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ORIGINAL ARTICLE

CLINICAL CO RELATION BETWEEN ARTERIAL VERSUS VENOUS AMMONIA LEVELS IN HEPATIC ENCEPHLOPATHY IN CIRRHOSIS OF LIVER

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ABSTRACT: INTRODUCTION: Hepatic encephalopathy is a reversible neuropsychiatry state that complicates liver disease. Pathogenesis of Hepatic Encephalopathy in chronic liver is function is widely accepted to be due to failure of hepatic clearance of toxins products from gut exact toxin involved remains controversial but ammonia is thought to be an important factor. Ammonia levels help both in diagnosis and serve as a guide in treatment. Diagnosis of Hepatic Encephalopathy can be done based on clinical criteria and the severity of Hepatic Encephalopathy can be graded by West Haven Criteria. This criterion is the simplest grading of Hepatic Encephalopathy based on clinical findings. **AIMS AND OBJECTIVES:** To correlate between Ammonia levels and clinical severity of Hepatic Encephalopathy in Cirrhosis of liver and correlate between Arterial versus venous ammonia levels with severity of Hepatic Encephalopathy. **RESULTS:** Male patients had higher incidence than females. Severity of hepatic encephalopathy was graded by West Haven grading. Arterial total ammonia and venous ammonia was correlated with the clinical severity of HE. Of the 50 patients 3 had grade 1, 18 had grade 2, 22 had grade 3 and 7 had grade 4. Arterial and venous ammonia levels co related with severity of HE. The highest level of arterial ammonia was seen in grade 3 and grade 4.It was seen that other lab parameters also increased with severity of HE. But were not significant. Serum albumin was inversely co related with severity of HE. **CONCLUSIONS**: Arterial total ammonia correlated better with the severity of Hepatic Encephalopathy as compared to venous ammonia levels. Venous total ammonia did not correlate with severity of Hepatic Encephalopathy and with arterial ammonia levels.

INTRODUCTION: Hepatic encephalopathy is a reversible neuropsychiatry state that complicates liver disease. Pathogenesis of Hepatic Encephalopathy in chronic liver is function is widely accepted to be due to failure of hepatic clearance of toxins products from gut exact toxin involved remains controversial but ammonia is thought to be an important factor.

Correlation between plasma ammonia levels and severity of Hepatic Encephalopathy is not consistent, but ammonia levels are used widely in diagnosis of Hepatic Encephalopathy in cirrhotic patients.[1] In a study done by Janus. P.ong it was seen that Ammonia levels increased with the severity of Hepatic Encephalopathy and that Arterial Ammonia levels correlated better with the severity as compared to Venous ammonia.[2]

Ammonia levels help both in diagnosis and serve as a guide in treatment. Diagnosis of Hepatic Encephalopathy can be done based on clinical criteria and the severity of Hepatic Encephalopathy can be graded by West Haven Criteria. This criteria is the simplest grading of Hepatic Encephalopathy based on clinical findings.[3]

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AIMS AND OBJECTIVES:

- To correlate between Ammonia levels and clinical severity of Hepatic Encephalopathy in Cirrhosis of liver.
- To correlate between Arterial versus venous ammonia levels with severity of Hepatic Encephalopathy.

METHODOLOGY:

SOURCE OF DATA: The Study will comprise established cases of hepatic encephalopathy admitted to KIMS hospital.

Total Number of cases: 50 Period of study from October 2004 -September 2006.

METHOD OF COLLECTION OF DATA: Method of collection of data is by Patient evaluation which will be done by (asking detailed history, clinical examination) and lab investigations through a proforma specially designed for this study. Informed consent will be taken from all patients who will be included in this study'.

INCLUSION CRITERIA:

ADULTS: Patient of chronic liver disease who are in hepatic encephalopathy.

EXCLUSION CRITERIA: Patient with mental status changes due to causes other than hepatic encephalopathy.

SAMPLE PROCEDURE: Comparative and prospective study.

STATISTICAL METHODS: Analysis of variance, Post hoc turkey test.

