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A STUDY OF ARTERIAL BLOOD GAS ANALYSIS IN PATIENTS WITH CIRRHOSIS OF LIVER AT A TERTIARY CARE CENTRE

Medical Science

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ABSTRACT

Background: Liver cirrhosis has become one of the major causes of morbidity and mortality. Patients with liver cirrhosis are known to develop hepatopulmonary syndrome.The liver is an important organ in acid base physiology. The objective of the study is to analyse arterial blood gas changes in cirrhosis of liver and to correlate arterial blood gas changes with severity of Child Pugh's scoring of chronic liver disease. **Methods:** Cross sectional study of 140 Patients admitted to hospitals attached to Bangalore Medical College and Research Institute from March 2022 to March 2023. Haemotological tests, ABG and Ultrasonography,LFT,PT and INR were done, Child- Pugh scores, were calculated and the data analyzed. **Results:** We noted that as the Child Pugh score increased mixed acid base disorders and respiratory alkalosis increased. Acid base disorders are less frequent in patients with Child Pugh Class A compared to patients in Child Pugh Class C. **Conclusion:** We concluded that acid base disturbances are common in cirrhotic cases, initially the acid base disturbances are most often mixed acid base disorder then they become metabolic acidosis then compensated metabolic acidosis and then respiratory alkalosis is the abnormality. These acid base disorders are predominantly observed in Child Pugh Class C more than Child Pugh Class A

KEYWORDS

Liver cirrhosis ; Acid base disorders; Child Pugh Class

INTRODUCTION

Liver cirrhosis has become one of the major causes of morbidity and mortality. The global burden of disease (GBD) reported that over 1 million people died due to cirrhosis in 2010 worldwide compared with 676,000 deaths in 1980[1]

Liver cirrhosis is defined as a condition marked by the diffuse transformation of the entire liver into regenerative parenchymal nodules surrounded by fibrous bands and variable degrees of vascular shunting[2]

Cirrhosis is usually irreversible. But when the underlying cause is removed, fibrosis can be reversed. This can be seen in chronic hepatitis C, hemochromatosis and alcohol liver disease.[3,4]

Compensated chronic liver disease refers to the progressive destruction of the liver parenchyma over a period extending more than six months which in turn leads to fibrosis and cirrhosis[5].

Decompensated chronic liver disease refers to the progressive destruction of the liver parenchyma over a period extending more than six months with decompensation due to various causes in the form of any of the following complications[6]

- Oesophageal variceal bleed
- Ascites
- Spontaneous bacterial peritonitis
- Hepatic encephalopathy
- Hepatorenal syndrome

In cirrhotic patients in addition to hepatocyte and kupffer cells dysfunction, circulatory anatomic shunt and ventilation/ perfusion (VA/Q)ratio abnormalities can induce decrease in partial pressure of oxygen in arterial blood PaO2 and SaO2 as well as various acid-base disturbances.As a result pulmonary resistance is impaired and patients are more likely to succumb to infections and adult respiratory distress syndrome[7]

Patients with liver cirrhosis are known to develop hepatopulmonary syndrome.The condition is characterized by resistant hypoxaemia (PaO2<70mmHg or 9.3kPa) and intrapulmonary vascular dilatation in patients with cirrhosis[8]

The liver is an important organ in acid-base physiology.It is metabolically active organ which may be either a significant net producer or consumer of hydrogen ions. The acid-base roles of the liver include:carbon dioxide production from complete oxidation of substrates,metabolism of organic acids anions such as lactate,ketone and amino acids.The conversion of ammonium(NH4) to urea in the liver,synthesis of plasma proteins:except immunoglobulins[9]

Acid base disturbances occur frequently in the setting of liver diseases.

As liver's metabolic function worsen particularly in the setting of renal dysfunction haemodynamic compromise, hepatic encephalopathy and acid base disorders ensue. Hence this study has been undertaken to assess acid base disturbances in patients with liver cirrhosis.

Liver Failure And The Child-pugh Classification[10]

The final stage of chronic inflammation in the liver is cirrhosis. Child´s grade is used to assess hepato-cellular function in cirrhosis based on these factors.

