



# Hospital Acquired AKI: When "Blame the Vancomycin" is not the Answer

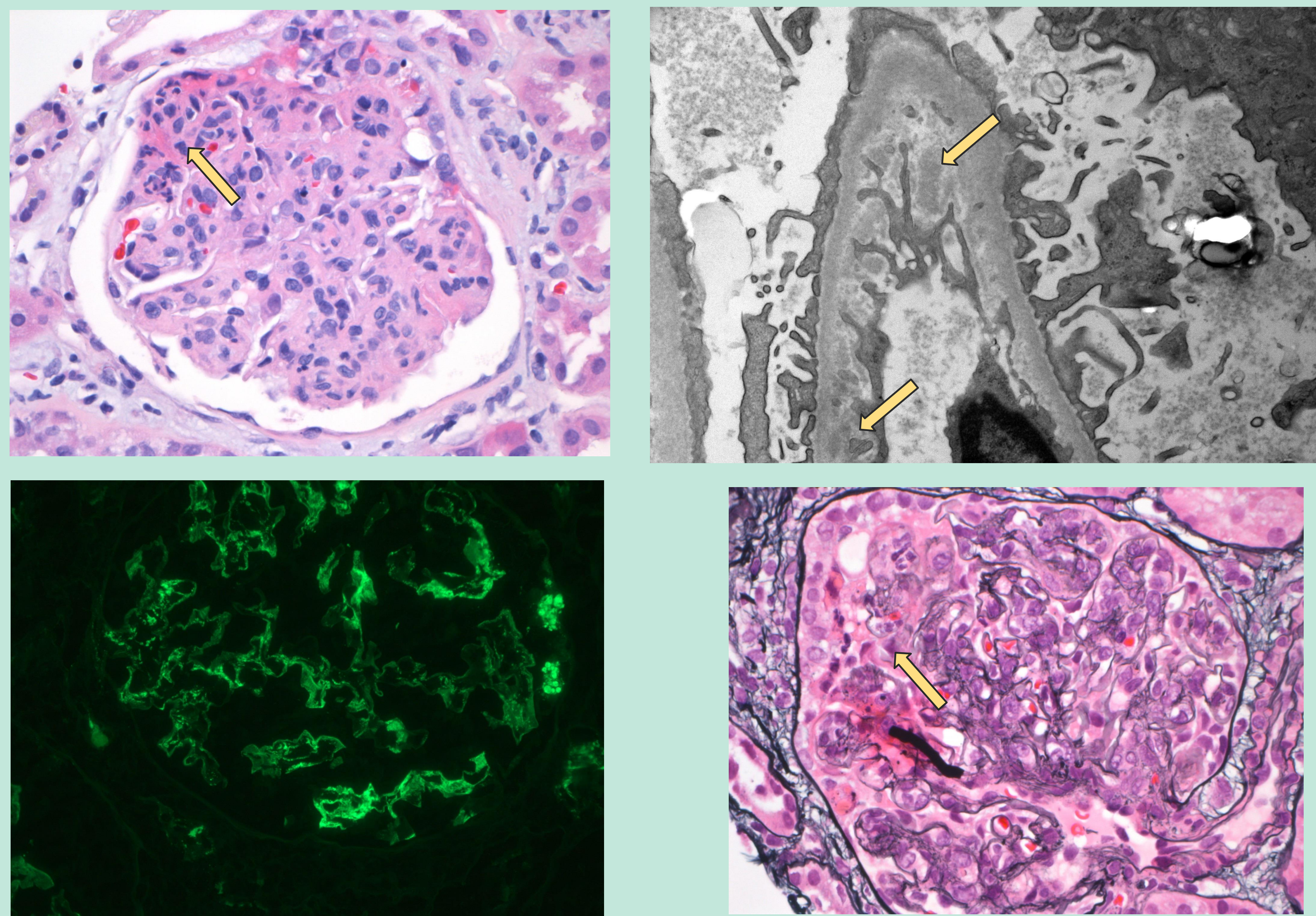
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## Introduction

The etiology of acute kidney injury (AKI) which occurs during a hospitalization can be difficult to assess due to many different overlapping risk factors that are common during hospital admission including radiologic contrast, exposure to new medications, and changes in renal perfusion based on volume status and underlying disease state. This case will highlight an uncommon cause of hospital acquired AKI and briefly review staphylococcus-associated acute glomerulonephritis.

