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ANTIBIOTIC-ASSOCIATED PSEUDOMEMBRANEOUS COLITIS IN A NIGERIAN- CASE REPORT

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INTRODUCTION

Psedomembranous enterocolitis (PMC) was first described in the 19th Century following occurrence of fatal diarrhoea in post-operative patients who were administered antibiotics[1]. Several risk factors in settings outside the use of antibiotics have since been identified. Some of these risk factors are colonic, gastric and pelvic surgeries. Others include intestinal obstruction, spinal fracture, colon carcinoma, leukaemia, severe burns, shock, haemolytic uraemic syndrome, heavy metal poisoning, ischaemic cardiovascular disease, Crohn's disease, severe infection, shigellosis, ischaemic colitis, Hirschprung's disease and neonatal necrotising enterocolitis[2]. In spite of the various causes, antimicrobial agents appears to be the commonest cause of pseudomembranous colitis. Virtually all drugs with antimicrobial properties have been associated. A recent report incriminated clindamycin as the commonest associated drug[3]

The responsible organism in PMC was initially thought to be *Staphylococcus aureus* and was then called *Staphylococcus aureus*-associated enterocolitis, until 1978, when the toxins of *Clostridium difficile* were implicated in the great majority of cases[1]. Detection of *Clostridium difficile* is possible by stool assay using enzyme immunoassay that can detect toxin A and B. Most cases of PMC usually respond to antibiotic cessation and use of metronidazole, vancomycin and supportive care. Notable complications are ileus and toxic megacolon.

The objective of this report was to sensitize all medical practitioners to this potentially fatal complication of use and abuse of antibiotics in clinical settings, so as to be cautious about use of multiple antibiotics and be able to recognize the condition and treat appropriately.

CASE REPORT

EP, a 42 year old man was referred to our Hospital with a history of profuse diarrhoea of 11 days duration which started a few hours after taking a popular stew called "isi ewu" (goat head) at a restaurant. Bowel motions were watery, non-mucoid, non-bloody and not associated with fever or abdominal pain. He presented in a private clinic on the day of onset of diarrhoea and was placed on amoxicillin/ampicillin and gentamicin. With no improvement, he presented in another hospital where he was placed on amoxicillin clavulanate, metronidazole, hyoscine hydrochloride and metochlopramide. Diarrhoea became more profuse and he later developed fever and abdominal cramps. He was then admitted and placed on cephalosporin, secnidazole and later azithromycin and loperamide. Bowel motions increased to about 25 per day and had to be referred to a tertiary health facility. He had had 2 previous episodes of diarrhoea after eating at similar restaurants but these episodes were self-limited and quite unlike the present one. There were no associated symptoms of arthritis or history of food allergy. He is a known hypertensive on amlodipine.

Clinical examination revealed a well nourished man, weak and moderately dehydrated with a tinge of jaundice. Temperature was 37.2^{0}C, with mild pitting ankle oedema.

Abdomen was slightly distended and tympanitic with diffuse tenderness and reduced bowel sounds. Liver was palpable 2 cm below the right costal margin, and was tender with a span of 14cm. The rectum was empty and smooth, gloved finger was stained with a thin film of brownish stool. Other aspects of clinical examination were essentially normal.

Stool microscopy did not reveal trophozoites or ova of any parasite and stool culture was negative after 72 hours. WBC count was 10,000 with 82% neutrophilia. Electrolytes and urea examination showed potassium of 3.2 Meq/L and of 87mg/dl. Serum bilirubin was 2.4mg/dl and alanine transaminase of 52 IU/L. Abdominal ultrasonography was normal. A clinical diagnosis of food poisoning complicated by septicaemia and pseudomembranous colitis was made. Proctosigmoidoscopy showed

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Inflamed colon with patchy yellowish exudates (Fig. 1) and the histology report of biopsy (Fig. 2) revealed fragments of ulcerated colonic tissue with a focal exuberant mucoid and fibrinosuppurative exudates, with prominent acute inflammation consistent with pseudomembranous colitis.

All previous antibiotics were discontinued except oral metronidazole at 250mg tds. He was hydrated, had electrolytes corrected intravenously and also placed on oral cholestyramine 4g tds. He was subsequently placed on vancomycin. Diarrhoea and abdominal pains resolved over a two week period and was discharged.

Figure1. **Endoscopic Picture Of The Lest Hemicolon Showing Severe Inflammation And Yellowish Exudate**

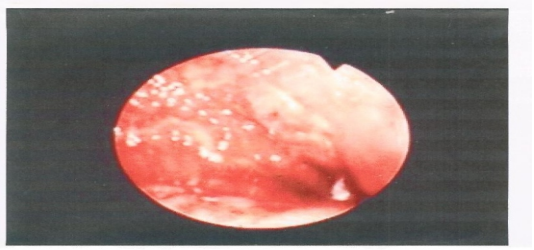
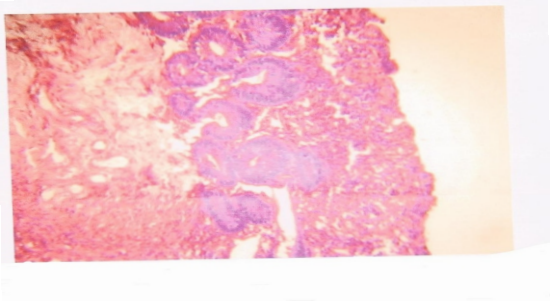


Figure II: **Low Power Micrograph Showing Adherent Pseudomembrane Of Fibrin, Mucus And Inflammatory Debris.**



DISCUSSION

A number of gastrointestinal disorders have been known to be associated with consumption of contaminated food eaten outside the home setting. This is particularly rife with roadside eateries where diseases like typhoid, cholera and food poisoning could easily be contracted. The case in point is a victim who developed food poisoning which was followed by a septicaemic illness. The overzealous use of antibiotics, predisposed him to the complication that brought him to our care. It is auspicious to postulate that a liver abscess, from colonic amoebiasis could have been responsible for the patient's condition and is a possible clinical diagnosis in this patient. This was considered but the ultrasonography of the liver easily excluded this. Histology of colonic biopsy in patients with PMC usually shows a plaquelike adhesion of fibinopurulent-necrotic debris suggestive of an infective origin, as seen in this patient`s biopsy features. These features may however be indistinguishable from those of an early stage of ulcerative colitis. Though confirmatory test for presence of clostridium difficile cytotoxin in stool sample could not be performed in this patient due to lack of laboratory facility, the clinical and colonic histological features were however highly suggestive in this patient.

Among the effective modalities of treatment of *C. Difficile* are oral vancomycin, metronidazole, cholestyramine which is known to chelate the toxin of the C. difficile and supportive care. *C. Difficile* antitoxin administration is also well known to be effective. Of all these metronidazole and cholestyramine were the most readily available in this environment. Vancomycin, which is the better of the antibiotics for management of PMC, is not readily available and had to be specially ordered for our patient. Clostridium antitoxin could not be sourced for this patient and was not used. Probiotics which are dietary supplements containing potentially beneficial bacteria and yeasts, are well known for their microbiological properties and have been used to treat gastrointestinal and vaginal mucosal infections. They have been proved to be beneficial in antibiotic associated enterocolitis[4] and is recommended as food supplement in patients with antibiotic-associated colitis.

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