

## Alcoholic Gastritis: Eminence of Clinical Pharmacist to Attenuate Quality of Life

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*Available Online: 25<sup>th</sup> August, 2019*

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### ABSTRACT

Gastritis is one among the many gastrointestinal conditions commonly found. The etiological cause of gastritis may be of various sources like food habits, toxicity from food etc.,. Alcoholic gastritis is where chronic abusers of alcohol face a serious condition with revilement possible only once the withdrawal of alcohol is done. Rehabilitation therapy is involved in engaging the patients to withdraw alcohol which is being abused by them. Presently a case series on alcoholic gastritis of three patients have been done. Culture examination of the patient's revealed helicobacter pylori found in two and Escherichia coli found in the other. Their amylase and lipase levels showed wide variations with USG and CECT reports confirming gastritis with etiological cause of alcohol. Pharmacotherapy was initiated with Bismuth subsalicylate 524mg, amoxicillin 500g, and metronidazole 500mg. additionally; antacids like Ranitidine 150 mg, antiemetic like ondansetron 8mg and vitamin supplements like B-complex were given. Rehabilitation counseling and necessary lifestyle modification has also been insisted upon. Cognitive and behavioral therapies were included within the counseling given. The intact role of the clinical pharmacist could be in the sectors of guiding with pharmacotherapy, counseling imposed on lifestyle changes to be done and rehabilitation therapy guidance with support. Eradication of the microorganism which gained access through food an alcohol, and withdrawal of alcohol would bring back the patient to normal life.

keywords: Alcohol, Abuse, Gastritis, Rehabilitation, case series, Patient counselling, Clinical Pharmacist.

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### INTRODUCTION

Gastritis occurs due to an inflammation or swelling to the lining of the stomach. Excessive consumption of alcohol is one among the number of potential causes of gastritis. Symptoms of gastritis include pain in the upper area of abdomen, abdominal bleeding, nausea and vomiting, unpleasant mouth, diarrhea, loss of appetite, unexplained weight loss etc.,<sup>1-3</sup> Alcohol is a toxin that irritates the stomach lining so that it becomes inflamed. It is more likely to occur if the individual regularly abuses alcohol. Frequent consumption does not allow the stomach to recover from the irritation to the lining<sup>4-6</sup>. NSAID may aggravate development of alcohol-induced acute gastric lesions causing hemorrhages which destroy parts of mucosa. Prolonged alcohol exposure however disturbs microcirculation and leads to progressive structural mucosal damage. Decreased formation of hormone substances, prostaglandins play crucial roles in alcohol-induced mucosal injury. Studies have proved that alcohol concentration of 10% and more disrupt the gastric mucosal secretion and increase mucosa's permeability<sup>7,8</sup>. Continued alcohol abuse may cause the stomach lining to bleed leading to the development of other stomach problems such as ulcer. Some medications such as Aspirin are to be used cautiously in case of symptoms of bleeding; some drugs are to be taken along with meal in order to reduce irritation<sup>9,10</sup>. Recreational drug use may increase

the risk of overdose or serious health complications because many people will binge on multiple drugs. Alcoholics have a significantly higher incidence of atrophy or gastric mucosa and decreased gastric secretory capacity<sup>11</sup>. Decrease in auto production reduce stomach's ability to destroy the bacteria that enter with foods and thus favors colonization of upper small intestine with potentially harmful micro-organisms at the same time<sup>12</sup>. Due to social situations people binge on drugs and alcohol and take multiple drugs at same time. Alcohol consumption can interfere with function of all parts of GI tract. Hypocalcaemia is commonly associated with acute gastroenteritis condition. Calcium regulation is critical for normal cell function, neural transmission, bone structure, membrane stability, blood coagulation, intracellular signaling. Hypocalcemic urgencies could lead to significant death<sup>13,14</sup>.

Complications of gastritis include;

Anemia which can result from chronic bleeding due to erosive gastritis.

Atrophic gastritis which occurs due to lose of both the stomach lining and glands.

Gastric or peptic ulcers which form in the lining of stomach epithelium and duodenum.

Growth in stomach lining which can be benign or cancerous. If the gastritis is caused by helicobacter pylori bacteria, if also increase risk of stomach ulcer.

