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**Original Article**

# Profile of biochemistry and haematology of patients having alcoholic and nicotine dependence syndrome

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**ABSTRACT**

Alcoholic and Nicotine dependence syndrome is as a cluster of physiological and psychological including behavioral and cognitive phenomena in which the use of a substance takes on much higher priority for individual than other behaviors that once had greater value. The major objective of this study is to compare and assess the biochemical and haematological markers observed in patients with alcohol and nicotine dependent syndrome in comparison to those with only alcohol dependent syndrome group. A total of 50 male patients with alcohol and nicotine dependent syndrome were included. As a control group, 50 male patients with alcohol dependent syndrome only and 25 male normal healthy volunteers also included. The liver function marker enzymes and haematological parameters were analyzed. The hepato function markers including alanine aminotransferases, aspartate aminotransferases, alkaline phosphatase and gamma glutamyl transferase were significantly elevated in patients with both the syndrome, in comparison to patients with alcoholic dependence syndrome and healthy controls. The haemoglobin, total RBC, polymorphonuclear cells were significantly decreased in only alcoholic dependence syndrome group compared to controls and MCV and ESR were elevated in patients with alcoholic and nicotine dependence syndrome. Not much variation is observed between patients with alcoholic - nicotine dependence syndrome and alcoholic dependence syndrome only. The evidence of changes in the biochemical and hematological parameters in patients were observed only on alcohol dependent syndrome and clearly depicting that it acts as markers of alcoholism.

**Keywords:** Biochemical markers, hematological markers, alcohol and nicotine dependence syndrome.

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## INTRODUCTION

Alcohol based disorders are very closely associated with several medical co-morbidities that adversely affecting multi organs including hepatic, neurological, gastrointestinal, cardiovascular, reproductive systems etc [1,2]. Syndrome related to alcohol and nicotine dependence is clinically characterized by any three or more of the following have been present together including craving, loss of control on substance use, physiological withdrawal state, evidence of tolerance, preoccupation with substance use and persistent substance use despite having medical or psychotic morbidity [3].

The usage of alcohol is often associated with the concomitant abuse of other addictive substances including nicotine [4,5]. People who drink alcohol often also smoke and vice versa. Several mechanisms may contribute to concurrent alcohol and tobacco use that include genes involved in regulating certain brain chemical systems, neurobiological mechanisms including cross tolerance and cross sensitization to both drugs [6,7]; conditioning mechanisms, in which cravings for alcohol or nicotine are elicited by certain environmental cues and psychosocial factors such as personality characteristics and coexisting psychiatric disorders [8-10]. The treatment outcomes for patients addicted to both alcohol and nicotine are generally worse than for people addicted to only one drug and many treatment providers do not promote smoking cessation during alcoholism treatment. Recent findings suggested that concurrent treatment for both addictions may improve treatment outcomes.

A strong association was observed between alcoholism and tobacco consumption [11]. Among alcohol consuming persons, the usage of tobacco was found prevalent (80%) [12,13] and also found that smokers have increased risk for developing alcohol related disorders [14]; same like AIDS and tuberculosis. Some studies highlighted that 80% of alcohol consumers have smoking habit regularly and the mortality rate get increases due to smoking related disorders rather than alcohol related diseases. It is also well analyzed and defined that the tobacco and alcohol consumers have increased risk in upper digestive tract and respiratory tract carcinoma and also it was clearly stated that the lung dysfunction is increased due to alcohol consumption [15-17]. It was also suggested that cigarette smoking and alcohol interact to cause head and neck cancers [13].

Mortality related to cigarette smoking and alcohol consumption individually is very high, as an estimated 400,000 deaths from tobacco and 100,000 deaths from alcoholism reported annually and the role of tobacco in abstinent alcoholics is also found [13].

Recently, newer biomarkers of alcoholism and alcoholic liver disease related to multiorgan dysfunction (MOD) to multiorgan failure (MOF) were identified [18,19]. Globally, the analysis of correlation found among biochemical and haematological profile in persons with alcohol and nicotine dependent syndrome. Thus, this study was aimed to evaluate the biochemical and haematological changes in patients with alcohol and nicotine dependent syndrome in comparison to patients with alcohol dependent syndrome alone.

