

Case Report



Does High Poultry Intake Serve as a Potential Risk Factor for Recurrent Guillain-Barré Syndrome in Childhood?

Dr. Ravi Prakash ¹, Dr. Arshi Shamim ^{*2}, Dr. Suhas Panditrao Kulkarni ³, Dr. Saiprasad Onkareshwar Kavthekar ⁴, Dr. Nivedita Balasaheb Patil ⁵

¹Junior Resident, Department of Pediatrics, D.Y. Patil Medical College, D. Y. Patil Education Society (Deemed to be University), Kolhapur (416003), Maharashtra, India.

²Junior Resident, Department of Pediatrics, D.Y. Patil Medical College, D. Y. Patil Education Society (Deemed to be University), Kolhapur (416003), Maharashtra, India.

³Professor, Department of Pediatrics, D.Y. Patil Medical College, D. Y. Patil Education Society (Deemed to be University), Kolhapur (416003), Maharashtra, India.

⁴Professor, Department of Pediatrics, D.Y. Patil Medical College, D. Y. Patil Education Society (Deemed to be University), Kolhapur (416003), Maharashtra, India.

⁵Professor and HOD, Department of Pediatrics, D.Y. Patil Medical College, D. Y. Patil Education Society (Deemed to be University), Kolhapur (416003), Maharashtra, India.

*Corresponding author: Dr. Arshi Shamim; arshishamim007@gmail.com

Abstract

Guillain-Barré syndrome (GBS) is characterized by the onset of weakness following a nonspecific gastrointestinal or respiratory infection. Generally, GBS cases are monophasic, with recurrences being a rarity. The exact mechanisms and causes behind recurrent GBS are still not completely understood. There are only a few comprehensive reports of individuals who have been infected with *Campylobacter jejuni* and subsequently developed GBS on both occasions. In this case report, we discussed a case involving a 17-year-old boy who experienced recurrent GBS, having first encountered the syndrome at the age of 5 years and later at 17 years of age (present episode) which was more severe with a noted history of excessive poultry intake.

Keywords: *Guillain-Barré Syndrome; peripheral neuropathy; Campylobacter Jejuni, poultry intake.*

Introduction

Guillain-Barré Syndrome (GBS) is the most common and most severe acute paralytic neuropathy, with about 1.1/100,000 people developing the disorder every year worldwide ^[1]. GBS is thought to result from an immune response to a preceding infection that cross-reacts with peripheral nerve components because of molecular mimicry ^[2]. *Campylobacter Jejuni* (C. *Jejuni*) infection is the most frequently identified precipitant of GBS ^[3]. An analysis of the first 1000 patients enrolled in the International GBS Outcome Study (IGOS), for which 768 bio samples were available, showed that C. *jejuni* caused GBS in 30% ^[3]. The European Food Safety Authority's Expert Opinion Assessment has determined that chicken meat consumption is responsible for 20% to 30% of campylobacteriosis cases in the EU. Additionally, it suggests that the overall chicken reservoir may contribute to 50% to 80% C. *jejuni* infections, highlighting that the incidence of campylobacteriosis linked to broiler meat production varies across different countries ^[4].

Most cases of GBS present as a monophasic disease, but recurrence has been reported in 2-5% of cases ^[2]. We report a 17-year-old boy who presented with a second attack of GBS after 12 years; this time, the level of paralysis extended to the diaphragm and resulted in subsequent respiratory failure, requiring intubation and a prolonged period of mechanical ventilation, and linked to high intake of poultry intake.

Case Report

A 17-year-old male with a history of progressive ascending weakness, dysphagia, and respiratory distress following a brief episode of fever and diarrhoea 2 weeks prior. On detailed diet and past-history, there was history of excessive poultry consumption and he had similar episode of acute flaccid paralysis at 5 years of age and was successfully managed with Intravenous globulin (IVIG) alone. The detailed central nervous system examination revealed flaccid quadriparesis, generalized areflexia, and bulbar involvement and

respiratory failure. The cerebrospinal fluid and magnetic resonance imaging of brain and spine were normal. The clinical diagnosis of acute flaccid paralysis likely to be GBS was kept. He was treated with IVIG in the dose of 1 gm /kg /day for 2 days, mechanical ventilation with tracheostomy and supportive care. His hospital course was complicated by ventilator associated pneumonia and treated with broad spectrum antibiotics. He was successfully weaned off the ventilator by day 86 and discharged on day 117 with minimum residual neurological deficit.

Discussion

A recent outbreak of Guillain-Barré Syndrome (GBS) in western Maharashtra has sparked considerable concern regarding the potential role of poultry consumption as an etiological factor [6]. In response, we conducted an extensive review of the literature to examine the relationship between GBS and poultry intake, particularly given the patient's notable history of substantial poultry consumption. *Campylobacter jejuni*, a bacterium ubiquitous in the gastrointestinal tracts of poultry, pigs, cattle, and humans, is frequently transmitted via cross-contamination, predominantly within domestic kitchens [7]. It is prevalently found in both wild and domesticated avian species, particularly broiler chickens, breeder flocks, and egg-laying hens, positioning poultry as a principal vector for human infections [8,9]. The risk of transmission is markedly exacerbated when raw poultry is mishandled. According to Bull *et al.* [7] *C. jejuni* contaminates 98% of retail chicken meat in the United States, with contamination rates in Europe ranging from 60% to 80%.

A retrospective analysis of our case revealed a discernible history of substantial poultry consumption, which could have served as a precipitating factor for the onset of GBS, potentially mediated by *C. jejuni* infection. Given this context, it is plausible that dietary exposure to *C. jejuni* played a critical role in initiating the GBS episode in our patient. This underscores the necessity for adherence to stringent food hygiene practices, including ensuring that poultry is cooked to optimal temperatures to mitigate the associated risks of infection [4,7].

Campylobacter jejuni BD67 has been implicated in the pathogenesis of severe axonal variants of GBS. Its lipooligosaccharide structure bears a significant resemblance to gangliosides present in peripheral nerve membranes, thus potentially triggering an autoimmune response. Ganglioside-specific antibodies selectively target various peripheral nerve components, with anti-GD1a antibodies binding to the neuromuscular junction, nodes of Ranvier, and paranodal myelin, while antibodies against GM1 and GQ1B predominantly target the neuromuscular junction and peripheral nerves. The presence of these multiple antigenic targets' likely accounts for the heterogeneity in GBS clinical manifestations [10]. Moreover, stool examination to exclude *C. jejuni* infection should have been systematically performed, a step regrettably overlooked in this instance, which underscores the need for a more thorough diagnostic approach in similar cases. Thus, eating chicken that has *C. jejuni* on it could result in GBS.

Conclusions

1. The recurrence of GBS is potentially linked to *C. jejuni* from poultry consumption, emphasizing the critical need for a comprehensive dietary history and stool analysis.
2. It advocates for further research to find link between the recurrence of GBS and infections caused by *C. jejuni*, as well as the consumption of poultry.

List of abbreviations

GBS: Guillain-Barré Syndrome
 PN: Peripheral Neuropathy
 CJ: *Campylobacter jejuni*
 PI: Poultry Intake

Declarations

Ethics approval

Not Applicable

Consent to participate

Taken

Data Availability

Not Applicable

Conflicts of Interest

None

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None

Authors' contributions

All authors have contributed, designed and approved the study

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