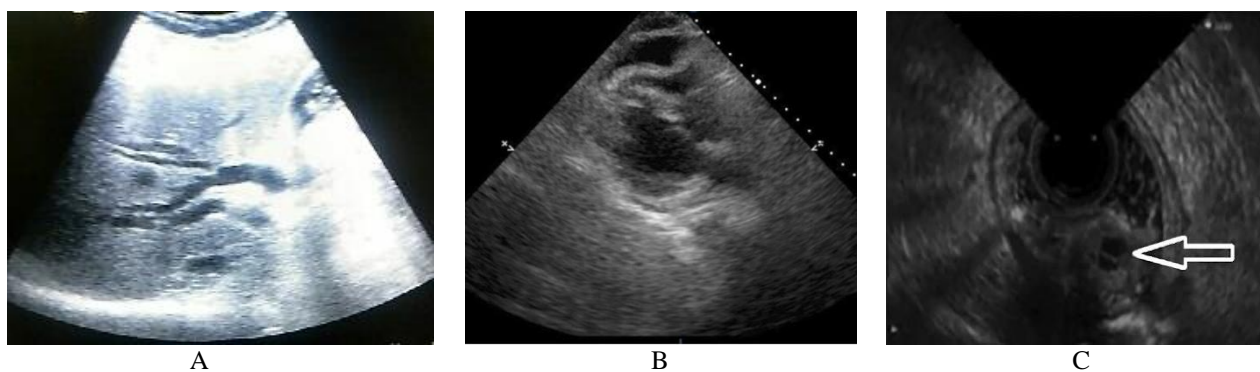


Figure 1: USG reports.

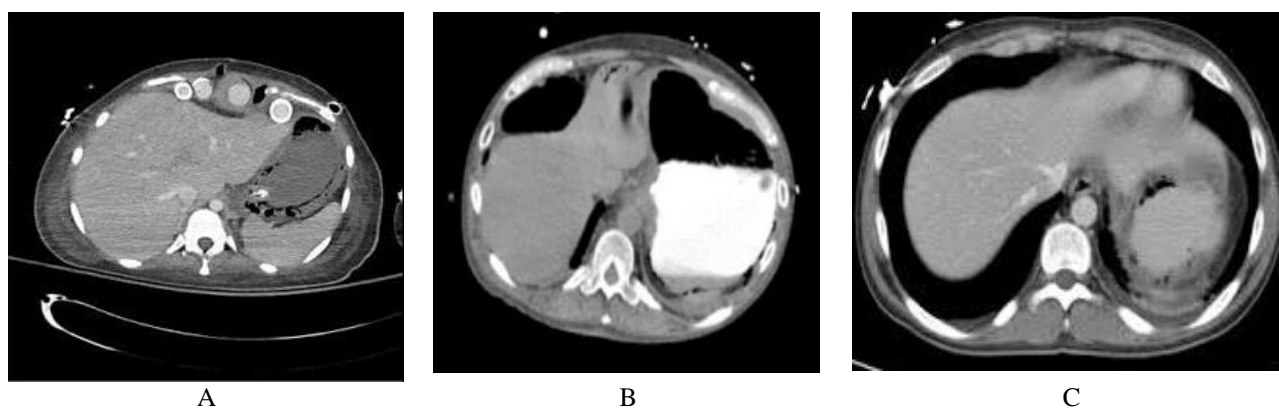


Figure 2: CECT reports.

A case series of three patients on Alcoholic gastritis with long term alcohol abuse have been presented below along with steps taken in alcohol rehabilitation and lifestyle changes possibly done.

**CASE 1:** A 48 year old male patient was admitted to the hospital with chief complaints of abdominal pain for past 10 days of sudden onset with intermittent attacks with aching abdominal pain in visceral region, which relieves on staying still for certain times, vomiting for past 3 days with episodes of five times, watery content usually sour smelling natural chills occasionally and loss of appetite for past 3 days. He was a functional alcoholic for the past 20 years and not a known case of DM/HTN/Epilepsy/Asthma/TB. He belonged to a low socioeconomic class category and had a poor diet with intake of caffeinated drinks. He had a family history of liver problems. On general examination the patient was conscious, oriented, and afebrile. On systemic examination his GI had an inflammation of mucosal layer of abdomen with P/A soft. He was habituated to chewing of nicotine gums. He was a daily wager and found difficulty in reducing his alcohol level with drinking habits at morning and night times during work. His Amylase-450 U/L, Lipase-366 U/L levels were high. His transabdominal USG indicated a bulky edematous abdomen with erosive central gastritis. CECT of abdomen showed emphysematous gastritis with gas in stomach wall along with portal venous gas. Pharmacotherapy and lifestyle modifications were initiated with recommendation of rehabilitation counseling for alcohol decrement.

