Journal of Medicine in Scientific Research

Volume 5 | Issue 3 Article 23

Subject Area:

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Recommended Citation

Elfaizy, Moheb W. and Ghatas, Therese S. (2022) "Study of arterial blood gases in different stages of chronic liver diseases," *Journal of Medicine in Scientific Research*: Vol. 5: Iss. 3, Article 23.
DOI: https://doi.org/10.4103/jmisr.jmisr_65_22

Original Article

Study of arterial blood gases in different stages of chronic liver diseases

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Abstract

Background
Early detection of arterial gas abnormalities and acid–base disturbances in patients with chronic liver disease is essential to improve prognosis on the basis of pulmonary and liver diseases. The aim of the current study was to detect arterial blood gas changes, acid–base disturbances, and prevalence of hepatopulmonary syndrome (HPS) in different classes of severity of chronic hepatic disease for achieving a higher survival rate.

Results
Patients with hypoxemia represented 38 (33.6%) cases: three (15.7%) of 19 patients in group A, 16 (33.3%) of 48 patients in group B, 12 (34.2%) of 35 patients in group C, and seven (63.6%) of 11 patients with encephalopathy. Highly statistically significant decreases in arterial blood partial pressure of O_2 and oxygen saturation were found with the increase in the severity of the hepatic disease and cirrhosis as assessed using the Child–Pugh score. Moreover, partial pressure of O_2 was lowest in patients with encephalopathy. HPS was present in 22 (19.4%) patients with hypoxemia. A total of 12 patients were Child class C, eight patients were Child class B, and two patients were Child class A. Acid–base disturbances were observed in 82 (72.6%) patients, respiratory alkalosis in 49 (43.4%) patients, metabolic alkalosis in 10 (8.8%) patients, metabolic acidosis in seven (6.2%) patients, respiratory acidosis in eight (7.1%) patients, and mixed disturbances in eight (7.1%) patients.

Conclusions
Patients with hypoxemia were found to have a higher score of Child–Pugh than patients not having hypoxemia. HPS was found in all three Child–Pugh classes but mostly in Class C, so it is recommended that every patient with chronic liver disease should be evaluated and assessed for therapeutic decision, control of symptoms, improving survival, and quality of life.

Keywords: Acid–base disturbances, cirrhosis, hepatopulmonary syndrome, hypoxemia, liver disease

Background

Arterial blood gas (ABG) analysis and interpretation is an investigation tool widely used to investigate and monitoring management of patients with acute or chronic diseases and is permitted through the perfect interpretation of multiple interrelated variables and a correct assessment of metabolic, respiratory, and circulatory alterations. Thus, blood gas analysis is considered essential to reach the final diagnosis, decide the adequate treatment, and monitor its effectiveness in management of the patients. It is beneficial to study the hepatic role in the maintenance of acid–base homeostasis and also the effects of acute or chronic hepatic disease on the acid–base disturbances. Thus, in clinical application and practice, physicians managing patients with acute or chronic hepatic cell failure should suspect the possibility of existence of lung–kidney–liver cross-talk. The interpretation of acid–base equilibrium cannot be analyzed perfectly if the respiratory gases exchange is not perfectly considered and detected. The interrelationship between pulmonary and hepatic disease is, actually, well known, and ventilation-perfusion mismatching, alveolar capillary oxygen disequilibrium, or intrapulmonary or extrapulmonary

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DOI: 10.4103/jmisr.jmisr_65_22

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Submitted: 15-Jun-2022 **Revised:** 24-Jul-2022 **Accepted:** 25-Jul-2022 **Published:** 23-Nov-2022

How to cite this article: Ghatas TS, Elfaizy MW. Study of arterial blood gases in different stages of chronic liver diseases. J Med Sci Res 2022;5:348-54.

shunting, or a combination of all of these finding has been described in previous literature to clarify the abnormalities of gas exchange mechanisms that occur in both acute and chronic hepatic diseases [1,2].

Abnormal findings of pulmonary gas exchange commonly occur in hepatic disease and cirrhotic patients. The natural history and course of the disease in these patients and hypoxemia are unknown. Because chronic liver diseases and cirrhosis usually progress gradually, there is suspicion that oxygen level in the blood also may be gradually impaired [3].