RESULTS:

Study Design: A Prospective clinical study consisting of 50 cirrhosis patients in Hepatic Encephalopathy is undertaken to study the correlation between Arterial ammonia and venous ammonia and severity of Hepatic Encephalopathy.

Age and Sex Distribution: 84% of the subjects (n=42) were male and 16% of subject (n=8) were female. The highest incidence of Encephalopathy was seen in the age group 41-50, 34% (n=17) and 51-60, 34%.

Age in years	Male	Female	All cases
Up to 40	6(14.3%)	3(37.5%)	9(18.0%)
41-50	16(38.1%)	1 (12.5%)	17 (34.0%)
51-60	14(33.3%)	3 (37.5%)	17(34.0%)
61-70	6(14.3%)	1 (12.5%)	7(14.0%)
Total	**42(100.0%)**	**8(100.0%)**	**50**

Table 1: Age distribution of study

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Sex	Number (N=50)	%
Male	42	84.00
Female	8	16.00

Table 2: Sex Distribution of the study

Etiology of Cirrhosis: 80% of the subjects (n=40) were Alcoholic, 12% of (he subjects (n=6) had chronic Hepatitis B, 4.0% of the subjects (n=2) had chronic Hepatitis C, 4.0% had (n=2) Cryptogenic Cirrhosis.

Etiology	Number	%
Alcoholic	40	80.0
Hepatitis -B	6	12.0
Hepatitis -C	2	4.0
Cryptogenic cirrhosis	2	4.0

Table 3: Etiology of cirrhosis

Presenting Symptoms: 96% of the subjects (n=48) presented with Altered sensorium, 72% of the subjects (n =06) had Ascites, 35% had Icterus (n=35). 60% had Edema (n=30), 6% had Hematemesis (n=3), 6% had Bleeding (n=3).

Presenting Symptoms	Number N=50	%
Altered sensorium	48	96.00
Ascites	36	72.00
Icterus	35	70.00
Edema	30	60.00
Hematemesis	3	6.00
Bleeding	3	6.00

Table 4: Presenting Symptoms

Presenting Signs: 86% of the subjects (n=43) had Flapping tremors, 74% of the subjects (n=37) had Fetor hepaticus, 22% had Dupuytren's contracture (n=l 1), 10% had Parotid Enlargement, 66% of the subjects (n=33) had Dilated veins, 18% of the subjects (n=9) had Umbilical hernia.

Presenting signs	Number (n=50)	%
Flapping tremors	43	86.0
Fetor hepaticus	37	74.0
Dupuytren's contracture	11	22.0
Parotid enlargement	5	10.0
Dilated veins	33	66.0
Umbilical hernia	9	18.0

Table 5: Presenting signs

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Precipitating factors: In 52% of the subjects (n=26) precipitating factor for 1 Hepatic Encephalopathy was Azotemia, 20.0% of the subjects (n=l0) had infection, 5 of the subjects (n-5) had constipation, another 10% of the subjects (n=$) had Gl bleed and 8.0% had (n=4) other unknown precipitating factors.

PPT factors	Number	%
Azotemia	26	52.0
Infection	10	20.0
Constipation	5	10.0
Gl bleed	5	10.0
Others	4	8.0

Table 6: Precipitating factors

West Haven Grading Of Hepatic Encephalopathy: 6.0% of the subjects (n=3) had Grade I, 36.0 % of the subjects (n=8) had Grade 2, 44.0% (n=22) had Grade 3 and 14% of the subjects (n=7) had Grade 4 - the most severe form of Hepatic Encephalopathy.

West haven grading	Number (n=50)	%
Grade I	3	6.0
Grade II	18	36.0
Grade III	22	44, 0
Grade IV	7	14.0

Table 7: West Haven grading

Pattern of Laboratory Parameters

Lab parameters	Range	Mean ± Sd
Arterial Ammonia	61-120	93.33±12.07
Venous Ammonia	56-100	76.63±10.75
Bilirubin	1.40-20.40	6.69±4.27
Albumin	0.80-4.40	2.49±0.78
Prothrombin time	19.70-44.70	29.1l±5.39

Table 8: Mean pattern of laboratory parameters

Association of Arterial Ammonia with West Haven Grading: Arterial ammonia levels increased ivilh increasing severity of Hepatic Encephalopathy i.e. Increased Grading- p value <0.001.

