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Guillain – Barre syndrome associated with *Helicobacter pylori* isolates from rectal swabs of Four Patients: University of Benin Teaching Hospital Experience

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Abstract

Background: A Gram-negative bacteria known as *Helicobacter pylori* (*H. pylori*) is thought to be the cause of autoimmune gastrointestinal diseases. Additionally, this pathogen has been connected to peripheral neuropathies and extra-gastrointestinal illnesses with autoimmune sequelae. *H. pylori* may be involved in the 30% of GBS cases that *Campylobacter jejuni* is blamed for. Guillain-Barré syndrome (GBS) typically has a post-infectious aetiology. The Aim of the study is to show that rectal swabs to isolate *Helicobacter pylori* as a cause of Guillain - Barre syndrome should be a routine instead of gastric biopsy.

Methods: The study was a Retrospective Cross Sectional Study. It involved a review of Case notes of Patients that were admitted in the Neurology Unit of UBTH from 2014-2019, been managed for Guillain-Barre syndrome associated with *H. pylori* 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 been the practice of the Neurology unit to do rectal swabs for patients with Guillain - Barre syndrome, this was done by the medical microbiology unit that co-managed the patient. Patients with incomplete or missing medical data were excluded from the study.

Results: Four case series of one male and three female patients, age ranged from 7 years to 23years with the mean age of 15 ± 8 years. The most common initial symptom was weakness in the extremities which was seen in all the patients. Motor deficit involved all four limbs in all the patients. The mode of onset was progressive in all the cases and ascending.

History of peptic ulcer disease was present in all four patient and *H. pylori* was isolated from rectal swabs from all the patients. Three (75%) of the patient showed marked improvement on commencement of Omeprazole and erythromycin, while one (25%) ,did well on amoxicillin and Omeprazole. One (25%) had complications of bilateral patchy lower limb skin exfoliation and desquamation. A case of death was recorded. On discharge complete functional recovery was noted in two (50%), while one (33%) had partial recovery.

Conclusion: This study of four case series highlights a relatively inexpensive and straightforward faecal specimen collection by rectal swab, and readily available faecal specimen compared with gastric biopsy specimen, in the laboratory diagnosis and isolation of *Helicobacter pylori*, a cause (among other causes) of Guillain-Barre Syndrome, which is a medical emergency in most cases.

In addition Guillain-Barre Syndrome linked with *H. pylori* is not always a Post-infectious diseases because medications used to clear the gastrointestinal tract of Bacterial usually lead to recovery from the paralysis caused by Guillain-Barre Syndrome.

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Introduction

Helicobacter pylori is a spiral-shaped Gram negative bacilli. *H. pylori* is associated with a myriad of diseases in humans including antral gastritis, chronic gastritis, intestinal metaplasia, gastric adenomas, gastric hyperplastic polyps, duodenal (peptic) ulcer disease, gastric ulcers, gastric carcinomas, gastric lymphomas, atrophic gastritis, Guillain-Barre syndrome, Fisher's syndrome, Bickerstaff brainstem encephalitis, short statue, polyposis coli, colonic adenomas, colonic adenocarcinomas and sudden infant death syndrome.[1,2,16,17,18,19]

H. pylori overlies gastric-type but not intestinal type epithelial cells.[2,9] The mechanisms by which *H. pylori* causes mucosal inflammation and damage are not well defined but probably involve both bacterial and host factors.[1]

Gastric biopsy specimens can be used for histologic examination or minced in saline and used for culture.[2,9] Blood is collected for determination of serum antibodies.[1]

The diagnosis of *H. pylori* infection can be made histologically and by isolation of the bacteria by culture[1]. A gastroscopy procedure with biopsy is required.[1,9] Gram stain, Giemsa or special silver stains can show the curved or spiraled organisms.[1]

H. pylori is present in the stomach of approximately half the World's population.[7,8,9,10,11,12] The majority of those infected however remain asymptomatic throughout life, although some may have gastritis if gastric biopsies are carried out.[9] The rate at which a population acquires *H. pylori* infection is greater in developing than in industrialized countries, with high prevalence rates associated with poverty, poor sanitation, and low social class.[8,9,10] While in developing countries infection is acquired early in life.[8]

Routes of infection include; faeco-oral, oro – oral, person to person transmission, occupational hazards like in cases of gastroenteroscopist, genetic subsceptibility like those with blood group O antigen secretors[8,13,14].

