

HEPATIQ Measure Of Hepatic Function: Threshold Function for Ascites and Death in Patients with Chronic Liver Disease (CLD)

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BACKGROUND

1. Gradual accumulation of hepatic fibrosis is the hallmark of progressive CLD eventually causing abnormal hepatic function, portal hypertension and carcinogenesis.

2. PHM correlates with the functional mass of the liver (1997 AmJGastro;92:2054) and PHM was predicted to correlate with clinical Outcomes. PHM was proven to predict in the quantitative Liver function test (QLFT) ancillary study of the prospective HALT-C (Hepatitis C Antiviral Long-term Treatment Against Cirrhosis) trial (2010 Hep 51:585).

3. HEPATIQ is a program that automates calculation of the PHM as a measure of Hepatic function.

4. We identified in this population the range of PHM in patients with/without ACO (adverse clinical outcome) and report the threshold of hepatic function for clinical problems.

AIMS

Determine the PHM threshold for treatable ascites (Rx-A), refractory ascites (Rf-A), and death (D)

PATIENTS

1. 389 sequential patients with Quantitative liver spleen scan (QLSS) enrolled
2. Liver Disease: normal 29, HBV 61, HCV 138, NASH 49, PBC 16, ACAH 16, PSC 3, ALD 13, abnormal AST/ALT 44, post liver transplant (LT) 6, CC 8, misc. 6,
3. Complications:

No current ascites: Normal - 29, CLD None current or past – 306, prior ACO with ascites recovered on treatment with no diuretics > 2 years – 10 and 6 recovered with LT, ACO without ascites - 5
Current Ascites: treatable 14, refractory 12, and death 6 (2 with metastatic HCC)

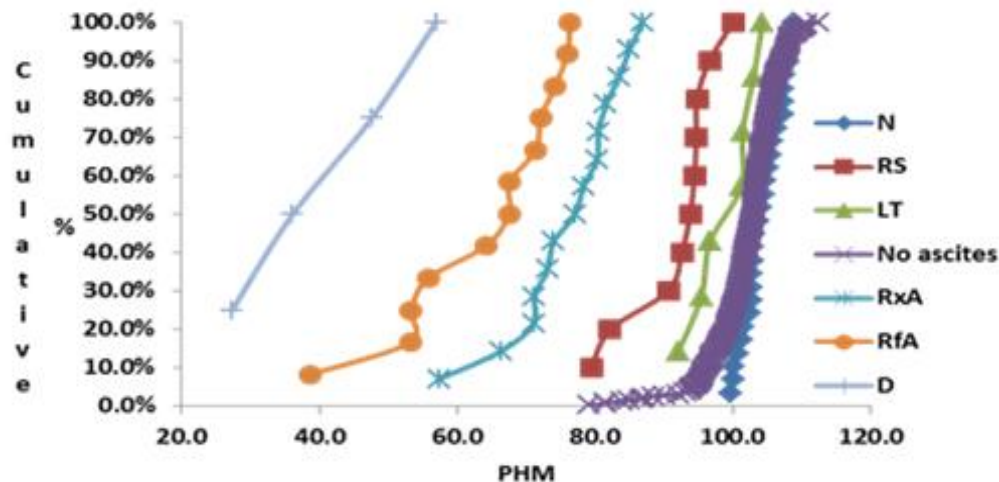
Groups

- never had a clinical outcome in past or present (# 308) plus 29 normals
- current adverse clinical outcomes with ascites (# 32)
- clinical abdominal fluid (scan only and untreated were excluded as ascites)
 - new onset ascites or controlled on treatment
 - refractory or difficult – requires repeat paracentesis for control (last paracentesis within 2 months of scan) or TIPPS
 - prior ACO – history of ascites > 2 yr prior to scan, but not requiring on-going treatment (# 10)
 - liver transplant prior to recent QLSS (# 6)

RESULTS

	#	PHM	SV/IBW Cc/lb IBW	Hb Gm/cc	Platelet X1000	INR	Albumin Gm%	Bilirubin Mg%	creatinine Mg%
Normal	29	104(3)	.9 (.6)	14(1)	253(91)	1(0)	4.5(.3)	.9(.1)	.8(.2)
No ACO	306	102(5)	1.4(1)	14(1.6)	205(69)	1(.1)	4.4(.4)	.7(.4)	.9(.4)
Prior ACO	10	92(6)	3.7(2.1)	13.3(1.9)	123(47)	1.1(0)	4.3(.3)	.9(.4)	1.8(.2)*
LT	7	99(4.5)	2.5(1.5)	13.4(2.6)	155(74)	1(0)	4.2(.4)	.6(.3)	1.3(.3)
ACO no ascites	5	92 (6)	2.1 (.8)	13.6(1.9)	161(85)	1.1(.1)	4.2(.6)	.6(.2)	.9(.4)
Act ACO	32	67(15)	5.1(2.5)	11.0 (1.7)	100(55)	1.4(.3)	3.4(.5)	4.8(7.7)	1.3(1.1)
Rx Asc	14	78(7) * +	4.5(2.3)	11.2(1.5)	91(55)	1.2(.1)	3.4(.6)	1.7(1.1)	1.0(.3)
Refr Asc	12	64(12) * +	6.4(3.1)	10.8(1.9)	92(45)	1.3(.2)	3.3(.5)	2.2(1.7)	1.5(1.5)
Death	6	50(16) * +	3.8(.9)	10.4(1.6)	112(59)	1.8(.4)	3.2(.5)	7.3(9.7)	1.8(1.3)

* P < .05 compared to no ACO or normal, + p < .05 Rx Asc vs Refr asc vs death



CONCLUSIONS

1. Adverse clinical outcomes are closely related to hepatic function as measured by PHM.
2. Range of PHM shifts to the left with increasing clinical problems.
3. Platelets, INR and Bilirubin are not as well correlated with clinical events except at the point of death.
4. Hepatic function can be measured precisely with HEPATIQ PHM and range of PHM correlates with severity of clinical problems.