LETTER TO THE EDITOR

An Additional Case of Minimal Change Disease Following the Pfizer-BioNTech COVID-19 Vaccine

To the Editor:

A recent case report by Lebedev et al[1] described a patient presenting with minimal change disease (MCD) within days of his first injection of the BNT162b2 vaccine (Pfizer-BioNTech mRNA-based vaccine against severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2]).[1] Here, we describe a similar case of a patient with new-onset MCD after this vaccine.

A man in his early 80s was admitted with a 2-week history of edema and increase of body weight of 12 kg. He had received the first injection of the Pfizer-BioNTech COVID-19 (coronavirus disease 2019) vaccine 7 days before onset of edema. His medical history mentioned venous thromboembolisms several years earlier. He used no medication. Blood pressure was 168/94 mm Hg, and physical examination showed generalized pitting edema including periorbital edema. Blood tests revealed serum creatinine, 1.43 mg/dL; albumin, 1.03 g/dL; and total cholesterol, 522 mg/dL. Proteinuria was 15.3 g/d. Additional studies revealed no signs of an underlying systemic disease or malignancy. Light microscopy of a kidney biopsy specimen showed no apparent abnormalities in all 23 glomeruli examined. Tubular epithelial cells showed prominent nuclei and vacuolization. Immunofluorescence studies were negative. Electron microscopy showed diffuse podocyte foot-process effacement. All findings were consistent with MCD. Treatment with prednisolone 80 mg daily was initiated. After 10 days of prednisolone treatment, urinary protein-creatinine ratio declined to 0.68 g/g and edema had disappeared.

Our case provides support for a potential association between the BNT162b2 vaccine and onset of MCD. Pharmacovigilance of COVID-19 vaccines will be important to determine the incidence of this potential adverse event.

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Support: None.

Financial Disclosure: The authors declare that they have no relevant financial interests.

Peer Review: Received May 7, 2021. Accepted May 7, 2021 after editorial review by an Associate Editor and a Deputy Editor.

 Published online May 13, 2021 with doi 10.1053/j.ajkd.2021.05.003

Reference

1. Lebedev L, Sapojnikov M, Wechsler A, et al. Minimal change disease following the Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis*. 2021;78(1):142-145.

RESEARCH LETTERS

Effect of Lanthanum Carbonate on Blood Pressure in CKD

To the Editor:

Serum phosphate concentrations rise as chronic kidney disease (CKD) progresses and higher concentrations are associated with vascular calcification, cardiovascular events, and all-cause mortality.[1,2] The relationship between serum phosphate and blood pressure (BP) is less established, but emerging data suggest that higher levels may induce microvascular dysfunction and increase BP.[3,4] Here we looked at the association between phosphate-lowering medications and BP in advanced CKD.

We evaluated data from COMBINE, a randomized, double-blind, placebo-controlled trial designed to test 2 medications intended to reduce dietary phosphate absorption, lanthanum carbonate (LC; a phosphate binder) and nicotinamide (an intestinal phosphate transport inhibitor), in 205 participants with CKD stage 3b-4 over 12-month follow-up.[5] The trial was approved by institutional review boards at each center and all participants provided informed consent. Participants randomized to active LC arms exhibited reduced 24-hour urine phosphate excretion (UPE), but serum phosphate remained unchanged.[5] As UPE is an indicator of dietary phosphate absorption,[6] we hypothesized that randomization to LC would lower BP compared to the non-LC arms.

As nicotinamide did not affect serum phosphate or UPE in COMBINE, our post hoc analysis evaluated participants by 2 groups, LC vs non-LC (instead of the original 4). Participant data were included if they presented for at least 2 follow-up visits (which occurred monthly for 3 months, and then at 6, 9, and 12 months postrandomization). Using an electronic BP device, BP was measured 3 times from the right arm while seated in a quiet room following 5 minutes' rest during baseline and follow-up visits.

We hypothesized that effects would manifest by month 3 since theoretically the phosphate-BP relationship is mediated acutely, so we modeled the "acute" (baseline to month 3) and "chronic" (months 3–12) stages separately using a 2-slope linear spline model. The 2 slopes were weighted by their proportional time spans to produce a composite measure for 12-month rates of change. Our primary model adjusted for age, sex, clinical center, and baseline estimated glomerular filtration rate (eGFR), BP, and number of antihypertensives. Two-tailed $P < 0.05$ was considered statistically significant.

Of 205 individuals randomized (Fig S1), mean age was 69 ± 12 (SD) years, 38% were women, and 34% were