Triple therapy with metronidazole plus amoxycillin or Erythromycin for 14 days eradicates *H. pylori* infection in 70-95% of patients.[2,21] An acid suppressing agent given for 4-6 weeks enhances ulcer healing.[1,2] Proton pump inhibitors directly inhibit *H. pylori* and appear to be potent urease inhibitors.[1] Either One week of a proton pump inhibitor plus amoxicillin or a macrolide or of amoxycillin and plus metronidazole also is highly effective.[1,22]

H. pylori infection leads to the release of several neurotransmitters, such as acetylcholine, adrenaline, noradrenaline, serotonin, and dopamine.[3,4]

Moreover, *H. pylori* infection leads to axonal/neuronal damage, production of free radicals and changes in neuropeptide expression, such as vasoactive intestinal peptide (VIP) and c – fos.[3]

Helicobacter pylori is considered to trigger autoimmune gastrointestinal diseases and peripheral neuropathies.[5,20] Guillain-Barre syndrome (GBS) is a serious autoimmune demyelinating disorder of peripheral nerves, usually with a post- infectious onset.[5,20] There is growing evidence suggesting the likely involvement of *H. pylori* infection in the development of GBS.[5]

Differential diagnosis of Guillain- Barre syndrome include: infections with *Helicobacter pylori*, *Campylobacter jejuni*, *Diphylobothrium latum, Penicious* anaemia, trauma to the spinal cord and type 2 immune hypersensitivity reactions.[1,6]

Case reports

Patient 1

An 18-year old female was admitted into hospital ward on 15th February, 2015 with strong history of peptic ulcer disease. Had three months history of weakness in the extremities and sudden ascending paralysis while in the kitchen. On examination, both lower limbs were hypotonic with a power of 0/5 each. Right upper limb had a power of 2/5 with left upper limb a power of 1/5. She was conscious but not oriented in time, place and person, no fever.

Rectal swab done with culture after five days of incubation aerobically at 37°c on chocolate, blood and Butzler's agars, showed numerous Gram negative 'gull winged' "s" shaped bacilli which was urease positive, catalase positive, oxidase positive and a positive motility test; characteristics of *Helicobacter pylori isolate*.

She was placed on Amoxycillin and omeprazole and regular physiotherapy. She started moving her toes on day four of therapy but died four weeks later after

admission due to industrial strike action by medical doctors with no medical attention further given to her.

Patient 2

A 15-year old female was admitted into hospital ward on 8th January, 2014 with family history of peptic ulcer disease and history of her six older siblings dying suddenly of peptic ulcer related illness. Had one month history of weakness in the extremities and ascending paralysis.

On examination, both lower limbs were hypotonic with power each of 0/5. Right upper limb had a power of 3/5 with the left upper limb a power of 2/5. She was conscious but very anxious. Well oriented in time, place and person. Epigastric tenderness was elicited with severe abdominal guarding, no fever.

Rectal swab done with culture after 6 days of incubation aerobically at 37°c on chocolate, blood and Butzler's agars; showed heavy growth of Gram negative 'gull winged' "s" shaped bacilli which was oxidase positive, catalase positive, urease positive and a positive motility test; characteristics of *Helicobacter pylori* isolate.

She was placed on Erythromycin and Omeprazole with regular active and passive physiotherapy. Had complications of bilateral patchy lower limb skin exfoliation and desquamation from prolonged lower limb neuropathy for which plastic surgery was done. Started walking using crutches after six months of admission and became fully ambulant at seven months of admission and was discharged home to be followed up at the outpatient Clinic.

