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Review Article

AstraZeneca COVID-19 vaccine and Guillain- Barré Syndrome in Tasmania: A causal link?

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ABSTRACT

The emergence of the coronavirus 2019 (COVID-19) pandemic has presented an unprecedented global challenge. Vaccines against COVID have been developed to date. Covid-19 has been linked with the development of Guillain-Barre Syndrome (GBS), a rare immune-mediated demyelinating neuropathy. We report three cases of Guillain-Barre Syndrome and one case of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), presenting to a Tasmanian hospital, and review 15 other reported cases and discuss likely immunopathology. Nearly all reported cases of post-COVID-19 vacciation inflammatory demyelinating polyneuropathy are linked to AstraZeneca vaccination and a variant with bifacial weakness is the most reported form of GBS globally.

1. Introduction

Guillain-Barré syndrome is an immune-mediated demyelinating polyneuropathy characterised by progressive symmetrical weakness of limbs and decreased or absent deep tendon reflexes (Yuki and Hartung, 2012; van Doorn, 2013). Most cases are self-resolving, but some cases experience life-threatening respiratory muscle paralysis requiring mechanical ventilation (Verboon et al., 2017). It typically occurs in the post-infectious phase following bacterial or viral illness with most cases preceded by respiratory or gastrointestinal symptoms (Willison et al., 2016; van Doorn et al., 2008). *Clostridium jejuni* infection is associated with about one-third of the cases (McCarthy and Giesecke, 2001), but other identified infectious causes include viral infections such as cytomegalovirus, hepatitis E, Epstein Barr virus and influenzae A virus, and bacterial infections such as *Mycoplasma pneumoniae* and *Haemophilus influenzae* (Yuki and Hartung, 2012). Although not completely understood, current studies suggest that pathogenesis is due to autoimmune destruction of the myelin sheath and/or axonal damage caused by autoantibodies leading to functional blockade of nerve conduction (Yuki and Hartung, 2012; Willison et al., 2016; van Doorn et al., 2008; Walling and Dickson, 2013).

Although rare, several studies have explored a possible association of GBS to vaccination, notably the increased risk of GBS with H1N1 influenza (Swine Flu) vaccination in 1976 (Schonberger et al., 1979). The vaccination campaign against 'Swine Flu' was halted after a spike in cases of GBS was noted, with an incidence as high as 1 per 100,000 vaccinations (Lunn et al., 2021). Other studies explored the occurrence of GBS during influenza vaccination campaigns in 1990–2005 but suggested a relatively low risk associated with influenza vaccination (Lehmann et al., 2010; Stowe et al., 2009).

Herein, we report four cases of inflammatory demyelinating polyneuropathy presenting to the same hospital in northern Tasmania 1–3 weeks following ChAdOx1 nCoV-19 vaccination (AstraZeneca vaccine, AZ).

2. Cases

Over the course of six weeks, four individuals presented with Inflammatory Demyelinating Polyneuropathy (IDP), one with Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) and three with GBS, commencing one to three weeks after receiving the AZ vaccine. These included three males (51, 66 and 72 years) and one female (65

Abbreviations: GBS, Guillain- Barré Syndrome; CIDP, Chronic inflammatory demyelinating polyneuropathy; IDP, Inflammatory demyelinating polyneuropathy; CSF, Cerebrospinal Fluid; IVIG, Intravenous immune globulin; ICU, Intensive care unit; AZ, AstraZeneca Vaccine/ ChAdOx1 nCoV-19 vaccine; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; ACE2 receptor, Angiotensin-converting enzyme 2 receptor; NCV, nerve conduction velocity.

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