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Case Report

Acute disseminated encephalomyelitis after SARS-CoV-2 vaccination

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ABSTRACT

Several central and peripheral nervous system complications associated with the severe acute respiratory syndrome coronavirus (SARS-CoV-2) infection have been recently described. An effective mass vaccination program is necessary to effectively reduce infection spread and, consequently, limit long-term sequelae, including those affecting the nervous system. Nevertheless, as more patients gain access to coronavirus disease 2019 (COVID-19) vaccines, it is important to report potential adverse events. Herein, we report a patient with previous history of post-infectious rhombencephalitis who developed an acute disseminated encephalomyelitis (ADEM) two weeks after being vaccinated for COVID-19.

1. Introduction

Acute disseminated encephalomyelitis (ADEM) is a demyelinating disorder of the central nervous system (CNS) that typically occurs in close temporal association with either an antecedent infection or, less frequently, following vaccination [1].

Herein, we report a patient with previous history of post-infectious rhombencephalitis who developed an ADEM-like, severe neuroinflammatory disorder of the CNS shortly after being vaccinated for SARS-CoV-2.

2. Case report

A 56-year-old female patient was referred for subacute onset over one week of unsteadiness of gait, predominantly on the left side, followed by clumsiness of left arm. The day before the onset of the symptoms, she experienced malaise and chills, without fever. Nasopharyngeal swab was negative for SARS-CoV-2 on reverse-transcriptase polymerase chain reaction (RT-PCR) assay. Two weeks prior to presentation, she received the first dose of the Pfizer-BioMTech COVID-19 vaccine (Comirnaty), without any acute allergic reactions.

Her medical history was relevant for a previous clinical episode

suggestive for post-infectious rhombencephalitis 5 years before this admission (Fig. 1). At that time, the patient manifested diplopia, mild ataxia with left-ward deviation of gait, and urinary retention requiring catheterization one week after a febrile episode characterized by gastroenteritis and erythematous rash. Nuchal rigidity was not present. Brain magnetic resonance imaging (MRI), performed 6 days after onset, was unremarkable. Cerebrospinal fluid (CSF) showed pleocytosis (80 cells/mm³), while protein and glucose levels were within reference ranges. Electroencephalogram (EEG) was abnormal due to the presence of intermittent slowing in the delta range, predominantly over the fronto-temporal regions. A comprehensive microbiological and autoimmune screening was unrevealing. The patient spontaneously recovered and underwent regular follow-up, including a control brain MRI four months later, also unremarkable. Between 2016 and 2020 she was seen for recurrent episodes of cutaneous herpes zoster with resulting neuropathic pain, effectively treated with pregabalin. Neurological examination was normal during all these consultations, and the patient never complained of episodes of weakness, incoordination, sensory abnormalities, or visual loss.

In the current admission, the patient was found to be alert and oriented. No cranial nerve deficits were documented, but horizontal gazeevoked nystagmus was present on lateral gaze. Mild weakness on left



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