photomicrograph of urine sediment with white blood cells



White blood cells in the urine sediment with nuclei and granular cytoplasm.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 75211 Version 2.0

Urine sediment showing renal tubular epithelial cells and a fragmented epithelial cell cast

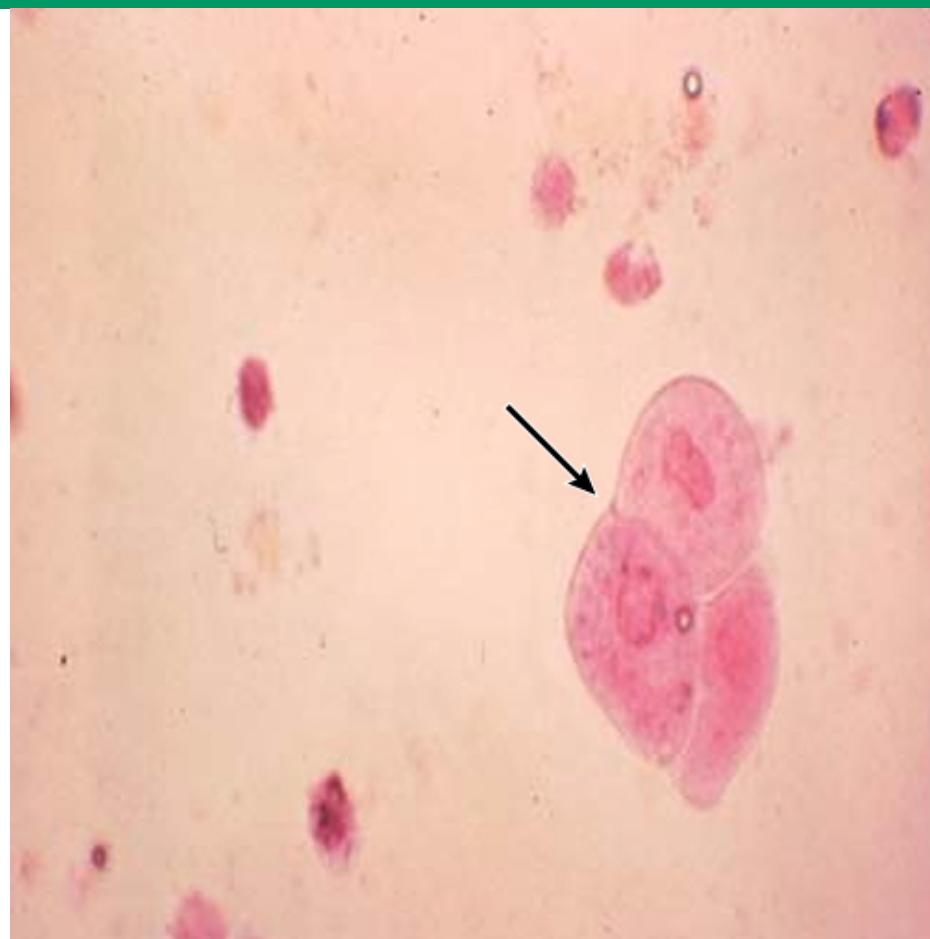


This slide shows renal tubular cells (arrows) found in the urine, together with a fragment of a tubular epithelial cell cast (arrowhead). The tubular cells are characterized by one central nucleus and many cytoplasmic granules.

