

Acute Hepatitis B Infection Presenting as Non-Lupus Full-House Nephropathy - A Case Report

Abstract

Non-lupus full-house nephropathy (FHN) is a rare and new entity, characterized by a histological kidney injury pattern resembling lupus nephritis without any extra-renal symptoms or serology suggestive of systemic lupus erythematosus (SLE). We report a male with nephrotic-range proteinuria. Kidney biopsy showed diffuse proliferative glomerulonephritis with a full-house pattern on immunofluorescence. The patient didn't have any clinical or laboratory criteria for SLE. He had an acute hepatitis B infection, and a diagnosis of acute hepatitis B-related non-lupus FHN was made. The patient was managed conservatively with antivirals and angiotensin receptor blockers. The patient recovered successfully without any lupus-related immunosuppressive therapy.

Keywords: Diffuse proliferative glomerulonephritis, Hepatitis B, Non-lupus full house nephropathy, Systemic lupus erythematosus

Introduction

Full-house immunofluorescence in a kidney biopsy is classically associated with lupus nephritis. A kidney biopsy showing full-house immunofluorescence in the absence of auto-antibodies and classical clinical features of systemic lupus erythematosus (SLE) is considered as non-lupus full-house nephropathy (FHN). Non-lupus FHN may be idiopathic or secondary to other disease processes.¹ We report a patient with acute hepatitis B infection-related non-lupus FHN, successfully managed with antivirals and angiotensin receptor blockers (ARBs).

Case Report

A 46-year-old male, with no known comorbidities, presented with swelling in the lower limbs that gradually progressed to anasarca over the next 2 months. Shortness of breath, orthopnea, or paroxysmal nocturnal dyspnea, or yellowish discoloration of the skin, eyes, or urine were absent. There was no history of fever, oral ulcers, rash, joint pains, or alopecia. On examination, he was afebrile; his pulse rate was 82/min, blood pressure was 118/70

mmHg in the right arm, and oxygen saturation was 98%. Bilateral pitting edema was present in the lower limbs. On systemic examination, the liver was enlarged with a span of 17 cm. The rest of the systemic examination was normal. Routine investigations revealed elevated liver enzymes and hypoalbuminemia [Table 1]. Chest X-ray was normal. Echocardiography of the heart was normal (Ejection fraction-65%). Urine routine microscopy showed protein 2+ and 64 RBCs/HPF. The 24-hour urine protein was 4417 mg. With edema, normal blood pressure, hypoalbuminemia, and nephrotic range proteinuria, a working diagnosis of nephrotic syndrome was made, and the patient was planned for kidney biopsy.

Simultaneously, in view of deranged liver enzymes, viral markers (HbsAg, HIV, and hepatitis C serology) were tested. HbsAg was positive. Further workup for hepatitis B revealed positive HbeAg, anti-Hbc IgM, and 130,500,000 IU/mL HBV DNA levels. USG whole abdomen revealed hepatomegaly (18 cm). The fibroscan didn't show any evidence of liver fibrosis. Acute Hepatitis B infection was diagnosed.

Table 1: Routine investigations

Lab parameters		At admission	At 4-week follow-up	Reference range
CBC	Hb (g/dL)	11.5	11.6	12-15
	TLC ($\times 10^3$ /cumm)	9.84	6.24	4-10
	Platelets (thous/cumm)	161	182	150-450
LFT	BT/BD (mg/dL)	0.26/0.16	0.30/0.16	0.2-1.2/0-0.5
	TP/ALB (g/dL)	5.79/2.82	7.2/3.6	6-8.3/3.5-4.6
	SGOT/SGPT (IU/L)	50/74	40/50	5-34/0-55
	ALP/GGT (IU/L)	205/107	160/80	40-150/9-36
KFT	Creatinine (mg/dL)	1.07	0.90	0.57-1.11
	Na/K (mEq/L)	139/4.6	138/4.5	136-146/3.5-5.1
	Ca/P (mEq/L)	8.41/4.4	8.52/3.8	8.4-10.2/2.3-4.7
PT/INR		13.3/1.24	12.2/1.1	10.7-12.4
APTT (seconds)		27.7	26.8	26.6-37
24-hour urine protein (mg/24 hours)		4417	121	<150

CBC: Complete blood count, LFT: Liver function test, KFT: Kidney function test, Hb: Hemoglobin, TLC: Total leucocyte count, BT/BD: Bilirubin total/bilirubin direct, TP/ALB: Total protein/albumin, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamate pyruvate transaminase, ALP: Alkaline phosphatase, GGT: Gamma glutamyl transferase, Na/K: Sodium/potassium, Ca/P: Calcium/phosphorus, PT: Prothrombin time, INR: International normalized ratio, APTT: Activated partial thromboplastin time.