The Child-Pugh classification is a modified grading system shown to be reliable in predicting survival of patients presenting with variceal bleedings but is also widely used as a method of assessing liver function.

The Child-Pugh score: Prognosis in chronic liver disease and cirrhosis[10]

Child-turcotte-pugh Classification For Severity Of Cirrhosis

Clinical and Lab criteria	Points*		
	1	2	3
Encephalopathy	None	Garde 1 or 2	Grade 3 or 4
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Billirubin(mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time	<4	4-6	>6
	<1.7	1.7-2.3	>2.3
*Child-Turcotte-Pugh class obtained by adding score for each parameter(total points)			
Class A= 5 to 6 points			
Class B= 7 to 9 points			
Class C= 10 to 15 points			

The Child-Pugh classification system correlates with survival in

patients, oneyear survival rates for patients with Child-Pugh class A, B, and C cirrhosis are approximately 100, 80, and 45 percent, respectively.

Objectives of the Study

- To analyze arterial blood gas changes in cirrhosis of liver.
- To correlate arterial blood gas changes with severity of Child Pugh's scoring of chronic liver disease.

Methodology

Study Design: Cross sectional study.
Study Period: March 2022 to March 2023
Place Of Study: Hospitals attached to BMCRI.
Sample Size: 140

Inclusion Criteria

1. Patient willing to give informed consent
2. Both sexes of age >18 years.
3. Cirrhosis of liver diagnosed by history, laboratory and ultrasonological Features Suggestive of cirrhosis of liver.

Exclusion Criteria

1. Patient not willing to give informed consent
2. Patients with coexisting primary pulmonary pathology.
3. Patients with pulmonary hypertension.
4. Patients with cardiovascular diseases.
5. Smokers.

Methodology

After obtaining approval and clearance from the institutional ethics committee, the patients fulfilling the inclusion criteria were enrolled for the study after obtaining informed consent.The study is a observational cross sectional study which was conducted between March 2022 and March 2023.Data was collected and analyzed for all the patients satisfying the inclusion and exclusion criteria.

Assessment Tools

History, physical examination to detect features of cirrhosis like signs of liver cell failure, ascites, splenomegaly, peripheral edema.

Hematological tests include Complete Blood Count, Liver Function Test, Prothrombin Time.

Arterial Blood Gas samples were obtained by percutaneous radial puncture with the subject in semi recumbent position breathing room air and analyzed in standard blood gas analyzer.

Partial pressure of oxygen in arterial blood, oxygen saturation of haemoglobin as well as various acid base disturbances were determined in all patients.

Hypoxemia is indicated by PaO2<80mmHg and Alveolar-arterial gradient of oxygen [A-a PO2] ≥20mmHg.Alveolar–arterial pressure gradient of O2 is calculated as P(Aa)O2=PaO2-150-1.25xPaCO2.

Liver disease was staged according to Child Pugh's scoring.

Ultrasonography was done for all patients to look for size and altered echotexture of liver,presence of ascites.

All patients were tested for Hepatitis B and Hepatitis C, 2D ECHO and Chest X-Ray.

Securing Blood Sample

After drawing the arterial blood it is transported in a heparinized syringe. This ensures that sample is fresh blood and the sample is immediately subjected to analysis.

Statistical Analysis

The data collected was entered in excel spread sheet.Descriptive statistics like mean, standard deviation were calculated.

Inferential statistics like chi-square test were calculated.

Analysis was done using SPPS version 19.

Sample Size Calculations

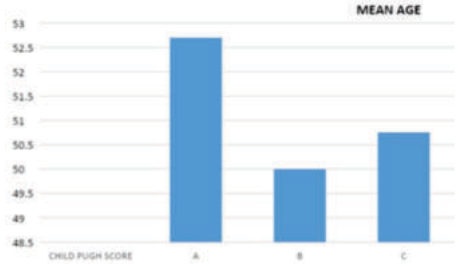
Total sample size:140

M	Your guess of Population M	82.46
S	Standard deviation of M	11.74
1-a	Set level of confidence(value<1.0)	0.95
Z1	Z value associated with confidence	1.96
d	Absolute precision (=Value<M)	2
n	Minimum sample size	140