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Graphic 86348 Version 1.0

Urine sediment showing a transitional epithelial cell



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Graphic 83886 Version 1.0

Urine sediment showing squamous epithelial cells

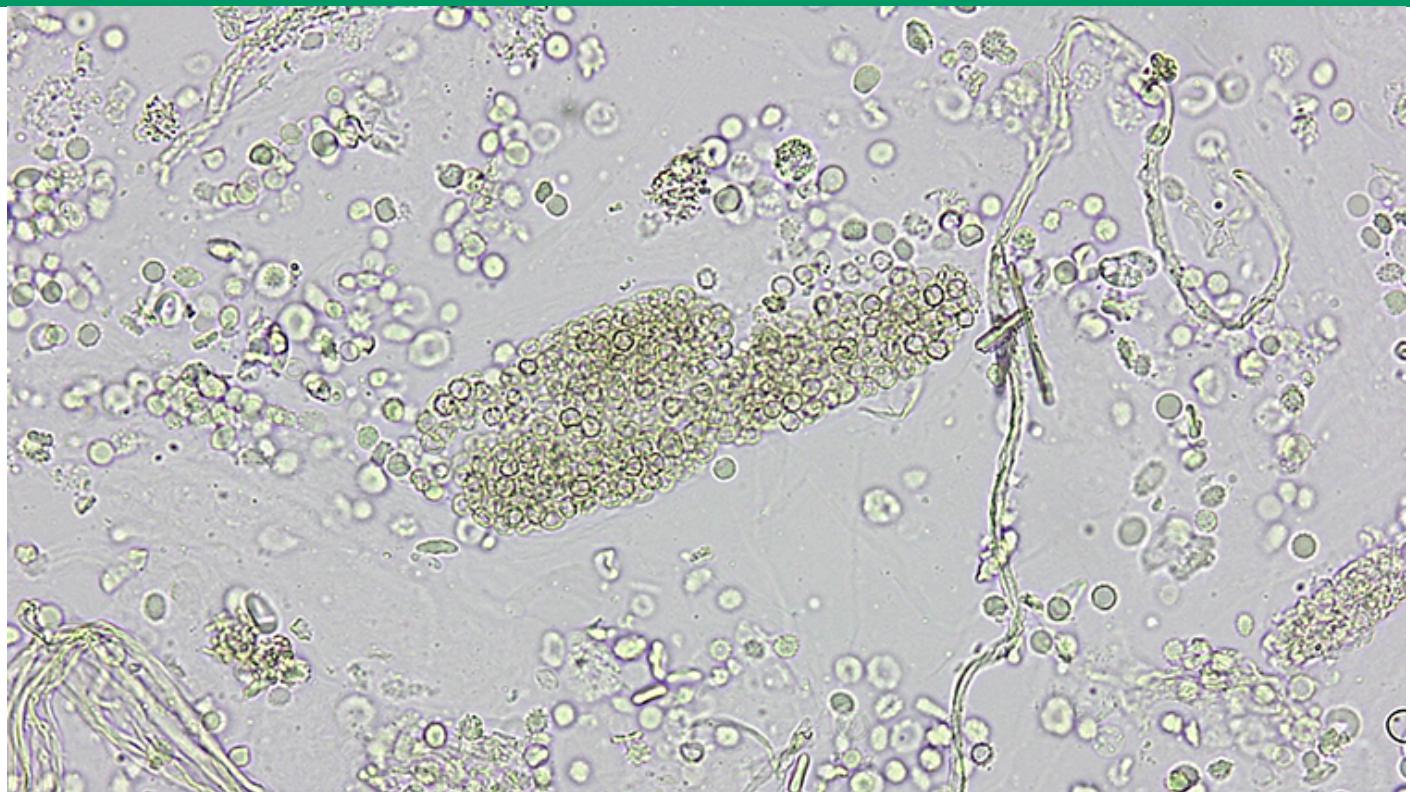


A group of squamous epithelial cells in urine. The cells are large and flat and have some granules in their cytoplasm. The central nucleus is approximately the size of a large lymphocyte. (Bright-field microscopy, 3160.)

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Graphic 83632 Version 1.0

Photomicrograph of urine sediment with a red cell cast



Urine sediment showing free red cells and a red cell cast that is tightly packed with red cells. It is more common for red cell casts to have fewer red cells trapped within a hyaline or granular cast. Red cell casts are virtually diagnostic of glomerulonephritis or vasculitis.

Courtesy of James F Simon, MD.