Development of ABG changes and hypoxemia in patients with chronic hepatic disease affects and modifies the decision and the line of management and treatment and worsens the course of the disease and prognosis [4].

Pulmonary symptoms and abnormalities occur in patients with hepatic disease and cirrhosis regardless of the cause. ABG changes also commonly occur and are found in as many as 45–50% of patients with liver cirrhosis [5].

Up to ~70% of patients with cirrhosis undergoing preparation and evaluation for a liver transplantation operation complain of difficulty of breathing and dyspnea. Hepatopulmonary syndrome (HPS) forms one of the main differential diagnosis for dyspnea and hypoxemia and ABGs changes in patients with chronic hepatic disease and it carries a worse prognosis [6].

End-stage liver cell failure and its complications are considered leading causes of death in adults. Liver plays a main and central role in health and homeostasis, so as a result, the diseased liver leads to many deleterious effects and complications on multiple organ systems, including the pulmonary system. A variety of etiologies for pulmonary dysfunction in hepatic disease have been recognized and identified, including cardiopulmonary disease, disorders not specifically related to hepatic disease, and unique complications that occur with the presence of hepatic disease and/or portal hypertension. Hepatic disease and portal hypertension can occur with pulmonary vascular problems, which include portopulmonary hypertension and HPS [7].

HPS is a frequent pulmonary complication of chronic hepatic disease and cirrhosis, characterized by hypoxemia triggered by intrapulmonary vascular dilatation. The prevalence of HPS was found in 4–47% of patients with chronic hepatic disease owing to the presence of different criteria and diagnostic tools used by different research studies. Angiogenesis and nitric oxide excessive production seem to be the main complicated mechanisms, result in ventilation/perfusion mismatch and intrapulmonary shunting. HPS classification according to hypoxemia severity has been considered and suggested. An effective management that can modify the natural history of the syndrome is performance of liver transplantation. Despite usually presenting without symptoms, HPS imparts a high risk of mortality in pretransplantation time, independently of the stage or severity of hepatic disease; however, there are many variable data about survival rates after operation of liver transplantation [8]. The aim of the current study was to detect ABG changes, acid–base disturbances, and prevalence of HPS in different classes of severity of chronic hepatic disease for achieving higher survival rates.

Patients and methods

This is a cross-sectional descriptive study that was carried out in the Department of Chest Diseases, Al Sahel Teaching Hospital, from January 2019 to January 2020. A total of 113 patients previously diagnosed with chronic liver disease were recruited in this study. Patients varied in their disease severity according to the Child–Pugh classification. Full clinical history, complete clinical examination, laboratory investigation (liver function test, level of albumin in serum, prothrombin time determination, complete blood count, viral markers, serum creatinine, and blood urea nitrogen) with arterial blood sampling for arterial gas analysis, and acid–base disturbance pulmonary function test were done for all patients. ECG, echocardiography, chest radiograph, and chest high resolution computed tomography (HRCT) were performed for every patient to exclude respiratory or cardiac diseases. Ultrasound of the abdominal was done for evaluation of liver and spleen, diameters of portal vein, and determination of ascites. Severity of the hepatic disease was assessed by calculating Child–Pugh scores. The work was approved by the local ethics committee of General Organization of Teaching Hospital and Institutes GOTHI. Patients participated in the study after giving written informed consent.

Inclusion criteria were all patients at different stages of chronic liver disease according to the Child–Pugh score [9].

Meticulous care was taken to exclude all other comorbidities that directly or indirectly affect the patient's arterial oxygen saturation (SaO_2), such as follows:

(1) Heart failure: patients with congestive heart failure before diagnosis of chronic liver disease or with history of coronary artery disease.
(2) Anemia or massive blood loss: all patients were screened with complete blood count. Patients with hemoglobin less than 10 g/dl were excluded from the analysis.
(3) Chronic lung disease: asthma standard roentgenograms and pulmonary function test were used for excluding suspected patients from the study.
(4) Hydrothorax or pneumothorax: standard roentgenograms and ultrasonography were done to exclude patients from the study with clinical finding of hydrothorax or pneumothorax.
(5) Acute or chronic renal failure: all patients were screened with renal function tests to exclude hepatorenal syndrome.
(6) Long-term oxygen therapy.

