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Minimal Change Disease With Severe Acute Kidney Injury Following the Oxford-AstraZeneca COVID-19 Vaccine: A Case Report

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We report a case of minimal change disease (MCD) with severe acute kidney injury (AKI) following the first injection of the ChAdOx1 nCoV-19 (AZD1222) vaccine from Oxford-AstraZeneca against coronavirus disease 2019 (COVID-19). A 71-year-old man with a history of dyslipidemia and a baseline serum creatinine of 0.7 mg/dL presented with nephrotic syndrome, AKI, and severe hypertension 13 days after receiving the Oxford-AstraZeneca vaccine. Refractory hyperkalemia and hypervolemia with oligoanuria prompted initiation of hemodialysis. His serum albumin was 2.6 g/dL and his urinary protein-creatinine ratio was 2,321 mg/mmol. Given a high suspicion for rapidly progressive glomerulonephritis, empirical glucocorticoid treatment was initiated (3 methylprednisolone pulses followed by high-dose prednisone). A kidney biopsy showed MCD and acute tubular injury. Kidney function and proteinuria subsequently improved, and hemodialysis was discontinued 38 days after the start of therapy. This case describes de novo MCD after the Oxford-AstraZeneca vaccine. It adds to the few published case reports of MCD after the Pfizer-BioNTech COVID-19 vaccine. Further reports and studies will be needed to elucidate whether MCD is truly associated with COVID-19 vaccination.

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Introduction

The coronavirus 2019 (COVID-19) pandemic has been associated with an increased mortality worldwide. New vaccines against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) were thus developed to limit contagion and mortality. Major side effects of these vaccines are uncommon. However, de novo minimal change disease (MCD) following the Pfizer-BioNTech COVID-19 vaccine has been reported.[1-3]

We report the case of a 71-year-old man with nephrotic syndrome and acute kidney injury (AKI) following the administration of the ChAdOx1 nCoV-19 (AZD1222) vaccine, which was developed by the University of Oxford, AstraZeneca, and the Serum Institute of India, and is based on a replication-incompetent chimpanzee adenovirus vector that expresses the SARS-CoV-2 spike protein. His kidney biopsy demonstrated MCD and acute tubular injury.

Case Report

A 71-year-old man presented to the emergency room with anasarca, AKI, and severe hypertension. He was only known to have dyslipidemia, which was treated with rosuvastatin, 20 mg daily. His usual blood pressure was 130/80 mm Hg. He had no prior history of SARS-CoV-2 infection or chronic kidney disease. His baseline serum creatinine was 0.7 mg/dL. He was not taking any other medications including nonsteroidal anti-inflammatory drugs. Thirteen days before presentation, he received the first injection of the Oxford-AstraZeneca COVID-19 vaccine. One day after vaccination, and 12 days before presentation, he developed new-onset hypertension (>190/>100 mm Hg) and progressive facial and upper extremity edema. He was seen by his family physician who prescribed amlodipine; he then presented to the emergency department when his edema worsened.

On admission, his blood pressure was 204/102 mm Hg. Physical examination revealed bilateral pitting edema in all extremities. His serum albumin and creatinine were 2.8 g/dL and 10.6 mg/dL, respectively. His serum urea nitrogen was 103.8 mg/dL. Serum sodium (138 mmol/L), calcium (9 mg/dL), and bicarbonate (25 mmol/L) were within reference ranges, but serum magnesium (2.9 mg/dL), potassium (5.5 mmol/L), and phosphorus (9.6 mg/dL) were elevated. Hemoglobin (13.4 g/L) and bilirubin (0.18 mg/dL) were within reference ranges, and haptoglobin was not decreased (3.05 g/L). The initial urinary protein-creatinine ratio (UPCR) was 2321 mg/mmol. On urine microscopy, 6 to 10 red blood cells per high-power field and granular casts were seen. Polymerase chain reaction testing for SARS-CoV-2 was negative. The clinical course and important laboratory studies are summarized in Figure 1.

To exclude infection and neoplasm, an abdominal ultrasound, thoracic computed tomography, and blood cultures were performed and were unremarkable. Negative results were found upon testing for antinuclear and anti–double-stranded DNA antibodies, antineutrophil cytoplasmic autoantibodies, as well as for antibodies to glomerular basement membrane and M-type phospholipase A_2 receptor (PLA_2R). Levels of complement factors C3 and C4 were normal. Tests for HIV and hepatitis B and C virus were negative. Serum free light chains levels and protein electrophoresis with immunofixation were normal.

One day after admission, the patient was oligoanuric and his hyperkalemia and hypervolemia became refractory to intravenous diuretics, prompting hemodialysis to be initiated. Given the presence of AKI, proteinuria,