

AZD-1222

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Thrombotic thrombocytopenia and diffuse arterial thrombosis: case report

A 69-year-old woman developed vaccine-induced thrombotic thrombocytopenia (VITT) and diffuse arterial thrombosis following the administration of AZD-1222 vaccine against COVID-19.

The woman was hospitalised to the stroke unit due to left hemiparesis and stroke occurred 9 days after the first dose of AZD-1222 [ChAdOx1 nCov-19; AstraZeneca; *dosage and route not stated*] vaccine injection against COVID-19. During this period, she complained of persistent headache initiated 2 days after the injection. She underwent hysterectomy twenty years prior to the admission, and she was treated with valsartan for arterial hypertension. Upon admission, a CT angiography revealed massive ipsilateral hemispheric oedema and the almost complete occlusion of the right internal carotid artery (ICA) and middle cerebral artery (MCA). A thromboaspiration under sedation was suspended after 2 hours because the removed thrombi were continuously replaced by newly formed ones, and she was then transferred to the ICU. On transfer to the ICU, she was drowsy but arousable with verbal stimulation. In addition, thrombocytopenia associated with reduced fibrinogen and elevated D-dimer levels was noted. Thus, VITT was suspected. The second total body CT angiography showed multiple thrombi in the descending aorta, celiac tripod, inferior mesenteric artery, and minor branches of the left pulmonary artery in association with small ischaemic areas in the left kidney, spleen, and liver, all in the absence of atherosclerotic plaques and venous thrombi. A diagnosis of VITT and diffuse arterial thrombosis was made secondary to vaccination.

The woman was treated with dexamethasone, immune globulin, argatroban and mannitol. On the next day, she appeared drowsy and a brain CT showed a right hemispheric infarction causing an initial brainstem compression. Argatroban was stopped. Subsequently, a decompressive craniotomy was performed. After the procedure, she recovered her consciousness but the left hemiparesis persisted. Argatroban was re-started. The immunoenzymatic assay was performed in the hospital in Italy (2 days after admission) showed high levels of anti-PF4/heparin IgG. In the following days, the clinical course characterised by neurological stability and progressive increase in platelet count. Repeat CT angiograms at 24 and 96 hours did not detect any venous thrombosis. Seven days later, argatroban was stopped. Her treatment changed to dabigatran etexilate. Fourteen days after the ICU admission, she was transferred awake with a left hemiparesis to a neurorehabilitation unit. A CT scan angiography obtained 16 days after the ICU admission showed the persistent occlusion of the right ICA whereas the right MCA territory was supported by leptomeningeal collateral activation; no venous thrombi were identified in the cerebral venous sinuses or elsewhere.