Arterial blood partial pressure of O_2 (PaO_2), SaO_2, and different acid–base changes were detected in every patient. Samples from arterial blood were withdrawn while patients were breathing room air ($FiO_2 = 21\%$) in a semi-seated position (after 10 min of rest). Patients were subjected to puncture from the radical artery. PaO_2, carbon dioxide tension, and SaO_2 of hemoglobin were immediately measured. Hypoxemia was considered to be present when arterial blood PaO_2) was less than 80 mmHg.

Statistical analysis was performed using SPSS for Windows (SPSS Inc., Chicago, Illinois, USA), version 23. Values were expressed as mean ± SD. Differences between groups were evaluated assessed using Student paired *t* test. Differences in variables between groups were assessed and evaluated by the χ^2 test. Values of *P* value less than 0.05 were considered statistical significance, and *P* value less than 0.01 were considered to be highly significant.

Results

A total of 113 patients with chronic liver disease, comprising 80 (70.8%) males and 33 (29.2%) females, were included in this study. The mean age of patients was 57.58 ± 2.92 years. There was no statistically significant difference regarding age or sex between the groups. Table 1 shows the characteristics of the 113 patients.

Patients were classified according to the Child–Pugh scoring system, and data of each group were collected and statistically analyzed. A total of 19 (16.8%) patients were of Child–Pugh Class A, 48 (42.4%) were of Class B, 35 (30.9%) were class C, and 11 (9.7%) had encephalopathy (Tables 1 and 2 and Fig. 1).

HPS was present in 22 (19.4%) patients with hypoxemia. A total of 12 patients were in Child C, eight patients found in Child B, and two patients were found in Child A.

The number of hypoxemic patients whose PaO_2 was less than the lower limit of normal was 38 (33.6%) patients, where patients with hypoxemia were three (15.7%) patients out of 19 in group A, 16 (33.3%) patients out of 48 in group B, 12 (34.2%) patients out of 35 in group C, and seven (63.6%) patients out of 11 in patients with encephalopathy.

There was a highly statistically significant decrease in the arterial blood PaO_2 and a statistically significant decrease in SaO_2 with increase in the severity of hepatic cirrhosis, as assessed using the Child–Pugh score. Moreover, PaO_2 was the lowest in patients with encephalopathy. Table 2 and Figs. 2 and 3 summarize the PaO_2 and SaO_2 in each group of patients, respectively.

In comparison of PaO_2 and SaO_2 levels between group A and group B, there was no difference of statistical significance between them (*P*= 0.193 and 0.456, respectively). However, comparison between both groups regarding PaO_2 denoted a highly statistically significant hypoxemia in group C (*P* = 0.007) and a statistically significant lowering in blood SaO_2 (*P* = 0.048), which indicate that hypoxemia prevalence increases in patients with chronic liver disease with more deterioration in liver functions. There was a highly significant decrease in PaO_2 levels in patients with

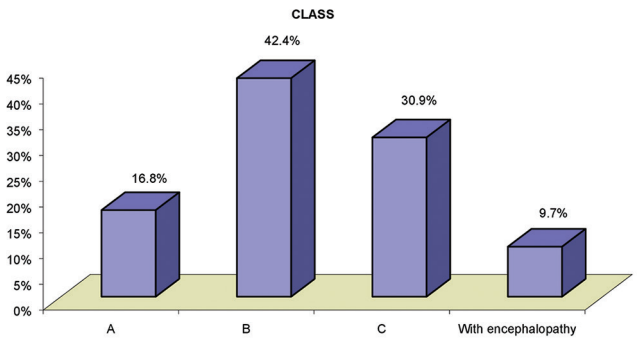

Figure 1: Classes of patients according to Child–Pugh scoring system.