## MATERIALS AND METHODS

***Patient profile:*** The subjects included in this investigation were diagnosed primarily confirmed by clinical psychiatrist based on the international classification of mental and behavioural disorders, diagnostic criteria for research of WHO [20]. Further this work was approved by institutional research board and certified by institutional ethics committee. The patients included in this study were grouped into three

1. Group 1: Patients with alcohol dependence syndrome (ADS) only
2. Group 2: Patients with alcohol dependence syndrome (ADS) and nicotine dependence syndrome (NDS)
3. Group 3: Control persons without any syndrome (non smoker and non alcoholic)

In group 1 and 2, 50 patients confirmed to be fit for the criteria each group and 25 control persons were also included.

***Study area and criteria:*** The study was performed in the Department of Biochemistry, Ponniah Ramajayam Institute of Medical Sciences, Kacheepuram. Patients with observable syndrome of ADS + NDS and ADS alone were included and patients with systemic illness including cancer, inflammatory diseases, infections and nonalcoholic dependent liver diseases were excluded. The written informed consent was obtained from all the participants after explaining the purpose, potential risk and nature of study.

***Samples and assay:*** Blood samples were collected from all the participants including control groups with and without EDTA for various hematological and biochemical parameters respectively. All the serum samples were impregnated to biochemical assay including total protein [21], total albumin [22], alanine amino transferase [23], aspartate amino transferase [24], gaama glutamyl transferase

[24], total bilirubin [25], alkaline phosphotase [26], urea [27] and creatinine [28] were assayed. Hematological parameters including haemoglobin, total RBC count, polymorphonuclear cells, lymphocytes and other WBC cells, ESR, MCV.

All the biochemical reagents were purchased from Roche diagnostics and performed on Biochemistry auto analyzer (Logotech Pvt. Ltd, India). All the data obtained are statistically analyzed using SPSS package.

## RESULTS

***Demographic data:*** The average time of dependence for alcohol and alcohol + nicotine was 17.5 years and 18.5 years respectively. The time of alcohol consumption was categorized into low (10 and below 10 years), moderate (11 – 20 years) and high (above 20 years) exposures. The detailed description of consumption with appropriate years was described in detail in table 1.

***Biochemical parameters:*** No significant changes found in the serum proteins and albumin in control and ADS group but elevation was observed in both serum protein and albumin in ADS+NDS cases. In the case of serum bilirubin, moderate elevation was observed in ADS group and heavy increase up to 1.9mg/dL was observed in most of the cases. The other detailed description related to the biochemical evaluation and its comparison are documented in table 2.

The hemoglobin levels, RBC count and PMN were reduced more among ADS-NDS group compared to ADS alone. The eosinophils between control, ADS and ADS+NDS group were determined and the percentage of eosinophils increased in ADS-NDS group. There were no significant differences in any of the haematological parameters between ADS and ADS+NDS group. MCV and ESR were increased significantly. Hemoglobin, RBC count and PMN cells were decreased significantly in ADS groups compared to controls whereas the ADS+NDS and control group, lymphocytes was reduced significantly. Further it was noticed that there were no significance in ADS-NDS group compared to controls. The other parameters with its reference value, increased and decreased status were impregnated in table 3.

**Table 1: Characterization of subjects – age and group**

| Characteristics | Control (n=25) | ADS alone (n=50) | ADS+NDS (n=50) |
|---|---|---|---|
| Age (mean±SD) | 37±6.2 | 47.3±11.12 | 42.6±9.96 |
| ***Alcohol dependence group*** | | | |
| Low (10years and below) | - | 11 (22) | 13 (26) |
| Moderate (11 – 20 years) | - | 27 (54) | 22 (44) |
| High (above 20 years) | - | 12 (24) | 15 (30) |

[Figure in parenthesis denoted percentages]

