Review Article
ISSN: 0974-6943

O. Azuonwu et al. / Journal of Pharmacy Research 2011,4(1),22-23

Available online through
http://jprsolutions.info

Hemorrhagic colitis and its public health implications: A review.

Azuonwu.O, Abbey.S.D, Wokem.G.N, Adegoke

[1]*Department of Medical Laboratory Sciences,Rivers State University of Science and Technology,P.M.B. 5080, Port Harcourt, Nigeria.*

Received on: 15-09-2010; Revised on: 18-10-2010; Accepted on:13-12-2010

ABSTRACT

Hemorrhagic colitis also known as Hamburger's disease is a type of food poisoning caused by a specific strain of *Escherichia coli*: *E. coli 0157:H7*. The causative agent produces toxins known as shiga-like toxins and also produces lesions causing hemorrhagic colitis with severe abdominal pains and cramps followed by bloody diarrhea. This strain of Escherichia coli infects the large intestine and produces toxins which kill the vascular endothelial cells with consequent epithelial necrosis. The disease is generally associated with ingestion of enterohemorrhagic *E.coli* (EHEC) in undercooked ground beef, unpasteurized fruit juices and milk. The pathogen is spread by the feacal-oral route, and an infectious dose appears to be only 500bacteria.Hemorrhagic colitis which is one of the worldwide diarrheal diseases is often misdiagnosed and causes 70,000 deaths annually. Several chemicals, toxins, drugs and infections can also cause hemorrhagic colitis. Oral administration of antibiotics, oseltamivir, sennoside- a laxative used for cleansing and treatment of constipation has been associated with hemorrhagic colitis. This tends to lead to certain complications such as high blood pressure. Early laboratory diagnosis (using Sorbitol MacConkey agar and other serological diagnosis) and a high sense of hygiene must be monitored carefully in slaughterhouses where contact of meat with faecal material can occur, and at homes where contaminated hands and utensils can aid the spread of the disease.

Key words: Hemorrhagic colitis, Public Health Implications, *E. coli* 0157:H7

INTRODUCTION

Enterohemorrhagic *Escherichia coli 0157:H7*, a world-wide human food-borne pathogen, causes gastroenteritis with mild to severe bloody diarrhea, hemorrhagic colitis and hemolytic-uremic syndrome (HUS) (1,2). The ability of this pathogen to persist in the environment contributes to its dissemination onto a wide range of foods and food processing surfaces (3). The strain of *E. coli* was first recognized as a pathogen during an outbreak of unusual gastrointestinal illness in 1982 in the United States (4). The outbreak was traced to contaminated hamburgers and the etiological agent of the illness was identified as a rare 0157:H7 serotype of *Escherichia coli* in 1983.

Several chemicals, toxins, drugs and infection can also cause hemorrhagic colitis. Oral administration of sennoside as a laxative [for cleansing and treatment of constipation] has been associated with sennoside- induced hemorrhagic colitis (5). Antibiotic-associated hemorrhagic colitis have also been reported with hemorrhagic colitis developing on the left colon after introduction of quinolones (6) and amoxicillin- induced hemorrhagic colitis developing on the right colon. (7,8). Oseltamivir, a strategic drug for the treatment of influenza A has been recognized as being associated with the risk of acute hemorrhagic colitis after being administered orally. (9).

Klebsiella oxytoca was recently described as the causative organism for antibiotic-associated hemorrhagic colitis (AAHC). (10). It is currently not known if this novel GIT infection exists in children. (7).

BACTERIOLOGY

The bacterium *E. coli* which is a normal inhabitant of the intestine of man and animals has several strains of which *E. coli 0157:H7* is one. This strain which produces shiga-like toxins (11) causes severe illness and is referred to by its toxin-producing capabilities. *E. coli 0157:H7* does not produce heat-stable or heat-labile toxins, it is non-hemolytic, sorbitol negative, ß-glucuronidase negative and does not grow at 45^{0}C in the presence of 0.15% bile salts, hence this serotype because of the latter characteristic cannot be isolated using standard faecal coliform method which includes incubation at 45^{0}C. (12). One of the virulence factors of *E. coli 0157:H7* is production of a periplasmic catalase enzyme (11). The perisplamic catalase enzyme is believed to be involved in virulence by providing additional oxidative protection when infecting the hosts (13).

