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ACUTE KIDNEY INJURY FOLLOWING VACCINE-INDUCED THROMBOTIC MICROANGIOPATHY

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ABSTRACT

Background: Within 10 months of the declaration of the coronavirus disease 2019 (COVID-19) pandemic, multiple vaccines, including messenger ribonucleic acid, adenoviral vector, and inactivated vaccines, were developed. Adverse events associated with vaccination are closely monitored. Thrombotic microangiopathy after administration of the Pfizer/BioNTech BNT162b2 vaccine is a rare but serious adverse event.

Case report: We report a 28-year-old healthy female who developed fever, arthralgias, myalgias, swelling of the feet, and bilateral loin pain 3 days after receiving the Pfizer/BioNTech BNT162b2 vaccine. She was diagnosed with acute kidney injury with raised creatinine and proteinuria. Kidney biopsy revealed a hilar thrombus, extensive glomerular basement membrane remodelling, and endothelial lesions attributed to vascular injury caused by the BNT162b2 vaccine. She responded well to conservative management and recovered fully from her renal failure.

Conclusion: Our patient's favourable outcome of renal-limited thrombotic microangiopathy with little lasting effects is unique compared to other cases leading to end-stage renal disease or death. A high index of suspicion is needed for early diagnosis. The study of vaccine adverse effects is paramount to improve safety and confidence surrounding COVID-19 vaccination.

KEYWORDS

COVID-19, mRNA vaccine, thrombotic microangiopathy, acute kidney injury, microvascular thrombosis

LEARNING POINTS

- Renal-limited thrombotic microangiopathy is a rare cause of acute kidney injury following vaccination.
- Renal recovery in renal-limited thrombotic microangiopathy is variable; conservative management with steroids is an initial option.
- Early comprehensive evaluation to exclude secondary causes of thrombotic microangiopathy, atypical haemolytic uremic syndrome, and autoimmune diseases is recommended.

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INTRODUCTION

Coronavirus disease 2019 (COVID-19), first detected in Wuhan, China, in December 2019, rapidly became a global pandemic, resulting in over 700 million infections and 7 million deaths[1]. Within 10 months, a massive international collaboration led to the development and approval of multiple vaccines, including messenger ribonucleic acid (mRNA) (Pfizer-BioNTech BNT162b2 and Moderna mRNA-1273), an adenoviral vector (Oxford/AstraZeneca ChAdOx1), and inactivated virus vaccines (Sinopharm BBIBP-CorV)[1]. The safety platform for emergency vaccines (SPEAC) monitors adverse events of special interest (AESIs), such as myocarditis and seizures[1]. Haematologic and renal events, including vaccine-induced thrombosis with thrombocytopenia (VITT), thrombotic thrombocytopenic purpura (TTP), and, rarely, thrombotic microangiopathy (TMA), have also been reported. Although uncommon, the severity of this case, the need for a high index of suspicion to establish the diagnosis, and subsequent recovery emphasize the importance of documenting it.

CASE DESCRIPTION

A 28-year-old previously healthy female received two doses of the BBIBP-CorV vaccine followed by a booster dose of BNT162b2. Three days after the booster, she developed fever, arthralgia, swelling of lower limbs, and severe bilateral

Test	Day 1	Day 3 - hospital admission	Day 7	Day 10	Day 30 - follow-up	Reference range
Haematology						
Haemoglobin (g/l)	121	–	–	–	–	115–165
Platelets (×10^9/l)	298	–	–	–	–	140–400
WBC count (×10^9/l)	12.3	–	–	–	–	3.6–11.0
Coagulation						
PT (s)	–	12.7	12.3		–	10–14
APTT (s)	–	28.9	26.1		–	24–37
D-dimer (ng/ml)	0.85	–	–	–	–	<500
Biochemistry						
Creatinine (µmol/l)	202	285	220	224	84	45–80
eGFR (ml/min/1.72m^2)	28	19	26	–	83	>60
Urea (mmol/l)	6.1	7.4	8.0	–	–	2.8–8.1
Sodium (mmol/l)	135	135	–	–	–	133–146
Potassium (mmol/l)	4.1	3.97	–	–	–	3.5–5.3
Albumin (g/l)	–	36	–	–	–	35–50
LDH (U/l)	–	–	199	–	–	135–225
ESR (mm/hr)	–	–	44	–	–	0–12
CRP (mg/l)	28.9	–	–	–	–	<5
CK (IU/l)	–	85	81	–	–	26–192
Urine studies						
Spot protein (g/l)	1.5	–	0.5	–	–	<0.3
24-h urine protein (mg)	–	–	694		–	<140
Protein (g/l)	–	–	–	–	0.05	<0.15