**Table 2: Descriptive evaluation of biochemistry in ADS, ADS+NDS and control groups**

| Variables | Reference value | Control (n=25) | ADS (n=50) | ADS+NDS (n=50) |
|---|---|---|---|---|
| Total protein | 6-8 g/dL | 7.35 (6.8-7.9) | 6.9 (6.7-7.1) | 7.6 (7.3-7.9) |
| Total albumin | 3.5-5.5 g/dL | 4.75 (4.1-5.4) | 4.95 (4.6-5.3) | 4.9 (4.5-5.3) |
| Total bilirubin | <1 mg/dL | 0.61 (0.32-0.9) | 1.18 (0.41-1.96) | 1.15 (0.36-1.95) |
| Alkaline amino transferase | 0-40 IU/L | 18 (16-20) | 62.9 (32.5-93.4) | 60.5 (30.6-90.4) |
| Aspartate amino transferase | 7-40 IU/L | 20 (17-23) | 91.4 (43.6-139.2) | 77.9 (41.3-114.6) |
| Gamma glutamyl transferase | 0-50 IU/L | 22.5 (12-33) | 156.8 (62.1-251.6) | 152.5 (63.9-241.2) |
| Alkaline phosphatase | 50-160 U/L | 92.5 (59-126) | 90.8 (73-108.6) | 90.7 (74-107.4) |
| Urea | 20-40 mg/dL | 29 (27-31) | 23.4 (14.6-32.2) | 17.7 (13.9-21.6) |
| Creatinine | 0.6-1.3 mg/dL | 1.0 (0.9-1.1) | 0.76 (0.7-0.82) | 0.83 (0.76-0.91) |

Data are median (min-max); Groups are compared and p value <0.05 is taken statistical significant

**Table 3: Descriptive evaluation of haematological parameters in ADS, ADS+NDS and control groups**

| Variables | Reference value | Control (n=25) | ADS (n=50) | ADS+NDS (n=50) |
|---|---|---|---|---|
| Hb gm% | 13.5-17.5 (M)<br>12-16 (F) | 13.9 (12.6-15.3) | 12.3 (10.6-14.1) | 10.8 (9.3-12.4) |
| RBC ($X10^6$ cells/ cu.mm) | 4.3-5.7 (M)<br>3.8-5.1 (F) | 5.05 (4.7-5.4) | 4.35 (3.8-4.9) | 4.1 (3.6-4.7) |
| PMN % | 4.5-11 | 62.6 (60.2-65.1) | 51.3 (43.1-59.6) | 50.5 (42.6-58.4) |
| Lymphocytes % | 23-33 | 33 (32-34) | 27 (23-31) | 23.5 (20-27) |
| Neutrophils % | 57-67 | 51.5 (35-68) | 54.5 (53-56) | 75 (57-93) |
| Eosinophils (%) | 1-3 | 1.5 (0-3) | 1.5 (3-4) | 4.5 (3-6) |
| Monocytes (%) | 3-7 | 5.5 (4-7) | 3 (2-4) | 2 (1-3) |
| Basophils (%) | 0-1 | 0.5 (0-1) | 0.5 (0-1) | 0.5 (0-1) |
| ESR (mm/h) | <15mm/h (M)<br><20mm/h (F) | 12.5 (11-14) | 8 (4-12) | 7.5 (4-11) |
| MCV (fl) | 80-100 | 90 (84-96) | 97.5 (91-104) | 106 (96-116) |
| Platelet ($X10^3$/ µl) | 150-450 | 397.5 (359-436) | 283.5 (241-326) | 260.5 (211-310) |

Data are median (min-max); Groups are compared and p value <0.05 is taken statistical significant

## DISCUSSION

Alcoholism and smoking are the most serious socioeconomic and health issues globally cause biochemical, physiological and hematological changes. The concentrations of alcohol verses various health hazards depends on the variety of factors including consumption, age, time of day, diet, genetic predisposition and liver infections [29]. But in this study, we concentrated much on biochemical and hematological changes among the subjects not sociodemographic analysis including predisposing factors.

Alcohol problems, particularly in their early stages, are not always easy to detect. The signs and symptoms of alcohol problems often mimic those of other conditions; patients may be resistant to discussing their drinking and may not understand the connections between their drinking and the problems they are experiencing [30]. Thus a prerequisite analysis done with the doctors suggested that they are not much aware about the analysis of stages of alcohol problems.

Some of the harm associated with alcohol arises from acute intoxication, some from long-term heavy consumption. There is a range of medical, psychological and social problems associated with alcohol dependence. All types of problems exist in varying degrees of severity. Despite their advantageous situation it has been a common theme for many years that doctors often fail to identify alcohol problems. In a study including six hospitals, the false negative rate ranged from 25-91% [31]. The same type of information also collected in this study predicted to fail in collected the degrees of severity. There is a failure to anticipate alcohol problems and to ask about alcohol in routine examinations. Where an alcohol problem is identified it does not necessarily follow that any constructive intervention takes place. There is evidence that some doctors may prescribe doubtful appropriateness to problem drinking patients, most obviously anti-depressants [32].