***Corresponding author.**
Azuonwu, Obioma.
Department of Medical Laboratory Science,
Rivers State University of Science and Technology,
Nkpolu, P.M.B 5080, Port Harcourt, Rivers State, Nigeria.
Tel: +234803519688.
Email: azuonwuobi@yahoo.com

SIGNS AND SYMPTOMS

The illness is characterized by severe abdominal cramps and diarrhea which initially is watery but becomes grossly bloody with occasional vomiting. (14). Fever is either low grade or absent (15). The illness is usually self-limiting with incubation period of 2-10 days. (15).

EPIDEMIOLOGY

The first *E. coli 0157:H7* outbreak was reported in 1982 and linked to ground beef, which remains the most common vehicle for food borne outbreaks (16). The etiology of the disease is universal and can be food borne or environmental (16). Epidemiological investigation has shown that the illness appeared to be associated with consumption of hamburgers (17), swimming in, drinking faecally contaminated water (18,19), consumption of contaminated sprouts, lettuce, unpasteurized milk (20), juice (21) eating undercooked/uncooked ground beef and through faecal-oral transmission from an infected person to a healthy individual due to unhygienic practices. (14,16).

Close to 75,000 cases of *0157:H7* infections are now estimated to occur annually in the United States (22). Another potential vector of *Escherichia coli 0157:H7* is filth flies which include house flies: *Musca domestica*. (23). Filth flies have been shown to be vectors of *Escherichia coli 0157:H7* using the polymerase chain reaction technique (23).

PATHOGENESIS

Production of a potent shiga-like toxin is essential for many of the pathological features as well as the life-threatening sequelae of STEC infection (24). Orally ingested STEC must initially survive the harsh environment of the stomach before competing with other gut microbes to establish intestinal colonization (25) as they are resistant to the acidity of the stomach (26). The rpoS factor regulates genes enabling *E. coli* organisms to survive for extended period before pH 2.5 (27). Certain STEC strains are capable of causing A/E lesions on enterocytes (28). Other adherence factors implicated in STEC pathogenicity include the fimbriae and lipopolysaccharides (29). Studies with rabbits have shown the direct enterotoxic properties of shiga-like toxins on the villus epithelial cells in the ileum (13) and intravenous injection of stx1 into rabbits caused diarrhea with edematous and hemorrhagic lesions in the mucosa and submucosa of the cecum (13). The toxin binds principally to distal convoluted tubules in adult kidney and to the glomeruli and convoluted tubules in infants (25). Tubular necrosis is also seen in a proportion of cases (30).

LABORATORY DIAGNOSIS

Laboratory examination of stool is likely to show the presence of *E. coli 0157:H7*, though the test is not done routinely.

After development of HUS however, there is likelihood of anaemia and thrombocytopenia (15,30). Strains of Verocytoxic *E. coli 0157* are cultured on sorbitol MacConkey medium for their selectivity (31). Detection of VTEC in faecal sample has also been used successfully as a diagnostic tool (24). Serological diagnosis of VTEC infection primarily in cases of HUS has also been described with serological responses and secretory immune response in Hemorrhagic colitis due to *E. coli 0157* appearing to have been little studied (31).

Enzyme-linked immuno-strip biosensor can also be used to detect *E. coli 0157:H7* (32). Urinalysis may reveal hematuria and proteinuria, elevated creatinine levels, slight leukocytosis and thrombocytopenia (33, 30).

COMPLICATIONS

About 5-10% of individuals with *E. coli 0157:H7* infections develop a potentially fatal complication called hemolytic-uremic syndrome (HUS) characterized by renal failure (14) occurring in 55-70% of patients and hemolytic anemia (30). This conditions usually occurs in younger children (10-15%) (2-3years) and can lead to permanent kidney damage or death (1, 30). Oliguria and edema may also set in some individual develop complications of the nerve or brain damage such as seizures and strokes (15).

PROGNOSIS AND TREATMENT

Majority of infections resolve completely (27). One-third of patients with HUS have abnormal kidney function many years later, 3-5% die, a few require long-term dialysis and another 8% develop other lifelong complications such as high blood pressure, seizures, blindness, paralysis and perhaps surgery (30).

Most people recover without antibiotics and antibiotics may precipitate kidney complications (6, 7, 8). Blood transfusion and kidney dialysis often may be required (14).

CONCLUSION AND RECOMMENDATION

Since hemorrhagic colitis is a type of food poisoning which is often being misdiagnosed, learning more about the ecology of the aetiological agent in cattle and other ruminants is an indispensable tool.

