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Oxygen desaturation in Patients with decompensated Cirrhosis measured at rest and after 6 minutes of walk

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A B S T R A C T

Background: Histologically, cirrhosis is characterised by distorted hepatocellular regeneration, aberrant fibrosis, and bridging fibrous septa that result in strange hepatocellular architecture. Clinically, this manifests in a variety of ways like lethargy, pruritus and jaundice etc. However, in end stage liver disease, a phase called decompensated liver cirrhosis appears which is manifested in the form of ascites, portal hypertension, splenomegaly, gastro-esophageal varices and in the worst cases, hepatic encephalopathy.

Objective: To determine the frequency of oxygen desaturation in patients with decompensated cirrhosis by using pulse oximetry at rest and after 6 minutes walk on a flat surface.

Methodology: This descriptive study of 139 patients were carried out at Medicine Department of Medicine, Khyber Teaching Hospital Peshawar, Pakistan from October 2019 to April 2020. To meet the diagnostic requirements of decompensated liver cirrhosis, including smaller liver, dilated portal vein, splenomegaly, and ascites, a liver ultrasound was conducted. All patients had their oxygen saturation checked using a Santa Medical Generation 2 pulse oximeter both at rest and after a 6-minute walk. All information was entered into a proforma that was created specifically for this use. To control variables and research bias, strict exclusion criteria was followed.

Results: In the current study, 139 patients were included, of whom 29% were between the ages of 30 and 50 and 71% between the ages of 51 and 70. The SD was ±10.85, and the mean age was 58. Among the study cases 34% were female and 66% were male. Only 25 patients (18%) among the study cases had oxygen desaturation throughout the six-minute walking test.

Conclusion: Frequency of oxygen desaturation (after 6 minutes walk on a flat surface) was 18% in patients presenting with decompensated liver cirrhosis.

Key words: Oxygen desaturation; Hepatic encephalopathy; Decompensated; Liver cirrhosis

Introduction

Cirrhosis is defined histologically by distorted hepatocellular regeneration, abnormal fibrosis, and bridging fibrous septa, all of which result in a peculiar hepatic architecture.[1] Clinically, this manifests in a variety of ways like lethargy, pruritus and jaundice etc. In end-stage liver disease, however, a phase known as decompensated liver cirrhosis develops, characterised by ascites, portal hypertension, splenomegaly, gastro-esophageal varices, and, in the worst cases, hepatic encephalopathy.[2]

Snell et al reported desaturation of arterial blood for oxygen in patients with cirrhosis of the liver in 1935, in addition to the clinical signs of decompensated cirrhosis noted above. The researchers came to the conclusion that arterial desaturation is caused by either a diminished diffusing capacity of the lungs or an increase in venous mixing. True anatomic venous admixture from intra-cardiac or extra-cardiac right-to-left shunts, as well as physiologic venous admixture due to perfusion of poorly or non-ventilated alveoli, are included in this group.[3]

The oxygen saturation of the blood in healthy people is greater than 94%. In COPD patients, a range of 88-92 % is considered ideal. Any oxygen saturation below 94 % in COPD patients and below 88% in COPD patients is regarded highly abnormal and requires oxygen treatment.[4,5] End-stage liver disease is linked to high blood levels of ammonia and nitrous oxide, which causes aberrant dilation of the arterio-venous (AV) junctions, resulting in shunting and venous mixing of blood in the lungs. In severe situations, this might appear clinically as dyspnea and cyanosis, and it can be detected by monitoring oxygen desaturation, especially on exertion.[6,7]

Hepatic cirrhosis is the 10th highest cause of mortality worldwide, with chronic viral hepatitis being the most common aetiology, according to local Pakistani statistics.[8,9]

The rationale of this study will be to document the frequency of oxygen desaturation by using pulse oximetry at rest and after 6 minutes' walk in patients with decompensated cirrhosis. This is because hypoxia is the earliest predictor of hepato-pulmonary syndrome and has been proven to be an indicator of mortality in such patients. Therefore, this study will aid in its timely identification and treatment with oxygen.

Objectives

Objective of the present study was to determine the frequency of oxygen desaturation in patients with decompensated cirrhosis by using pulse oximetry at rest and after 6 minutes walk on a flat surface.

Methodology

After getting approval from the hospital ethical committee this descriptive cross sectional study was carried out at Medicine Deptt of Khyber Teaching Hospital Peshawar from October 2019 to April 2020. The study comprised a total of 139 patients of either gender with decompensated liver cirrhosis and cirrhotic patients falling into either Child Pugh Class A, B, or C.

Patients with obstructive airway disease, pleural effusions, heart failure, venous thromboembolic illness including pulmonary embolism, fluid overload owing to nephrotic syndrome, parenteral nutrition, as well as those with primary pulmonary problems, Patients taking drugs like amiodarone, nitrofurantoin and methotrexate, which can impair lung function as well as those with conditions that interfere with the measurement of oxygen saturation, such as henna application, nail polish over natural or artificial fingernails, dyshemglobinemia and anaemia were excluded from the study.

To meet the diagnostic requirements of decompensated liver cirrhosis, including smaller liver, dilated portal vein, splenomegaly, and ascites a liver ultrasound was conducted. Statistical analysis was done using SPSS 27.0. P-value ≤0.05 was considered as significant.

