

**Case Report****Amphetamine Induced Acute Interstitial Nephritis with RBC Casts**

Suma Raju\*

**ABSTRACT**

**Background :** Intravenous amphetamine drug use is known to cause acute renal failure due to rhabdomyolysis / hyperpyrexia, and rarely due to necrotizing angitis and acute interstitial nephritis.

We report a case of 46 y/o Caucasian male who presented with nausea, vomiting and abdominal pain two days post intravenous amphetamine use, found to have acute renal failure, with urinalysis showing red blood cell casts. All serologies being negative, renal biopsy showed evidence of acute interstitial nephritis.

**Conclusion :** Amphetamine intravenous drug abuse can cause acute interstitial nephritis and RBC casts can be seen in the setting of acute interstitial nephritis.

**Key words :** Amphetamine, Acute renal failure, Acute Interstitial Nephritis, RBC Casts

**Introduction -**

Literature review shows that Amphetamine Intravenous Drug Abuse (IVDA) is well known to cause acute renal failure (ARF) most commonly due to hyperpyrexia, rhabdomyolysis, occasional reports of myoglobinuric renal failure and necrotizing angitis<sup>1,2,3,4,5,6</sup> but there has been only one case report of amphetamine IVDA causing acute interstitial nephritis so far<sup>7</sup>. Here we report yet another case of amphetamine IVDA induced acute renal failure (ARF) present with hematuria and RBC urine casts due to acute interstitial nephritis (AIN) which is quite rare in AIN<sup>8</sup>.

**Case Report -**

43 year old Caucasian male with unremarkable past medical history except for Intra venous drug abuse 10 yrs ago presented to the emergency room with complaints of nausea, vomiting, diffuse abdominal pain and dark urine for 10 days. On further questioning patient admits to IV methamphetamine use two days prior to his symptoms. He reported that

following methamphetamine use, he experienced episodes of confusion, myalgia, palpitation, severe retching, heat intolerance and diaphoresis. He denied any chest pain, Shortness of breath, Leg swelling, joint pain, or skin rashes. Initial physical examination was unremarkable. His BP on admission was 152/71 mmhg, HR 81 regular temp 98.8° F, Respiratory Rate 20/min. His admission labs showed Sodium 138 mEq/L, potassium 5.5 mEq/L, chloride 95 mEq/L, bicarbonate 14 mEq/L, BUN 193 mg/dL, Cr 16 mg/dL, phosphorus 12.4 mg/dL, WBC 7.7 cumm, Hemoglobin 13.8 g/dL, and hematocrit 39.0%, Creatine kinase 2920 mg/dL. Urinalysis revealed large blood, negative protein, specific gravity 1012, WBC 4-10/hpf, RBC>50 / HPF with many RBC casts (**Fig 1**), negative for eosinophils by Wright giemsa stains. All other serologies were negative including ANA, hepatitis B, hep. C, HIV, Rheumatoid factor, ASO titer, Rapid Plasma Reagin, and cryoglobulin. Serum complements reported within normal limits.

Renal ultrasound showed slightly enlarged kidneys measuring 14.1 cm on the right and 13.8cm on the left with increased echogenicity bilaterally.

The patient was oliguric on presentation & in view of his elevated Blood urea Nitrogen & serum creatinine, hemodialysis was done consecutively for 2 days and then renal biopsy was performed.

\*Consultant Nephrologist,  
Associate Professor of Nephrology,  
Raja Rajeshwari Institute of Medical Science  
Bangalore, India.

**Address for Correspondence -**

Dr. Suma Raju  
E-mail : sumaraju@gmail.com

**Light Microscopy :** (*Fig 2,3*) findings were as follows -

The principal abnormality was the finding of patchy interstitial lymphoplasmacytic infiltrates, with a few eosinophils seen. No granulomas seen. There was also interstitial edema. Tubules contain scattered eosinophilic granular casts, probably representing necrotic cells, and there were few foci of flattened, regenerative tubular lining cells. There were few foci of tubular atrophy and interstitial fibrosis. The few small arteries present showed no evidence of vasculitis.

**Immunofluorescence :** negative for IgG, C3, & C1q

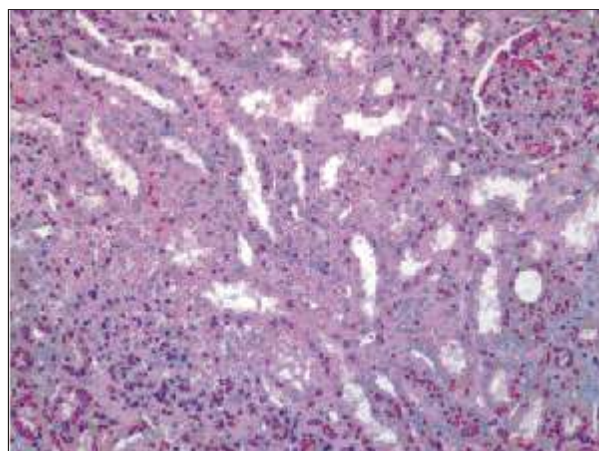
**Electron Microscopy -**

Showed good preservation of ultrastructural detail. The glomerular basement membranes were of normal thickness and configuration. No basement membrane electron-dense deposits were seen. The mesangial region was also unremarkable, without electron-dense deposits. There was mild segmental effacement of foot processes. No tubulo-reticular structures were seen in endothelial cells. Examination with polarized light was negative for talc powder.