Good and proper hygienic practices should be implemented in the handling and processing of meats, vegetables and juices by food handlers and day-care centres, as this decrease in the incidence of hemorrhagic colitis would decrease to a larger extent the development of HUS and other complications.
Other methods such as the serological approach for the detection of *E. coli 0157:H7* should also be utilize.

ACKNOWLEDGEMENT

We wish to thank the Rivers State University of Science and Technology ICT center who provided the enabling environment and infrastructure for the materials to be sourced for; their staff were also on hand to give technical services that led to smooth sourcing of the information. We equally wish to thank Mr. Adeyemo Emmanuel who did the typesetting and who took time to correct the mistakes that were initially observed.

REFERENCES

1. Ingrid Chou Koo (2009): Bloody diarrhea or hemorrhagic colitis caused by E. coli 0157:H7. Centre for Disease Control and Prevention. Division of Foodborne, Bacterial and Mycotic Diseases.
2. Eaton K.A., Friedman D.I., Francis G.J., Tyler J.S., Young V.B., Haeger J., Abu-Ali G., and Whittam T.S., (2008): Pathogenesis of Renal Disease due to EHEC in Germ-Free Mice. Journal of Infection and Immunity **76**:3054-63.
3. Puttamreddy S., Cornick N.A., and Minion F.C., (2006): Genome-wide transposon mutagenesis reveals a role for p0157 Genesin biofilm development in E. coli 0157:H7. Journal of Infection and Immunity. **78**(6): 2377-2384.
4. Riley L., Remis R., Helgerson S., McGee H., Wells J., David B., Blake P. Hebert R., Olcott E., Johnson L., and Cohem M., (1983): Hemorrhagic colitis associated with a rare E. coli Serotype. New England. Journal of Medicine;. 3**08**(12): 681-685.
5. Ozdil B., Kece C., and Cosar A., (2010): Acute Hemorrhagic colitis following administration of sennosides for colon cleansing. Annuals of. Pharmacotherapy; **44**(4). 770-1.
6. Kwun K. A., Ham J.H., Kim E.J., Chung I.K., Kim H.S., Park S.H., Lee M.H., and Kim S.J., (2001). Antibiotics – Associated hemorrhagic colitis: A report of two cases. Korean Journal of Gastrointestinal Endoscopy; **22** (1): 45-49.
7. Hoffmann K.M., Deutschmann A., Weitzer C. Joainig M., Zechner E., Hogenauer C., and Hauer A., (2010): Antibiotic-associated hemorrhagic colitis caused by cytotoxin-producing Klebsiella oxytoca. Pediatrics **125** (4): 960-963.
8. Kim H.S., Cho Y.S., Koh Y.S., Shim K.H., Choi Y.K., Kim H.K., Kim C.N., Kim S.S., Chae H.S., Han S.W., Lee C.D., and Choi K.Y., (2004): A case of hemorrhagic colitis complicated by thromborytic thrombocytopenic purpura. Korean Journal of Gastrointestinal Endoscopy; 29(6) 529-533.
9. Matsushita M., Nishihara H., Nishiyama R., and Kobayashi Y., (2007): Acute hemorrhagic colitis associated with oral administration of oseltamivir for the treatment of influenza A., Journal of Infection and Chemotherapy; **13**(4): 267-269(3).
10. Hogenauer C., (2006). Klebsiella oxytoca a causative organism of antibiotic associated hemorrhagic colitis. New England Journal Medicine; **355**:2418-2426.
11. Whittam T.S., Wachsmuth I.K., and Wilson R.A., (1988): Genetic evidence of clonal descent of E. coli 0157:H7 associated with hemorrhagic colitis and hemolytic-uremic syndrome. Journal of Infectious Diseases; **157**(6): 1124-33.
12. Winn W., (2006). Textbook of Diagnostic Microbiology; 6th edition. Lippincott Williams and Wilkins.
13. Mizuguchi M., Tanaka I., Fuji H., Tanizawa Y., Suzuki T., Igarashi T., Yamanaka T. and Miwa M. (1998): Neuronal and vascular pathology produced by verocytotoxin 2 in the rabbit central nervous system. Journal of Neuropathology; **91**:254-262.
14. Centre for Disease Control and Prevention Information Brochure (1997): Produce Handling and Process Practices. Journal of Emerging Infectious Disease**;** **3**(4).
