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Review Article

Nigella Sativa Therapy on Acute Alcoholic Gastritis

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ABSTRACT

Alcohol is a predominant factor in about 60 types of diseases and injuries worldwide. Almost 5.1% of all deaths occurring universally are attributed to consumptions of alcohol (WHO 2014). There is a progressive trend as being witnessed in the form of increase in youth addiction to alcohol consumption. Hence, there is an urgent need to pursue this chronic problem and to device an enduring solution. Consumption of alcohol leads to a proportionate increase in malnutrition in addition to clinical manifestation like fatty liver, wernicke's encephalopathy etc. Alcoholics required to be integrated into the mainstream society by providing suitable rehabilitation. In the due course of rehabilitation, alcoholics suffer from severe withdrawal symptoms and gastritis. On the basis of extensive perusal of research reports regarding the clinical benefit of nigella sativa, there is a substantial avenue to explore and to augment knowledge on the healing benefits of the herb.

Keywords: Nigella sativa, Gastritis, Histamine , Mucosal ,H2receptor Antagonist, Proton Pump Inhibitor

1. INTRODUCTION

Alcohol is one of the widespread drink consumed in social gathering. According to world health organization 2014 report.[1] Alcohol consumption is world's third largest risk factor for diseases. A National household survey of drug use reported that marked variation in alcohol use prevails in different state of India.[2] Similarly study of John et al (2009) showed that 14.2% of population had hazardous alcohol consumption in southern states of India.[3] Alcohol consumption leads to negative consequence for the drinker, to his immediate environment and the

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society. Social problems such as traffic accidents, workplace related issues, domestic violence and interpersonal misdemeanour have been the goals of research attention in the current era.

Due to habitual intake of drinks, the productivity at workplace is greatly reduced. Such people are irregular to work also resort to uninformed leave thereby causing imbalance in the work environment and reduction in the company performance. Klingemann & Gmel (2001) have found there is a strong association between heavy drinking or alcohol abuse and unemployment. According to industry association sources in India, 15% to 20% of absenteeism and 40% of accidents at workplace has been reported due to consumption of alcohol).

It has been proved that drinking severely impair the individual functioning in various social roles. Alcohol consumption adversely affects the drinkers' immediate family. Maternal consumption during pregnancy leads to foetal alcohol syndrome in children, and parental drinking is correlated with child abuse. It also adversely imparts the child's environment in many social, psychological and economical ways (Gmel & Rehm, 2003). Drinks are consumed after spending considerable money; this has a strong impact upon the family resources particularly for a economically backward family, leaving the family members a destitute. Specified intoxicated events leads to a lasting consequences, pronouncedly through home accidents and family violence (Room, 1998; Room et al., 2002).[4]

2. GASTROINTESTINAL DISORDERS IN ALCOHOLICS

When heavy amount of alcohol is ingested, the human body is adversely affected by such consumption. The alcohol metabolite acetaldehyde has deleterious effect on various organs. Primary among them being the brain and the gastrointestinal system.[5] Alcohol can alter functioning of parotid gland and its secretion, decrease lower oesophageal sphincter pressure and inhibit the peristalsis of the distal esophagus.[6] It is also considered as major cause of both acute and chronic pancreatitis. A study in–vivo (using pancreatic tissue from patients with alcohol-induced chronic pancreatitis and from animal models of experimental pancreatitis) and in-vitro indicate that alcohol exposure activate pancreatic stellate cells (psc) through mitogen – activated protein kinase pathway as the mediators of pancreatic fibrosis.[7,8,9]

According to world Health Organization excessive alcohol consumption may lead to development of spectrum of liver disease, including hepatic steotosis, alcoholic hepatitis, alcoholic fibrosis, cirrhosis and hepato cellular damage.[10,11] Recent studies indicate that alcohol plays a strategic role in gastric carcinoma and colon cancer. Possibly by immune suppression action of alcohol and augment the magnitude of free radical.[12] Ultimately alcohol also irritates the stomach, a condition known as gastritis[13] in which inflated production of gastric acid can be observed, which can contribute to peptic ulcers and potential bleeding.[14]