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Grade	Arterial Ammonia	
	Range	Mean ± Sd
Grade I	61-85	71.33±12.34
Grade 11	62-100	84.98±9.I5
Grade III	90-f 10	98.64+4.24
Grade IV	98.30-120.0	106.90±7.9
Significance	F=28.895, P<0.001	
Pair wise significance	Grade I vs. Grade 11 P=0.023* Grade! vs. Grade III P<0, 001** Grade 1 vs. Grade IV P0.001** Grade II vs. Grade III P<0.00J** Grade 11 vs. Grade IV P<0.001** Grade II vs. Grade IV P=0.068+	

Table 9:Association of grade of HE with Arterial Ammonia

+ Suggestive of significance * Moderately significance ** Strongly significance

Association of Venous ammonia levels with the severity of Hepatic Encephalopathy: Venous ammonia does not correlate statistically with the increasing severity of Hepatic Encephalopathy and with the increased grading in West Haven criteria, p =0.123.

Grade	Venous Ammonia	
	Range	Mean ± Sd
Grade 1	67.4-99.0	78.S0tl7.54
Grade 11	60.0-92.0	72.49±9.29
Grade III	56.0-98.0	77.74±9.70
Grade IV	60.0-100.0	83.14±12.48
Significance	F=2.031, P=0.I23	
Pair wise significance	Grade 1 $v_{s.}$ Grade 11 P-G.75S Grade1 vs. Grade III P=0.998 Grade I vs. Grade IV P=0.930 Grade II vs. Grade III P=0.380 Grade II vs. Grade IV P=0.109 Grade III vs. Grade IV P=0.634	

Table 10: Association of grade of HE with venous Ammonia

+ Suggestive of significance * Moderately significance ** Strongly significance

Correlation of other laboratory parameters with the severity of Hepatic Encephalopathy and the West Haven Grading: Among other laboratory parameters only serum albumin was inversely related the severity of Hepatic Encephalopathy with p value of 0.014, serum total bilirubin and prothrombin time did not correlate with the severity.

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Grade	Total bilirubin	Albumin	Prothrombin time
Grade 1	8.40±6.03	3.86±0.48	27.40±l.25
Grade II	7.14±0.97	2.36±0.2I	28.83±I.18
Grade 111	5.66±0.64	2.44±0.12	28.56i0.84
Grade IV	8.06± 1.92	2.49=0.22	32.27±3.68
P value	**0.458**	**0.014***	**0.403**

Table 11: Correlation of other laboratory parameters with severity of Hepatic Encephalopathy

The correlation of Venous ammonia with Arterial ammonia is small (0.338). Venous ammonia does not correlate either with the grading or with the Arterial Ammonia levels. Therefore there is a need to do the arterial ammonia to find the severity of HE.

DISCUSSION: Hepatic encephalopathy is a reversible neuropsychiatric state that complicates liver disease. Pathogenesis of Hepatic Encephalopathy in chronic liver dysfunction is widely accepted to be due to failure of hepatic clearance of toxins products from gut. Exact toxin involved remains controversial but ammonia is thought to be an important factor. Correlation between plasma ammonia levels and severity of Hepatic Encephalopathy is not consistent, but ammonia levels are used widely in diagnosis of Hepatic Encephalopathy in cirrhotic patients. Ammonia levels help both in diagnosis and serve as a guide in treatment. Diagnosis of Hepatic Encephalopathy can be done based on clinical criteria and the severity of Hepatic Encephalopathy can be graded by West Haven Criteria. This criterion is the simplest grading of Hepatic Encephalopathy based on clinical findings.