**CASE 2:** A 38 year old male patient was admitted to the hospital with chief complaints of loss of appetite for past 3 days, gastric mucosal bleeding, vomiting with 2 episodes at morning and night times, giddiness for past 3 days. He belonged to a middle class socioeconomic class category with no history of DM/Htn/Asthma/TB/seizures. He was a functional alcoholic for the past 10 years and a chain smoker too. He worked in a clerical post at a government office and met with increased day to day stress, which made him to drink alcohol on daily basis as a stress buster, according to his consideration. He had a diet with fast and spicy food and increased caffeinated drinks and carbonated products added upon. On general examination he was conscious, confused and afebrile. On systemic examination of his abdomen was found to be distended and contracted with tenderness associated. Bowel and bladder habits were irregular with motility of sphincters altered. His Amylase level was about 489U/L and Lipase was about 400U/L. On transabdominal USG taken; it indicated a perforation of his fundus region with bleeding associated. Chronic unnoticed condition of gastritis had resulted in such condition. CECT of the abdomen revealed accumulation of gas in the inner walls of stomach. Pharmacotherapy and rehabilitation counseling were insisted upon with proper follow up.

**CASE 3:** A 74 years old male patient was admitted to the hospital with chief complaints of abdominal cramping, bloody diarrhea, vomiting with signs of dehydration for the past 5 days. He had a past history of diabetes, hernia repair and prostate surgery. He was a farmer and belonged to an average socioeconomic class category. He was a functional

alcoholic for the past 30 years and was on limit for the next few years. Recently he had again begun to follow his habit of becoming himself addict to alcohol without which he felt shaky in his hands and legs with no strength. He was leading a stressful life due to his wife's death and no caring from his children. He had a family history of colorectal cancer in his mother. On general examination the patient was conscious, oriented, afebrile, drowsy. On systemic examination of his abdomen it was protuberant, soft and tender in the mid-epigastric region. Feeling of fullness of stomach was associated with loss of weight. Amylase level was about 425U/L and Lipase levels of 399U/L. Transabdominal USG indicated a severe gastritis in the base and bulky edematous pancreas with surrounding fat standing and fluid collection. CECT abdomen showed gastritis and acute pancreatitis with acute fluid collection and minimal left pleural effusion. Rehabilitation care was initially insisted for the person and normal lifestyle counseling was started on.

From culture sensitivity test of their stools, it indicated the presence of *Helicobacter pylori* in two patients and *Escherichia coli* in the other. Pharmacotherapy in all the three patients was initiated with Bismuth subsalicylate 524mg, amoxicillin 500g, and metronidazole 500mg. additionally antacids like Ranitidine 150mg, antiemetic like ondansetron 8mg and vitamin supplements like B-complex where eradication of the microorganism was focused initially.

## DISCUSSION

Alcohol induced damage to the mucosal lining of esophagus increases risk of esophageal cancer. Alcohol interferes with gastric acid secretion and with the activity of muscles surrounding the stomach. Alcohol may impair the muscle movement in the small and large intestines contributing to diarrhea, frequently observed in alcoholics<sup>15,16</sup>. Alcohol inhibits absorption of nutrients in the small intestine and increases the transport of toxins across the intestinal walls, effects that may contribute to the development of alcohol related damage to the liver and other organs. Direct contact of alcoholic beverages with the mucosa that lines the upper GI tract can induce numerous metabolic and functional changes<sup>17</sup>. It leads to marked mucosal damage resulting in wide range of acute and chronic disease such as acute gastrointestinal bleeding and diarrhea. Beverages with high alcohol concentration appear to inhibit gastric motility and thus delay emptying of stomach. Thus, due to increased gastric transit time bacterial degradation of food may begin, the resulting gastric may lead to feelings of fullness and abdominal discomfort<sup>18</sup>.

Alcoholic beverages with low alcohol content increase gastric acid secretion and release of gastrin, In contrast beverages with high alcohol content stimulate neither gastric acid secretion nor gastric release<sup>19,20</sup>. With increased level of stress, the individuals may start to develop physical symptoms such as gastritis. Avoiding of excessive alcohol consumption by sticking to the recommended levels of safe drinking; (i.e.) one drink per day for women and two drinks per day for men would be a

healthy routine. Clinical pharmacist could be effective in insisting on the rehabilitation points to the patients and their family members.

## CONCLUSION

Chronic abuse of alcohol could lead to various improper functioning of each system within the body, especially the gastrointestinal tract experiences with wide problems among which gastritis are common. Alcohol remains as a toxin at its highest consumption rate and poses many issues. Rehabilitation considerations and patient counseling on withdrawal of alcohol slowly along with life style modification counseling could be enhanced in order to avoid further complications and to improve the quality of life.