By this study, it have been experienced with a strong desire or sense of compulsion to take the substance, difficulties in controlling behavior related to substance intake in terms of its onset, termination or levels of use, analysis of psycho-physiological during withdrawal symptoms, evidence of tolerance ie, increasing the doses, progressive motivation of alternative pleasures or interests because of the substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects, persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the internal organs, depressive mood states or impairment of cognitive functioning and narrowing of the personal repertoire or patterns of substance use has also been described as a characteristic feature, for example, a tendency to drink alcoholic drinks in the same way on weekdays and weekends regardless of social constraints that determine appropriate drinking behavior.

Nicotine dependence is consistently associated with increased neutrophil counts [33] leads to the risk for coronary events [34] that contribute to ischemic heart disease through the release of oxygen derived free radicals, proteases and leukotrienes [35]. These mediators can result in endothelial cell injury, aggregation and activation of platelets. The neutrophil count gets reduced after cessation of smoking [36,37]. This study also supported the same of increasing neutrophil count in most of the cases. Alcohol and nicotine dependency also produces hypercoagulable state that associated with platelet activation and other changes in clotting factors [38]. Enhanced viscosity is directly

contributed by increased fibrinogen levels and increased red cell mass [39]. This hyper coagulation condition induced the release of platelet factors that promote smooth muscle cell migration leads to atherosclerosis [40]. Compared to normal healthy controls, the platelet count also reduced significantly.

## CONCLUSION

It was concluded that the absolute requirement of biochemical and hematological parameters to detect the early stage of alcohol dependence for better treatment to avoid multi organ dysfunction (MOD) to multiorgan failure (MOF). However, improved medical training is a very important component of a better response to this major public health problem. There is now in existence a large body of knowledge on alcohol problems and effective methods of treatment. There is a consensus that new insights and interventions, both psychosocial and pharmaco-therapeutic, can contribute to an improved medical response to alcohol problems and its dependency syndromes.

## REFERENCES


1. Albano E. Oxidative mechanisms in the pathogensis of alcoholic liver disease. Mol Asp Med 2008; 29: 9-16.
2. Shivanand K et al. Medical comorbidities in patients with alcohol dependence – does nicotine matter? Int J Emerg Ment Hlth Hum Res 2010; 17: 616-7.
3. World Health Organization. Classification of Mental and behavioral disorders, 10th ed.; World Health Organization: Geneva, 1992.
4. Arnaout B, Petrakis IL. Diagnosing co-morbid drug use in patients with alcohol use disorders. Alc Abuse Alcohol 2008; 31: 148-54.
5. Patkar AA et al. Relationship between nicotine smoking and medical symptoms among cocaine, alcohol and opiate dependent patients. Am J Addict 2005; 14: 43-53.
6. Li TK. Pharmacogenetics of responses to alcohol and genes that influence alcohol drinking. J Stud Alcohol 2000; 61: 5-12.
7. Lopez MF et al. Alcohol tolerance and nicotine cross tolerance in adolescent mice. Addic Biol 2001; 6: 119-27.
8. Hoffman JH et al. Co-occurrence of alcohol and cigarette use among adolescents. Addic Beh 2001; 26: 63-78.
9. Carter BL, Tiffacny ST. Meta-analysis of cue-reactivity in addiction research. Addict 1999; 94: 327-40.
10. Bobo JK, Husten C. Sociocultural influences on smoking and drinking. Alc Res Hlth 2000; 24: 225-32.
11. Bein TH, Burge R. Smoking and Drinking: A review of the literature. Int J Addict 1990; 25: 263-70.
12. Bobo JK. Nicotine dependence and alcoholism epidemiology and treatment. J Psychoactive Drugs 1990; 24: 123-9.
13. Miller NS, Gold MS. Comorbid cigarette and alcohol addiction: epidemiology and treatment. J Addict Dis 1998; 17: 55-66.
14. Grant BF et al. Nicotine dependence and psychiatric disorders in the United States: results from the national epidemiologic survey on alcohol and related conditions. Arch Gen Psych 2004; 61: 1107-15.
15. Bagnardi V et al. Alcohol consumption and the risk of cancer: a meta-analysis. Alc Res Hlth 2001; 25: 263-70.
16. Korte JE et al.. Dose specific meta-analysis and sensitivity analysis of the relation between alcohol consumption and lung cancer risk. Am J Epidemiol 2002; 155: 496-506.
17. Strom K et al. Alcohol, smoking and lung disease. Respr 1996; 63: 66-72.
18. Das SK et al. Biomarkers of alcoholism: an updated review. Scand J Clin Lab Invest 2008; 68: 81-92.
19. Honnamurthy JB et al.. Biochemical and haematological profile in patients with alcohol dependence syndrome (ADS) co-morbid with nicotine dependence syndrome (NDS). Int J Biochem Res Rev 2016; 13: 1-10.
20. Skinner SA, Allen BA. Alcohol dependence syndrome: measurement and validation. J Abnor Psychol 1998; 91: 199-209.
21. Gornall AG et al. Determination of serum proteins by means of the Biuret reaction. J Biol Chem 1949; 177: 751-66.
22. Doumas BT, Peters T. Serum and urine albumin: a progress report on their measurement and clinical significance. Clin Chim Acta 1997; 58: 3-20.
23. Bergmeyer HU et al. Approved recommendations on IFCC methods for the measurement of catalytic concentration of enzymes: part 1: Alanine Aminotransfereas. J Clin Chem Clin Biochem 1985a; 24: 481-95.