## Case Presentation

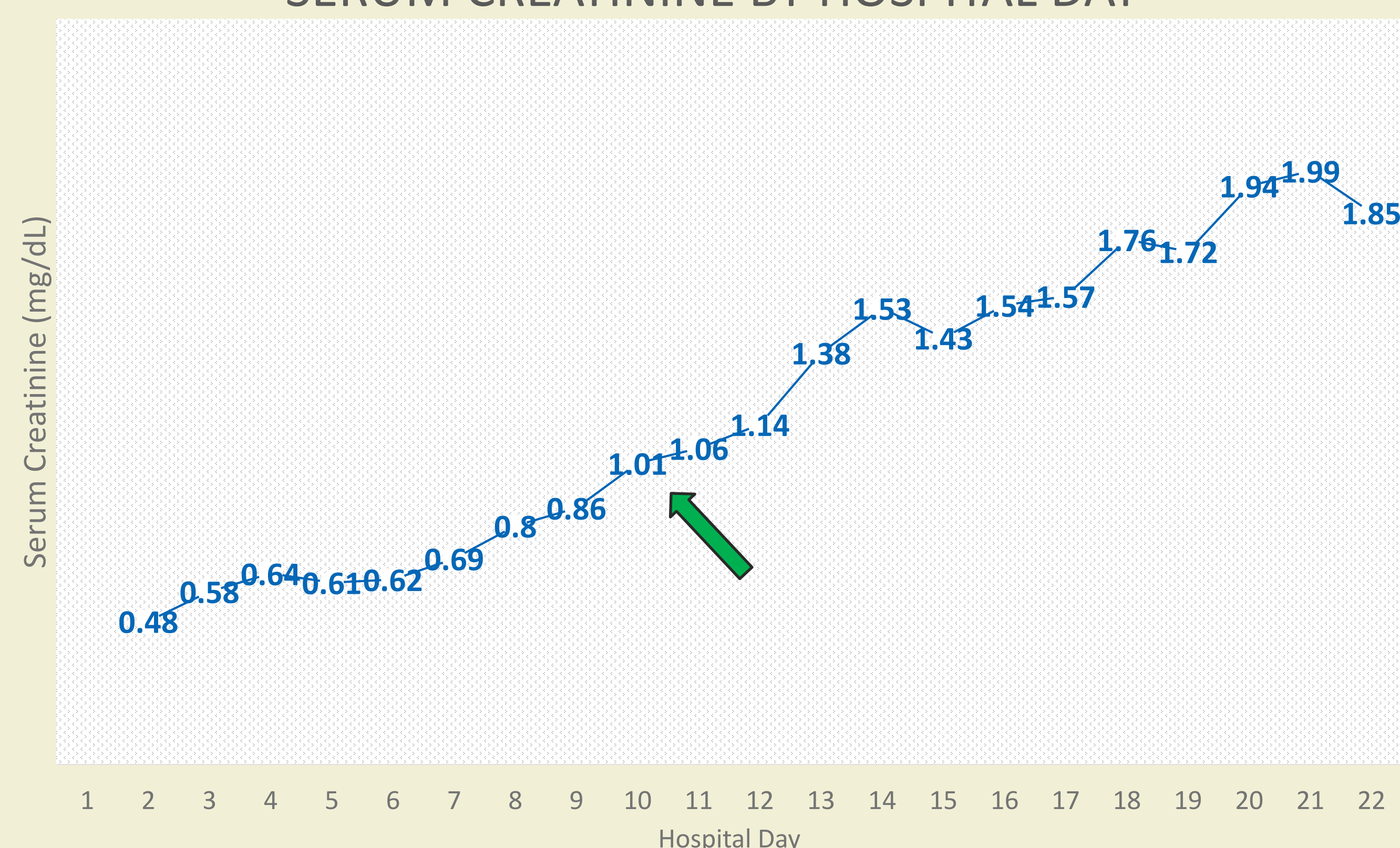
Our patient is a 54 year-old homeless man with active polysubstance abuse including IVDU, PE on anticoagulation with rivaroxaban, and history of treated hepatitis C who presented with low back pain, leg pain, and fever after a reported traumatic injury and was found to be septic with MRSA bacteremia, sacral osteomyelitis, and paraspinal and gluteal muscular abscesses.