Table 1: Characteristics of patients

	n=113 [*n* (%)]
Sex	
Female	33 (29.2)
Male	80 (70.8)
Age (years)	
Mean±SD	57.58±2.92
Range	50–64
BMI	
Mean±SD	29.47±1.46
Range	24.3–32.8
Class	
A	19 (16.8)
B	48 (42.5)
C	35 (30.9)
With encephalopathy	11 (9.7)
PaO_2 (mmHg)	
Mean±SD	78.07±8.67
Range	55.1–88.7
SaO_2 (%)	
Mean±SD	95.24±3.24
Range	86–99
PH	
Mean±SD	7.43±0.05
Range	7.32–7.56
$PaCO_2$ (mmHg)	
Mean±SD	39.27±7.55
Range	29.7–61.4
Arterial HCO_3 (mmol/l)	
Mean±SD	24.38±3.85
Range	17.6–34.1
Acid–base disorder	
Normal	31 (27.4)
Metabolic alkalosis	10 (8.8)
Respiratory alkalosis	49 (43.4)
Metabolic acidosis	7 (6.2)
Respiratory Acidosis	8 (7.1%)
Mixed	8 (7.1%)
Bilirubin (mg/dl)	
Mean±SD	2.98±1.05
Range	1.3–6.2
Albumin (g/dl)	
Mean±SD	3.04±0.48
Range	2.5–4.5
INR	
Mean±SD	2.19±0.55
Range	1–3.3
Creatinine (mg/dl)	
Mean±SD	1.04±0.15
Range	0.7–1.3

INR, international normalized ratio; $PaCO_2$, carbon dioxide tension; PaO_2, partial pressure of O_2; SaO_2, oxygen saturation.

Table 2: Characteristics of each group of patients

	Group A [*n* (%)] *n*=19	Group B [*n* (%)] *n*=48	Group C [*n* (%)] *n*=35	Encephalopathy group [*n* (%)] *n*=11	Test value	*P*	Significance
Sex							
Female	6 (31.6)	14 (29.2)	10 (28.6)	3 (27.3)	0.078*	0.994	NS
Male	13 (68.4)	34 (70.8)	25 (71.4)	8 (72.7)			
Age (years)							
Mean±SD	58.16±3.22	56.94±2.59	58.11±3.32	57.64±2.11	1.427•	0.239	NS
Range	50-62	52-63	52-64	55-61			
Class							
A	19						
B		48					
C			35	11			
PaO_2 (mmHg)							
Mean±SD	82.48±6.88	79.64±8.02	76.25±8.61	69.40±7.67	7.452•	0	HS
Range	64.8-88	60.6-88.7	59.1-83.5	55.1-80.3			
SaO_2 (%)							
Mean±SD	96.37±2.24	95.73±2.94	94.57±3.84	93.27±2.94	3.145•	0.028	S
Range	91-99	87-99	86-99	89-97			
PH							
Mean±SD	7.42±0.03	7.42±0.04	7.45±0.06	7.47±0.06	6.553	0	HS
Range	7.37-7.47	7.34-7.47	7.32-7.56	7.32-7.55			
$PaCO_2$ (mmHg)							
Mean±SD	39.84±4.22	40.00±7.03	38.22±9.53	38.47±7.54	0.447•	0.72	NS
Range	34.2-46.9	32.8-58.7	29.7-61.4	30.2-49.9			
Arterial HCO_3 (mmol/l)							
Mean±SD	24.66±2.83	24.05±3.22	23.92±4.52	26.75±5.07	1.738•	0.163	NS
Range	18.9-30.1	17.6-34.1	18.1-33.5	18.2-32.6			
Acid–base disorders							
Normal	11 (57.9)	20 (41.7)	0	0	31.13	<0.001	HS
Metabolic alkalosis	1 (5.3)	3 (6.2)	4 (11.4)	2 (18.18)	2.181	0.535	NS
Respiratory alkalosis	4 (21.1)	17 (35.4)	22 (62.9)	6 (54.5)	11.061	0.011	S
Metabolic acidosis	1 (5.3)	2 (4.2)	3 (8.6)	1 (9.1)	0.867	0.833	NS
Respiratory acidosis	1 (5.3)	3 (6.2)	3 (8.6)	1 (9.1)	0.332	0.953	NS
Mixed	1 (5.3)	3 (6.2)	3 (8.6)	1 (9.1)	0.332	0.953	NS
Bilirubin (mg/dl)							
Mean±SD	1.78±0.41	2.45±0.39	3.95±0.52	4.26±1.10	105.823•	0	HS
Range	1.3-2.8	1.8-3	3.1-5.2	2.7-6.2			
Albumin (g/dl)							
Mean±SD	3.62±0.34	3.23±0.35	2.60±0.10	2.58±0.09	75.195•	0	HS
Range	3.1-4.5	2.5-4.4	2.5-2.8	2.5-2.7			
INR							
Mean±SD	1.34±0.20	2.03±0.22	2.78±0.26	2.52±0.24	176.018•	0	HS
Range	1-1.6	1.7-2.3	2.4-3.3	2.1-2.8			
Creatinine (mg/dl)							
Mean±SD	0.96±0.15	1.06±0.14	1.03±0.16	1.11±0.14	3.048•	0.032	S
Range	0.7-1.2	0.7-1.3	0.7-1.3	0.9-1.3			

