

# A Study of Arterial Blood Gas (ABG) Values in Liver Cirrhosis and Ascites.

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## ABSTRACT

**Background:** In cirrhotic patients, in addition to hepatocyte and Kupffer cells dysfunction, portopulmonary shunt, intrapulmonary arteriovenous shunt and  $V_A/Q$  inequality can induce a decrease in  $PaO_2$  and  $SaO_2$  as well as acid base disturbances. The present study was done to analyse arterial blood gas changes, prevalence of hypoxemia and acid base disturbances as well as to correlate grading of hypoxemia with different aetiologies and Child-Pugh score in patients with liver cirrhosis and ascites. **Methods:** The present correlative cross sectional study was conducted on 100 patients with cirrhosis and ascites for a period of 24 months. Arterial blood gas samples obtained by percutaneous radial puncture were analysed for various acid base abnormalities and arterial blood gas oxygenation. **Results:** Acid base disturbances observed were: respiratory alkalosis in 39 cases (39%), metabolic alkalosis in 20 cases (20%), metabolic acidosis in 11 cases (11%), metabolic acidosis with respiratory alkalosis in 10 cases (10%) and no acid base disturbance in 20 cases (20%). Mean values of  $PaO_2$  was  $75.85 \pm 7.8$  mmHg,  $PaCO_2$  was  $35.27 \pm 5.13$  mmHg, pH was  $7.44 \pm 0.115$  and  $HCO_3^-$  was  $23.65 \pm 3.85$  mmol/l. Alcoholic cirrhotics had hypoxemia in 42% cases in contrast to hypoxemia in other aetiologies (Hepatitis C 18%, Hepatitis B 5%). **Conclusion:** Metabolic abnormalities, hypoxemia and hypocapnia are commonly found in cirrhotics. Hypoxemia is more common in alcoholic cirrhotics but has no correlation with Child-Pugh score.

**Keywords:** Arterial blood gas; cirrhosis; hypoxemia; ascites

## INTRODUCTION

Cirrhosis of liver is diffuse hepatic fibrosis where normal liver architecture is replaced by nodules.<sup>[1]</sup> There are various causes of liver disease leading to cirrhosis eg. alcoholic liver disease, hepatitis, non alcoholic liver disease etc. Cirrhosis leads to a variety of complications as variceal haemorrhage, ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepato-cellular carcinoma, hepatorenal syndrome, hepato-pulmonary syndrome (HPS), portopulmonary syndrome etc.<sup>[3]</sup> Hepato-pulmonary syndrome is characterised by a triad of liver disease, impaired oxygenation, Intrapulmonary vascular abnormalities known as intrapulmonary vascular dilatations (IPVDs).<sup>[4]</sup>

Patient present with dyspnea, platypnea, orthodeoxia and hypoxia is also a common but non-specific finding.<sup>[5]</sup> HPS-related hypoxemia is due to intrapulmonary dilatations (IPVDs).<sup>[6]</sup> Shunting through these collaterals leads to ventilation-perfusion mismatch and oxygen diffusion limitation. Commonly used laboratory investigations to assess liver function are enzyme assay like serum glutamic-oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphate (ALP), gamma-glutamyl transferase (GGT), serum bilirubin, serum albumin, prothrombin time, ultrasonography, transient elastography.<sup>[2]</sup> The diagnosis of hepatopulmonary syndrome (HPS) is made with impaired oxygenation with pulse oximetry level  $<96\%$  and detection of impaired oxygenation status requires arterial blood gas analysis. The most sensitive measure of impaired oxygenation is an elevated alveolar-arterial (A-a) oxygen gradient, defined as  $\geq 15$  mmHg when breathing room air.<sup>[7]</sup> An arterial oxygen tension ( $PaO_2$ ) of  $<80$  mmHg also indicates impaired oxygenation. The right-to left shunt fraction is a measure of the degree to which oxygenation is impaired.<sup>[8]</sup> PPH (portopulmonary hypertension) is

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defined as a mean pulmonary artery pressure (MPAP) 25mmHg associated with pulmonary vascular resistance (PVR) 240 dyn/s/cm<sub>5</sub> and pulmonary artery wedge pressure (PAWP) <15mmHg. The presence of HPS or POPH increases mortality in affected patients. Arterial blood gas (ABG) analysis is a test that measures the oxygen tension (PaO<sub>2</sub>), carbon dioxide tension (PaCO<sub>2</sub>), acidity (pH), oxyhemoglobin saturation (SaO<sub>2</sub>), and bicarbonate (HCO<sub>3</sub>) concentration in the arterial blood.