**Top Left:** Light microscopy, Glomerulus with mesangial and endocapillary hypercellularity with intracapillary neutrophils (H+E stain).  
**Bottom Left:** IgA immunofluorescence (IF).  
**Top Right:** Electron microscopy, glomerular capillary with subendothelial deposits.  
**Bottom Right:** Light microscopy, Glomerulus with segmental endocapillary hypercellularity with neutrophils and a segmental cellular crescent (Jones silver stain).  
**Important Events insert:** C3 IF.  
**Final Pathology Report:** Native Kidney Biopsy: IgA-dominant acute glomerulonephritis, mild acute interstitial nephritis and acute tubular injury, moderate arteriosclerosis.

Special thanks to Dr. Mazdak Khalighi of the OHSU Department of Pathology for the renal biopsy images and descriptions.

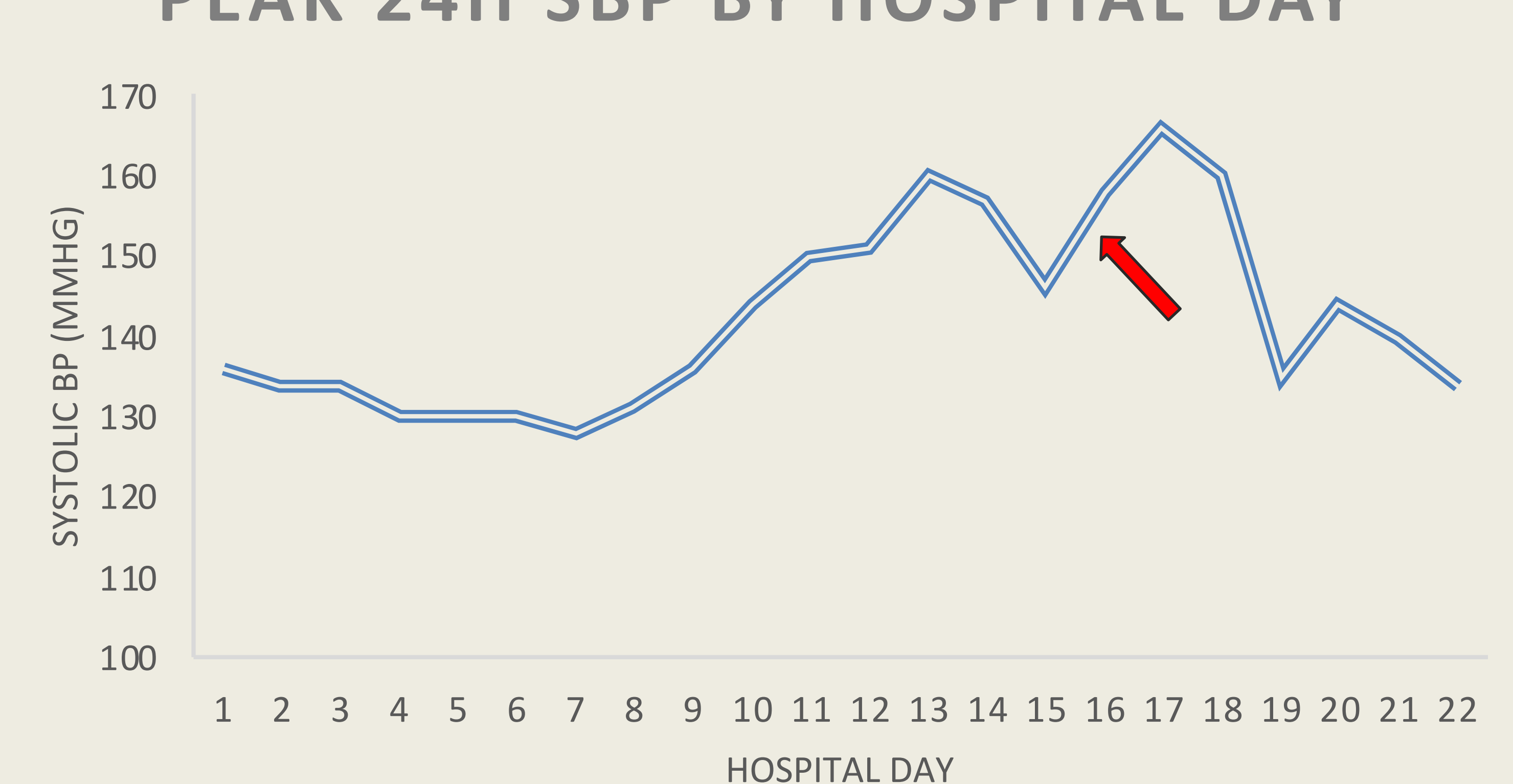
SERUM CREATININE BY HOSPITAL DAY



## Important Events in Hospital Course

- Initial blood cultures with MRSA, Pip/Tazo stopped HD#2
- Clearance of blood cultures HD#8
- Elevated vancomycin trough of 37 mg/dL on HD#10
- Recognition of AKI on HD#10 (Green arrow, left)
- Transition to daptomycin on HD#15
- Development of gross hematuria on HD#16 (Red arrow, right)
- Amlodipine started HD#16
- Renal Biopsy HD#21

PEAK 24H SBP BY HOSPITAL DAY



## Labs:

- C3 26mg/dL (normal), C4 119mg/dL (normal)
- RF <10 IU/mL
- Hepatitis C PCR -> 73,000 IU/mL
- Anti-nuclear antibodies (ANA), Anti-neutrophil cytoplasmic antibodies (ANCA), and Anti-glomerular basement membrane antibody negative