Graphic 55778 Version 4.0

Photomicrograph of urine sediment with white blood cell cast (I)



White cell cast in which blue stained white cells (arrow) are contained within a granular cast.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 54319 Version 3.0

Photomicrograph of urine sediment with white blood cell cast

(II)

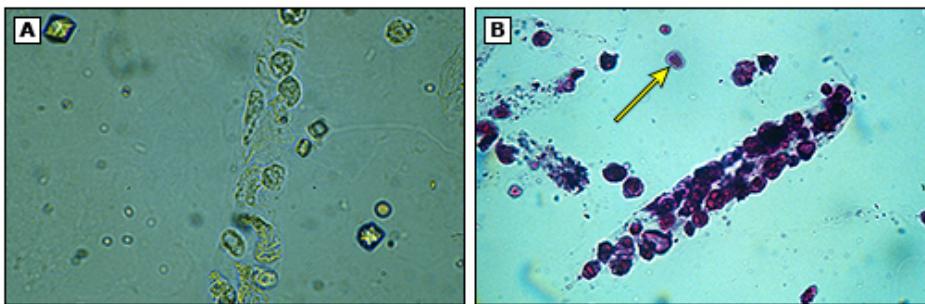


A white blood cell cast, three-quarters of which is filled with leukocytes.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 68147 Version 2.0

Photomicrograph showing tubular epithelial cell casts



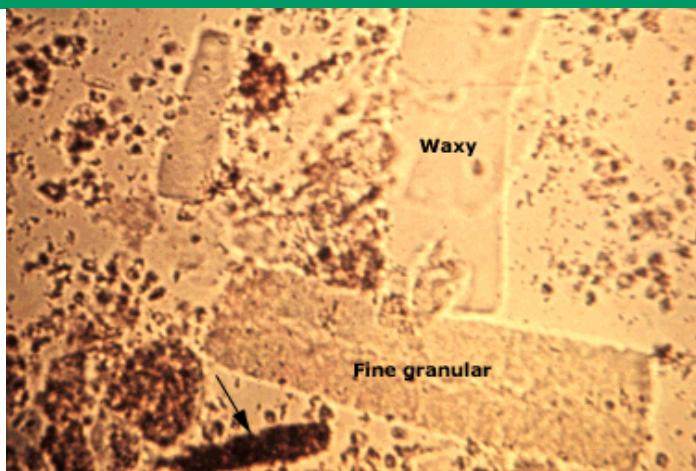
(A) Epithelial cell cast containing cells that are larger than white cells.

(B) Epithelial cell cast with free epithelial cells (arrow) in the urine sediment. Renal tubular epithelial cells are larger than white cells and have a single, large central nucleus.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 65729 Version 6.0

Granular and waxy casts



Urine sediment showing waxy and fine and coarse (arrow) granular casts. The broader casts are thought to form when there is stasis (due to advanced renal failure) in the wider collecting tubules into which many nephrons drain.

Courtesy of Harvard Medical School.

Graphic 59811 Version 1.0

Urine sediment showing a hyaline cast



Hyaline cast

Representative photomicrographs of unstained elements in urine.

Reproduced from: Brunzel NA. Fundamentals of urine and body fluid analysis, 2nd ed, WB Saunders, Philadelphia, 2004. Illustration used with the permission of Elsevier Inc. All rights reserved.