15. David C. Dugdale (2008): Life-threatening cases associated with outbreaks of hemorrhagic colitis. Journal of Clinical Medicine**;** **54**:1125-1126.
16. Rangel J.M., Sparling P.H., Crowe C., Griffin P.M. and Swerdlow D.L., (2005): Epidemiology of E. coli 0157:H7 outbreaks, United States 1982-2002. Journal of Emerging Infectious Disease; **11**(4): 603.
17. Pai C.H., Gordon R., Sims H.V., and Bryan L.E. (1984): Sporadic cases of hemorrhagic colitis associated with E. coli 0157:H7. Annuals of Internal Medicine; **101**(6): 738-42.
18. Keene W.E., McAnulty J.M., Hoesly F.C, Williams L.P., Jr., Hedberg K., Oxman G.L, Barrett T.J., Pfaller M.A., and Fleming D.W., (1994): A swimming-associated outbreak of hemorrhagic colitis caused by E. coli 0157:H7 and Shigella sonnei. New England Journal of Medicine; **331**(9) 579-84.
19. Ackman D., Marks S.P., Mack M., and Root T., (1997): Swimming-associated hemorrhagic colitis due to E. coli 0157:H7 infection evidence of prolonged contamination of a freshwater lake. Journal of Infectious Disease Epidemiology: **38**:1125-7.
20. Keene W.E., Hedberg K., Heriott D.E., Hancock D.D., McKay R.W., Barett T.J., and Fleming D.W., (1997): A prolonged outbreak of E. coli 0157:H7 infections caused by commercially distributed raw milk. Journal of Infectious Diseases; **176** (3): 815-8.
21. Cody S.H, Glynn M.K., Farrar J.A. Cairns L.K., Griffin P.M., Kobayashi J., Fyfe M., Hoffmann R., King A.S., Lewis J.H., Swaminathan B., and Vugia D.J., (1999): An outbreak of E. coli 0157:H7 Infection from unpasteurized commercial apple juice. Annuals of Internal Medicine; **130**:202-209.
22. Mead P.S., (1999). Food-related illness and death in the United States Journal of Emerging Infections; **5**: 607-625.
23. Brazil S.M., Steelman C.D., and Szalanski A.L., (2007): Detection of pathogen DNA from filth flies (Diptera: Muscidae). The Journal of Agricultural and Urban Entomology; **24** (1).
24. Paton J.C., and Palm A.W., (1998). Clinical Microbiology Reviews: Pathogenesis and diagnosis of shiga-toxins producing E. coli infections. American Society for Microbiology **25**:1128-35.
25. Gourmelon M., Montet M.P., and Lozach S., (2006): Pathogenesis and diagnosis of shiga-toxin producing E. coli infections. Journal of Applied Microbiology **100**(1): 85-97.
26. Goodson M., and Rowbury R.J., (2002): Habituation to normally lethal acidity by prior growth of E. coli at a sublethal acid pH value. Journal of Applied Microbiology **8**:77-79.
27. Greatorex J.S., and Thorne G.M., (1998): Humoral immune responses to shiga-like toxins and E. coli 0157:H7 lipopolysaccharides in HUS patients and healthy subjects. Journal of Clinical Microbiology; **32**:1172-78.
28. Pamela J.S., Kenneth G.C., and John M.L., (2007): Interaction of Enterohemorrhagic E. coli with mammalian cells. Journal of Infections and Immunology; **57**:1290-98.
29. Rendon M.A., (2007). Commensal and Pathogenic E. coli **104**(25): 10637-42.
30. Fivush B., Jabs K., and Primack W., (2009): Complications associated with hemorrhagic colitis. Kidney and Urologic Disease Control Publication Number 09-4570. American Society of Pediatric Nephrology.
31. Chapman P.A., Siddon C.A., Zadik P.M., and Jewes L., (1991): An improved selective medium for isolating E. coli 0157:H7. Journal of Medical Microbiology; **35**:107-110.
32. Sojung P., Kim H., Se-Hwan P., Hong J.W., and Kim Y.K., (2008): Enzyme-linked immuno-strip biosensor to detect E. coli 0157:H7. Journal of Clinical Medicine; **55**:1126-30.
33. McMillan R. (2007). Hemorrhagic Disorders: Abnormalities of Platelet and Vascular Functions. Cecil Medicine. 23rd edition, Philadelphia Pa: Saunders Elseviers. Chapter 179.

Source of support: Nil, Conflict of interest: None Declared