INR, international normalized ratio; $PaCO_2$, carbon dioxide tension; PaO_2, partial pressure of O_2; SaO_2, oxygen saturation. χ^2 test one way analysis of variance test. *P* value more than 0.05, nonsignificant; *P* value less than 0.05, significant; *P* value less than 0.01, highly significant. HS, highly significant; NS, non significant; S, significant.

encephalopathy when compared with patients without encephalopathy (*P*= 0.000) (Figs. 4 and 5).

P(A-a) O_2 was highly significantly elevated in patients with encephalopathy and advanced liver cirrhosis than in patients with less advanced stage of the disease according to Child–Pugh classification (*P* = 0.00) (Figs. 6 and 7).

pH was statistically significantly higher in patients with encephalopathy than in those without encephalopathy (Fig. 8).

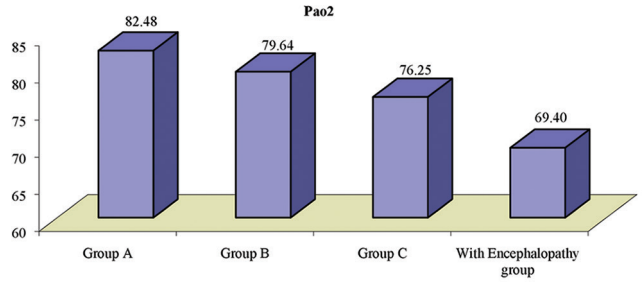

Figure 2: Arterial blood partial pressure of O_2 in each group of patients.

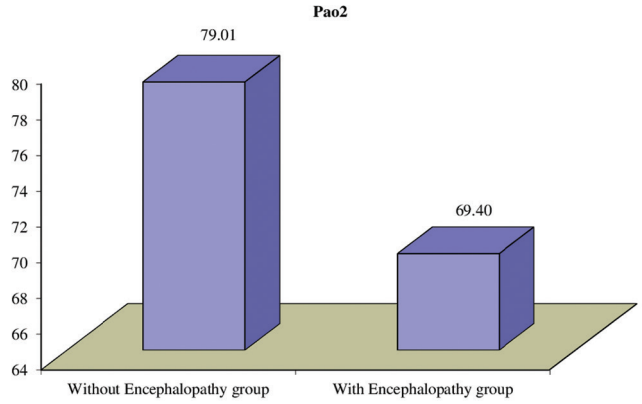

Figure 4: Comparison between patients without encephalopathy and patients with encephalopathy regarding partial pressure of O_2 (PaO_2).

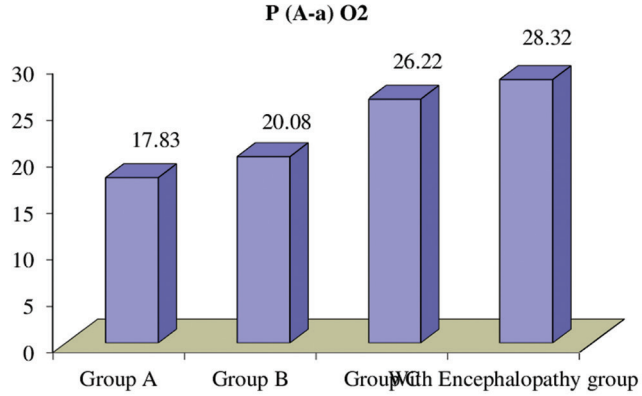

Figure 6: P(A-a) O_2 in each patient group.

