



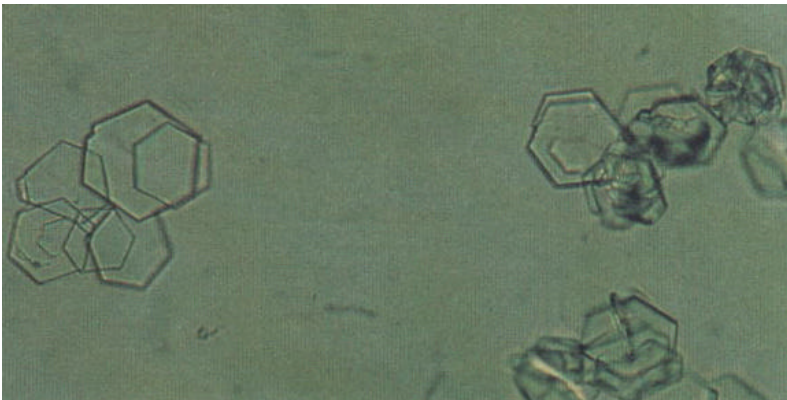
# Holy C.O.W.!

It's...

Clinical Question of the Week #16  
October 13th, 2008 through October 20th,  
2008

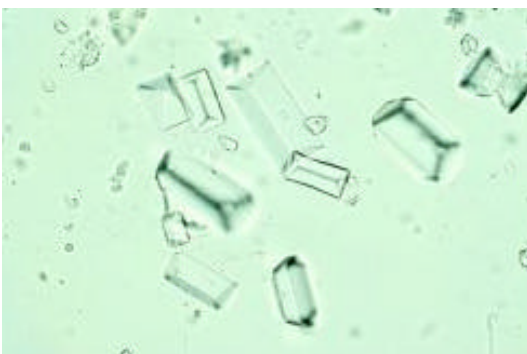
Please e-mail your answers to Kuo, Tim, Wendy, and Kevin ([klian@mednet.ucla.edu](mailto:klian@mednet.ucla.edu); [tprovias@mednet.ucla.edu](mailto:tprovias@mednet.ucla.edu); [wsimon@mednet.ucla.edu](mailto:wsimon@mednet.ucla.edu); [kbreger@mednet.ucla.edu](mailto:kbreger@mednet.ucla.edu)) by 0800 on Monday, October 20th, 2008. The resident or intern with the most correct answers at the end of each month will receive a prize!

**Case 1:** A 24-year-old man presents with nephrolithiasis. Examination of the urine sediment is shown below. You take the specimen to the clinical lab (RR B-403) where you and the tech alkalinize the sample, then add a few drops of sodium cyanide, followed by a few drops of sodium nitroprusside. After this, the specimen turns pinkish red.



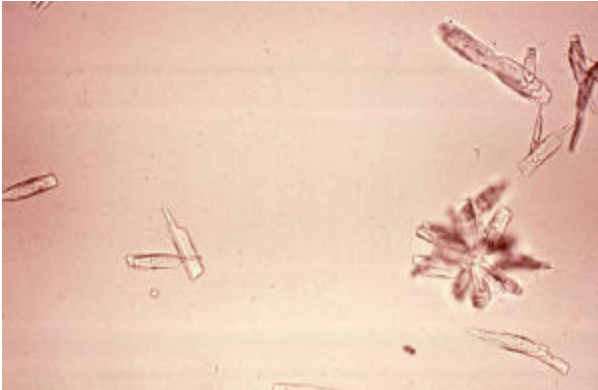
Case 1 urine sediment

**Case 2:** A 30-year-old woman with history of spina bifida presents to IMS with nephrolithiasis and recurrent urinary tract infections due to chronic indwelling urinary catheter. You take a specimen to the microscope for sediment analysis after spinning the sample. The urine sediment is shown.



Case 2 urine sediment

**Case 3:** A 63-year-old woman presents with gradual onset of abdominal pain and “achy bones.” She is also found to have nephrolithiasis as well as acute renal failure on evaluation. After spinning the sample in the clinical lab, you and your on call team look at the sediment, which is shown below.



Case 3 urine sediment

**Questions:**

**1. What is the diagnosis and stone composition in Case 1?**

Cystinuria, a rare autosomal recessive cause of kidney stones. Patients with this disorder have impaired renal and intestinal cysteine transport, resulting in increased filtered and excreted cysteine. Various phenotypes are identified based on the amounts of cysteine excreted by patients and causative genetic mutations in *SLC3A1* and *SLC7A9* are noted. Diagnosis is based on history, phenotype of kidney stones, and urine sediment analysis, which reveals pathognomonic hexagonal crystals. Treatment is based on maintaining the concentration of cysteine in the urine below its solubility limit, including measures of high fluid intake and urine alkalinization. (1)

**2. Name the stone composition in Case 2 and one associated infectious risk factor.**

Struvite stones are composed of magnesium ammonium phosphate, calcium carbonate-apatite, or a combination of both. They may occur de novo after development of upper urinary tract infection, with an increased frequency (3:1) among women. Urease producing bacteria are usually implicated, including *Proteus* and *Klebsiella* species. Other risk factors include neurogenic bladder and/or urinary diversion; in the latter, staples and mucus in the loop may serve as a nidus for crystal formation with characteristic “coffin-lid” shapes. (1)

**3. What is the diagnosis and stone composition in Case 3?**

Hyperparathyroidism with resultant hypercalcemia, resulting in the classic findings of “stones, bones, abdominal groans, and psychic moans.” Seen in about 15-20% of patients with hyperparathyroidism, other symptoms caused by hypercalcemia include anorexia, constipation, nausea, polyuria, and polydipsia. Other renal/urinary manifestations of hyperparathyroidism include hypercalciuria, nephrocalcinosis, chronic renal insufficiency, and decreased concentrating ability of the renal tubules. While most stones formed in hyperparathyroidism are calcium oxalate stones, a slightly alkaline urine may favor the precipitation of calcium *phosphate* stones (shown in the image in Case 3). (1, answers with multiple myeloma also accepted)