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**A CASE OF GUILLAIN-BARRE SYNDROME-A RARE AUTOIMMUNE MEDIATED  
INFLAMMATORY NEUROPATHY PRECEDED BY CAMPYLOBACTER JEJUNI.**

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**ABSTRACT**

autoimmune diseases are becoming quiet common among the population. these diseases are difficult to diagnose but, once diagnosed they are curable in most cases when treated earlier. here, we aRE GOING TO DISCUSS A CASE OF GUILLAIN-BARRE SYNDROME,AN AUTOIMMUNE DISEASE AND THE EARLY TREATMENT WHICH HELPS IN THE CURE.

**KEYWORDS:** GBS, CMV, EBV, IVIg.



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## INTRODUCTION

Peripheral nerve is susceptible to wide range of categories of disease including inflammation, trauma, toxins, etc.... Guillain-Barre syndrome is a life-threatening disease of the peripheral nervous system which is considered to be autoimmune in nature. Infections with *Campylobacter jejuni*, CMV (cytomegalovirus), EBV (Epstein-Barr virus) have been shown to have a significant epidemiological association with GBS. The incidence is approximately 1 in 3500 cases per year in the U.S. and Canada, which implies its rarity. Here we are going to discuss a case of *Campylobacter jejuni* induced GBS.

## CASE REPORT

A 17 year old female presented to the ER with complaints of rapid onset of disability in walking and tingling sensation in the lower limbs ascending in nature, since the past 3 days. She also complained of intermittent low back pain and deep aching pain in both the calf muscles. She was afebrile and her vitals were normal. Her bladder and bowel movements were normal. She also recently suffered from sudden onset of fever and diarrhoea, was admitted and was diagnosed to have infected with *Campylobacter jejuni*. This triggered us the doubt of GBS (Guillain-Barre syndrome). She was admitted under the general physician. The physician's examination revealed areflexic motor paralysis with hypoesthesia of both lower limbs, below the hip symmetrical in nature. Routine and certain specific investigations like ANA (anti-nuclear antibodies) and CSF (cerebro-spinal fluid) analysis were asked for. The abnormal results are as follows.

1. Absence of ANAs.

2. CSF analysis : protein- 7g/L, no pleocytosis.

## DIAGNOSIS

On the basis of the patient's history, clinical presentation and the laboratory investigations, she was diagnosed to be suffering from an autoimmune mediated neuropathy, Guillain-Barre syndrome. The absence of ANAs and other normal investigations helped us to rule out the other causes for neuropathy.

## DISCUSSION

Guillain-Barre syndrome is a rare lethal condition, if untreated, results in death. GBS results from immune responses to non-self antigens (infectious agent in this case) that misdirect to host nerve tissue through a resemblance-of-epitope (molecular mimicry) mechanism. The neural targets are likely to be the gangliosides. Antiganglioside antibodies, most frequently to GM1, are common in GBS, particularly in those precede by *C.jejuni* infection. Based on the history of the patient, clinical examination and the lab investigations, she was diagnosed to be suffering from GBS following infection with *C.jejuni* resembling the study conducted by *Mishu B. Blaser et al*<sup>1</sup>, *Noel McCarthy et al*<sup>2</sup> and *Tam CC et al*<sup>3</sup>. Unlike the study conducted by *Jeffery C Kwong et al*<sup>4</sup>, the patient had no history of any recent influenza vaccination. After she was diagnosed, she was immediately started on high-dose intravenous immunoglobulins (IVIg) for 5 days at a total dose of 2g/kg. As she started responding dramatically, unlike the study conducted by *Nobuhiro Yuk*<sup>5</sup>, she was discharged with review after a week.

## REFERENCES

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