Gullain – Barre syndrome associated with *Campylobacter jejuni* isolatefrom Rectal swab of a Patient; a University Teaching Hospital, Benin City Experience.

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ABSTRACT

Background: The unusual immune-mediated acute polyradiculo-neuropathy known as Guillain-Barré syndrome (GBS) is also known as post-infectious polyneuropathy due to the fact that it often manifests after a previous respiratory or gastrointestinal infection.

Although GBS has a wide spectrum of clinical manifestations, the condition most frequently shows up as increasing weakness of the limb, axial, face, or respiratory muscles, along with or without sensory or autonomic abnormality.

Numerous infectious organisms, vaccines, and other circumstances have been found to be GBS triggers.

C. jejuni and *Mycoplasma pneumoniae* are by far the most frequent bacteria associated with GBS.

However there have been several reported cases of GBS triggers which are not as a result of infection or vaccination

This case not only demonstrates the clinical features of Guillain-Barre syndrome but also its severity,

Method: The study was a Retrospective Cross Sectional Study. This involved a review of Case notes of Patients that were admitted in the Children Emergency Room (CHER) of UBTH from January 2019 to December 2019 ,been managed for Gullain-Barre syndrome associated with *Campylobacter jejuni* infection and also a review of their Medical microbiology laboratory test records during management especially their rectal swab Microscopy , Culture and Sensitivity results. It has never being the practice of the Children emergency unit to do rectal swabs for patients with Guillain - Barre syndrome, this was done by the medical microbiology unit upon invitation to see these patients. Patients with incomplete or missing medical data were excluded from the study.

Results: A case report of a male, patient aged 15years. He had recurrent episodes of passage of bloody diarrhoea. The most common initial symptom was weakness in the extremities.

Motor deficit involved all four limbs in this patient. He had ascending progressive paralysis within 48hours of onset of symptoms. There was no history of peptic ulcer disease and no Family history of peptic ulcer disease. No clinical features on examination was suggestive of peptic ulcer disease. There was a history of collection of poultry droppings from a nearby poultry for manure in family farm behind the house. There was history of dyspnoea and tachypnoea respiratory cycle was 63 cycles per minute, Rectal Swab showed Gram negative " gull wing" "s" shape bacilli, urease negative, Catalase positive, Oxidase positive characteristic of *Campylobacter jejuni*. There was heavy growth on Blood agar and Butzler's medium within 6 days of aerobic incubation sensitive to genticin, Erythromycin, Azithromycin and clarithromycin. He was placed on Erythromycin 250 mg 6 hourly for 14 days and recovered fully with Ambulation within 5 days on Erythromycin.

Conclusion: This case report emphasises a relatively inexpensive and straightforward faecal specimen by rectal swab, free of procedural complications like bowel perforation and readily available faecal specimen in the laboratory diagnosis and isolation of *Campylobacter jejuni ,* a cause (among other causes) of Gullain-Barre Syndrome, which is a medical emergency in most cases.Also that with the use of antibiotic therapy, most patients with GBS associated *Campylobacterjejuni* infection if they present early ,make full recover from enteric campylobacter infections.

Keyword: Gullain – Barre syndrome, *Campylobacter jejuni,* Rectal swab, Benin City.

INTRODUCTION

The unusual immune-mediated acute polyradiculo-neuropathy known as Guillain-Barré syndrome (GBS) is also known as post-infectious polyneuropathy because it often manifests after a previous respiratory or gastrointestinal illness [1,2]. It can also be brought on by immunisations and affects both children and adults. Although GBS has a wide range of clinical manifestations, the condition most frequently shows up as increasing weakness of the limb, axial, face, or respiratory muscles, along with or without sensory or autonomic dysfunction [1,2].

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Numerous infectious organisms, vaccines, and other circumstances have been found to be GBS triggers. Although it might undulate, the disease history is often monophasic. Up to one-third of instances involving respiratory muscles require artificial breathing, and up to twenty percent of those cases do so.

According to reports, the prevalence of GBS is 1-2/100,000/y [3]. Worldwide, an estimated 100,000 new cases are diagnosed each year. Males are afflicted more commonly than females [3). Every 10 years of age increase results in a 20% increase in incidence [2].

The majority of times that GBS occurs, infections are the cause [2].