The present study was a prospective clinical study consisting of 50 patients admitted to department of medicine, Kempegowda Institute of Medical Sciences, Bangalore from October 2004-September 2006, who were diagnosed to have Hepatic Encephalopathy. It was done to correlate whether serum ammonia levels increases with increasing severity of Hepatic Encephalopathy, and to find out whether arterial ammonia measurements are a better indicator of Hepatic Encephalopathy when compared to venous ammonia levels.

AGE DISTRIBUTION: In the present study, it was observed that highest incidence of Hepatic Encephalopathy was seen with the age group 41-50, 38% (n=16) & 51-60, 33.3 % (n= 17) in males and in the age group 51-60, 37.5% (n= 3) in the females. There was no significant age difference of the mean age between males and females patients.

These results were in correlation, with the other studies done by Tarun Kumar [4]et al; Chakrabarti et al, [5] Nanda Kumar et al[6] & Janus P Ong et al.[2] In these studies also incidence of Hepatic Encephalopathy in chronic liver disease was more common in the age group 41-60.

	Janus P Ong et al study[2]	Tarun Kumar et al study[4]	Nanda Kumar et al study[6]	Chakrabarti et al study[5]	Present study
Mean age of presentation (years)	54±10	44.5±6, 72	47.5±8.7	49.8±12.9	48.6±12.32

Table 12: CORRELATION OF AGE DISTRIBUTION WITH OTHER STUDIES

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In the study done by Mark et al[7] alcoholics developed cirrhosis in ten years duration with the presenting mean age of about 42.8±9, whereas according to Guptan[8] et. al. The mean age of presentation in patient with HBV infection was 41.6± 11. So the mean age of presentation of chronic liver disease is in accordance with the previous studies.

SEX DISTRIBUTION: In the present study 84% (n=42) were males and 16% (n=8) were females. This was in correlation with the other studies conducted by Tarun Kumar et al in Bihar, Nanda Kumar et al in Bombay and Bhagwan Singh et al[9] in Bombay.

In a study conducted by Bhagwan Singh et al it was found that women have increased incidence of developing chronic liver disease i.e. cirrhosis with Hepatic Encephalopathy in the case of Hepatitis B and C virus infection, whereas males had more incidence with alcoholic liver disease. In the present study majority of patients were male patient with history of alcohol consumption that could be the reason for male patient more than the females patient, moreover the incidence of alcoholism in our setup among females is low.

	Tarun Kumar et al study	**Nanda Kumar et al study**	**Bhagwan Singh et al study**	**Present study**
PERCENTAGE OF FEMALES	10%	12.6%	12.66%	16%

Table 13: PERCENTAGES OF FEMALES IN OTHER STUDIES AS COMPARED TO PRESENT STUDY

ETIOLOGY OF CIRRHOSIS: In the present study it was seen that etiology of cirrhosis was Ethanol consumption 80 % (n=40), Hepatitis B virus Infection in 12% (n=6), Hepatitis C virus 4.0% (n=2) and Cryptogenic cirrhosis 4.0 % (n=2).These results regarding etiology of cirrhosis was in correlation with the other studies done by Janus P Ong et al, Nanda Kumar et al, Tarun Kumar et al, .and Bhagwan Singh et al.

STUDY	**Alcohol**	**HBV infection**	**HCV infection**	**Cryptogenic cirrhosis**	**Primary biliary cirrhosis**	**Nonalcoholic steatohepatitis**	**Autoimmune disease and others**
Janus P Ong et al	54%	4%	14%	8%	4%	4%	12%
Nandakumar et al	65%	15%	10%	5%	-	5%	-
Tarun Kumar study	85%	15%		-	-	-	-
Bhagwan Singh et al	40%	14%	12%	18%	-	6%	10%
Present Study (Bangalore)	80%	12%	4%	4%			

TABLE 14: SHOWING ETIOLOGY OF CIRRHOSIS IN THE PRESENT STUDY IN RELATION TO THE OTHER STUDIES

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In a study conducted by Deepak Kumar.et.al[10] in Delhi, it was found that that there is decline in prevalence of HBV infection as a cause of chronic liver disease in the past five years. In this study it was found that overall distribution of HBV declined from 42.47%- 24.85% in the last five years.

