

# Fibrillary Glomerulonephritis Presenting as Acute Kidney Injury

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## Introduction

Acute nephrotic syndrome encompasses a variety of discrete syndromes that often present very similarly. The defining feature is proteinuria, specifically of >3.5 grams per day, combined with low serum albumin, and the resultant edema, secondary to glomerular injury. The specific cause of nephrotic syndrome can be suggested by the patient's history and physical exam, including HIV status, medication usage, and the presence of systemic disease. Lab values including fractional excretion of sodium, in addition to BUN and creatinine, can suggest intrinsic renal pathology. However, renal biopsy is required for specific diagnosis.

Fibrillary glomerulonephritis is a rare cause of nephrotic syndrome, and as such not much is known about its pathophysiology. It classically presents as proteinuria, hypertension, and renal insufficiency, and renal biopsy will demonstrate evidence of immunoglobulin deposit as well as widespread microfibrils randomly arranged and deposited in the mesangium and glomerular membrane. Here we present a case of nephrotic syndrome as a cause of acute renal failure.

## Case Presentation

58 year-old female presented to the ED after receiving a call from her PCP regarding abnormal lab values:

- Two week history of progressive lower extremity edema which had spread to her abdomen and lower arms
- Also reported intense itching of the upper back, nausea, vomiting, and decreased appetite
- Denied fever, jaundice, hematuria, dysuria
- History of IV drug use (heroin/cocaine) most recently one year ago
- Past medical history of hypertension, myocardial infarction, and hepatitis C

## Pertinent Physical Exam Findings

- Significant edema of bilateral lower extremities and abdomen
- Exquisite tenderness to palpation over edematous areas
- Lungs clear to auscultation bilaterally
- Excoriations of upper back
- Abdomen non-tender
- No signs of inflammation near injection site

## Laboratory Evaluation

BUN	100 (H)	Urine Protein	>500 mg/dL
Creatinine	14 (H)	Urine Albumin	>15,00 mg/dL
GFR	3	Potassium	4.6
HCO3	13	Serum Light Chain	Positive
Venous pH	7.11 (H)	Lupus Anticoag	Negative
Corrected Ca	7.24 U/L	ANA Screen	Positive
Hep C Load	436,907	Blood Cultures	Negative

## Hospital Course

- Tunneled catheter placed four days after admission, and hemodialysis was begun
- Acute renal failure was diagnosed, and workup revealed positive ANA of 1:160 and positive serum light chain
- Left kidney biopsy was performed with a final diagnosis of fibrillary glomerulonephritis
- Creatinine trended down to 8.15, GFR increased to 6, ionized calcium increased to 1.08
- Edema decreased slightly, pain improved, and patient was discharged with plans for outpatient hemodialysis

## Discussion: Nephrotic Syndrome and Fibrillary GN

- Although the current literature states that the accepted treatment for glomerulonephritis is corticosteroids and cytotoxic agents, recent studies have indicated that these modalities are relatively ineffective in FGN, and over half of all patients will progress to end-stage renal disease within four years. As such, prompt, accurate diagnosis by kidney biopsy is essential.
- Recent studies have suggested an association between FGN and HCV infection, which was present in this patient untreated.
- One case reported improvement of renal function with administration of alpha-interferon. This would suggest that FGN is not a primary renal disease, but rather a secondary manifestation of an unknown systemic issue.

## Discussion: Our Patient

Our patient presented with symptoms of acute renal failure, as well as findings suggestive of chronic glomerular disease, including hypertension. Initial management included compression stockings, which the patient used with no success as her edema worsened. The patient's findings of positive serum light chains and positive ANA were initially suggestive of amyloidosis, which can be another cause of glomerular damage. However, as with all nephrotic syndrome, renal biopsy was necessary to confirm the diagnosis.

This is an interesting case that allows us to explore management of nephrotic syndrome, as well as possible association between hepatitis C virus and fibrillary glomerulonephritis. Further research is necessary to establish a definitive link.

## Conclusion

This case demonstrates nephrotic syndrome and glomerular damage as a cause of acute renal failure, in this case severe edema. While a rare cause of renal disease, fibrillary glomerulonephritis can be confirmed by renal biopsy, and its classification as a primary renal versus secondary systemic disease should be studied further.

## References

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