The pathophysiology of GBS has been linked to bacteria and viruses. Accordingly, in a study in approximately two-thirds of cases, GBS is preceded by a symptomatic infection such as *Campylobacter jejun*i (*C. jejuni*), Epstein-Barr virus (EBV), cytomegalovirus (CMV), or influenza [4). *C. jejuni* and *Mycoplasma pneumonia*e are by far the most frequent bacteria that cause GBS [5]. CMV, Zika, and dengue are the three viruses that cause GBS most frequently [6].Humans typically contract C. jejuni through food consumption, especially through the eating of raw or undercooked poultry meat, tainted, unpasteurized milk, or water-based environmental sources [*7]. C. jejuni* colonises the distal ileum and colon after uptake [7)

Human *C. jejuni* infection post-infectious morbidity is significantly influenced by the shape of the pathogenic Lipo-oligosaccharide (LOS,), which activates the innate immune system via Toll-like receptor (TLR)-4 signalling [8]. TLRs (Toll-Like Receptors) identify specific molecular patterns linked with pathogens. They have a crucial role in inflammation, immune-cell modulation, survival, and proliferation as well as the initial line of defence against invasive infections. TLR1, TLR2, TLR4, TLR5, TLR6, and TLR11 are found on the cell surface, whereas TLR3, TLR7, TLR8, and TLR9 are found in the endosomal/lysosomal compartment. There are currently 11 members of the TLR family known to exist. The "molecular mimicry" between surface LOS antigens and ganglioside antigens (ceramides) on the surface of myelin sheaths or the axolemma is what causes GBS to develop through *C. Jejuni*.(9).

There have been several reported cases of GBS triggers which are not as a result of infection or vaccinations. Angio-immunoblastic T-cell lymphoma was the cause of GBS in a male patient in his 80s [10]. Although the pathophysiological rationale remained obscure, it was hypothesised that a tumour, a paraneoplastic condition, or an autoimmune reaction brought on by a typical exposure could have caused the immunological dysregulation [10]. GBS has also been described as a condition following a kidney donation [11).

Additionally identified as rare GBS triggers are intravenous ganglioside injection for trauma, surgery, stroke, peripheral neuropathy, and surgery [12].

According to culture studies, many Guillain-Barre syndrome patients also have *C jejuni* infection in their stools when their neurologic symptoms first appear. We present a case of Guillain-Barre syndrome linked to a *C.jejuni* infection in the gastrointestinal tract. This case not only demonstrates the clinical features of Guillain-Barre syndrome but also its severity.

CASE REPORT

A 15-year-old boy came to the hospital complaining of weakness in the lower extremities. Ten days earlier he had developed nausea, vomiting, and recurrent bloody diarrhoea associated with fever and chills after collection of poultry droppings from a nearby poultry for manure in family farm behind the house. The gastrointestinal symptoms subsided after 5 days but recurred after 2 days. His gastrointestinal symptoms did not resolve, necessitating his presentation and admission in the Children emergency ward of University of Benin Teaching Hospital. He had ascending progressive paralysis within 48hours of onset of symptoms. There was no history of peptic ulcer disease and no Family history of peptic ulcer disease.

There was history of dyspnoea and tachypnoea respiratory cycle was 63 cycles per minute, When the patient was admitted into the ward, 48 hours after being seen in the emergency department, he was paralyzed in all four limbs. He had grade 2/5 power in his upper extremities and grade 2/5 power in his lower extremities at the time of admission, and all deep tendon reflexes were absent.

Results of laboratory studies at the time of admission included a white blood cell count of 8,700/uL with a normal differential (presumably as a result of previously administered antibiotics).Rectal Swab showed Gram negative " gull wing" "s" shape bacilli, urease negative, Catalase positive, Oxidase positive characteristic of *Campylobacter jejuni* . There was heavy growth on Blood agar and Butzler's medium within 6 days of aerobic incubation sensitive to genticin, Erythromycin, Azithromycin and clarithromycin.

DISCUSSION

Campylobacter jejuni pathogen is highly susceptible to heating, but infections can be acquired by eating undercooked meat or through cross-contamination of kitchen utensils and surfaces. Poultry is the most important reservoir, and several studies indicate that 50 to 70% of sporadic human infections are caused by *Campylobacter species.* (13).

Person-to-person transmission is uncommon, but there are a few exceptions. One is perinatal transmission from mother to child, as a result of the infant's passage through the birth canal.in these instances the mothers are not necessarily symptomatic. (14). The second is when carers are exposed to faeces from infants or other incontinent people. Except for homosexual guys, adults rarely transmit diseases to one another.