Acid–base disturbances were observed in 82 (72.6%) patients, respiratory alkalosis in 49 (43.4%) patients, metabolic alkalosis in 10 (8.8%), metabolic acidosis in seven (6.2%), respiratory acidosis in eight (7.1%), and mixed disturbances in eight (7.1%) patients. Normal acid–base balance was founded in 31 (27.4%) patients. Respiratory rate was within the normal limits in all patients studied. So, acid–base disturbances could not be attributed only to abnormalities in respiratory rate, like respiratory alkalosis in patients with tachypnea (Fig. 9).

Discussion

Hypoxemia in patients with chronic hepatic disease worsens the prognosis of the disease and modifies the lines of management.

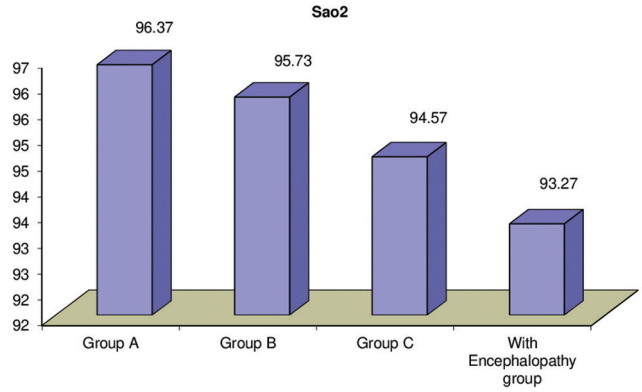

Figure 3: Oxygen saturation (SaO_2) in each group of patients.

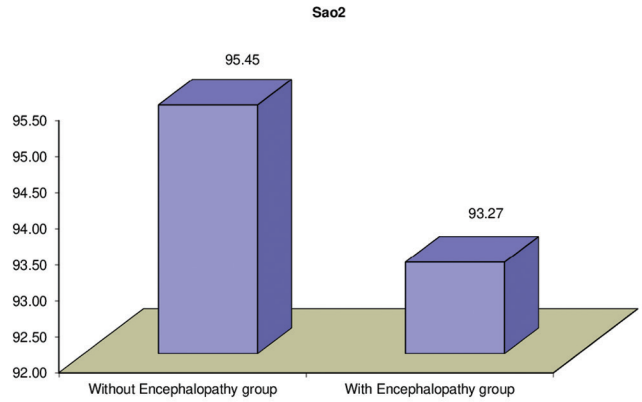

Figure 5: Comparison between patients without encephalopathy and patients with encephalopathy regarding oxygen saturation (SaO_2).

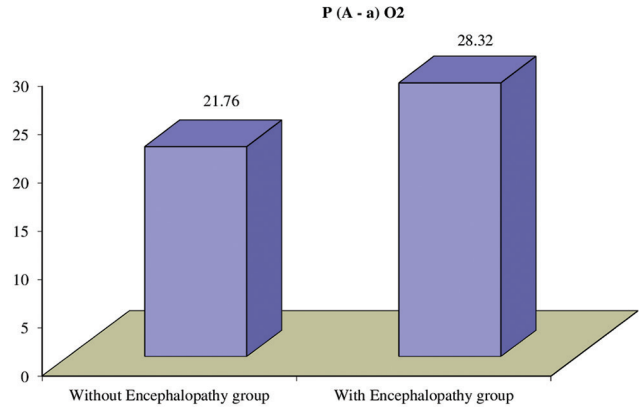

Figure 7: Comparison between patients without encephalopathy and patients with encephalopathy regarding P(A-a) O_2.