In the present study decreased incidence of HBV associated chronic liver disease can be attributed to this. In our study incidence of cirrhosis was 16% with both HBV and HCV infection when taken together, while alcohol consumption was the cause in 80%.

So according to the above a mentioned result there is a need to create more awareness among general population regarding adverse-effect of alcohol consumption.

PRECIPITATING FACTORS: In the present study it was found that the most common precipitating factor of Hepatic Encephalopathy was Azotemia 52 % (n=26), Infection 20 % (n=10), GI Bleed 10% (n=5), and rest unknown cause in 8 % (n= 4).In a study done by Janus P Ong et.al2 it was seen that precipitating factor for Hepatic Encephalopathy were Azotemia 28 %, Infection 21 %, GI bleed 19 %, Constipation 5%.lactulose noncompliance 5% and miscellaneous 22%.

Precipitating Factors	Janus P Ong Et al study2	Present study
Azotemia	28%	52%
Infection	21%	20%
GI Bleed	19%	10%
Constipation	5%	10%
Lactulose noncompliance	5%	-
Miscellaneous	22%	8%

Table 15: PERCENTAGE OF PRECIPITATJNG FACTORS IN HEPATIC ENCEPHALOPTHY AS COMPARED TO OTHER STUDY

SYMPTOMS: In the present study, it was found that the most common presenting symptoms altered sensorium, distention of abdomen, yellowish discoloration of skin and swelling of the feet.

These results were similar to the results seen in studies done by Tandon et al, 58 where patient with chronic liver disease with Hepatic Encephalopathy had presented with similar symptoms.

SIGNS: In the present study signs of Hepatic Encephalopathy and chronic liver disease were seen like Fetor Hepaticus 74%, Asterisks 86%, altered sensorium 96 Ascites 72 S. and Parotid Enlargement 0% were seen in the patients.

In a study done by Guptan et al it was seen that fetor hepaticus, altered sensorium, ascites and other signs of Portal Hypertension were commonly noted signs.

WEST HAVEN GRADING: In the present study severity of Hepatic Encephalopathy was assessed by West Haven Grading system and it was found that 6.0 % were in Grade 1, 36% were in Grade 2> 44%

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were in Grade 3and 14% were in Grade 4 the most severe form of Hepatic Encephalopathy. In the present study maximum number of patient were in Grade 3.

In the studies done by Jalan et al,[11] Tandon et al had also mentioned in their study use of West Haven Criteria for assessing the severity of Hepatic Encephalopathy. Janus P Ong et al[2] in their study had used West Haven Grading for assessing the severity of Hepatic Encephalopathy, in their study 25 % were in Grade 1, 22 % in Grade 2, 23 % in Grade 3and 11% in Grade 4.

West Haven Grading	Janus P Ong et al study2	Present study
Grade 0	25%	-
Grade 1	22%	6.0%
Grade 2	9%	36%
Grade 3	23%	44%
Grade 4	11%	14%

Table 16: CORRELATION OF WEST HAVEN GRADING OF PRESENT STUDY WITH OTHER STUDY

SERUM ARTERIAL AMMONIA AND VENOUS AMMONIA LEVELS: In the present study it was observed that serum total Ammonia increased with the severity of Hepatic Encephalopathy. But it was found that increase in Arterial ammonia increase correlated statistically with the severity of Hepatic Encephalopathy. Venous ammonia had also increased but the increase was not statistically significant. These suggested that Ammonia definitely plays an important role in the pathogenesis of hepatic Encephalopathy and also suggested that Arterial ammonia measurements are superior to Venous ammonia while assessing the severity of Hepatic Encephalopathy.

Stahl et al[8] in his study had found that estimation of Arterial ammonia in drowsy with suspected liver failure was of diagnostic significance

Janus. P. Ong et al[2] in their study found that all four measures of ammonia increased with the severity of Hepatic Encephalopathy i.e. arterial and venous ammonia and partial pressure of ammonia (arterial and venous).but arterial ammonia had the highest correlation coefficient (rs=0.6l) with the grade of Encephalopathy..