Graphic 86339 Version 1.0

Urine sediment showing a waxy cast

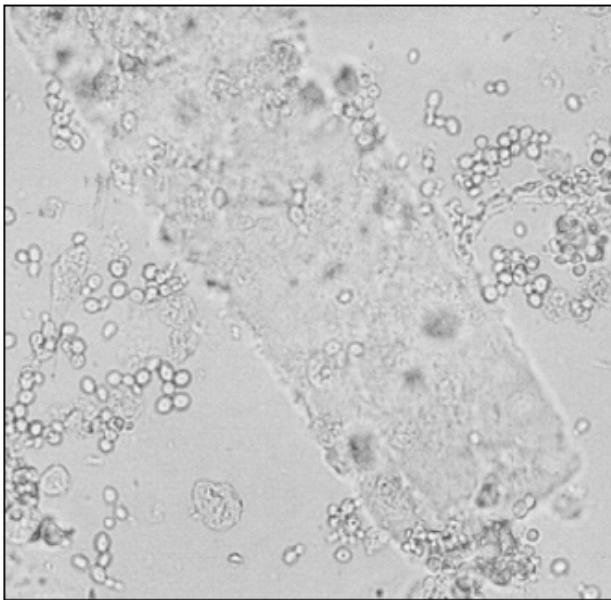


Waxy cast

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Graphic 83885 Version 1.0

Urine sediment showing a broad cast

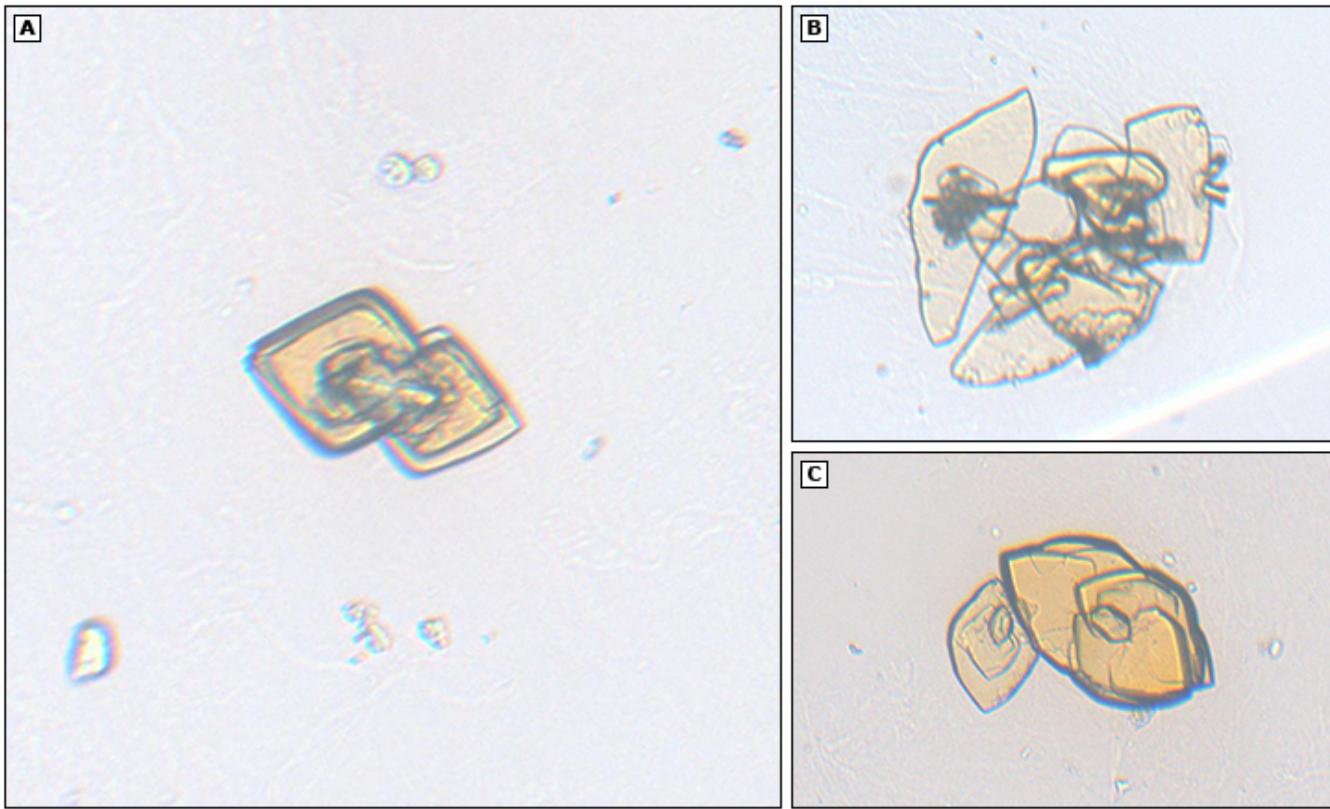


Broad waxy cast

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Graphic 86555 Version 1.0

Uric acid crystals in the urine

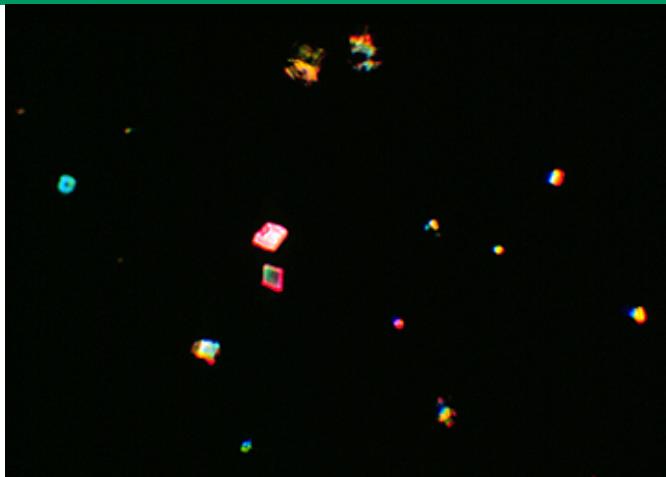


These crystals are pleomorphic, most often appearing as rhombic plates or rosettes. They are yellow or reddish-brown and form only in an acid urine (pH 5.5 or less).

Courtesy Gary C Curhan, MD, ScD.

Graphic 61827 Version 4.0