In the current study of 113 patients with different stages of chronic liver disease, 38 patients had hypoxemia ($PaO_2 < 80$ mmHg). Arterial hypoxemia in the present study was seen in 33.6% of patients with liver cirrhosis. Previous studies performed by Helmy and Awadallah [7], Hakan *et al*. [10], and Rao *et al*. [4] found a prevalence of 18, 43.8, and 13.9%, respectively. Mild hypoxia presents in about 33% of patients with chronic hepatic disease. Pulmonary symptoms and abnormalities

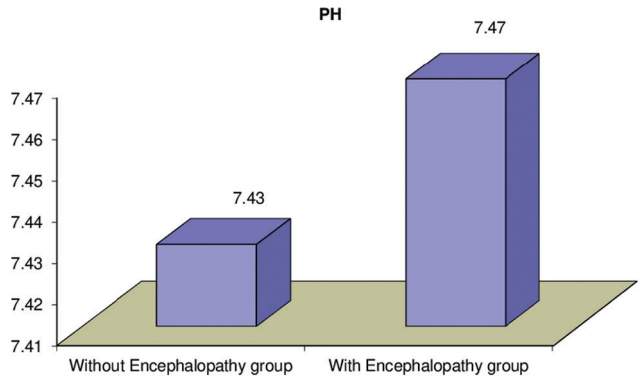

Figure 8: Comparison between patients without encephalopathy and patients with encephalopathy regarding PH.

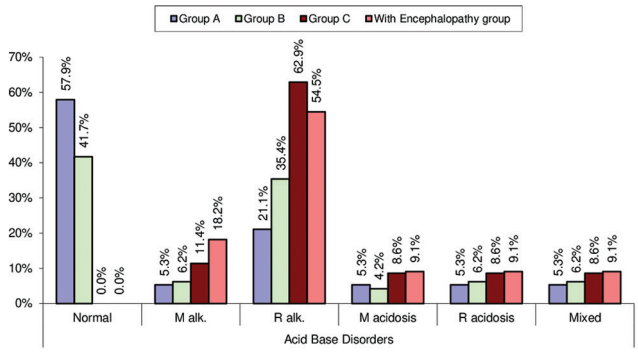

Figure 9: Acid–base disturbances in each group of patients.

are common in patients with chronic hepatic disease [11]. Presence of deficiency of oxygen in patients with chronic hepatic disease modifies the approach and management and worsens the prognosis of the disease [4]. The prevalence of hypoxemia in chronic hepatic disease and cirrhotic patients varies among previous studies [3,4,12], ranging from 13.9 to 60%. This discrepancy found among studies can be explained and interpreted by a difference in clinical profiles and the disease severity in the group of patients studied.

In the current study, there was a highly statistically significant decrease of arterial blood PaO_2 and a statistically significant decrease of SaO_2 in patients with chronic liver disease, and nonhypoxemic patients had a lower Child–Pugh score than patients with hypoxemia. Moreover, PaO_2 was the lowest in patients with encephalopathy. A previous study in 2014 stated that arterial hypoxemia was presents in chronic liver disease and cirrhotic patients assessed and evaluated by Child–Pugh score, and evaluation of its severity revealed an increase of hypoxemia in hepatic patients having advanced stage of chronic liver disease. They showed the presence of multiple pulmonary complications in patients with liver cirrhosis. Early diagnosis and identification of any pulmonary dysfunctions or complications in chronic liver disease and cirrhotic patients is essential, as it guides the decision and the further management through speeding up the recommendations of operation of orthotopic liver transplantation, and also early diagnosis and detection of pulmonary complications affects the prognosis [7].

When there is no lung disease, the major etiology of ABG changes, and hypoxemia in patients with chronic liver disease was suspected to be intrapulmonary vascular abnormalities, pulmonary arterio-venous shunting, portopulmonary shunting, and/or ventilation-perfusion mismatching. Alternatively, patients with chronic hepatic disease with cirrhotic liver usually become hyperkinetic, and in a few patients with cirrhosis, the expected hypoxemia presence may be compensated by the increased circulation and hyperventilation. Moreover, in some hepatic patients with cirrhosis and hypoxemia, no cause of hypoxemia can be demonstrated [13]. A previous study by Ghayumi *et al*. [3] stated that there was a lack of correlation between the Child–Pugh score and the degree of hypoxemia; all of these findings elevate the possibility and suspicion that some other unknown mechanisms and factors are still present in the pathogenesis and mechanisms of the occurrence of hypoxemia in patients with chronic hepatic disease and cirrhosis [3].