RESULTS AND ANALYSIS

Table 1: Mean Age

CHILD PUGH SCORE	MEAN AGE
CHILD PUGH SCORE A	52.70 years
CHILD PUGH SCORE B	50.00 years
CHILD PUGH SCORE C	50.75 years

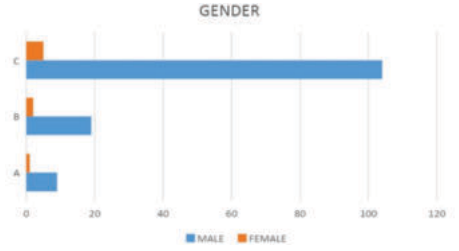


Graph 1: Mean Age

In CHILD PUGH SCORE A the mean age was 52.70 years, in CHILD PUGH SCORE B mean age was 50.00 years, in CHILD PUGH SCORE C mean age was 50.75 years

Table 2: Gender

GENDER	MALE	FE-MALE	TOTAL	MALE	FE-MALE	TOTAL
CHILD PUGH SCORE A	9	1	10.00	6.43%	0.71%	7.14%
CHILD PUGH SCORE B	19	2	21.00	13.57%	1.43%	15.00%
CHILD PUGH SCORE C	104	5	109.00	74.29%	3.57%	77.86%

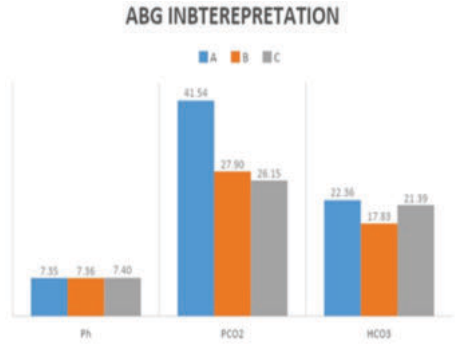


Graph 2: Gender

In the present study we had 132 (94.28 %) males and 8 females (5.71 S%).

Table 3: ABG Interpretation

ABG INTERPRETATION	Ph	PCO2	HCO3	PO2
CHILD PUGH SCORE A	7.35	41.54	22.36	83.60
CHILD PUGH SCORE B	7.36	27.90	17.83	68.19
CHILD PUGH SCORE C	7.40	26.15	21.39	68.27

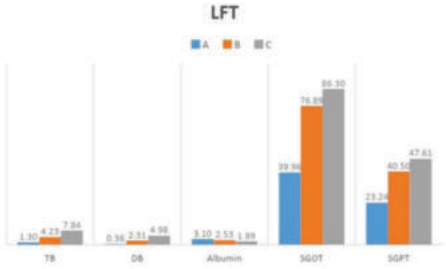


Graph 3: ABG Interpretation

The mean pH was 7.35, 7.36, and 7.40 in the Child pugh score A, Child pugh score B group and Child pugh score C, the mean PCO2 was 41.54, 27.90, 26.15 in the Child pugh score A, Child pugh score B group and Child Pugh Score C. The mean HCO3 levels were 22.36, 17.83 and 21.39. the mean PO2 was 83.60 68.19 and 68.27 in the Child pugh score A, Child pugh score B group and Child pugh score C.

Table 4: LFT

LFT	TB	DB	Albumin	SGOT	SGPT
CHILD PUGH SCORE A	1.30	0.36	3.10	39.96	23.24
CHILD PUGH SCORE B	4.23	2.31	2.53	76.89	40.50
CHILD PUGH SCORE C	7.84	4.98	1.89	86.30	47.61
P Value	0.02	0.02	0.044	0.02	0.02



Graph 4: LFT

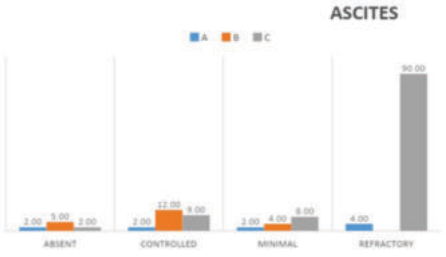
The mean total bilirubin was 1.30 mg/dl, 4.23 mg/dl, and 7.84 mg/dl in the CHILD PUGH SCORE A, CHILD PUGH SCORE B group and CHILD PUGH SCORE C,there was a statically significant difference in the grades of CHILD PUGH SCORE and the bilirubin was statistically significant with a p value 0.02, the correlation was also positive as the grade of CHILD PUGH SCORE increased the bilirubin concentration also increased.