Patient 3

V. O., a female 23 years old was admitted into hospital ward on 11th September, 2019 with strong history of peptic ulcer disease, married for two years but with primary infertility. Had two weeks history of weakness in the extremities and ascending paralysis. On examination, her right lower limb had a power of 2/5, with left lower limb having a power of 1/5. Her both upper limbs had a power of 4/5 each. She was conscious but very anxious and well oriented in time, place and person. Epigastric tenderness was elicited with severe abdominal guarding, no fever.

Rectal swab was taken and cultured on blood, chocolate and Butzler's agars and after 4 days of incubation aerobically at 37oc; revealed heavy growth of Gram negative 'gull winged' "s" shaped bacilli which was catalase positive, oxidase positive, urease positive and a positive motility test; characteristics of *Helicobacter pylori.*

She was placed on Erythromycin and Omeprazole with gradual rat tailing of her previous steroids therapy with passive and active physiotherapy. She recovered fully and became ambulant after seventy – two hours on Erythromycin and Omeprazole which was completed for fourteen days before she was discharged home with follow up at the outpatient clinic.

Patient 4

P. M., a male 7 years old boy from Cross Rivers state, Nigeria admitted into the intensive care unit with history of peptic ulcer disease; from the relatives and one month history of weakness in the extremities and ascending paralysis. On examination, he was placed on positive pressure ventilation, conscious but orientation in time, place and person could not be elicited due to the fact that he was on the ventilator. He was globally hypotonic, with both lower and upper limbs flaccid and hypotonic.

Rectal swab was taken and cultured on chocolate, blood and Butler's agar and incubated aerobically at 37°c. After 7 days of incubation there was growth of Gram negative 'gull winged' "s" shaped bacilli, catalase positive, oxidase positive, urease positive and a positive motility test; characteristics of *Helicobacter pylori*.

He was weaned off the ventilator after 48 hours on Erythromycin and Omeprazole and discharged home from hospital after three weeks of hospital admission with follow up at the outpatient clinic after completing fourteen days on Erythromycin and Omeprazole.

Discussion

Helicobacter pylori is known to be associated with a myriad of diseases including Gullain – Barre syndrome and peptic ulcer disease.[1] Specimen usually used to isolate this bacteria is gastric biopsy which can only be gotten by gastroenteroscopy which make it a difficult specimen to collect ith potential complication of gastric perforation and needs for expensive facilities and a trained

gastroenteroscopist. In this study, rectal swab was used to collect faecal specimen. This study demonstrates a well-documented case series of isolating *Helicobacter pylori* from faecal specimens collected by rectal swabs from a University Teaching Hospital in Benin City, Nigeria. Findings from this study suggest that *H. pylori* can also survive in intestinal mucosa contrary to other studies.[1,2] Also, this study suggests that Gullain-Barre syndrome associated with *Helicobacter pylori* infection is not usually a post-infectious disease as the bacteria clearance from the gastrointestinal tract using drugs usually lead to recovery from the paralysis caused by GBS as compared to other studies.[5]

Usually, our Neurology unit does not request for rectal swabs for patients with Guillain - Barre syndrome, this was done by the medical microbiology unit upon consultation to co-manage these patients. In the 4 patients, *H. pylori* isolated was differentiated from their close relative *Campylobacter jejuni*, by biochemical test. *Campylobacter jejuni* isolates are urease negative, while *H. pylori* isolates are strongly urease positive but both bacteria are catalase and oxidase positive and both show as Gram negative "gull winged" "S" shaped bacilli on Gram stain.

Conclusion

This study of four case series Guillain-Barre syndrome highlights a relatively cheap and easy -to-collect faecal specimen by rectal swab compared with gastric biopsy specimen for the laboratory isolation of *H. pylori* as a cause of Guillain – Barre syndrome which is a life threatening medical emergency.

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