

LETTER TO THE EDITOR

First case of atypical haemolytic uraemic syndrome following COVID-19 vaccination with BNT162b2

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A broad range of glomerular diseases is associated with different severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) vaccinations [1]. In this context, atypical haemolytic uraemic syndrome (aHUS) due to glomerular thrombotic microangiopathy (TMA) has not been reported in the literature so far following SARS-CoV-2 vaccination with Comirnaty (BioNTech, Mainz, Germany).

We herein describe a 60-year-old woman who was admitted to a rural hospital because of general discomfort. She was then transferred to our institution for suspicion of aHUS. On admission, she presented with a serum creatinine of 2.1 mg/dL, a platelet count of 42 g/L, a haemoglobin of 7.1 g/dL and lactate dehydrogenase of 1700 U/L and schistocytes were detectable on a peripheral blood smear (21 ppt). Haptoglobin was not detectable and the activity of a disintegrin and metalloprotease with thrombospondin type 1 motif 13 was 53%, ruling out the presence of thrombotic thrombocytopenic purpura. Her medical history and laboratory workup were unremarkable with regard to potential causes of secondary TMAs such as active cancer, hypertensive emergency, drugs, infections or autoimmune diseases [2, 3]. Of note, after suffering from mild coronavirus disease 2019 (COVID-19) some 6 months ago, she received her first SARS-CoV-2 vaccination (Comirnaty) about 2 weeks before the onset of symptoms of her current illness. She did not carry pathogenic variants in CFH, CFI or CD46. However, she showed the CFH-H3 haplotype (heterozygous), which results in slightly reduced plasma levels of factor H and is strongly associated with a risk for aHUS. A kidney biopsy showed a glomerular TMA

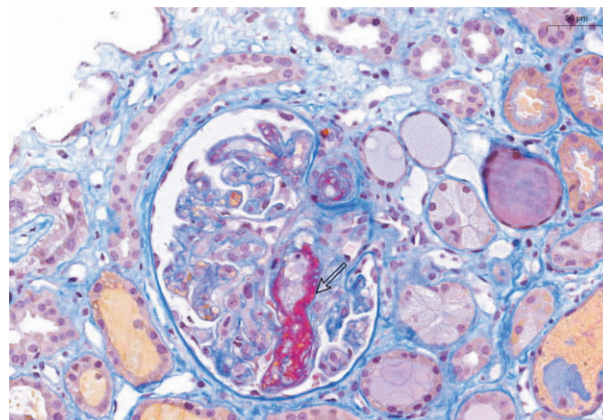


FIGURE 1: Kidney biopsy showing glomerular thrombotic microangiopathy (arrow). Staining: acid fuchsin orange G, bar = 50 µm, original magnification × 80.

(Figure 1). She responded well to plasma exchange and currently has preserved renal function.

In summary, we propose a clinical diagnosis of aHUS, triggered by the messenger RNA (mRNA) SARS-CoV-2 vaccination. Causality, in this case, is based on a temporal association and we cannot demonstrate a direct link with vaccination. However, our observation finds support with reports describing renal TMA lesions in kidney biopsies in two cases with minimal

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change disease not presenting with aHUS following vaccination with Comirnaty [4, 5]. Furthermore, vaccination with the vector-based ChAdOx1 nCoV-19 vaccine led to aHUS in another case described by Ferrer et al. [6]. Finally, thrombotic thrombocytopenic purpura may also be associated with SARS-CoV-2 vaccination [7]. Thus mRNA- and vector-based COVID-19 vaccines seem to represent important trigger factors for the rare development of different TMA entities. In our case, the competent authority made an exception to full immunization with a second and subsequent booster vaccinations given the potential risk for recurrence of aHUS.

PATIENT CONSENT

The patient gave informed consent to publish this case.

CONFLICT OF INTEREST STATEMENT

None declared.

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