The mean total albumin was 3.10 mg/dl, 2.53 mg/dl, and 1.89 mg/dl in the CHILD PUGH SCORE A, CHILD PUGH SCORE B group and CHILD PUGH SCORE C respectively there was a statically significant difference in the grades of CHILD PUGH SCORE and the albumin was statistically significant with a p value 0.044, the correlation was negative as the grade of CHILD PUGH SCORE increased the albumin concentration also decreased.

There was a statically significant difference in the grades of CHILD PUGH SCORE and the liver enzymes which was statistically significant with a p value 0.02, the correlation was also positive as the grade of CHILD PUGH SCORE with liver enzymes increased the liver enzymes concentration also increased.

Table 5: Ascites

ASCITES	Child Pugh Score A	Child Pugh Score B	Child Pugh Score C	Child Pugh Score A	Child Pugh Score B	Child Pugh Score C
ABSENT	2.00	5.00	2.00	1.43%	3.57%	1.43%
CONTROLLED	2.00	12.00	9.00	1.43%	8.57%	6.43%
MINIMAL	2.00	4.00	8.00	1.43%	2.86%	5.71%
REFRACTORY	4.00	0.00	90.00	2.86%	0.00%	64.29%
TOTAL	10	21	109	7.14%	15.00%	77.86%

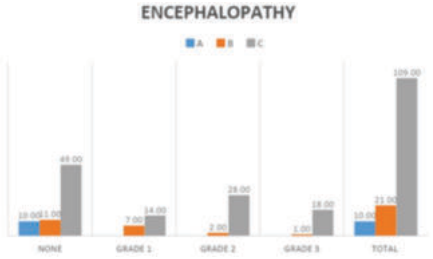


Graph 5: Ascites

No ascites was seen in 2.00 (1.43%), 5.00 (3.57%) 2.00 (1.43%) in the Child pugh score A, Child pugh score B group and Child pugh score C respectively. Controlled ascites was seen in 2.00 (1.43%), 12.00 (8.57%) 9.00 (6.43%) in the Child pugh score A, Child pugh score B group and Child pugh score C respectively. Minimal ascites was seen in 2.00 (1.43%), 4.00 (2.86%) 8.00 (5.71%) in the Child pugh score A, Child pugh score B group and Child pugh score C respectively. Refractory ascites was seen in 4.00 (2.86%) 90.00 (64.29%) in the Child pugh score A, Child Pugh score B group and Child pugh score C respectively.

Table 6: Encephalopathy

ENCEPHA-LOPATHY	CHILD PUGH SCORE A	CHILD PUGH SCORE B	CHILD PUGH SCORE C	CHILD PUGH SCORE A	CHILD PUGH SCORE B	CHILD PUGH SCORE C
NONE	10.00	11.00	49.00	7.14%	7.86%	35.00%
GRADE 1	0.00	7.00	14.00	0.00%	5.00%	10.00%
GRADE 2	0.00	2.00	28.00	0.00%	1.43%	20.00%
GRADE 3	0.00	1.00	18.00	0.00%	0.71%	12.86%
TOTAL	10.00	21.00	109.00	7.14%	15.00%	77.86%

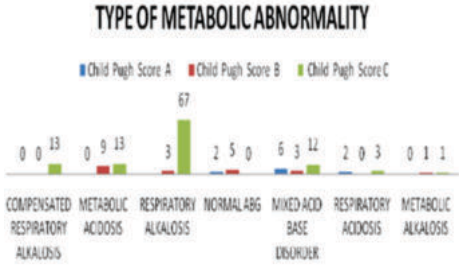


Graph 6: Encephalopathy

Encephalopathy was not seen in 10 00 (7.14% %), 11.00 (7.86%), 49.00 (35 %) in the Child pugh score A, Child pugh score B, group and Child pugh score C respectively. Grade 1 encephalopathy was seen in 7.00 (5%), 14 cases (10.00%) in the Child pugh score B group and Child pugh score C respectively. Grade 2 encephalopathy was seen in 2 (1.43%), 28 cases (20.0 %) in the Child pugh score B group and Child pugh score C respectively. Grade 3 encephalopathy was seen in 1 (0.71%), 18 cases (12.86%) in the Child pugh score B group and Child pugh score C respectively.