## COMPLIANCE WITH ETHICAL STANDARDS

Written informed consent was obtained from the patient for publication of the case study, inclusion of the accompanying images. Copies of written consent may be requested for review from the corresponding author.

## CONFLICT OF INTEREST

The authors declare no conflicts of interest concerning the content of this case report.

## REFERENCE

1. Musher, D.M. and B.L. Musher. "Contagious acute gastrointestinal infections". *N. Engl. J. Med.* 2004; 35(1):2417-27.
2. Gangarosa, R.E, R.I. Glass, J.F. Lew, *et al.* "Hospitalizations involving gastroenteritis in the United States, 1985: the special burden of the disease among the elderly". *Am. J. Epidemiol.* 1992; 135(5): 281-90.
3. Garey, K.W. and D. Pharm. "Prevalence of Diarrhea at a University Hospital and Association with Modifiable Risk Factors". *The Annals of Pharmacotherapy.* 2006; 40(6): 1030-34.
4. Bovee-Oudenhoven, I.M, M.L. Lettink-Wissink, Van W. Doesburg, *et al.* "Diarrhea caused by enterotoxigenic *Escherichia coli* infection of humans is inhibited by dietary calcium". *Gastroenterol.* 2003; 125(5): 469-76.
5. Westblom, T.U. and T.W. Milligan. "Acute Bacterial Gastroenteritis Caused by *Hafnia alvei*". *Clinical Infectious Diseases.* 1992; 14(6): 1271-72.
6. Turner, M.J., J.B. Angel, K. Woodend, *et al.* "The efficacy of calcium carbonate in the treatment of protease inhibitor-induced persistent diarrhea in HIV-infected patients". *HIV Clin Trials.* 2004; 5(1): 19-24.
7. Heubil, J.E., J.C. Partin and W.K. Schubert. "Hypocalcemia and steatorrhea-Clues to etiology". *Digestive Diseases and Sci.* 1983; 28(2): 124-128.
8. Hirshhorn, N. "The treatment of acute diarrhea in children: A historical and physiological perspective". *The American J. Clinical Nutrition.* 1980; 33: 637-63.
9. Bode, J.C. "Alcohol and the gastrointestinal tract". *Advances in Internal Medicine and Pediatrics.* 1980; 45:1-74.

10. Gentry, R.T.; Baraona, E.; and Lieber, C.S. Agonist. "Gastric first pass metabolism of alcohol". *Journal of Laboratory and Clinical Medicine*. 1994; 12(3):21-26.
11. Levitt, M.D. Antagonist. "The case against first-pass metabolism of ethanol in the stomach". *Journal of Laboratory and Clinical Medicine*. 1994; 12(3):28-31.
12. Salaspuro, M. "Bacteriocolonial pathway for ethanol oxidation: Characteristics and implications". *Annals of Medicine*. 1996; 28:19(5)-200,.
13. Sato, N, and Kitamura T. "First-pass metabolism of ethanol: An overview". *Gastroenterology*. 1996; 11(1):1143-1150.
14. Dupont HL. "The Practice Parameters Committee of the American College of Gastroenterology". Guidelines on acute infectious diarrhea in adults. *Am J Gastroenterol* 1997; 92: 1962-75.
15. Dupont HL, Reves R, Galindo E, et al. "Treatment of travelers' diarrhea with trimethoprim/sulfamethoxazole and with trimethoprim alone". *N Engl J Med*. 1982; 30(7): 841-4.
16. Bhan M, Rai P, Levine M, et al. "Enteroggregative *Escherichia coli* associated with persistent diarrhea in a cohort of rural children in India". *J. Infect Dis*. 1989; 15(9): 1061-4.
17. George W, Nakata M, Thompson J, et al. "Aeromonas-related diarrhea in adults". *Arch Intern Med*. 1985; 14(5):2207-11.
18. Guerrant RL, Van Gilder T, Steiner TS, et al. "Practice guidelines for the management of infectious diarrhea". *Clin Infect Dis*. 2001; 31:331-51.
19. Park SI and Gianella RA. "Approach to the patient with acute diarrhea", *Gastroenterol Clin North Am*. 1993; 22:483-97.
20. Dryden MS, Gabb RJ, Wright SK. "Empirical treatment of severe acute community-acquired gastroenteritis with ciprofloxacin". *Clin Infect Dis*. 1996; 22:1019-25.