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Collapsing Glomerulopathy Following Moderna COVID-19 Vaccine and Response to Treatment

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1. Background

Over 13 billion doses of the COVID-19 vaccine have been administered worldwide. Though these vaccines have been proven safe and effective by large clinical trials, there is an emerging evidence of rare adverse events. We report here case of de-novo nephrotic range proteinuria and acute kidney injury caused by collapsing focal segmental glomerulosclerosis in a previously healthy seventy-five-year-old female, 5 weeks following the second dose of Moderna COVID-19 vaccine.

2. Case

A 75-year-old Caucasian female presented to her primary care physician's office with gradual worsening of bilateral lower extremity edema, weight gain and new onset hypertension. Her pertinent medical co-morbidities included well controlled migraine headache, allergic rhinitis and essential tremor. Her medications were propranolol and vitamin D supplements.

She received the second dose of Moderna COVID-19 vaccine 5 weeks prior to symptom onset. Evaluation and treatment of above symptoms included a transthoracic echocardiogram which showed an estimated left ventricular ejection fraction of 55 to 60% and grade 2 diastolic dysfunction; bilateral lower extremity dopplers were negative for deep vein thrombosis.

Lisinopril and hydrochlorothiazide were initiated. Her baseline serum creatinine (SCr) was 0.9 mg/dL. Blood work revealed an increase in serum creatinine to 8 g/dL.

Urine analysis revealed microscopic haematuria, pyuria, proteinuria and normoglycemic glucosuria. 24-hour urine collection showed proteinuria of 5161 mg. Serum albumin was 2.6g/dL. Serological work-up was unremarkable as noted in Table 1. Renal ultrasound showed mild caliectasis of left renal pelvis with some increased cortical echogenicity bilaterally with cortical thinning.

She progressed to oliguric renal failure and required dialysis support. Renal biopsy showed collapsing focal segmental glomerulosclerosis (FSGS) with interstitial nephritis. Immunofluorescence was positive for IgM and C1q. Electron microscopy demonstrated diffuse podocyte effacement. She received pulse dose steroids followed by prednisone 1mg/kg and diuretics.

Over the next 12 weeks, renal function gradually improved and dialysis support was discontinued. 24-hour urine collection showed protein 1.785 g; measured creatinine clearance of 25ml/min/1.73m^2; serum creatinine improved to 1.9 mg/. Prednisone was tapered to 5 mg daily in the subsequent three months. At six months, proteinuria increased to 4570 mg/day Serum creatinine remained stable at 1.8 mg/dL (eGFR 27.1 mL/min/1.73 m^2). The patient declined an increase or addition of further immunosuppression. Over the next 6 months her proteinuria worsened, and serum creatinine increased to 2.9 mg/dL. She was started on Mycophenolate mofetil (MMF) (chosen over CNIs since her eGFR was less than 30ml/min/1.73m^2) and oral prednisone. Her renal function and proteinuria improved in 1 month. The patient achieved partial remission in 2 weeks and complete remission in 10 months. SCr stabilized at 1.4 mg/dL and eGFR ~ 40 ml/min/1.73m^2.

Table 1:

Test	Ref Range	Patient's value
ANTINUCLEAR ANTIBODIES (ANA), IGG BY ELISA W REFLEX TO ANA, HEP-2 SUBSTRATE, IGG BY IFA	Not detected	Not detected
Double-Stranded DNA (dsDNA) Ab IgG IFA	<1:10	<1:10
Anti-Neutrophil Cytoplasmic Ab, IgG	<1:20	<1:20
GBM Antibody, IgG by IFA	Negative	Negative
Myeloperox Antibodies, IgG	0 - 19 AU/mL	0
Serine Protease3, IgG	0 - 19 AU/mL	0
Rheumatoid Factor Qualitative	Negative	Negative
C3 Complement	67 - 154 mg/dL	113
C4 Complement	16 - 66 mg/dL	48
IgG	624-1,766 mg/dL	282
IgA	82 - 460 mg/dL	243
IgM	22 - 293 mg/dL	50
Hepatitis B Surface Antigen	Negative	Negative
Hepatitis C Antibody	Negative	Negative
HIV Combo AB/AG	Negative	Negative

3. Discussion

Focal segmental glomerulosclerosis (FSGS) is a frequent cause of end stage renal disease (ESRD) in United States with annual incidence rates ranging from 0.2 to 1.8/100,000 population per year [1]. Collapsing FSGS is characterized by collapse and sclerosis of the entire glomerular tuft, rather than segmental injury. It presents with more severe nephrotic syndrome and greater kidney function impairment than other forms of FSGS. Affected patients are frequently resistant to therapy and often have a rapid progression to ESRD. Collapsing FSGS has been reported to occur in association with several disorders including viral infections, drugs, autoimmune conditions. Most cases are idiopathic. The mainstay of treatment in any primary FSGS is immunosuppressive medications including glucocorticoids or glucocorticoid-sparing medications in addition to supportive measures.

Several reports of immune mediated glomerulopathies following vaccinations against COVID-19 have been reported. Other vaccines such as pneumococcus, hepatitis B and influenza have also been associated with such phenomena [2-4,6]. The pathogenesis of COVID-19 vaccine-related glomerular pathology remains poorly understood. Available evidence suggests that the T-cell mediated adaptive immune response following the vaccination results in production of pro-inflammatory cytokines including tumor necrosis factor, interleukin – 1B among others that cause autoimmune glomerular damage. This autoimmune response could be related to molecular mimicry between the SARS-CoV-2 spike protein and host protein in genetically susceptible individual. Both mRNA and inactivated COVID-19 vaccine have been implicated after either the first or the second dose. mRNA vaccine can produce a more potent humoral and cell mediated immune response compared to other types of vaccine and is more frequently reported to be associated with glomerulonephritis. This may also be from the more widespread use of mRNA vaccines in a short period of time. To date minimal change disease, IgA nephropathy and membranous nephropathy represent the bulk of glomerular diseases associated with COVID-19 vaccine [3]. There are cases of de novo and relapsing FSGS in native and transplanted kidneys following ChAdOx1 nCoV-19, Pfizer [7,8,9]. Sirpal et al reported a case of tip lesion variant FSGS after a second dose of Moderna COVID-19 vaccine that improved with prednisone [10]. Khan et al reported a case of collapsing focal segmental glomerulosclerosis after Moderna mRNA COVID – 19 vaccine in African-American male with Human Immunodeficiency Virus (HIV) [11].

4. Conclusion

Nephrotic syndrome after COVID-19 vaccine administration is described in literature. Our case appears to be the first reported case of collapsing FSGS following the Moderna COVID-19 vaccine in an otherwise healthy individual. Some cases of collapsing FSGS were described after Pfizer mRNA vaccine, which could be related to higher incidence in Pfizer vaccine administration as compared to other Covid-19 vaccines. COVID-19 vaccines have excellent safety profile and mass vaccinations has largely proven to be immensely beneficial in reducing the incidence and severity. Surveillance for vaccine related immediate, intermediate and long-term adverse effects are essential. This case stresses the importance of early recognition, evaluation and treatment in order to improve chances of meaningful renal recovery. Our patient showed significant recovery with immunosuppressive therapy.

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