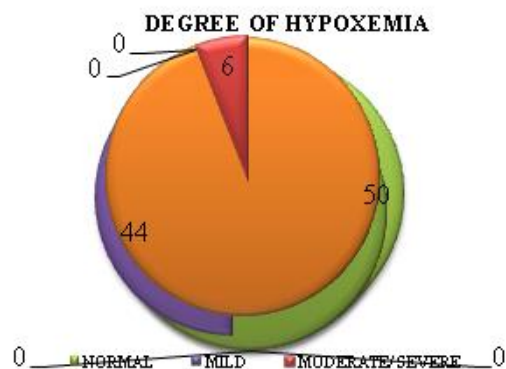
### MATERIALS AND METHODS

After the approval of ethical committee, the present correlative cross sectional study was conducted on 100 patients of age 17-72 years of either sex admitted to Guru Nanak Dev Hospital, Amritsar with cirrhosis and ascites for a period of 24 months and informed consent was taken. Cirrhosis was diagnosed by history, clinical examination, laboratory findings, and liver biopsy. From a clinical point of view the patients' conditions varied from moderate to severe according to the Child-Pugh classification (B, C class). Partial pressure of oxygen in arterial blood, oxygen saturation of haemoglobin as well as various acid base disturbances were determined in all patients. Arterial blood gas samples were obtained by percutaneous radial puncture with subject in semi-recumbent position breathing room air, and analysed in standard blood gas analyser (Cobas b 221 analyser and Nova Stat profile pHox Ultra). Those excluded from the study were patients with coexisting primary pulmonary pathology, coexisting intrinsic heart disease and active smokers. The aim of the study was to analyse arterial blood gas changes in liver cirrhosis and ascites, to determine the prevalence of hypoxemia among patients with liver cirrhosis and ascites, to correlate various etiologies and Child Pugh score with hypoxemia in liver cirrhosis and ascites. The data so collected was analysed by frequency, percentage, mean standard deviation (S.D), t test and chi-square test.

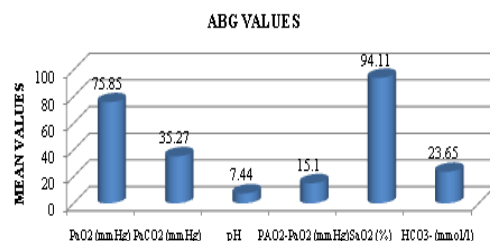
### RESULTS

The present study was a Correlative cross sectional study carried out on 100 patients with mean age of onset of cirrhosis 46.43±9.92 years and with sex distribution of M:F was 81:19 and Child Pugh score of B (31%) and C (69%). The study was done to analyse arterial blood gas changes in patients with liver cirrhosis and ascites for a period of 24 months admitted to Guru Nanak Dev Hospital, Amritsar. Patients presented with symptoms of abdominal distension (100%), haematemesis/ malena (55%), altered sensorium (65%), fever (26%), abdominal pain (30%), dyspnea (10%), platypnea (6%) and