24. Bergmeyer HU et al. Approved recommendations on IFCC methods for the measurement of catalytic concentration of enzymes: part 2: Aspartate Aminotransfereas. J Clin Chem Clin Biochem 1985b; 24: 497-510.
25. Pearlman FC, Lee RTY. Detection and measurement of total bilirubin in serum, with use of surfactants as solubilizing agents. Clin Chem 1974; 20: 447-53.
26. Robert BM, George NBJ. Assay of alkaline phosphatase. Clin Chem 1972; 18: 97-104.
27. Bretaudiere JP et al. Direct enzymatic determination of urea in plasma and urine with a centrifugal analyzer. Clin Chem 1976; 22: 1614-7.
28. Larsen K. Creatinine assay by a reaction kinetic principle. Clin Chim Acta 1972; 41: 209-17.
29. Oduola T et al. Drinking patterns; biochemical and haematological findings in alcohol consumers in Ile-Ife, Nigeria. Afr J Biotechnol 2015; 4: 1304-8.
30. Walsh RA et al. Smoking cessation interventions in Australian drug treatment agencies: a national survey of attitudes and practices. Drug Alc Rev 2005; 24: 235-44.
31. Dawson NV et al. The effect of patient gender on the prevalence and recognition of alcoholism on a general medicine inpatient service. J Gen Intern Med 1992; 7: 38-45.
32. Deehan A et al. How do general practitioners manage alcohol misusing patients? Results from a national survey of GPs in England and Wales. Drug Alc Rev 1998; 17: 259-66.
33. Petitti DB, Kipp H. The leucocyte count: association with intensity of smoking and persistence of effect after quitting. Am J Epidemiol 1986; 123: 889-95.
34. Grimm RH et al. Prognsotic importance of the white blood cell count for coronary, cancer and all cause mortality. JAMA 1985; 254: 1932-7.
35. Hansen PR. Role of neutrophils in myocardial ischemia and reperfusion. Circul 1995; 91: 1872-85.
36. Bain BJ et al. Acute Changes in haematological parameters on cessation of smoking. J Royal Soc Med 1992; 85: 80-2.
37. Neal LB, Steven GG. Cardiovascular toxicity of nicotine: implications for nicotine replacement therapy. J Am College Cardiol 1997; 29: 1422-31.
38. FitzGerald GA et al. Cigarette smoking and hemostatic functions. Am Heart J 1998; 115: 267-71.
39. Ernst E et al. The measurement of plasma and blood viscosity. Brit J Haematol 1990; 15: 84-8. .
40. Fuster V et al. The pathogenesis of coronary artery diseases and the acute coronary syndromes New Engl J Med 1992; 326: 242-50.