In the current study, of 113 patients with different stages and severity of chronic hepatic disease, 22 patients had HPS. Thus, HPS was present in 19.4% of the patients. HPS was found in all three classes of Child–Pugh but mostly in Class C. This is in agreement with the study of Younis *et al*. [14]. Studies on the prevalence of HPS in patients with chronic hepatic disease and cirrhosis done by Helmy and Awadallah [7], Kari *et al.* [15], Thevenot *et al*. [16], and Zhang and Fallon [11] showed a prevalence of 12, 24, 20, and 5–32%, respectively. Mohan and colleagues stated that HPS had been shown to occur in ~4–19% of patients with chronic liver disease and cirrhosis. Increased alveolar-arterial gradient or hypoxemia in patients with liver cirrhosis should be examined and HPS presence should be evaluated as a routine procedure [17].

Alongside kidneys and lungs, the liver has been known as an important regulator of acid–base homeostasis. Interestingly, the literature and studies on disorders of acid–base in chronic hepatic disease are very limited. Patients with liver disease and disorders often show acid–base disturbances. The most common (acid–base) disorder in patients with chronic hepatic disease is respiratory alkalosis [18].

In the current work, pH was statistically significantly elevated in patients with encephalopathy than in patients without encephalopathy. Acid–base disturbances were observed in 82 (72.6%) patients, respiratory alkalosis in 49 (43.4%) patients, metabolic alkalosis in 10 (8.8%), metabolic acidosis found in seven (6.2%), respiratory acidosis found in eight (7.1%), and mixed disturbances in eight (7.1%) patients. No acid–base changes were founded in 31 (27.4%) patients. Respiratory rate was within the normal rates in all patients studied. So, acid–base disturbances could not be attributed to only abnormalities in the respiratory rate, like respiratory alkalosis, which occurs in patients with tachypnea. Jimenez *et al.* [19] found that acid–base disorders are frequent and common in patients with chronic and liver cell failure; the underlying pathogenesis and mechanisms are complex and represent diagnostic and therapeutic challenges to the physician. They stated that several

complications of chronic liver disease and cirrhosis and its therapeutic interventions foster homeostatic disturbance of acid–base balance. The clinical applications and therapeutic interventions of acid–base disorder must focus on the etiology and not on the metabolic disorder *per se* [19].

Conclusion

Decreases in SaO_2 and PaO_2 as well as various acid–base disorders are common in patients with chronic hepatic disease and cirrhosis. Hypoxemic patients have a higher Child–Pugh score than those not suffering from hypoxemia. HPS was found in all three classes of Child–Pugh but mostly in Class C, so it is recommended that every patient with chronic liver disease should be evaluated and assessed for therapeutic decision, quality of life, control of symptoms, and survival. HPS is considered a high-risk factor of pretransplantation mortality, independently of the stage or severity of hepatic disease. HPS screening among patients with hepatic disease is essential in the guidance of achieving a higher rate of survival, through diagnosis and appropriate prioritization of patients with HPS for whom operation of orthotopic liver transplantation is curative.

Recommendations

Use of ABG sampling, especially in patients candidates for liver transplantation, is the most prudent approach. As decreases in SaO_2 and PaO_2 as well as various acid–base disorders result in impaired pulmonary resistance, patients are more likely susceptible to adult respiratory distress syndrome and infections. So, prognosis in these patients is poor on the basis of pulmonary and hepatic disease. Further studies are needed to detect the desired frequency of this investigation and testing in patients with chronic hepatic disease and cirrhosis who are listed for liver transplantation.

Further research studies are needed to clarify the precise mechanisms that induce (HPS) pathogenesis and secondary subclinical and clinical vital organ interactions.

Acknowledgements

The authors acknowledge the late Dr Essam Anwar, MD, consultant of hepatic diseases, who provided great and valuable input in this work. May God rest his soul in peace.

Authors contributions: T.S.G.: concept, acquisition of data, writing, reviewing, and publishing. M.W.E.: acquisition of data and reviewing. The authors have read, reviewed, and approved the final manuscript.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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