Table 7: Type Of Metabolic Abnormality

TYPE OF METABOLIC ABNORMALITY	CHS A	CHS B	CHS C	Total	CHS A	CHS B	Total
COMPENSATED RESPIRATORY ALKALOSIS	0	0	13	13	0.00	0.00	9.29
METABOLIC ACIDOSIS	0	9	13	22	0.00	6.43	9.29
RESPIRATORY ALKALOSIS		3	67	70	0.00	2.14	47. 86
NORMAL ABG	2	5	0	7	1.43	3.57	0.00
MIXED ACID BASE DISORDER	6	3	12	21	4.29	2.14	8.57
RESPIRATORY ACIDOSIS	2	0	3	5	1.43	0.00	2.14
METABOLIC ALKALOSIS	0	1	1	2	0.00	0.71	0.71
total	10	21	109	140	7.14	15.00	77.86

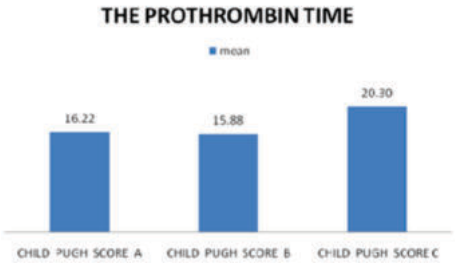


Graph 7: Type Of Metabolic Abnormality

We noted as the grade of child pugh score increased mixed acid base disorder and respiratory alkalosis increased. Acid base disorders are less frequent in patients with Child Pugh Class A compared to patients in Child Pugh Class C.

Table 8: The Prothrombin Time

PT	CHILD PUGH SCORE A	CHILD PUGH SCORE B	CHILD PUGH SCORE C
mean	16.22	15.88	20.30
SD	5.55	6.35	7.24



Graph 8: The Prothrombin Time

The prothrombin time in Child Pugh Score A group 16.22 sec was Child Pugh Score B group was 15.88 sec, Child Pugh Score C group was 20.30 sec.

DISCUSSION

Oxygen saturation of arterial blood in normal man at rest is remarkably constant. Though subnormal arterial concentrations can be traced to obvious pulmonary or cardiac disorders, it is also noted that relative arterial unsaturation is common among patients with chronic liver disease. In such cases, ascites and oedema did not appear to account for decreased saturation and its pointed out that in these case hypoxemia must have resulted due to interference with gas exchange in lungs. Mild hypoxia has been seen in approximately one third of patients with chronic liver disease.

Development of hypoxemia in patients with chronic liver disease, modifies the line of management and worsens the prognosis of disease. Its early detection, if it could be corrected can be helpful in preventing complications. Hence an early detection of hypoxemia in these patients is essential. The easiest way to detect hypoxemia in such patients is by arterial blood gas analysis. Hence this study intends to analyse arterial blood gas changes in chronic liver disease with their clinical correlation.

CHILD PUGHS CLASS	pH	Frequency	Percent
A	LOW	54	38.57%
B	NORMAL	54	38.57%
C	HIGH	32	22.86%
	TOTAL	140	100.00%

CHILD PUGHS CLASS	PaCO2	Frequency	Percent
A	LOW	128	91.43%
B	NORMAL 35 to 45	1	0.71%
C	HIGH	11	7.86%
	TOTAL	140	100.00%

CHILD PUGHS CLASS	HCO3	Frequency	Percent
A	LOW	68	48.57%
B	NORMAL mmol/L21-27	46	32.86%
C	HIGH	26	18.57%
	TOTAL	140	100.00%

CHILD CLASS	PUGHS	PaCO2	Frequency	Percent
A		less than 70	60	42.86%
B		70-90	28	20.00%
C		more than 90	52	37.14%
		TOTAL	140	100.00%

Medha Y Rao [11] et al 6 OF 43 patients with cirrhosis had developed hypoxemia. H.S. Hira et al[12] 30 patients with 17% had PaO2<70 mmHg. The study showed presence of hepatopulmonary syndrome and intrapulmonary vascular dilatation among patients with cirrhosis.