Uric acid crystals under polarized light

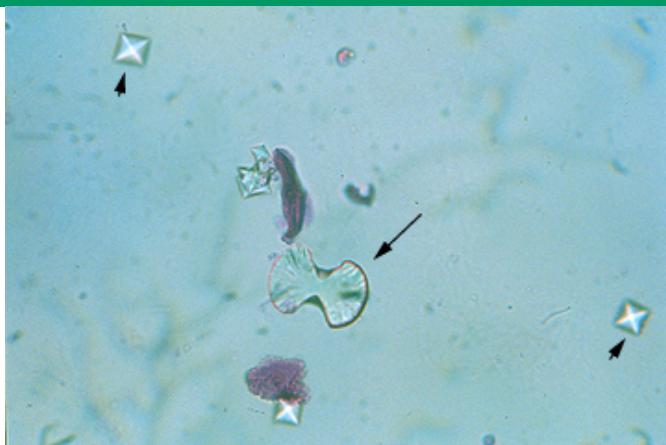


Urine sediment showing uric acid crystals viewed under polarized light.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 73642 Version 2.0

Calcium oxalate crystals in the urine

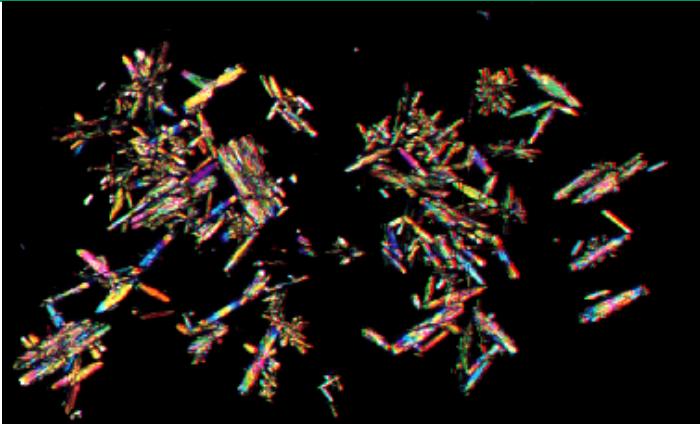


Urine sediment showing both dumbbell-shaped calcium oxalate monohydrate (long arrow) and envelope-shaped calcium oxalate dihydrate (short arrows) crystals. Although not shown, the monohydrate crystals may also have a needle-shaped appearance. The formation of calcium oxalate crystals is independent of the urine pH.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 65169 Version 2.0