3. PATHOPHYSIOLOGY OF GASTRITIS IN ALCOHOLICS

Alcohol after ingestion through mouth, passes through esophagus and reaches stomach walls into blood stream.[15] Alcohol consumption results in production of more gastric acid, which can exacerbate gastritis and contribute to peptic ulcers and potential bleeding.[16] Alcoholic gastritis have been included in ICD-10 code bearing the number K 29.2.[17] Moderate drinkers have acute gastritis and heavy drinkers may have chronic gastritis. Acute gastritis is a term covering a broad spectrum of entities that induce inflammatory aetiologies.[13] Acute gastritis can be classified into two categories, erosive (superficial erosion) and non-erosive (caused by helico-bacter pylori).[18] Gastritis accounts for 20% of all upper gastrointestinal bleeding

in that population.[13] In a study by Rin Yoshinda (2007), alcoholics were observed for two weeks post regular alcohol consumption. The study was focused on the presence and the degree of esophageal and gastric inflammation. During the upper digestive tract endoscopy revealed that 39% patients had gastritis in gastric corpus and 98% had in the antral part of the stomach. In microscopic examination gastritis was confirmed in gastric corpus and antrum respectively in 62 and 94% of patients.[19]

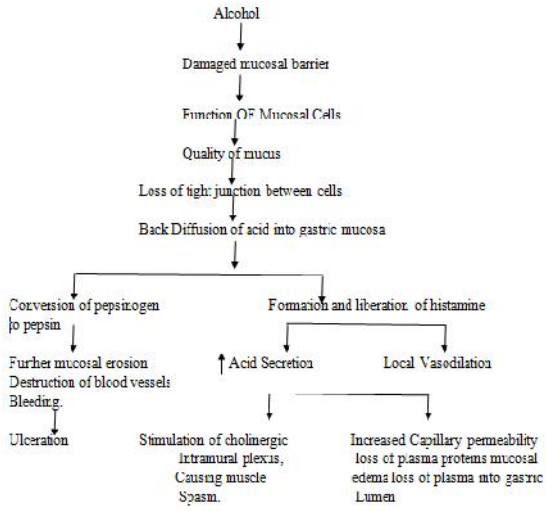

Fig 1: Paradigm of Pathology of Gastritis

(SOURCE- Kathyrn.L.Mccance, Sue.E.Huether., Pathophysiology the biologic basis for disease in adults and children, seventh Edition, (2014)., Elsevier Mosby Pp – 1438). [20]

4. MANAGEMENT OF ALCOHOLIC GASTRITIS THROUGH PHARMACOTHERAPY

There are two pharmacologic methods for reducing gastric acid content. The first entails the neutralization of gastric acid through the use of antacids, and the second a decrease in gastric acid production through the use of H2- receptor antagonist that block gastric acid secretion stimulated by histamine, gastrin and acetylcholine.[13] Histamine H2 –receptor antagonist decreases acid secretion and endorses healing.[20] In acute hemorrhagic gastritis, intravenous proton pump inhibitors are given for few days and thereafter switched to oral proton pump inhibitors.[21] The proton pump inhibitors block the final stage of hydrogen ion secretion by blocking the action of the gastric parietal cell proton pump.[22]

5. TREATMENT OF ALCOHOLIC GASTRITIS WITH NIGELLA SATIVA

Nigella Sativa is a potent healer of several biological systems in human body. Gastritis is treated with pharmacological agents, a tradition continued from good old days. Alternatively, rural medical professionals have used folk medicines, particularly some herbs, functional foods, spices and condiments etc. Conventional drugs such as omeparazole, Rantidine, Pirenzepine is competent to curtail the escalation of gastric acid secretion, inflammation and neutralize mucin individually. The intake of nigella in measured quantity addresses the above discussed theme holistically.

Nigella induce anti–inflammatory prostaglandins which in turn lead to secretion of increased mucin content, bicarbonate and thereby neutralize acid in gastric tissue[23], thus acting as a potent antacid agent.

In several other studies, a prominent component thymoquinone was isolated from Nigella sativa seeds and its impact on alcoholic gastritis had been evaluated. Thymoquinone can augment mucin content and glutathione level and a significant decline in mucosal histamine content in the stomach.[24] In another study, administration of Thymoquinone promoted ulcer index by increasing thiobarbituric acid – reactive substances (TBARS) (an index of lipid peroxidation), glutathione content (GSH), enzyme gastric superoxide dismutase (SOD), glutathione transferase (GST) and radical scavenging activity.[25]

The injection of thymoquinone on lab animal, increased glutathione, total nitric oxide and superoxide. Indeed higher dosage of thymoquinone corrected the altered parameters in comparable to the function of proton pump inhibitor drug omeparazole.[26]

Intake of alcohol leads to gram positive bacterial growth and increase in endotoxin. This condition eventually results in non-erosive acute gastritis. Such a condition can be effectively addressed by the use of nigella sativa.[27] Thymoquinone primarily present in nigella sativa exhibits strong anti-microbial properties that inhibits the growth of both gram positive and gram negative bacteria except certain strains pseudomonas pyocyanea.[28]