- Urine Protein/Creatinine 24.34 mg/mg (HD#16)
- Urine RBCs 123 (HD#13), 514 (HD#16)
- Fractional Excretion of Sodium (FENa) 1.78% with 280cc urine output (HD#14)

## Imaging:

Ultrasound Kidney and Bladder (HD# 16): Increased renal parenchymal echogenicity suggestive of underlying renal dysfunction. No hydronephrosis. No obvious stone disease.

## Discussion

Our initial concern for AKI was vancomycin-induced due to high trough levels, but review of his hospital course revealed:

- Decreased serum albumin compared to outpatient labs
- Rising systolic BP
- Microscopic then macroscopic hematuria

Work-up suggested an intrinsic cause of renal injury with serologic work-up negative. Biopsy was performed demonstrated staphylococcus-associated acute glomerulonephritis (SAAGN).

- Risk factors include male sex, older age, previous DM
- Sometimes compared to post-infectious GN associated with streptococcal infection.
- In one case series, 23% progressed to ESRD, and 14.5% died.
- Biopsy shows mesangial and endocapillary proliferation and IgA-dominant or co-dominant deposition on IF.

## Teaching Points

1. SAAGN occurs during active infection with MRSA or MSSA.
2. It presents as an acute or subacute GN, with proteinuria, hematuria and AKI.
3. Treatment is aimed at cure of infection.
4. SAAGN is a highly morbid complication of staphylococcal infection.

## References

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