Abbreviations: WBC, white blood cell; PT, prothrombin time; aPTT, activated partial thromboplastin time; eGFR, estimated glomerular filtration rate; LDH, lactate dehydrogenase; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; CK, creatine kinase.

Table 1. Laboratory investigations.

loin pain. On hospital presentation, she was afebrile and clinically stable. Laboratory blood tests *(Table 1)* revealed acute kidney injury. Urinalysis and 24-hour urine collection revealed sub nephrotic-range proteinuria; no erythrocytes, hyaline, or white cell casts were observed. Renal Doppler showed normal perfusion bilaterally. Haematological studies and coagulation studies were within normal range. A raised D-dimer level was noted. No evidence of haemolysis was observed on the peripheral smear, and ADAMTS13 activity was in an acceptable range. Autoimmune screening for lupus, vasculitis, rheumatoid factor, and antiphospholipid antibodies was negative, and complement levels were within normal range. The COVID-19 polymerase chain reaction test was negative.

In the absence of standard guidelines, following a multidisciplinary meeting, intravenous methylprednisolone 750 mg was administered daily for 3 days, alongside intravenous furosemide 40 mg twice daily and oral amlodipine 5 mg once daily to control hypertension. Creatinine plateaued at 220 µmol/l (285) during hospitalization.

A kidney biopsy was performed. Light microscopy revealed hilar thrombi in two glomeruli, a recent organizing mural thrombus in one interlobular artery, and mild focal acute tubular injury *(Fig. 1)*. Immunofluorescence demonstrated no significant immune complex deposits. Electron microscopy revealed extensive segmental thickening, accompanied by wrinkling and splitting of the glomerular basement membrane, and swollen endothelial cells indicative of acute endothelial injury *(Fig. 2)*.

These clinical features support a diagnosis of renal-limited TMA precipitated by the BNT162b2 vaccine, confirmed on kidney biopsy. The patient made a full renal recovery at 1-month follow-up *(Fig. 3)*.

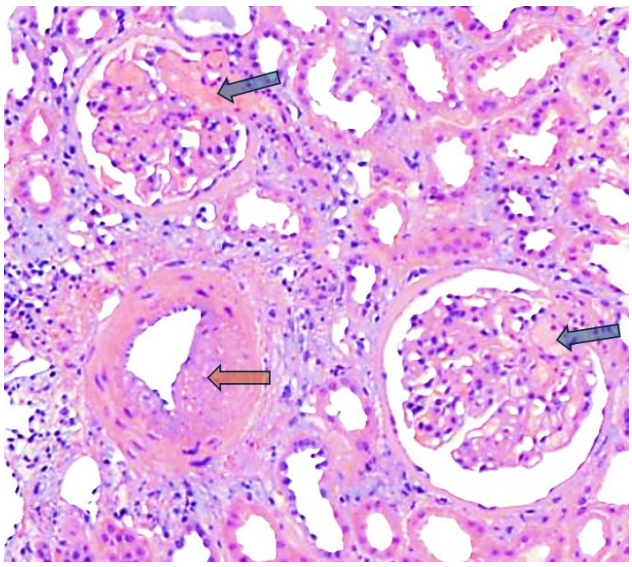


Figure 1. *Two glomeruli display hilar thrombi (blue arrows) with segmental shrinkage of the capillary loops. The interlobular artery shows an organising mural thrombus (red arrow). There is acute tubular injury. Haematoxylin and eosin, magnification 20×.*