Nigella sativa seeds and oil extracts are effective anti-inflammatory substances. The Nigellone (Polythymoquinone) subsisting in nigella seeds controls inflammation by adapting following mechanisms:

1. By increasing the secretions 5- lipoxenase and 5-hydroxy eicosateterenoic acid probably due to anti-oxidant action thus ameliorating inflammatory disease.[29, 30,31,32,33]

2. By inhibiting histamine release induced by antigen and calcium ionophores.[28]

3. By restraining nitric oxide production.[34]

6. CONCLUSION

Research on alcoholic gastritis is the need of the hour due to the phenomenal consumption of alcohol globally. As more and more population gets hooked to alcohol, particularly the youths in general, with a chunk of the patients confined to the developing nations of Asia and the African populace in particular, as a consequence of globalization. Worldwide alcoholics suffer from several clinical manifestations, this phenomena continue to haunt them even when they undergo rehabilitation. In the due course of rehabilitation, malnutrition associated with hemorrhagic gastritis will alleviate the withdrawal symptoms. For such group, an interventional diet will be of great support in the treatment of the gastritis. A humble effort has been endeavoured to find a cost effective and naturally available and endurable product devoid of any artificial additives or preservatives. A very encouraging result emerges on the basis of discourse of research already available on the positive aspect of the outcome of nigella sativa on clinical studies on patients suffering from cardio-protective, nephro-protective, hepato-protective to name a few. A scientific based nutrition supplement program has to be inculcated with the regionally available diet of the patient which needs to be cost effective and available in its pristine form to cater to the less fortunate populaces. Of all the products available, the modern research on Nigella Sativa is found to be very promising.

7. REFERENCES

1. Vladimir Poznyak, Dag Rekve, Jurgen Rehm, Kevin Shield,Gretchen Stevens,World Health Organization , Global Status Report on Alcohol and Health 2014, Chapter -3,Pp-46.

2. Pramitha Murthy, N.Manunatha, B.N.Subodh, Prabhat Kumar Chand, Vivek Benegal, Substance use and addiction research in India, Indian journal of Psychiatry.Org.,2011,IP: 210.212.203.211.

3.John A, Barman A, Bal D,Chandy G, Samuel J,Thokchom M, et al Hazardous alcohol use in rural Southern India, Nature Prevalence and risk factors, National Medical journal, India, 2009; 22:123 -5.

4. Catherine Le Gales -Camus. et al,Global status report on alcohol Department of mental health and substance abuse, World Health Organisation, 2004 Pp-35 – 62.

5. Eastwood., Principles of Human Nutrition II, Edition, Black Well Publishing, (2006), Pp – 224-238.

6. Singer M.V, Bernner.D.A, Alcohol and the Gastro – intestinal tract, S. Kanger, 2005, 8114-9, Pp – 31.

7. Mccarroll JA, Phillips PA,Park.S, Dohetry E, Pirola RC, Wilson JS, Aptemv Pancreatic stellate cell Activation by ethanol and acetaldehyde: Is it mediated

by the mitogen – activated protein kinase signalling pathway pancreas,2003; 27 (2): 150 – 160.

8. Masamune A,Satoh A, Watanabe T,Kikuta K, Satoh M, Suzuki N, Satoh K, shimosegawa T. Effects of ethanol and its metabolities on human pancreatic stellate cells, Dig ,Dis sci 2010 J; 204 – 11,

9. Masamune A,Kikuta K, satoh M, satoh A, Shimosegawa T.,Alcohol activates activator protein -1 and mitogen – atctivated protein kinases in rat pancreatic stellate cells, Jounal Pharmacol Exp Ther, 2002; 302 (1): 36 – 42.

10 World Health Organization Global Status Report on alcohol and health, WHO Press 2011, Pp -20 – 37.

11. Sugmund VT, Dooley S, Brenner DA, Molecular Mechanisms of alcohol induced hepatic fibrosis, Dig Dis. 2005; 23(3-4) : 264-74.

12.Thomas D.Boyer, Michael P.Mans, Arun J. Sanyal, Zakim and Boyer s Hepatology A Textbook of liver disease , 6th edition, Elsevier saunders , Pp – 493 - 525.

13.Bernadette madara, Vanessa pomarico-denino 2008 Pathophysiology, II Edition,Jones and Barlets Publishers Pp – 368.