Urinary calcium oxalate monohydrate crystals under polarized light

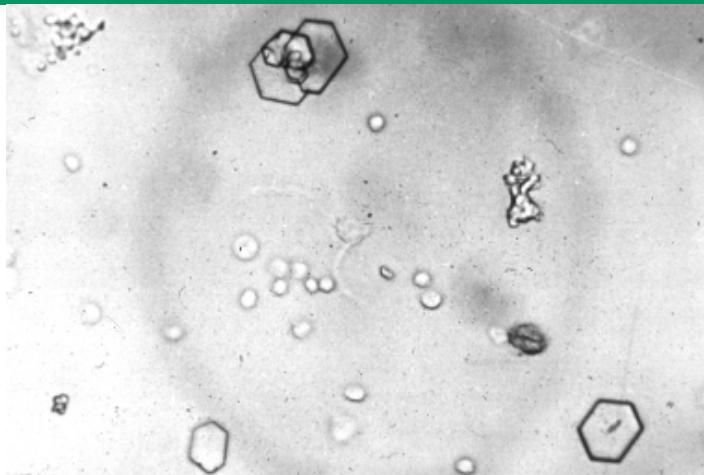


Urine sediment viewed under polarized light showing coarse, needle-shaped calcium oxalate monohydrate crystals. These crystals have a similar appearance to hippurate crystals.

Courtesy of W Merrill Hicks, MD.

Graphic 67694 Version 2.0

Urine sediment showing cystine crystals



Urine sediment showing hexagonal cystine crystals that are essentially pathognomonic of cystinuria.

Courtesy of Harvard Medical School.

Graphic 56834 Version 2.0

Urine sediment showing struvite (magnesium ammonium phosphate) crystals



Urine sediment showing multiple "coffin lid" magnesium ammonium phosphate crystals (struvite) that form only in an alkaline urine (pH usually above 7.0) caused by an upper urinary tract infection with a urease-producing bacteria.

Courtesy of Harvard Medical School.

Graphic 54594 Version 6.0

Urine sediment showing bacteria, budding yeast, and hyphae

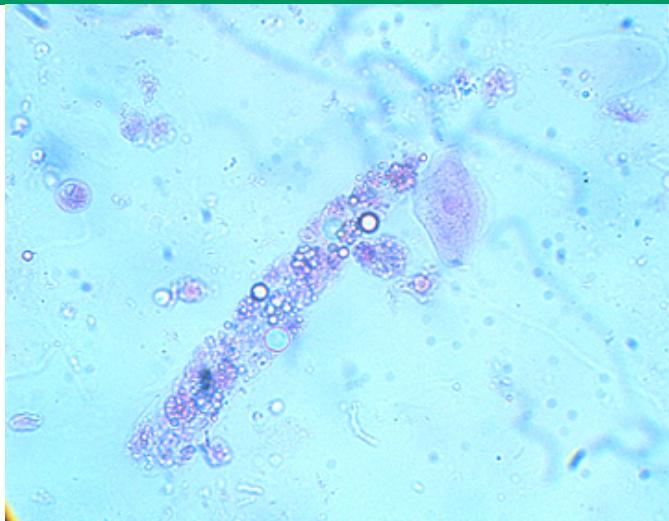


The background contains budding yeast and hyphae (white arrow), as well as a bacteria (yellow arrows). There is also a broad hyaline cast. (Bright-field microscopy, 3100.)

