INTRODUCTION

Acute renal cortical necrosis (ACN) is an uncommon but often catastrophic cause of acute kidney injury (AKI). Although the exact incidence of ACN is unknown, it is reported to account for 1% to 2% of all cases of AKI in developed countries. Commonly, ACN occurs as a result of catastrophic obstetric complications. We describe an unusual case of AKI due to renal cortical necrosis attributed to \textit{N}-methylamphetamine (‘crystal meth’) use.

CASE PRESENTATION

A 27-year-old Caucasian homeless woman presented to the emergency department with symptoms of vomiting, poor oral intake, lower back pain, and decrease in urine output of 2 days’ duration. The patient reported frequent use of nonsteroidal anti-inflammatory drugs for the past 4 years. Her past medical history was significant for chronic hepatitis C infection and a spontaneous second-trimester miscarriage. She denied any history of clotting disorders or autoimmune disease. The patient had a history of i.v. drug use for the past 10 years, and her previous toxicology screens had been positive for cocaine, marijuana, and opiates. She reported frequent use of both oral and i.v. crystal meth (methamphetamines) for the past 2 months. On examination, she was conscious and oriented. She was afebrile with a heart rate of 63 bpm, respiratory rate of 16 breaths/min, and blood pressure of 119/70 mm Hg. Her remaining physical examination findings were unremarkable except for anasarca. Laboratory evaluation revealed serum creatinine of 6.4 mg/dl and blood urea nitrogen of 34 mg/dl. No recent baseline creatinine value was available, and she denied any past history of kidney disease. The rest of the laboratory data were as follows: leukocyte count 13,700/µl, hemoglobin 11.9 g/dl, platelets 96,000K/UL, bicarbonate 23 mEq/dl, and creatinine phosphokinase 108 IU/l. Aspartate aminotransferase and alanine aminotransferase were mildly elevated at 219 IU/l and 82 IU/l, respectively. A pregnancy test result was negative. Urine examination showed large blood, protein 100 mg/dl, 24 red blood cells per high-power field, and no leukocytes. Renal ultrasound revealed normal sized kidneys without any hydronephrosis. Blood culture results were negative. With differential diagnosis of acute glomerulonephritis and acute interstitial nephritis, further a workup was performed. A component of prerenal AKI was suspected, and treatment was initiated with isotonic i.v. fluids. However, on day 2, the patient remained anuric with worsening renal function and refractory hyperkalemia, following which emergent dialysis had to be initiated.

Serological workup showed mildly decreased complement C3 at 82 mg/dl (normal range, 88–206 mg/dl), normal complement C4, and positive rheumatoid factor at 23.9 IU/ml. Antinuclear antibody and anti-neutrophil cytoplasmic antibody serology results were negative. Both hepatitis B core antibody and hepatitis B surface antibody results were positive, whereas hepatitis B surface antigen and hepatitis C RNA result were negative. Serum cryoglobulin results were also negative. Anticardiolipin IgM was mildly elevated at 21.1 IgM phospholipid units and lupus anticoagulant results were negative.

Kidney biopsy was performed on day 4. Sections for light microscopy had 19 glomeruli, 2 of which were globally sclerotic. Viable glomeruli were of normal overall size and cellularity. There was widespread coagulative necrosis of the renal cortex (Figure 1). There was also a sharp demarcation between areas of coagulative necrosis and the surrounding inflamed but nonnecrotic tissue (Figure 2). No vasculitis or evidence of thrombotic microangiopathy was seen in the viable kidney. Immunofluorescence staining was negative.
Toluidine blue–stained sections revealed extensive coagulative necrosis. Transesophageal echocardiography showed that the patient’s ejection fraction was 55% with no evidence of emboli, vegetation, or endocarditis. The patient’s further hospital course was complicated by line-associated bacteremia, for which she was successfully treated with i.v. antibiotics. She has remained dialysis dependent during 2 months of follow-up.

**DISCUSSION**

Acute renal cortical necrosis is a rare cause of AKI, with a reported incidence of about 2.0% in developed countries. In contrast, in developing countries, the incidence is higher, and about 6% to 7% of AKI cases are attributed to ACN. Pregnancy related complications remain the most common cause of ACN. About 60% to 70% of ACN cases are sequelae of obstetric complications such as septic abortion, puerperal sepsis, abruptio placentae, postpartum hemorrhage, and eclampsia. The remaining 30% to 40% are attributed to nonobstetric causes, with fulminant sepsis and hemolytic uremic syndrome being the most common ones. Other causes of nonobstetric ACN include snake bites, malaria, renal trauma, acute pancreatitis, diabetic ketoacidosis, and hyperacute renal transplant rejection. With improvement in obstetric health care, and a marked decline in septic abortion, the incidence of obstetric ACN has decreased significantly. However, novel causes of non-obstetric ACN have emerged in the literature, which include prescription drugs (bisphosphonates, tranexamic acid) and drugs of abuse (synthetic cannabinoids). Table 1 lists the known causes of ACN. To the best of our knowledge, this is the first case report of N-methylamphetamine (crystal meth)—induced ACN as a non-obstetric cause of AKI in a young white woman.