Results

A total of 139 study cases were included in this study. By

Table 1. Age and Gender distribution of the study cases

Number	Frequency	Percentage
Age wise distribution		
30-50 years	40	29%
51-70 years	99	71%
Gender wise distribution		
Male	92	66%
Female	47	34%

Mean age was 58 years with SD ± 10.85

Table 2. Grades of child pugh & hepatic encephalopathy

Grade	Frequency	Percentage
Child pugh		
Grade A	25	18%
Grade B	53	38%
Grade C	61	44%
	Encephalopathy	
Grade 1	48	35%
Grade 2	74	53%
Grade 3	17	12%

age distribution of 139 patients found that 40 (29%) were between the ages of 30-50 and 99 (71%) were between the ages of 51-70. The median patient age was 58 years, with a standard deviation of 10.85 years. There were 92 (66%) male and 47 (34%) females (Table 1). Duration of illness of the study cases showed that 63(45%) patients had duration of illness ≤3 years while 76 (55%) patients had duraiton of disease >3 years. The disease lasted an average/mean of 3 years, with an SD of ±3.29. Size of liver also different among study cases and results showed that 93 (67%) of cases showed their liver size ≤8 cm whereas 46 (33%) patients had liver size >8 cm. Mean liver size was 8 cm with SD ± 3.841. Spleen size ≤16 cm was noted in 43 (31%) while 96 (69%) patients had spleen size >16 cm. Mean spleen size was 15 cm with SD ± 2.994. 25 (18%) had child pugh grade A, 53 (38%) had child pugh grade B and 61 (44%) had child pugh grade C respectively. 48 (35%) had hepatic encephalopathy of grade 1, 74 (53%) had hepatic encephalopathy of grade 2 and 17 (12%) patients had hepatic encephalopathy of grade 3 (Table 2). Mild ascites was found in 45 (32%) of the patients, 58 (42%) had moderate ascites and 36 (26%) had severe ascites (Table 3).

At rest, none of the 139 patients showed oxygen desaturation, while 25 patients (18%) did so during the six-minute walking test (Table 4).

Discussion

A variety of factors including alcohol and viral hepatitis, can lead to cirrhosis. Any kind of ongoing liver injury can lead to cirrhosis. Hepatic parenchymal fibrosis is the end outcome of the common pathway . In most individuals, 80 to 90% of the liver parenchyma must be damaged before clinical signs of liver failure appear.10,11 Hepatic function declines or the liver parenchyma is physically disturbed and reorganised as a result of cirrhosis complications. One etiological agent may produce a range of morphological manifestations, making it challenging to categorise merely on the basis of morphology. Micro-nodular, macro-nodular, and mixed-nodular cirrhosis are the three subtypes. It only comes to light after an autopsy and affects 30 to 40% of people. As a result, the actual incidence of disease cannot be determined.12,13

In current study a total of 139 patients were analyzed in which 29% patients were in age ranged 30-50 years while 71% patients were in age ranged 51-70 years Mean age was 58 years with SD ± 10.85. Sixty six percent patients were male whle 34% patients were female. All the 139 patients didn't had oxygen desaturation at rest but 25(18%) patients had oxygen desaturation at 6 minutes walking test.

The oxygen saturation of the blood in healthy people is greater than 94%. In COPD patients, a range of 88-92 % is considered ideal. Any oxygen saturation below 94 % in COPD patients and below 88 % in COPD patients is regarded highly abnormal and requires oxygen treatment.14,15 End-stage liver illness is linked to high blood levels of ammonia and nitrous oxide, which causes aberrant dilation of arterio-venous (AV) junctions, resulting in shunting and venous mixing of blood in the lungs. In severe situations, this might appear clinically as dyspnea and cyanosis, and can be detected by

Table 3. Degree of ascites among study cases

Degree of ascites	Frequency	Percentage
Mild	45	32%
Moder	58	42%
Severe	36	26%

Table 4. Oxygen desaturation among the cases

Oxygen desaturation	Frequency	Percentage
Oxygen desaturation at rest		
Yes	0	0%
No	139	100%
Oxygen desaturation at 6 minutes walk		
Yes	25	18%
No	114	82%

monitoring oxygen desaturation, especially during exertion.

Hepatic cirrhosis is the tenth leading cause of death worldwide, with chronic viral hepatitis being the most common aetiology, and local Pakistani statistics support these findings.16 A study conducted by Gary A in 2003 found that arterial hypoxemia caused by intrapulmonary vasodilation occurs in approximately 10% of patients with cirrhosis.17 Another similar study conducted by Hopkins et al found that arterial hypoxemia occurs in approximately 10% of patients with cirrhosis.18

Moller J et al reported in a study that mean Po2 in kPa was 11.3 in Child class A, 10.8 in class B, 10.6 in class C, and 10.6 in patients with encephalopathy (p value 0.05).19 In class A, B, C & patients with encephalopathy, the proportion of patients with Po2 below the lower normal limit of 9.6 kPa was 10%, 28%, 25%, and 43%, respectively (p value 0.05). Oxygen saturation (So2) in these groups was respectively: 96%, 96%, 96%, and 93% (NS). So2 was below the lower limit of 92% in 0%, 9%, 7%, and 24% ($p<0.05$).

A multivariate regression study in patients without hepatic encephalopathy demonstrated that a high arterial carbon dioxide tension, a low systemic vascular resistance, and a low indocyanine green clearance were all independent determinants of a low Po2 (p value 0.0001). In patients without encephalopathy, the prevalence of arterial hypoxaemia in cirrhosis is around 22%, but it varies from 10% to 40% depending on the degree of hepatic dysfunction.20 Arterial hypoxaemia appears to be less common than previously thought in patients with various degrees of cirrhosis, and patients with severe arterial hypoxaemia are uncommon.

Conclusion

Our study concludes that frequency of oxygen desaturation (after 6 minutes walk on a flat surface) was 18% in patients presenting with decompensated liver cirrhosis.

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