ASSOCIATION OF GRADING OF HEPATIC ENCEPHALOPTHY WITH AMMONIA LEVELS: In the present study it was observed that increased ammonia levels correlated with the severity of Hepatic Encephalopathy and highest levels were seen in the Grade 3 and Grade 4, This correlated with the study done by Janus P.Ong2 where maximum Arterial ammonia levels were seen in the Grade 3 and Grade 4 of the West Haven Grading

In the study done by Nicolao F et al.[12] also it was seen that highest ammonia levels were seen in the Grade 3 and Grade 4 groups.

LIVER FUNCTION TESTS: In the present study it was observed that Serum Albumin statistically correlated inversely with the severity of Hepatic Encephalopathy. It was also observed that even

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though Serum total Bilirubin increased with the severity of Hepatic Encephalopathy it was not statistically correlating.

In the study of Janus P Ong[2] et al it was observed that Bilirubin correlated with the severity of Hepatic Encephalopathy whereas other parameters like serum Albumin did not correlate with the severity of Encephalopathy.

In the study done by Guptan et al it was observed that in patient with chronic liver disease with HE, had significantly more deranged Liver function especially lower Serum Albumin and increased serum Bilirubin.

	Janus P Ong.et al[2]	**Guptan et al study**	**Present study**
Serum Bilirubin	p<0.005	p<0.001	**0.458**
Serum Albumin	p<0.005	p<0.001	**p<0.014**

Table 17: CORRELATION OF LIVER FUNCTION TEST IN OTHER STUDIES WITH THE PRESENT SYUDY

PROTHROMBIN STUDY: In the present study it was observed that even though Prothrombin time increased with the severity of Hepatic Encephalopathy, this correlation was not statistically significant.

In the study of Janus P Ong[2], it was observed that Prothrombin was statistically increased with the severity of Hepatic Encephalopathy with the P value<0.005.

	Janus P Ong et al study [2]	**Present study**
P value of Prothrombin time	p<0.0Q5	**P<0.403**

Table 18: CORRELATION OF PROTHROMBIN TIME IN PRESENT STUDY WITH OTHER STUDIES

ARTERIAL AMMONIA VERSUS VENOUS AMMONIA LEVELS: In the present study it was observed that Venous ammonia levels did not correlate with the Arterial ammonia levels. The correlation of Venous ammonia with Arterial ammonia is small (0.338), hence the Venous ammonia does not correlate either with the grading or with the Arterial ammonia levels. Therefore there is a need to do the Arterial ammonia to assess the severity of HE.

CONCLUSION:

- Incidence of Hepatic Encephalopathy was more in males than females.
- Majority of patient were in Grade 3 as assessed by West Haven Grading.
- Arterial total ammonia correlated better with the severity of Hepatic Encephalopathy as compared to venous ammonia levels.
- Venous total ammonia did not correlate with severity of Hepatic Encephalopathy and with arterial ammonia levels.
- Other parameters like Serum Bilirubin and Prothrombin time did not correlate with arterial ammonia levels and severity of Hepatic Encephalopathy.

- Serum albumin correlated inversely with severity of Hepatic Encephalopathy.
- Arterial ammonia levels measurement is sufficient while assessing the severity of Hepatic Encephalopathy.

SUMMARY: This study was done in 50 patients of cirrhosis with Hepatic Encephalopathy to study the clinical correlation between arterial ammonia versus venous ammonia levels with severity of Hepatic Encephalopathy.

Males had higher incidence of HE. Severity of HE was assessed by West Haven grading and majority of patients belonged to Grade 3.

It was found that Arterial total ammonia levels correlated with severity of HE and venous total ammonia did not correlate with severity of HE and with Arterial ammonia levels.

It was also seen that other parameters like serum bilirubin and prothrombin time did not correlate with severity of HE.

Serum albumin was inversely correlated with severity of HE.

Results of this study indicates that ammonia levels correlate with the severity of Hepatic Encephalopathy and Arterial total ammonia levels measurement will be a better indicator in assessing the severity of Hepatic Encephalopathy when compared to Venous total ammonia levels.

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