Acute tubular necrosis is a major cause of acute renal failure. Acute renal failure that is caused by crystal deposition can result from drug toxicity, lymphoplasmacytic neoplasms, ingestion of industrial organic solvents, or intratubular obstruction due to degenerated red blood cells and red blood cell casts. We herein present an uncommon case of acute renal failure in a 57-year-old woman showing an unusually massive accumulation of variable-sized, round, ellipsoid or rhomboid, pale-pink, refractile bodies in the proximal and distal tubular epithelial cells, interstitial macrophages and Bowman’s spaces. These bodies were electron dense with a maximum diameter of \(3 \mu m\). The information we gathered from the patient history, the laboratory data and the various histochemical and immunohistochemical analyses failed to reveal the exact nature of these crystal-like structures.

**Key Words:** Kidney Failure, Acute-Kidney Tubular Necrosis, Acute-Crystal-like Structures

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**CASE REPORT**

A 57-year-old woman was admitted to our hospital due to renal failure; her creatinine level at that time was 15.2 mg/dL and her BUN level was 152.4 mg/dL. She was also suffering from anemia. Her BUN and creatinine levels soon increased up to 84 mg/dL and 15.2 mg/dL, respectively. Her medical history was significant as she’d had pulmonary tuberculosis and bronchiectasis 30 years and 4 years prior to her hospital admission, respectively. She denied any history of medication, diarrhea, dehydration or other possible causes for the acute decline in her renal function. The hemoglobin and hematocrit levels were 9.9 g/dL and 30.4%, respectively. Urinalysis revealed microscopic hematuria, but there was no proteinuria event. A renal biopsy was performed, and the following biopsy analysis showed:

1. **Light microscopy** demonstrated mild ischemia in the glomeruli. The tubules showed extensive necrosis with sloughing of the epithelium into the lumen, and there was rupture of the tubular basement membrane. The proximal and distal tubular epithelial cells were swollen and stuffed with variable sized (up to 3 \( \mu m \)) round-to-ovoid, pale pink refractile but non-birefringent structures that stained red on aldehyde fuchsin orange G stain, but these structures did not stain with periodic acid schiff (PAS) and Prussian blue. The interstitium showed...
mild fibrosis and it was infiltrated by macrophages; it also contained particles similar to those seen in the tubules (Fig. 1).

Immunofluorescence study demonstrated no specific findings either in the glomeruli or in the interstitium. No light chain restriction was demonstrated by a immunohistochemical analysis for kappa and lambda light chains.

Electron microscopy was then performed on the paraffin-embedded renal tissue. The particles were electron-dense with occasional clefts, and they were round, ellipsoid or rhomboid in shape (Fig. 2). They were present not only in the tubular epithelial cells and interstitium, but also in the glomerular endothelial cells. However, no clear relationship between these particles and the intracellular organelles could be demonstrated.

The patient was treated with conservative care including hemodialysis for 40 days. Her general condition began to improve, and her BUN and creatinine levels dropped to 104 mg/dL and 3.2 mg/dL, respectively. The patient was discharged on the 47th hospital day.

**DISCUSSION**

A variety of causative agents can result in crystal deposition which can cause acute renal failure; such causes are drug toxicity (e.g. acyclovir, sulfonamide, methotrexate and triamterene), lym

phoplasmacytic neoplasm, ingestion of industrial organic solvents (e.g. perchloroethylene) or intratubular obstruction due to degenerated red blood cells and red blood cell casts.

Our case showed an unusually extensive deposition of round, ovoid, ellipsoid or rhomboid, pale pink refractile bodies in the tubular lumens, tubular cell cytoplasm, glomerular endothelial cells, and interstitium. On electron microscopy, these refractile bodies appeared as round, ellipsoid, rhomboid or needle-like electron dense particles that were located in glomerular endothelial cells, tubular epithelial cells and interstitium.

Crystals that accumulate due to drug toxicity take on variable shapes including acidophilic and amorphous deposits on hematoxylin and eosin sections. These crystals stain black on von Kossa
stain. They are often birefringent on polarized microscopy, and sometimes a foreign body type giant cell reaction is evident. However, we eliminated this possibility as the patient denied any history of drug use or prior medication, and the crystals were not birefringent under polarized microscopy. Moreover, no foreign body type giant cell reaction was elicited around the crystals on the histological examination.

Crystals associated with lymphoplasmacytic neoplasms, and especially plasma cell myeloma, are deposited in the tubular epithelium, glomeruli and the interstitium; they are generally needle-like or rhomboid in shape. These crystals have been associated with a foreign body-type giant cell response. Such crystals are, in fact, immunoglobulin secreted by tumor cells, and they are PAS-positive. An ultrastructural examination of these crystals has typically demonstrated linear lattices with regular linear arrays of fibrils. They have also been demonstrated in various other organs, including bone marrow, lung, heart, spleen, lymph node, and bowel. In our present case, there was no laboratory evidence for supporting a diagnosis of a lymphoplasmacytic neoplasm in spite of the one-year clinical follow up that was done. The crystal-like structures were PAS-negative, and they did not demonstrate light chain restriction on kappa and lambda immunohistochemistry. They were also not associated with a giant cell reaction, and no ultrastructural evidence for linear lattices with regular linear arrays of fibrils was found; this made the diagnosis of lymphoplasmacytic neoplasm even less likely.

Crystal-like structures may also be seen in ATN due to intratubular obstruction by red blood cells. Crystal-like structures have been demonstrated following erythropagocytosis in the tubular cells. Similar inclusions have been found in the proximal and distal tubular cells of rat kidney in which heparinized autologous blood was injected using micropuncture techniques. However, this is unlikely to have occurred in our case as our patient presented with microscopic hematuria; such previously reported cases have been associated with gross hematuria. There was also no evidence of any hemosiderin-laden macrophage accumulations around the crystal-like structures, or any red blood cell casts, or pigmented casts.

We report here on an unusual case of acute renal failure associated with extensive deposition of crystal-like structures. The exact nature of the structure was not revealed by the patient’s clinical history, the histological findings, histochemical or immunohistochemical analyses, or the ultrastructural findings.

**REFERENCES**