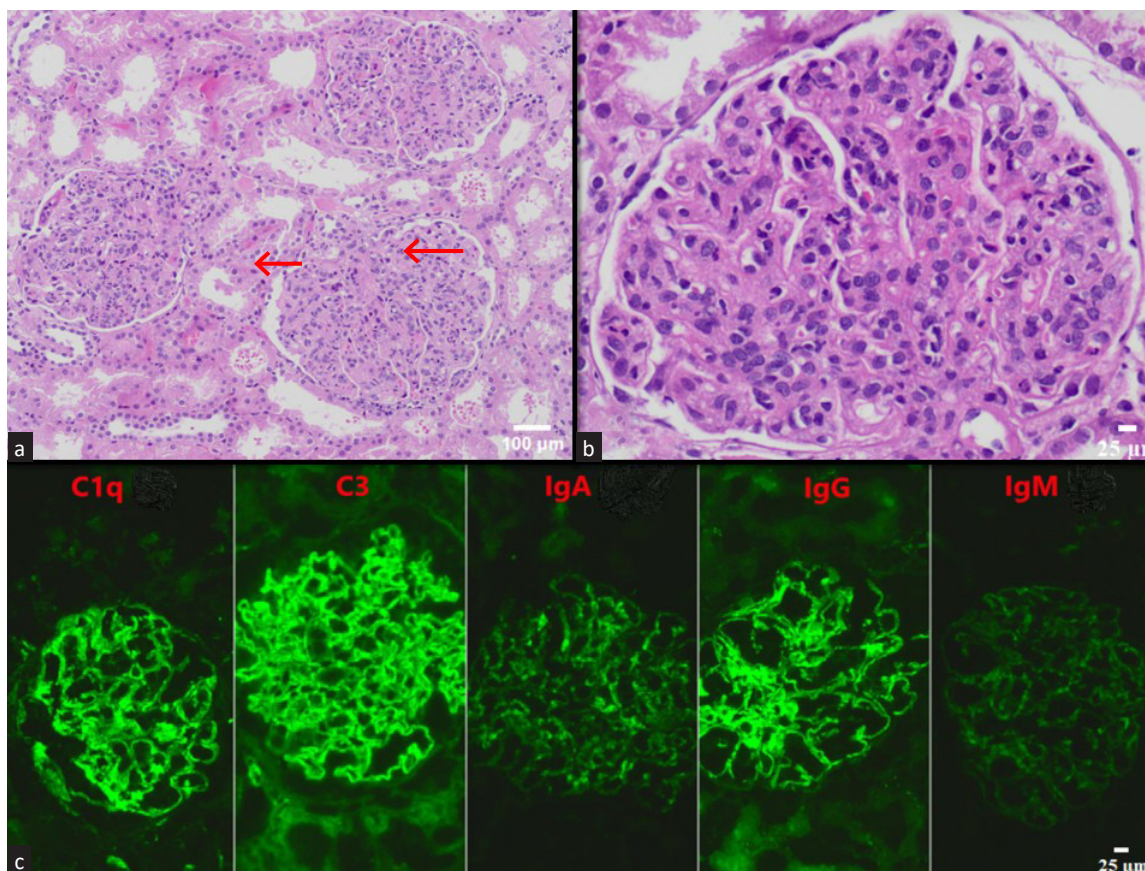


Figure 1: (a) Low power field microscopy (10x, Hematoxylin and eosin stain) showing segmental to global endocapillary hypercellularity along with neutrophilic inflammation in the glomerulus (red arrows). (b) High power field microscopy (40x, Hematoxylin and eosin stain) showing lobular accentuation and endocapillary hypercellularity (red arrow) in the glomerulus, suggestive of diffuse proliferative glomerulonephritis. (c) Immunofluorescence showing full house pattern with immunoglobulin and complement deposits (IgG, IgA, IgM, C3, C1q) along glomerular capillary wall and mesangial deposits.

Renal biopsy showed diffuse proliferative glomerulonephritis (DPGN) on light microscopy with “full-house” effect on immunofluorescence (IgG, IgA, IgM, C1q, C3) [Figure 1]. A detailed autoimmune workup was done. ANA by immunofluorescence, ANA profile, anti-ds DNA, c-anca, and p-anca were negative. Complements were low (C3- 734 mg/L, C4- 40 mg/L). The patient didn't have any clinical or laboratory features suggestive of lupus. Non-lupus full-house nephropathy with acute hepatitis B was diagnosed. The patient was started on a nucleoside reverse transcriptase inhibitor (NRTI), tenofovir alafenamide (25 mg) once daily, and ARB, telmisartan (20 mg) once daily, and discharged. A follow-up 24-hour urine protein was done at 4 and 8 weeks, which was 121 mg/day and 105 mg/day, respectively. The edema subsided.

Discussion

Full-house immunofluorescence in a kidney biopsy was previously synonymous with lupus nephritis. It was considered that FHN may be the first manifestation of lupus nephritis, and patients were classified as seronegative lupus nephritis when antibodies were negative for SLE.¹ However, most patients did not develop any clinical or serological features of lupus on follow-up.

Such patients with histological features resembling lupus nephritis without any extrarenal manifestations or serology suggestive of SLE are now classified as non-lupus FHN. Non-lupus FHN may be the first or only SLE manifestation, or a new SLE-unrelated pathology (idiopathic or secondary).² In a systematic review done by Uzzo *et al.*, 50% had idiopathic FHN, 44% had a secondary cause, with infections being the most common (41%), and 6% developed SLE over a median 5-year follow-up.³ In a study done by Wani *et al.* in India, the non-lupus FHN prevalence was 19.4%, and on the basis of light microscopy the most common cause of non-lupus FHN was membranous nephropathy (25.9%), followed by IgA nephropathy (22.2%), membranoproliferative glomerulonephritis (14.8%), DPGN (12.3%), crescentic glomerulonephritis (12.3%), amyloidosis (8.6%), and C1q nephropathy (3.7%).¹ Our patient had DPGN on light microscopy with FHN.

Membranous nephropathy is the most common renal manifestation of hepatitis B, followed by membranoproliferative glomerulonephritis.⁴ Our patient had DPGN with FHN, which, although classically seen with lupus nephritis, can very rarely be seen with hepatitis B infection. KDIGO recommends antivirals as the treatment of choice for hepatitis B-related nephropathy.⁵ The role

of steroids is controversial. It may be used with antiviral therapy for <6 months to control proteinuria.⁶ Our patient responded with antivirals and ARBs without the need for steroids.

It is important to recognize non-lupus FHN, which can prevent them from being classified as lupus nephritis and being subjected to immunosuppression protocols. However, it is equally important to follow up these patients because a minority classified as non-lupus FHN can seroconvert over the years to lupus nephritis.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.

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