14. Priscillia, Medical surgical Nursing, Critical thinking in client care,Karen Burke ,(2008), Pp – 677.

15. Vasudevan D.M., Sreekumari S, Text Book of Biochemistry, Third Edition Jaypee Brothers Publishers, 2001, Pp – 282.

16. Peter L. Meyers and Richard E. Isralowitz, " Health and medical issues today – Alcohol, Library of congress cataloguing in publication data, 2011,Pp – 49 – 50.

17. Laine L, Weinstein WM, "Histology of alcoholic hemorrahagic gastritis a prospective evaluation, Gastroenterology, 1998 ; 94(6) : 1254- 62.

18 Paul Insel, Don Ross, Kimberly McMchan ,Melissa Bernstein Nutrition, Jones and Barlett publishers ,2011 Pp – 314 – 316.

19 Rin Yoshinda, Trends Alcohol Abuse and Alcoholism Research, Nova Science Publishers, 2007, Pp - 77 .

20.Kathyrn.L.Mccance,Sue.E.Huether.Pathophysiology the biologic basis for disease in adults and children, seventh Edition, (2014)., Elsevier Mosby Pp – 1438.

21 Gerald Zernig et al.,Hand Book of Alcoholism, CRC Press, 2000,Pp- 179

22.K.D.Tripathi., Essentials of Medical Pharmaocology, Sixth Edition,Jaypee Brothers Medical

27 Akhtar A.H, Ahmad K.D, Gilani S.N and Nazvi .A, Antiulcer effect of aqueous extracts of Nigella sativa and Pongamia pinnato in rats fitoterpia,1996,Pp -67 :195- 199,

23 K.D.Tripathi.,Essentials of Medical Pharmaocology, Sixth Edition,Jaypee Brothers Medical Publishers, 2008, Pp-631-632.

24.El-Dakhakhny.M,Barakath.M,M.El.Halim and S.M.Aly ,Effects of Nigella Sativa oil on Gastric sectretion and ethanol – induced ulcer in rats, Journal ethanopharmacol, 2000,72 – 299 – 304.

25. Kanter.M, Demir.H, Karakaya.C, Ozbek.H, Gastroprotective activity of Nigella Sativa L oil and its constituent, thymoquinone against acute alcohol – induced gastric mucosal injury in rats, world journal gastroenterol, 2005; 11 (42) : 666..

26. Magdy MA, Hanan el – A, Nabila el – M, Thymoquinone Novel Gastroprotective Mechanisms, European Journal Pharmacol, 2012; 697 (1- 3): 126 – 131.

27. Aftab Ahmad, Asif Husain, Mohd Mujeeb, Shah Alam Khan, Abul Kalam Najmi, Nasir Ali Siddique Zoheir A, A review on therapeutic potential of Nigella sativa: A miracle herb, Asian Pacific Journal of Tropical Biomedicine,2013; (13): 60075-1 .

28. K.V.Peter,Hand book of Herbs and Spices ,Second Edition, Wood Head Publishing Limited (2012) Pp- 391- 415.

29. Houghton P .J, Zarka.R, De – Las – Heras.B and Hoult.JR, Fixed oil of Nigella sativa and derived thymoquinone inhibit eicosnoid generation in leukocytes and membrane lipid peroxidation , planta Med ,1995; 61: 33 – 39.

30. Mutabagani.A. and S.A.M.El - Mehdy, A Study of the anti – inflammatory activity of nigella sativa and thymoquinone in rats, Saudi Pharmaceutical Journal ,5 1997; 5: 110 – 113.

31. El-Dakhakhny.M,Barakath.M,M.El.Halim and S.M.Aly ,Effects of Nigella Sativa oil on Gastric sectretion and ethanol – induced ulcer in rats,Journal ethanopharmacol, 2000; 72: 299 – 304.

32. El – Mahmoudy, AH.Matsuyama, M.A.El – Sayeed, N.Minamoto and J.Jakewaki, Thymoquinone suppresses expression of inducible nitric oxide synthase in rat macrophages int. Immunopharmacol., 2002; 2: 1603 – 1611.

33. Mahmood.M.S, A.H.Gilani, A.Khwaja, A.Rashid and M.K.Ashfaq, The in vitro effect of aqueous extract of nigella sativa seeds on nitric oxide production, phytotherapy 2007; 17: 921 – 924.

34. El – Mahmoudy, AH.Matsuyama, M.A.El – Sayeed, N.Minamoto and J.Jakewaki, Thymoquinone suppresses expression of inducible nitric oxide synthase in rat macrophages int. Immunopharmacol., 2002;2:1603 – 1611.

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