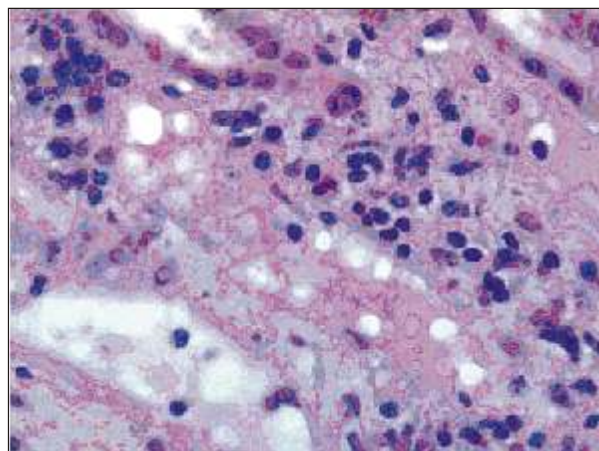
The patient was put on Methylprednisolone (solumedrol) pulse 500 mg IV q day for 3 days then switched to prednisone 60 mg once a day. Patient responded well and dialysis was stopped after few treatments as urine output improved. On discharge serum creatinine was 1.4mg/dl.



**Fig 1 : RBC cast in urine**



**Fig 2 : Kidney biopsy showing normal glomeruli, intact tubules and evidence of interstitial inflammatory infiltrate**



**Fig 3 : Interstitial infiltrate under higher magnification**

### Discussion -

There are multiple causes of acute renal failure due to amphetamine use, most commonly due to rhabdomyolysis, hyperpyrexia<sup>1,4,5</sup> necrotizing angiitis<sup>1,2,3</sup> (which is slightly less common and most often occurs in association with hepatitis antigen) and acute interstitial nephritis evidenced by single case report so far<sup>7</sup>.

Although patient reported that he did experience heat intolerance and myalgias after amphetamine intravenous drug abuse, he had mild elevation in Creatine Kinase on admission and he was afebrile, kidney biopsy did not show any evidence of acute tubular necrosis commonly seen from

rhabdomyolysis and hence in this case the possibility of rhabdomyolysis / hyperpyrexia contributing to acute renal failure is less likely, however can not be excluded entirely.

Necrotizing angitis seemed unlikely as there was no evidence of other organ involvement; hepatitis antigen was negative and no histological evidence of vessels involvement on the kidney biopsy.

To rule out a possibility of amphetamine contaminated with talc powder which may have induced acute interstitial nephritis we did polarized microscopy which was negative.

In our case renal biopsy demonstrated classic interstitial infiltrate with lymphocytes and eosinophils along with interstitial edema with no evidence of vasculitis and the interesting feature being the presence of RBC casts in the urine. Literature review shows a single case report of RBC casts in AIN secondary to possible hydralazine / Thiazide use<sup>8</sup>

Electron Microscopy and immunofluorescence failed to show evidence of any glomerular involvement to explain RBC casts in urine and all serologies were negative. Hence it's most likely that RBC casts were due to acute interstitial nephritis.

Classic urinary findings of Acute Interstitial Nephritis (AIN) include WBC, WBC casts, eosinophils but rarely RBC casts. In our case there were no eosinophils detected using Giemsa Wright stains perhaps due to low sensitivity of Giemsa stains for eosinophils.

Although possible role of rhabdomyolysis in ARF cannot be excluded, certainly this patient did have direct drug effect based on biopsy.

### Conclusion -

43 y/o Caucasian male presented with acute renal failure and hematuria with RBC casts after IV amphetamine use. Renal biopsy shows evidence of

acute interstitial nephritis. This is another case report to suggest that RBC casts can be seen with acute interstitial nephritis in absence of any glomerular injury due to amphetamine IV Drug abuse.

**Conflict of Interest :** Nil reported

### References :

1. Richards JR, Johnson EB, Stark RW and Derlet RW : Methamphetamine Abuse and Rhabdomyolysis in the ED : A 5-year Study. *Am J Emerg Med* 1999; 17 : 681-685.
2. Bingham C, Beaman M, Nicholls AJ and Anthony PP : Necrotizing renal vasculopathy resulting in chronic renal failure after ingestion of methamphetamine and 3, 4-methylenedioxymethamphetamine ('ecstasy'). *Nephrology Dialysis Transplantation* 1998; 13 : 2654-2655
3. Koff RS, Widrich W, and Robbins AH : Necrotizing angitis in a methamphetamine user with hepatitis B-angiographic diagnosis, five month follow-up results and localization of bleeding site. *New England Journal of medicine* 288 : 946-947, 1973.
4. Curry SC, Chang D, Connor D : Drug and toxin-induced rhabdomyolysis. *Annals of Emergency Medicine* 1989; 18 : 1068-1084.
5. Scandling J, Spital A : Amphetamine-associated myoglobinuric renal failure. *Southern Medical Journal* 1982; 75 : 237-240.
6. Cadnapaphomachai P, Taher S, McDonald FD : Acute drug associated rhabdomyolysis : An examination of its diverse renal manifestations and complications. *American Journal of Medical Science* 1980; 280 : 66-72.
7. Richard J Foley, MD, K. Kapatkin, R. Verani and EJ Weinman: Amphetamine-induced acute renal failure. *Southern Medical Journal* 77 : 258-260, 1984.
8. Sigala et.al Red Blood Cell Casts in Acute Interstitial Nephritis *Arch Intern Med.* 1978; 138 : 1419-1421.