Infection with Campylobacter occurs year-round, increasing in the summer in the majority of developed nations. Men and women contract the disease at comparable rates, however some data suggests women may be somewhat more at risk. Young children, especially those under 1 year old, have the highest age-specific attack rates, whereas those between the ages of 15 and 29 see a second wide peak in attack rates. In developing nations, where children may have 10 or more infections in their first two years of life, infection is considerably more prevalent. As people get older, attack rates drop and more infections go clinically quiet.

Bacteremia, which is seldom reported in part because of the organism's fastidious nature and the fact that blood cultures are frequently skipped, it is most frequent in patients who have impaired immune systems and are quite old.(15).Three main patterns have been identified for extraintestinal C jejuni infection. Days after the patient has recovered, the first condition is, transitory bacteremia in a previously healthy host, which is frequently identified by isolation from blood cultures. These patients typically do not require therapy. The second is persistent bacteremia, or seeding to an extraintestinal location in a healthy host together with enteritis. Responses to antibiotic therapy and drainage techniques are typically positive. Third, immunocompromised hosts are susceptible to bacteremia or profound infections. Some

of these infections are fatal, and they frequently last for a long time or come back. For Campylobacter infections Meninges, endovascular tissue, including the heart valve, peritoneal fluid, and soft tissues are a few of the other frequent extraintestinal locations, but many other sites have been documented as well. Hepatitis, interstitial nephritis, reactive arthritis in those who are HLA-B27 positive (16), a truncal macular rash, and Guillain-Barre syndrome are nonsuppurative consequences of *C.jejuni* infections.

In 1984, the first instance of *C .jejuni*-related Guillain-Barre syndrome was reported; since then, numerous other reports have been made public.(17,18). The only bacterial infection that consistently precedes Guillain-Barre syndrome is *C .jejuni*, which is currently believed to be the most frequent antecedent infection to the condition. (19) According to the description, the organism is a gram-negative bacterium belonging to the Campylobacter genus and related genera. This species seems to commonly colonise the intestines of people, other mammals, and birds. The likelihood that C jejuni infections play a part in the development of Guillain-Barre syndrome is supported by the high prevalence of these infections as well as by their propensity to penetrate tissue and cause inflammation. (20, 21).

In 50 and 75 percent of all instances of Guillain-Barre syndrome, jejuni infection has been found to be responsible. (20,21) Anecdotal reports are the first type of proof that Campylobacter infection causes Guillain-Barre syndrome. Serologic tests have revealed anti-C .jejuni antibodies in serum from Guillain-Barre syndrome patients, a finding that is consistent with a recent infection. This is the second line of evidence. A large percentage of Guillain-Barre syndrome patients had *C jejuni* in their faeces at the time of the onset of neurologic symptoms, according to culture studies. (22,23) Additionally, it has been noted that when C jejuni infection precedes Guillain-Barre syndrome, neurologic symptoms are more severe and more likely to be curable. (24). The risk of developing Guillain-Barre syndrome might be higher after infection with *C.jejuni* type 0: 19.(25).

In the treatment of Campylobacter-associated Guillain-Barre syndrome, plasma exchange was found to reduce the requirement for hospital treatment, with cost savings that outweighed the price of the medication.

Several antibiotics, such as macrolides, tetracyclines, quinolones, the aminoglycosides, chloramphenicol, and nitrofurantoin are effective against C jejuni in vivo. (26,27). Whether the infections arise naturally or as a result of antibiotic therapy, almost all patients make a full recovery from enteric Campylobacter infections. There have occasionally been fatalities in developed nations, particularly in elderly or immunocompromised people. The high morbidity and mortality rates linked to diarrheal diseases in underdeveloped nations are likely caused by a significant proportion of infections that occur very early in life before immunity takes over. This case highlights several interesting features of Guillain-Barre syndrome and C jejuni infection. Motor deficit involved all four limbs in this patient. He had ascending progressive paralysis within 48hours of onset of symptoms. There was no history of peptic ulcer disease and no Family history of peptic ulcer disease .No clinical features on examination of peptic ulcer disease. There was a history of collection of poultry droppings from a nearby poultry for manure in family farm behind the house before onset of symptoms.

CONCLUSION

This case report emphasizes a relatively inexpensive and straightforward faecal specimen by rectal swab, free of procedural complications like bowel perforation and readily available faecal specimen in the laboratory diagnosis and isolation of *Campylobacter jejuni*, a cause (among other causes) of Gullain-Barre Syndrome, which is a medical emergency in most cases. Also, that with the use of antibiotic therapy, almost all patients make full recovery from enteric campylobacter infections.

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