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Case report

Guillain-Barré syndrome after AstraZeneca COVID-19-vaccination: A causal or casual association?

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ABSTRACT

We report a case of Guillain-Barré syndrome (GBS) following the first dose of Oxford/AstraZeneca COVID-19 vaccine with papilledema as atypical onset. As the COVID-19 vaccination campaign progresses worldwide, GBSs vaccine-related have been increasingly reported. After reviewing the available literature, considering the annual incidence of GBS, in this historical moment, the public health systems cannot afford an unjustified distrust in vaccines, caused by misinterpretation of epidemiological data. Nonetheless, it is important for clinicians to promptly recognize neurological complications potentially associated with COVID-19 vaccinations and report them to pharmacovigilance agencies.

1. Introduction

Guillain-Barré syndrome (GBS) is an acquired inflammatory polyradiculoneuropathy that frequently occurs after recent infections [1]. GBS has been described during or after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. Moreover, GBS is also associated with any vaccination [1]. As the COVID-19 vaccination campaign progresses worldwide, GBSs vaccine-related have been increasingly reported. GBS with time-closely association to COVID-19 vaccinations has been described for both messenger-RNA vaccine [2] and adenovirus-vectored COVID-19 vaccine [3–5]. Herein, we report a case of GBS following the first dose of Oxford/AstraZeneca COVID-19 vaccine with visual impairment as atypical onset.

2. Case report

A 62-year-old Caucasian man with a previous medical history relevant for blood pressure hypertension referred to Emergency Department because of the onset of visual discomfort lasting from two days. His neurological examination was normal except for absent deep tendon reflexes and severe bilateral optic disc edema on fundus examination. Ten days before symptoms onset he received the first dose of the chimpanzee adenovirus-vectored COVID-19 vaccine, ChAdOx1. No previous flu-like or gastrointestinal episodes were reported. Nasopharyngeal SARS-CoV2 swab tested negative. Brain computed tomography scan with multiphasic and venous angiography and magnetic resonance imaging (MRI) with venous angiography were normal. His hospitalization was complicated by lower back pain and then progressively worsening sensory ataxia. Three days after the admission, his neurological examination revealed a new ascending tetraparesis with proximal predominant involvement (MRC sum-score 34/80), bilateral facial weakness, dysphagia, urinary retention, and distal paresthesia. Spinal cord MRI comprehensive of STIR and post contrast T1-weighted images was normal. Cerebrospinal fluid (CSF) examination showed albumin-cytologic dissociation (total protein count 101 mg/dl, five white blood cells) with high opening pressure (29 cms H_2O) and normal glucose at day-six from symptoms onset. Real-time PCR for herpes simplex virus, varicella-zoster, cytomegalovirus, Epstein-Barr virus, enterovirus and adenovirus and CSF microscopy and culture were negative. Serology for *Campylobacter jejuni*, *Mycoplasma pneumomiae*, Lyme syphilis and human immunodeficiency virus were normal. Four days after admission electrophysiologic study showed severe sensorimotor mixed polyneuropathy (demyelination with predominant axonal changes) (see Table 1). Needle electromyography did not show pattern of acute denervation. The antiganglioside antibodies test (line blot assay, Generic Assay GmbH - Dahlewitz, Germany) was positive for IgG GM1 (titer 39). A GBS diagnosis was performed and modified Erasmus GBS outcome score– (mEGOS) was 8. Intravenous immunoglobulins (2

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