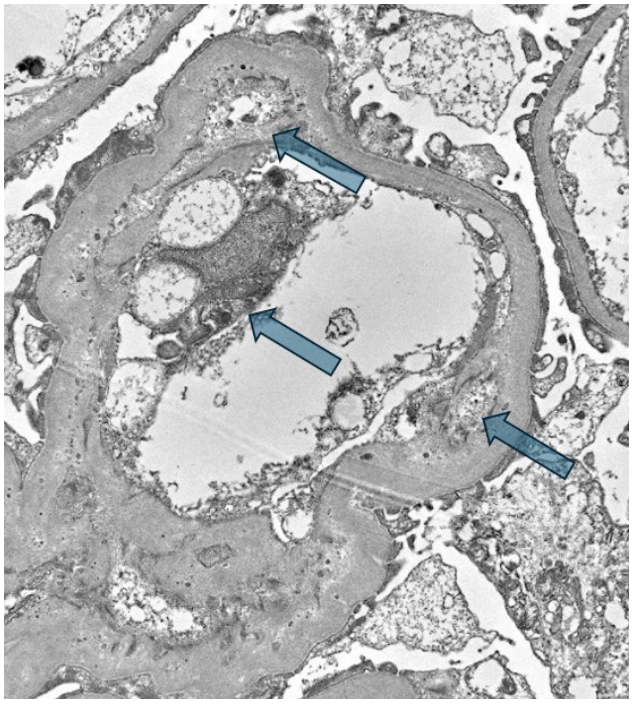


Figure 2. *Endothelial cells are markedly swollen with loss of fenestrations and intrusion into the underlying, thickened glomerular basement membrane (arrows). The podocyte foot processes are segmentally effaced. Electron microscopy, magnification 4500×.*

DISCUSSION

TMA encompasses a group of rare, life-threatening conditions characterized by microangiopathic haemolytic anaemia (MAHA), thrombocytopenia, and subsequent arteriolar and capillary thrombosis, causing end-organ damage. In MAHA, haemolysis occurs alongside platelet activation and consumption[2]. Common sites of end-organ thrombosis include the kidneys, liver, and brain. The aetiology of TMA is varied. TTP, Shiga toxin-mediated haemolytic uraemic syndrome (STHUS), and autoimmune disorders, including lupus and antiphospholipid syndrome, are implicated in precipitating TMA. However, TTP and STHUS are more commonly associated with this condition[2]. TMA has recently been linked to COVID-19 vaccination, with a particular preponderance for adenoviral vector vaccines compared to mRNA vaccines[3].

The pathophysiology underlying TMA after BNT162b2 vaccination is unclear. A hypothesis proposes that spike protein-encoding mRNA exhibits molecular mimicry with human proteins. Following vaccination, the spike protein elicits an immune response. This, alongside the circulation of unbound antigens and peptide fragments, can trigger endothelial inflammation and injury, leading to the formation of microthrombi[4].

In predisposed individuals, vaccines can induce a dysregulated complement activation, which can precipitate endothelial injury and subsequent thrombotic microangiopathy. At sub lytic levels, the C5b-9 membrane attack complex, formed as the terminal product of the classical, alternative, and lectin complement pathways, can stimulate endothelial prothrombinase and tissue factor activity, resulting in a pro-thrombotic endothelial cell surface. Additionally, C5b-

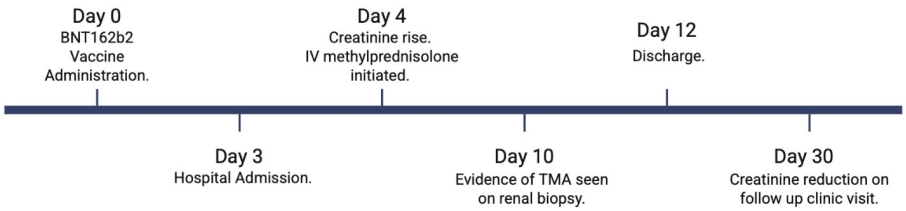


Figure 3. A timeline indicating key events in the case trajectory. Abbreviations: IV, intravenous; TMA, thrombotic microangiopathy.