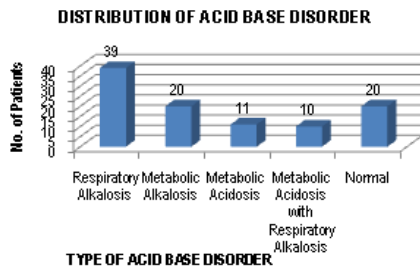
signs jaundice (67%), ascites (100%), cyanosis (25%), clubbing (25%), spider nevi (41%), asterixis (50%) and other signs (51%). The distribution of hypoxia with 50% patients was normal, 44% patients had mild hypoxia and 6% had moderate to severe hypoxia. Patients had various etiological factors like alcohol in 79%, hepatitis C in 37%, hepatitis B in 13% and others in 7% patients. Serum electrolytes level were serum sodium 129.39±4.47, serum chloride 99.74±1.93, serum potassium 5.06±.496. Patients had arterial blood gas parameters as PaO<sub>2</sub> 75.85±7.8 mmHg, PaCO<sub>2</sub> 35.27±5.13 mmHg, pH 7.44±.115, PAO<sub>2</sub>-PaO<sub>2</sub> gradient 15.10±5.78 mmHg, SaO<sub>2</sub> 94.11±5.15 % and HCO<sub>3</sub>. 23.65±65 mmol/l. Acid base disorder distribution was found as respiratory alkalosis in 39%, metabolic alkalosis in 20%, metabolic acidosis in 11%, metabolic acidosis with respiratory alkalosis in 10% patients. The presence of hypoxemia in various aetiologies was as in alcoholic patients 36% normal, 37% had mild and 6% had moderate to severe hypoxia. Hepatitis C patients 19% were normal, 17% with mild, 1% with moderate to severe hypoxia. Patients with hepatitis B had 8% normal, 5% had mild hypoxia, in other aetiology 3% normal, 3% with mild, 1% with moderate to severe hypoxia. The distribution of hypoxemia according to Child Pugh score was as in patients with Child Pugh score B 17% normal, 10% had mild, 4% had moderate to severe hypoxia and in score C 33% normal, 34% had mild, 2% had moderate to severe hypoxia. Results had been shown in graphs 1,2,3,4,5.



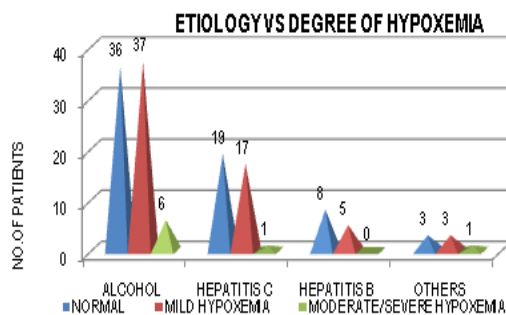
Graph 1: Showing the distribution of hypoxia in patients with cirrhosis



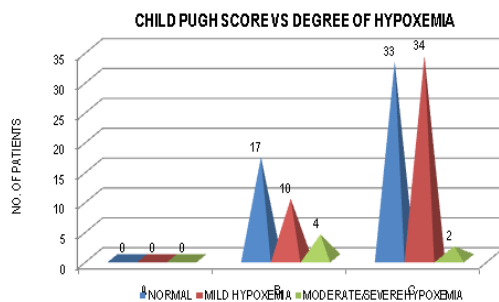
Graph 2: Showing the mean values of arterial blood gas parameters in cirrhotic patients



Graph 3: Showing the Distribution of Acid Base Disorder in Cirrhotic Patients:



Graph 4: Showing the Grading Of Hypoxemia In Patients with Different Etiologies



Graph 5: Showing the Grading of Hypoxemia in Patients with different Child Pugh Score

## DISCUSSION

Cirrhosis represents a late stage of progressive hepatic fibrosis characterized by distortion of the hepatic architecture and the formation of regenerative nodules. It is generally considered to be irreversible with only treatment option being liver transplantation. Ascites is defined as the accumulation of fluid in the peritoneal cavity resulting from liver cirrhosis. In patients suffering from severe liver cirrhosis and ascites, portopulmonary shunt, intrapulmonary shunt and VA/Q inequality may induce a decrease in PaO<sub>2</sub> and SaO<sub>2</sub> in association with various acid-base disturbances. In the present study mean age of cirrhotic presentation was 46.43 years as shown in the study done by Kamal A et al<sup>[6]</sup> with male dominance 81% similar in the study done by Ordiales Fernandez JJ et al,<sup>[9]</sup> symptomatic