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Graphic 83631 Version 1.0

Fatty cast

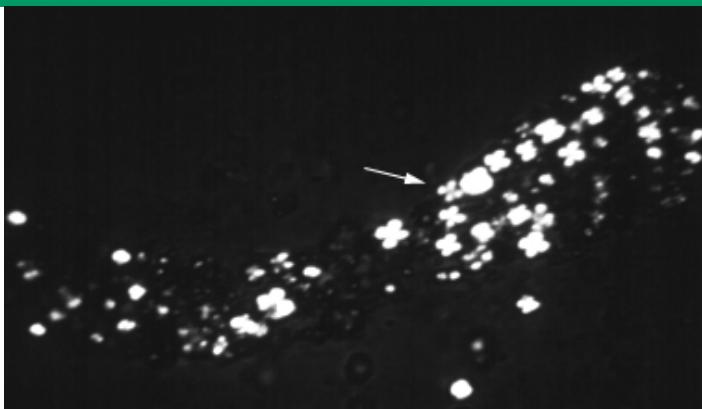


Urine sediment showing a fatty cast. The fat droplets (or globules) can be distinguished from red cells (which also have a round appearance) by their variable size (from much smaller to much larger than a red cell), dark outline, and "Maltese cross" appearance under polarized light.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Graphic 69603 Version 1.0

Fatty cast



Urine sediment showing fatty cast under polarized light. The fat droplets have a characteristic "Maltese cross" appearance (arrow).

Courtesy of Harvard Medical School.

Graphic 79604 Version 1.0

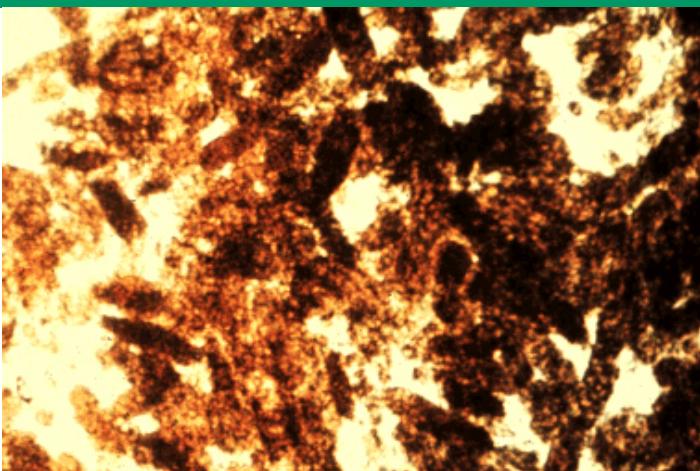
Urinary patterns associated with different kidney diseases

Urinary pattern	Kidney disease suggested by pattern
Hematuria with dysmorphic red blood cells, red blood cell casts, varying degrees of albuminuria	Proliferative glomerulonephritis (eg, IgA nephropathy, ANCA-associated vasculitis, lupus nephritis)
Heavy albuminuria with minimal or absent hematuria	Nonproliferative glomerulopathy (eg, diabetes, amyloidosis, membranous nephropathy, focal segmental glomerulosclerosis, minimal change)
Multiple granular and epithelial cell casts with free epithelial cells	Acute tubular necrosis in a patient with underlying acute kidney injury
Isolated pyuria	Infection (bacterial, mycobacterial) or tubulointerstitial disease
Normal urinalysis with few cells, no casts, and no or minimal proteinuria	In presence of acute kidney injury: prerenal disease, urinary tract obstruction, hypercalcemia, acute phosphate nephropathy, myeloma cast nephropathy In presence of chronic kidney disease: ischemic nephropathy, hypertensive nephrosclerosis, urinary tract obstruction, hepato renal disease, cardiorenal disease

IgA: immunoglobulin A; ANCA: antineutrophil cytoplasmic antibody.

Graphic 56160 Version 10.0

Photomicrograph showing urine sediment with muddy brown granular casts



Urine sediment showing multiple muddy brown granular casts. These findings are highly suggestive of acute tubular necrosis in a patient with acute kidney injury.

Courtesy of Harvard Medical School.

Graphic 56438 Version 6.0