The typical histological feature of ACN is total ischemic necrosis of the affected area of the renal cortex (glomeruli, blood vessels, and tubules). Two types of cortical necrosis are recognized on the basis of renal

![Figure 1](image1.png)

**Figure 1.** Necrotic tissue with ghost outlines of glomeruli and tubules with loss of cellular detail (hematoxylin and eosin stain; original magnification ×200).

![Figure 2](image2.png)

**Figure 2.** Sharp demarcation between eosinophilic necrotic parenchyma and inflamed but viable tissue (hematoxylin and eosin stain; original magnification ×100).

Table 1. Causes of acute renal cortical necrosis

| Obstetric                        | Non-obstetric                          |
|----------------------------------|----------------------------------------|
| Septic abortion                  | Drugs                                  |
| Puerperal sepsis                 | Nonsteroidal anti-inflammatory drugs   |
| Abruptio placentae               | Tranexamic acid                        |
| Postpartum hemorrhage            | Polyethylene glycol                    |
| Eclampsia                        | Quinine                                |
|                                  | Bisphosphonates                        |
| Drugs of abuse                   |                                        |
| Synthetic cannabinoids          |                                        |
| Alcohol                          |                                        |
| Diethylene glycol                |                                        |
| Poisonings                       |                                        |
| Snake bite                       |                                        |
| Wasp sting                       |                                        |
| Organophosphorus poisoning       |                                        |
| Laundry detergent ingestion      |                                        |
| Infections                       |                                        |
| Malaria                          |                                        |
| Streptococcal pharyngitis        |                                        |
| Sepsis                           |                                        |
| Acute gastroenteritis            |                                        |
| Protein S deficiency following varicella |                         |
| AIDS                             |                                        |
| Hemolytic uremic syndrome        |                                        |
| Trauma/hemorrhagic shock         |                                        |
| Burns                            |                                        |
| Pancreatitis                     |                                        |
| Diabetic ketoacidosis            |                                        |
| Glucose-6-phosphate dehydrogenase|                                        |
| deficiency with intravascular hemolysis |                      |
| Hyperacute kidney transplant rejection |                                    |
| SLE-associated antiphospholipid syndrome |                  |

SLE, systemic lupus erythematosus.
Acute renal cortical necrosis is a rare cause of acute kidney injury. Methamphetamine and other related drugs of abuse are emerging as an important cause of non-obstructive acute renal cortical necrosis. The most common presentation of acute cortical necrosis is anuric acute kidney injury that often requires initiation of renal replacement therapy. Renal biopsy is considered the gold standard in the diagnosis of acute renal cortical necrosis. Although acute renal cortical necrosis is partially reversible in 20% to 40% of cases, patients frequently require long-term renal replacement therapy. To the best of our knowledge, this is the first case illustrating N-methylamphetamine as a cause of acute renal cortical necrosis, and clinicians should be aware of its associated potential complications.

Table 2. Teaching points

- Acute renal cortical necrosis is a rare cause of acute kidney injury.
- Methamphetamine and other related drugs of abuse are emerging as an important cause of non-obstructive acute renal cortical necrosis.
- The most common presentation of acute cortical necrosis is anuric acute kidney injury that often requires initiation of renal replacement therapy.
- Renal biopsy is the gold standard for diagnosis of acute renal cortical necrosis.
- Although acute renal cortical necrosis is partially reversible in 20% to 40% of cases, patients frequently require long-term renal replacement therapy.
- To the best of our knowledge, this is the first case illustrating N-methylamphetamine as a cause of acute renal cortical necrosis, and clinicians should be aware of its associated potential complications.

Table 2 shows the teaching and interesting points of our case. Our case highlights that renal cortical necrosis remains an important differential in the diagnosis of acute kidney injury, especially with emergence of an epidemic of drug abuse. As in this case, N-methylamphetamine may not be used in isolation, so the use of other drugs may contribute to its effects. Although the patchy variety may be partially reversible, the more common outcome is long-term dialysis dependence.

**DISCLOSURE**

All the authors declared no competing interests.
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