presentation of abdominal distension in 100%, jaundice in 67%, haematemesis and malena in 55% patients which was in harmony with study by R Maskey et al<sup>[10]</sup> and Lorenzo-Zunga et al.<sup>[11]</sup> Alcohol consumption was the most common etiological agent found in the present study similar in the study conducted by Singh C et al.<sup>[12]</sup> However in the studies done by Kamal A et al,<sup>[6]</sup> Schenk P et al,<sup>[13]</sup> hepatitis C was the most common cause of cirrhosis, which accounted for only 37% cases of cirrhosis in our study. Hepatitis B was uncommon cause (13%) in our study which is in tune with the study done by Singh C et al.<sup>[12]</sup> Alcohol was the most common etiological factor in the present study due to increased prevalence of alcohol consumption in the northern part of India i.e. Punjab. Also cirrhosis and its complications occur at a much earlier stage than it occurs with hepatitis B and hepatitis C. Also there was considerable overlap between different aetiologies due to increased incidence of I.V drug abusers in Punjab which predisposes them to hepatitis B and hepatitis C infection. In the present study, grading for advanced cirrhosis was done using Child-Pugh grade and most of the patients were categorised into Child-Pugh grade C group and Child Pugh grade B group and none of the patient was in Child-Pugh grade A group similar results in studies done by Schenk P<sup>[13]</sup>, Lorenzo-Zunga et al<sup>[11]</sup>, Funk GC et al<sup>[14]</sup>. Our study has electrolyte imbalance in the form of hyponatremia (mean 129.29±3.47) with normokalemia (5.06±.496) were the most common findings in accordance with similar results in the studies done by Papper S,<sup>[15]</sup> and Papadakis MA et al<sup>[16]</sup>. The results of arterial blood gas analysis in our study were: Respiratory alkalosis (39%), metabolic alkalosis (20%), metabolic acidosis (11%), metabolic acidosis with respiratory alkalosis (10%), normal (20%) similar results shown by Charalabopoulos K et al<sup>[17]</sup> and Funk GC et al<sup>[14]</sup>. Respiratory alkalosis can be explained by direct stimulation of respiratory centre by retained amines or due to other humoral factors like progesterone which is a respiratory stimulant, whereas metabolic alkalosis is mainly seen in the patients on loop diuretics causing hypovolemia induced secondary hyperaldosteronism which further lead on to hypokalemia and alkalemia.<sup>[17]</sup> Metabolic acidosis alone or in combination with respiratory alkalosis is seen in patients with advanced stages of liver disease or fulminant hepatic failure due to inability to metabolise excess lactate, increased production of hydrogen ions as explained by Oster JR et al.<sup>[18]</sup> The present study results on arterial oxygen saturation (PaO<sub>2</sub>) showed normal (50%), mild hypoxemia (44%), moderate to severe hypoxemia (6%) with an average hypoxemia of 75.85 mmHg which is statistically highly significant (p value<<.001) and similar results have been shown in study done by Charalabopoulos K et al,<sup>[17]</sup> Moller S et al,<sup>[19]</sup> Lorenzo-Zunga et al<sup>[11]</sup>.

Hypocapnea is a frequent finding in our study which is similar to that observed by Charalabopoulos K et al.<sup>[17]</sup> The degree of hypoxemia is not related to severity of liver disease (described in terms of Child-Pugh scoring) which is consistent with the results concluded by Lorenzo-Zunga et al,<sup>[11]</sup> Maruyama S1et al<sup>[20]</sup> and Charalabopoulos K et al.<sup>[17]</sup> Hypoxia was more common (79%) and severe in alcoholic group when compared with cirrhosis due to other aetiologies (p value<.001) and the similar results have been depicted by Charalabopoulos K et al<sup>[17]</sup> and Maruyama S1et al.<sup>[20]</sup> This can be explained by an increased oxygen demand due to excess alcohol consumption leading to hyperdynamic and hyper-metabolic state. Dyspnoea, clubbing and spider naevi usually accompanied moderate to severe hypoxemia which can be explained by the development of HPS in these subset of patients and similar findings have been suggested by Singh C et al.<sup>[12]</sup>

Arterial venous anastomosis and communications between the portal and arterial circulation as well as between bronchial and pulmonary veins, portopulmonary shunts and  $V_A/Q$  inequality are more likely to be functional in patients with cirrhosis and contribute to hypoxemia.<sup>[17]</sup>

## CONCLUSION

In the present study, it has been concluded that arterial blood gas abnormalities in patients with liver cirrhosis and ascites include various metabolic abnormalities, hypoxemia and hypocapnoea. In addition, hypoxemia can be used for grading severity of HPS and its subsequent prognosis and management in the form of liver transplantation.

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