9 promotes expression of leucocyte adhesion molecules, which drives further platelet localisation. In parallel, complement activation releases C3a and C5a, small cleavage fragments that are potent anaphylatoxins[5]. The presence of these molecules increases procoagulant tissue factor expression, exposes the basement membrane, increases vascular permeability, and fuels platelet and leucocyte adhesion, creating a highly procoagulant state that favours microvascular thrombosis[5].

A fatal case of TMA occurring two days after BNT162b2 vaccination was reported in a 72-year-old woman with controlled lymphoma and hyperthyroidism. Platelet count, coagulation studies, and ADAMTS13 activity were unavailable. Autopsy revealed platelet-rich glomerular and arteriolar thrombi, attributed to endothelial injury and platelet activation, without the characteristic fibrin-rich thrombi of disseminated intravascular coagulation, a finding similar to that in our patient. Conversely, thrombi were widespread—affecting the myocardium, liver, pancreas, and adrenals—causing sudden cardiac death at home before treatment could be initiated[6]. Comparing our case to this, we can observe heterogeneity in post-BNT162b2 vaccine TMA in terms of presentation and outcome.

Thrombosis after the COVID-19 vaccine has also been attributed to VITT, an autoimmune condition characterized by the production of anti-platelet factor 4 (PF4) antibodies[7]. Similar to heparin-induced thrombocytopenia, it causes a consumptive coagulopathy, leading to thrombosis of larger vessels, such as the cerebral venous sinuses and splanchnic veins, in contrast to the microvascular thrombosis that occurs with TMA. A case of VITT was reported in a healthy 19-year-old male who was found unresponsive 7 days after receiving the ChAdOx1 vaccine. Imaging showed sagittal sinus thrombosis and bifrontal haematoma. He had thrombocytopenia, normal coagulation studies, and a positive ELISA for anti-PF4 antibodies, suggestive of VITT[8]. In contrast in our case, markers of haemolysis seen in TMA were absent; there is no definitive link between VITT and haemolytic anaemia. Notably, there is an almost exclusive association between VITT and adenoviral vector vaccines, rather than mRNA vaccines, which makes our case less suggestive of VITT.

TTP has been reported after influenza, pneumococcal, and, more recently, COVID-19 vaccines, with a slight preponderance for mRNA or vector vaccines. It is defined by less than 10% activity of the ADAMTS13 protein. Molecular mimicry, triggered by an immunological stimulus such as a vaccine, can precipitate the formation of autoantibodies against ADAMTS13, leading to MAHA, thrombocytopenia, and microvascular thrombi[8]. TTP often causes cerebral microthrombi, altering neurological status, compared to TMA's prevalence for renal microthrombi, worsening acute kidney injury. This, alongside regular ADAMTS13 activity, makes our case less suggestive of TTP[2].

Notably, our patient employed a mixed vaccination approach, receiving 2 doses of BBIBP-CorV and a booster dose of BNT162b2. A study from Iraq showed no significant increase in side effects with the mixed BBIBP-CorV and BNT162b2 vaccination approach compared to the matched vaccination approach. Larger studies investigating these two groups are recommended to obtain an accurate assessment[9].

Treatment for thrombotic microangiopathy depends on its underlying cause. In cases of suspected immune TTP, immediate intervention with plasma exchange and corticosteroids is necessary to target ADAMTS13 autoantibodies. For suspected VITT, prompt administration of intravenous immunoglobulin (IVIG) with or without plasma exchange has been proposed to attenuate complement activity by amplifying C3 convertase and neutralizing activated complement products. C5 inhibitors may also be considered in situations involving extensive or refractory life-threatening complement-mediated injury[10].

Supportive care, which encompasses renal replacement therapy and fluid management, plays an essential role in patient management. Clinical outcomes can exhibit significant variability, ranging from complete recovery to dialysis dependence and, in some instances, mortality. Our patient exhibited renal-limited microthrombosis alongside normal ADAMTS13 activity, ultimately resulting in a full recovery.

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