Florence Vachiery et al included 12 patients with cirrhosis and without cardiopulmonary disease 14% of patients had hypoxemia with PaO2 <70mmHg. In our study we had 42.86% that had hypoxemia

Konstantinos Charalabopoulos et al 22% of patients had acid base disturbances.3 In the present study 133 cases (95%) had acid base disturbances. Mohamed Alaa El-Din Nouh et al involving 60 cirrhotic patients showed multiple and mixed acid base disorders in cirrhotic patients[9] our study also mixed disorders were common.Vachiery and colleagues [13] showed that hypoxaemic patients had a significantly higher child pugh score PterSchenket al [14] Twenty-seven patients (24%) had HPS.

CONCLUSION

We concluded that acid base disturbances are common in cirrhotic cases, initially the acid base disturbances are most often mixed acid base disorder then they become metabolic acidosis then compensated metabolic acidosis and then respiratory alkalosis is the abnormality. These acid base disorders are predominantly observed in Child Pugh Class C more than Child Pugh Class A.

REFERENCES

1. Byass. P. The Global Burden of Liver Disease:a challenge for methods and for public health. BMC Med.vol.12,no.159,2014,doi:10.11861/S 12916-014-0159-5.
2. Robbins and Cortans. Liver and Gallbladder. In: Vinay kumar, Abdul K Abbas, Jon C Aster.(eds.)Pathologic basis of Disease. South Asia Edition.Volume 2,Saunders Elsevier, 2015. p. 827
3. Schuppan D, Afdhal NH. Liver cirrhosis. The Lancet. 2008 Mar 8;371(9615):838-851.
4. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. The Lancet. 2014 May 17;383(9930):1749-61.
5. Ginés P, Quintero E, Arroyo V, Terés J, Bruguera M, Rimola A, Caballería J, Rodés J, Rozman C. Compensated cirrhosis: natural history and prognostic factors. Hepatology. 1987 Jan;7(1):122-8.
6. D'amico G, Morabito A, Pagliaro L, Marubini E. Survival and prognostic indicators in compensated and decompensated cirrhosis. Digestive diseases and sciences. 1986 May 1;31(5):468-7
7. Charalabopoulos K, Peschos D, Zoganas L, Bablekos G, Golias C, Charalabopoulos A et al. Alteration in arterial blood parameters with liver cirrhosis and Ascites. Int J Med Sci.2007; 4(2):94-97.
8. Q.M. Anstee. D.E.J. Jones. Liver and Biliary tract disease. In: Brian R. Walker, Nicki R. Colledge Stuart H. Ralston Ian D. Penman.(eds.)Davidson's
9. Principles &Practice of Medicine 22nd edition, Churchill Livingstone Elsevier;2014.p. 975
10. El-Din Nouh M, Mohamed H, Masoud B, Yassin A. Study of Acid –Base Disturbances in Patients with Liver Cirrhosis. Afro –Egypt J Infect Endem Dis 2015;5(1):24-32.
11. Durand F, Valla D. Assessment of the prognosis of cirrhosis: Child–Pugh versus MELD. Journal of hepatology. 2005 Apr 1;42(1):S100-7.
12. Hira HS, Kumar J, Tyagi SK, Jain SK.A Study of Hepatopulmonary Syndrome among patients of cirrhosis of liver and portal hypertension. Indian J Chest Dis Allied Sci 2003;45:165-171.
13. Vachiéry F, Moreau R, Hadengue A, Gadano A, Soupison T, Valla D, Lebrec D. Hypoxemia in patients with cirrhosis: relationship with liver failure and hemodynamic alterations. J Hepatol 1997; 27: 492–5
14. Henriksen J, Bendtsen Moller S. Acid-base disturbance in patients with cirrhosis: relation to hemodynamic dysfunction. Eur J Gastroenterol Hepatol 2015; 27: 920-27
15. Abrams GA, Nanda NC, Dubovsky EV, Krowka MJ, Fallon MB. Use of macro aggregated albumin lung perfusion scan to diagnose hepatopulmonary syndrome: a new approach. Gastroenterology 1998; 114: 305–10.